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Epidemic size determines population-level effects of fungal parasites on *Daphnia* hosts

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Abstract Parasites frequently reduce the fecundity, growth, and survival of individual hosts. How often do these virulent effects reduce the density of host populations? Spectacular examples show that recently invaded parasites can severely impact host populations—but what about parasites persisting long-term in host populations? We have addressed this issue using a zooplankton host (*Daphnia dentifera*) that becomes infected with a fungal microparasite (*Metschnikowia bicuspidata*). We combined observations of epidemics in nine lakes over 6 years, fine-scale sampling of three epidemics, and a mesocosm experiment. Most epidemics remained small (<10% maximum prevalence) and exerted little influence on host densities. However, larger epidemics more severely depressed the populations of their hosts. These large/severe epidemics started and peaked earlier than smaller/benign ones. The larger epidemics also exerted particularly negative effects on host densities at certain lags, reflecting the delayed consequences of infection on fecundity reduction and host mortality. Notably, negative effects on the juvenile stage class manifested later than those on the adult stage class. The results of the experiment further emphasized depression of host density by the fungus, especially on the density of the juvenile stage class. Consequently, this common parasite reduces the density of host populations when conditions foster larger outbreaks characterized by an earlier start and earlier peak. Given these considerable effects on host density seen in a number of large epidemics, parasitism may sometimes rank highly among other factors (predation, resource availability) driving the population dynamics of these hosts.

Keywords *Daphnia* · *Metschnikowia* · Epidemic size · Host–parasite interaction · Multivariate auto-regressive models · Population effects of disease

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Introduction

By definition, parasites harm their hosts. Depending on the particular host–parasite interaction, these individual-level effects can range from negligible (or, at least, immeasurable) to rapid castration and/or greatly reduced survivorship (Scott and Dobson 1989; Minchella and Scott 1991). Given this variation, crafting predictions linking parasites to the regulation of host populations seems difficult (Ebert et al. 2000). Results from early studies often suggested that parasites should have little influence on host density. This prediction stemmed from the assumption that parasite fitness would decline if epidemics excessively damaged host

populations (Grundman et al. 1976; Price 1980). However, field evidence documenting extreme population drops following increases in pathogen prevalence subsequently challenged this view (Scott and Lewis 1987; Gulland 1995; Mutze et al. 1998; Hochachka and Dhondt 2000; LaDeau et al. 2007). Current thinking, grounded in theory (May and Anderson 1978; Anderson and May 1979) and experimental evidence (Scott 1988; Dobson and Hudson 1992; Hudson et al. 1998; Albon et al. 2002), suggests that parasites can occasionally depress the population densities of their hosts.

But under which circumstances are parasites able to reduce the densities of their hosts? When can we observe parasite effects on population dynamics against a background of other biotic factors (predators, competitors) or abiotic factors that drive host populations? Providing definitive answers to these questions remains challenging for several reasons. For example, parasites may virulently affect particular stages of hosts, yet these direct effects on the particular stage can still indirectly alter the densities of other age or stage classes. Parasites also interact with resources and predators of hosts non-linearly; such non-linearity might defy a simple connection between disease and host dynamics (Holmes 1995; Tompkins and Begon 1999; Hall et al. 2005). Moreover, many host–parasite systems cannot be experimentally manipulated. For those systems that are tractable, several field experiments have demonstrated that parasites alone cannot produce the observed fluctuations in host dynamics (Singleton et al. 1995; Redpath et al. 2006). As a result, individual-level effects of parasites may not translate into observable effects on host populations (Gulland 1995; Ebert et al. 2000; Duffy and Hall 2008).

Daphnia (Crustacea: Cladocera) is an ideal model system for examining links between parasite epidemics and population dynamics of their host. First, in nature, *Daphnia* densities can fluctuate over orders of magnitude (Lampert and Sommer 2007). Traditional explanations for these fluctuations implicate predation, resource competition, and climatic fluctuations (e.g., Mills et al. 1987; McCauley et al. 2008). Could this variation in density also stem from parasite outbreaks? *Daphnia* species can become infected with a variety of parasites that reduce the fecundity and survivorship of their hosts (Ebert 2005). In laboratory and mesocosm experiments, these parasites have been observed to depress *Daphnia* densities (Ebert et al. 2000; Bittner et al. 2002; Duffy 2007). Yet, in natural populations, the severity of microparasite epidemics in *Daphnia* varies considerably in space and time (Bittner et al. 2002; Decaestecker et al. 2005; Ebert 2005; Johnson et al. 2006, 2009; Hall et al. 2010). For example, epidemics of a chytrid fungus range interannually from 1 to 34% peak prevalence (Johnson et al. 2009). Spatio-temporal variation in the distribution and magnitude of epidemics, coupled with ease of experimental manipulation, provide an opportunity to examine the connection between outbreaks of these virulent parasites and the population dynamics of hosts.

In the study reported here, we explored this opportunity in a *Daphnia* host–fungal parasite system. Based on laboratory assays and field observations, we know that infection by the fungus (*Metschnikowia bicuspidata*) exacts severe consequences on individual hosts (*Daphnia dentifera*), such as decreasing the lifespan of infected hosts by up to 80% (Ebert et al. 2000; Duffy and Hall 2008; Hall et al. 2006, 2009a, b). Infection also elevates per capita mortality of infected hosts due to selective predation by fishes (Johnson et al. 2006; Duffy and Hall 2008). These effects might influence the densities of adults in particular. The adult stage class is also more susceptible than the juvenile stage class, all else being equal, because the larger bodied adult class comes into contact with more spores per unit time via higher feeding rates and consumes spores for longer periods of time (Hall et al. 2007). Additionally, the fungus reduces the fecundity of infected hosts by 25–50% (Duffy and Hall 2008; Hall et al. 2009a, b). Therefore, infection should indirectly depress the densities of the less vulnerable juvenile stage as well, mainly through enhanced mortality and reduced fecundity inflicted on the adults giving birth to them. This effect of fungal epidemics on the juvenile stage may arise with some dynamical delay, since virulence on the adult stage takes some time to manifest in the juvenile stage.

Thus, through these direct and indirect effects, the fungus could depress populations of their host. To examine these possibilities, we used data from observations of epidemics conducted since 2002 (Hall et al. 2010). These data revealed considerable spatio-temporal variation in the size of epidemics, but only larger epidemics exacted negative effects on host dynamics. In contrast, smaller epidemics sometimes even increased as host density increased. A closer examination (via time series analyses) of three more finely sampled epidemics confirmed this relationship between size and negative effects on host populations. Moreover, these analyses revealed the importance of time-lagged effects of epidemics on juvenile and adult stage classes. Finally, an in situ mesocosm experiment conducted in a stratified lake confirmed that the fungus itself can depress host populations.

Materials and methods

Field data

To determine the frequency and magnitude of disease, we monitored epidemics of *Metschnikowia* in nine populations

of *D. dentifera* (Barry and Kalamazoo Counties, MI) from 2002 to 2008 (Cáceres et al. 2006; Hall et al. 2010; Duffy et al. 2010). Every 2 weeks during August through October, we collected six bottom-to-surface zooplankton tows with a 153-μm Wisconsin bucket net. Two samples were created by pooling sets of three tows into each sample. One sample was used for visual diagnosis of live animals (400+ *D. dentifera*). The other sample was preserved in >70% ethanol for later estimates of host density. In addition, we carried out fine-scale monitoring (every 3–4 days) of epidemics during 2003 and 2004 (in Baker, Bassett, and Warner Lakes, Barry County). These data allowed characterization of the links between parasite prevalence and density of both juvenile and adult stage classes.

To analyze these field data, we coupled regression with cross-correlational and autoregressive approaches. Using data collected every other week, we calculated regression coefficients from linear models relating disease prevalence (independent variable) with host density (dependent variable) after first standardizing the prevalence and density data. (To standardize prevalence data, one divides data for each date of a lake-year by the standard deviation of prevalence for all dates in a lake-year; the same approach was used for density data). The standardized coefficients $\left(\hat{\beta}\right)$ enabled a more useful comparison of regression slopes among epidemics of different sizes [see Appendix of Electronic Supplementary Material (ESM) for more information; Fig. A3]. We also plotted standardized coefficients and peak prevalence versus centered day of maximum prevalence. We centered data (i.e., subtracted the annual mean) because epidemics start at different mean times each year. Then, with more finely scaled time series data, we first calculated cross-correlations between total host density and infection prevalence at different lags, ranging from one to four sampling visits (i.e., a lag of four visits means that infection prevalence now correlates with host density four sampling dates (12–16 days) from this time onwards). Multivariate autoregressive (MAR) models were used to quantify interaction strengths between infected hosts (I) and two stage classes of uninfected hosts, namely, adults (A_U) and juveniles (J_U) [following Ives et al. (2003) and especially Hall et al. (2009c); see ESM for details of this analysis].

Mesocosm experiment

We conducted an enclosure experiment during July and August 2006 in which we created fungal epidemics in four of eight replicate *D. dentifera* populations. The mesocosm experiment was conducted in Tamarack Lake (Barry County, Michigan). This small, oligotrophic, hardwater lake was dominated by *D. dentifera* during early July when the experiment started. We suspended polyethylene bags (diameter 1 m, length 6 m) from floating wooden frames on 1 July and covered them with 1-mm fiberglass window screen lids to keep out ovipositing insects. The bags were filled with filtered (130-μm mesh) lake water using a pump. After 3 days (5 July), we added zooplankton collected at night from the lake into each bag (8 tows of the 13-cm-diameter Wisconsin net used in field sampling). Plankton were first sampled (6-m tows) on 11 July and twice a week thereafter. Zooplankton were sampled at night using the Wisconsin net and live counted to entirety. Chlorophyll *a* was estimated using day-time casts of a Turner fluorimeter attached to a Hydrolab (Minisonde model) at 0.5-m intervals (calibrated with chlorophyll *a* measured from chilled ethanol extractions; Welschmeyer 1994). The chlorophyll *a* data presented here are the average values over these depths, from the surface to a depth of 5 m. On 14 July, fungal spores reared in vivo in Tamarack lake-collected hosts were added at a concentration of 5,000 spores/L level and mixed into the water column with a Secchi disk. Hosts in the spore addition treatment were then visually diagnosed for infection.

We used the REPEATED statement in PROC MIXED (SAS 9.1; SAS Institute, Raleigh, NC) to assess potential treatment differences on host density, host egg ratio, density of the congener *D. pulicaria*, and chlorophyll *a* concentrations. Degrees of freedom were estimated with the Kenward and Roger correction (Littell et al. 2002). The best-fit covariance structure was first determined by comparing actual Akaike Information Criterion values of compound symmetry in unstructured, Toeplitz, and first-order autoregressive models; compound symmetry provided the best fit. When significant treatment × day interactions were identified, we used the SLICE option to test the treatment effect on each day. Significant effects are reported for each model (see ESM for ANOVA tables; Table A1).

Results

Epidemics varied considerably in size among lake-years, and this variation in epidemic size mattered to host populations (Fig. 1; see also ESM Fig. A1). When epidemics stayed small (maximum prevalence <10%), host density and infection prevalence were often positively related (e.g., $\hat{\beta} > 0$; Whitford Lake in 2006; ESM Fig. A2). In cases of higher disease prevalence, host density declined as epidemics increased (e.g., $\hat{\beta} < 0$; Baker Lake 2002; ESM Fig. A2). Among lake-years, we observed pronounced relationships between epidemic size (i.e., maximum prevalence), severity (as indexed by the sign and size of the standardized regression coefficients), and timing of the

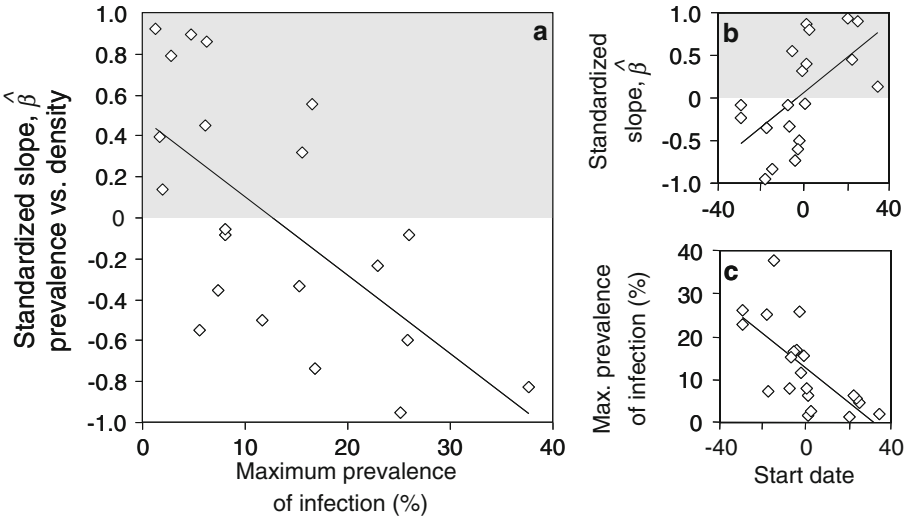

Fig. 1 Variation in the impact of a virulent fungus (*Metschnikowia bicuspidata*) on host populations of *Daphnia dentifera* in several lakes, 2002–2006. **a** Larger epidemics (i.e., higher maximum prevalence) exerted larger negative effects on host populations (i.e., have more negative $\hat{\beta}$) than smaller epidemics. Additionally, more severe epidemics (**b**) and larger epidemics (**c**) start earlier than less severe/smaller ones. Start days were centered prior to plotting and analysis (see text for explanation). *Grey shading* Positive standardized regression coefficients

start and peak prevalence. More specifically, most epidemics larger than approximately 8% maximum prevalence depressed host populations, whereas smaller epidemics did not ($R = 0.48$, $P = 0.026$; Fig. 1a). Furthermore, epidemics that were more severe (in terms of effects on host density) started earlier ($R = 0.47$, $P = 0.033$; Fig. 1b) and peaked earlier ($R = 0.48$, $P = 0.026$; ESM Appendix Fig. A4). Similarly, epidemics with a higher maximum infection prevalence started earlier ($R = 0.69$, $P = 0.002$; Fig. 1c) and peaked earlier ($R = 0.48$, $P = 0.042$; ESM Fig. A4). Finally, epidemics that started sooner reached their peak sooner ($R = 0.58$, $P = 0.006$; ESM Fig. A4).

The more finely sampled epidemics permitted closer characterization of prevalence-density links. The epidemics in Bassett and Warner Lakes in 2004 reached high (approx. 40%) and moderate (15%) infection levels, respectively, while the epidemic in Baker Lake in 2003 peaked at about 8% prevalence (Fig. 2). In Bassett Lake, *Daphnia* populations fluctuated twofold before the epidemic became large (Fig. 2a); as infection prevalence increased, the population density dropped to almost 50% of the early epidemic peak—but in a temporally lagged manner. We found the strongest negative cross-correlation between prevalence and host density at lag 3, or 9 days ($R = -0.79$, $P < 0.001$; Fig. 2b), although negative relationships emerged from other lags [lag, R (P value): lag 0: −0.49 (0.008); lag 1: −0.65, lag 2: −0.73, lag 4: −0.75 (all three <0.001)]. Interestingly, when infection prevalence was low, the lagged host population fluctuated, but once it surpassed approximately 10% infection prevalence, host density declined until peak infection was crossed (Fig. 2a, b). The more modest epidemic in Warner Lake still showed a correlated decline in host density (i.e., density was halved; Fig. 2c). We saw a strong negative cross-correlation between density and prevalence at lag 3 again, but the increase in density after the epidemic was steeper than the decline during the epidemic (Fig. 2d). Once these two parts of the dynamics had been separated, very strong negative relationships between host density and prevalence emerged [$R = -0.84$, $P < 0.001$ for white, pre-peak points in Fig. 2d; $R = -0.98$, $P = 0.02$ for grey, post-epidemic points in Fig. 2d; other lags, R (P value): lag −2: −0.407 (0.075); lag −1: −0.54 (0.012); lag 0: −0.59 (0.004); lag 1: −0.52 (0.016); lag 2: −0.44 (0.051); lag 3, overall: −0.40 (0.09); lag 4: 0.27 (not signficant)]. Finally, no relationship between host density and prevalence arose at any lag in the smaller epidemic in Baker Lake (Fig. 2e, f).

More sophisticated MAR models showed the differential effects of infection on stage structure between the three epidemics. In Bassett Lake (Fig. 3a), the density of the infected hosts had strong effects on uninfected juvenile and adult stage classes. This negative effect on the juvenile stage appeared stronger at higher lags (significant at lags 2–4 sampling visits), while that on the adult stage was significant even at lag 1 (significant at lags 1–3; Fig. 3d). In Warner Lake, we found stronger evidence for the negative effects of infected density on uninfected adults (Fig. 3b), with only a significant effect of infection on the juvenile stage only at lag 4 (i.e., 12 days later; Fig. 3e). Furthermore, this effect on the uninfected juvenile stage class appeared to be weaker than that seen in Bassett Lake (i.e., confidence intervals for lag four in Warner Lake do not overlap the estimates of significant parameters in Bassett Lake; Fig. 3d, e). However, strong, negative effects of infection on the adult stage class were found at lags 2–4 (6–12 days; Fig. 3e). The coefficients rivaled those from Bassett in magnitude. Finally, no significant negative interaction coefficients were found in the small epidemic in Baker Lake; rather, we found a positive coefficient at lag 4

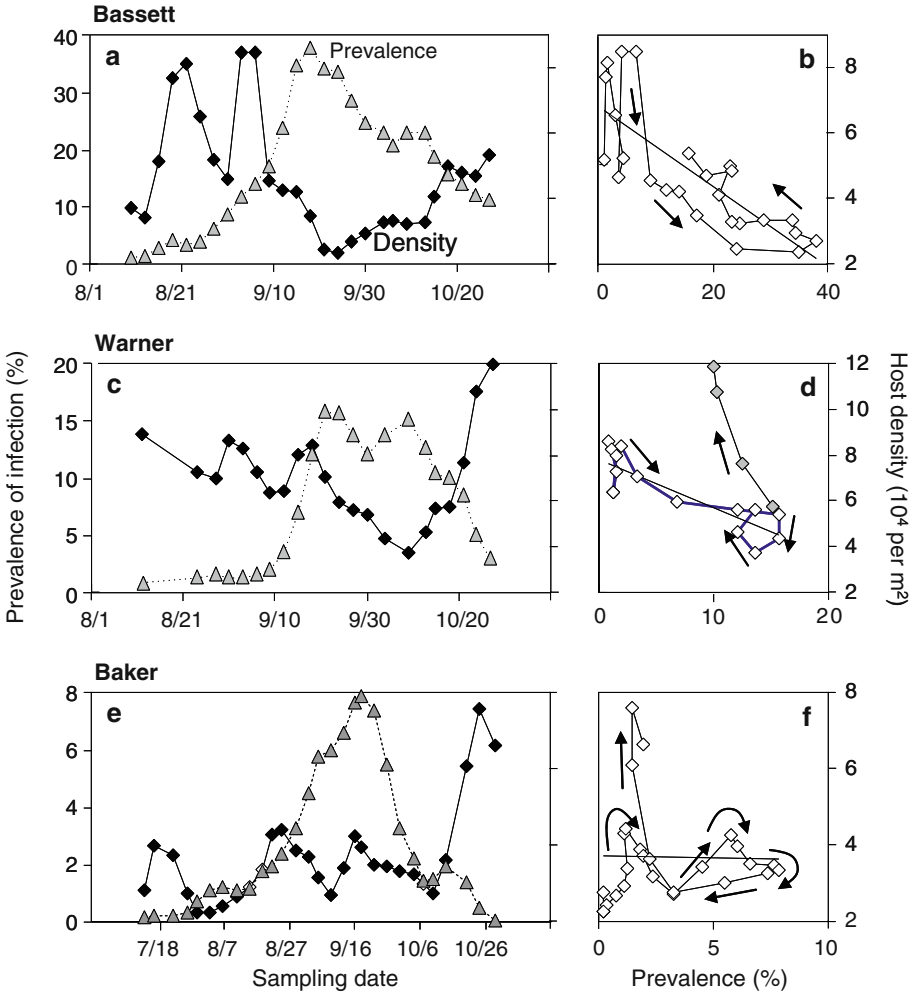

Fig. 2 Time series (**a, c, e**) and phase plots (**b, d, f**) of prevalence of infection by a virulent fungal parasite, *M. bicuspidata*, and density of its zooplankton host, *D. dentifera*, in three lakes: Basset (**a**, **b**) and Warner (**c**, **d**) Lakes with epidemics in August–October 2004, and Baker Lake (**e**, **f**) surveyed during July–October 2003. The phase plots show how epidemics move in time through prevalence-lagged density space ($\tau = 3$; 9 days for the 2004 epidemics, 12 days for the 2003 epidemic). **a, c, e** *Black diamonds*, *solid lines* denote host density, *gray triangles*, *dotted lines* denote infection prevalence. **b, d, f** *Arrows* point in the direction of time, *different colors* (*white* pre-peak, *gray* post-peak) in **e** are described in the text. Data were smoothed prior to analysis

(16 days) of the infected hosts on the stage class of adult uninfected hosts (Fig. 3c, f).

In the in situ mesocom experiment, spore addition resulted in a peak prevalence of infection of 13%—a moderate epidemic (Figs. 1, 4a). As expected, infected individuals were not observed until about 1 week after the spores were added. Following peak infection, prevalence declined and remained between 1 and 7%. As the infection became established in the population, the densities in the two treatments began to differ, resulting in a significant time × treatment effect on total densities (Fig. 4b; $F_{7,42} = 2.82$, $P = 0.017$, time $F_{7,42} = 29.3$, $P < 0.0001$; see ESM Appendix for full ANOVA tables for this model and all others). Total densities between the two levels of the parasite treatment differed significantly on days 14 and 17 (detected via slicing: $F_{1,22.4} = 10.13$, $P = 0.004$; $F_{1,22.4} = 6.47$, $P = 0.018$, respectively). On day 14, densities in the no spore treatment had risen to nearly twofold that of the spore addition treatment. Among treatments, the juvenile stage class comprised 64–80% of the total population—higher than typically seen in the field (Figs. 3, 4c). As with total densities, the juvenile stage class began diverging in the two treatments on day 10; by day 14, mean densities in the no spore treatment were nearly twofold those of the spore treatment (time × treatment $F_{7,42} = 3.94$, $P = 0.002$; Slice Day 14: $F_{1,22.8} = 13.24$, $P = 0.001$; Slice Day 17: $F_{1,22.8} = 11.15$, $P = 0.003$). Densities of the uninfected adult stage class were highly variable among replicates; consequently, no significant treatment or time × treatment effect was detected (data not shown, $P > 0.49$).

All populations, regardless of infection status, declined precipitously between day 17 and 21 and remained low at the final sampling (day 24). Three lines of evidence suggest that this decline was due, at least in part, to food limitation in both treatments. First, densities of the competitor *D. pulicaria*, which completely resists *Metschnikowia* infection (Hall et al. 2009c), increased in both treatments over time (Fig. 4d; time $F_{7,42} = 24.7$, $P < 0.0001$). However, densities of this species peaked prior to the final sampling date in both treatments. Second, in both treatments, egg ratios of uninfected *D. dentifera* began

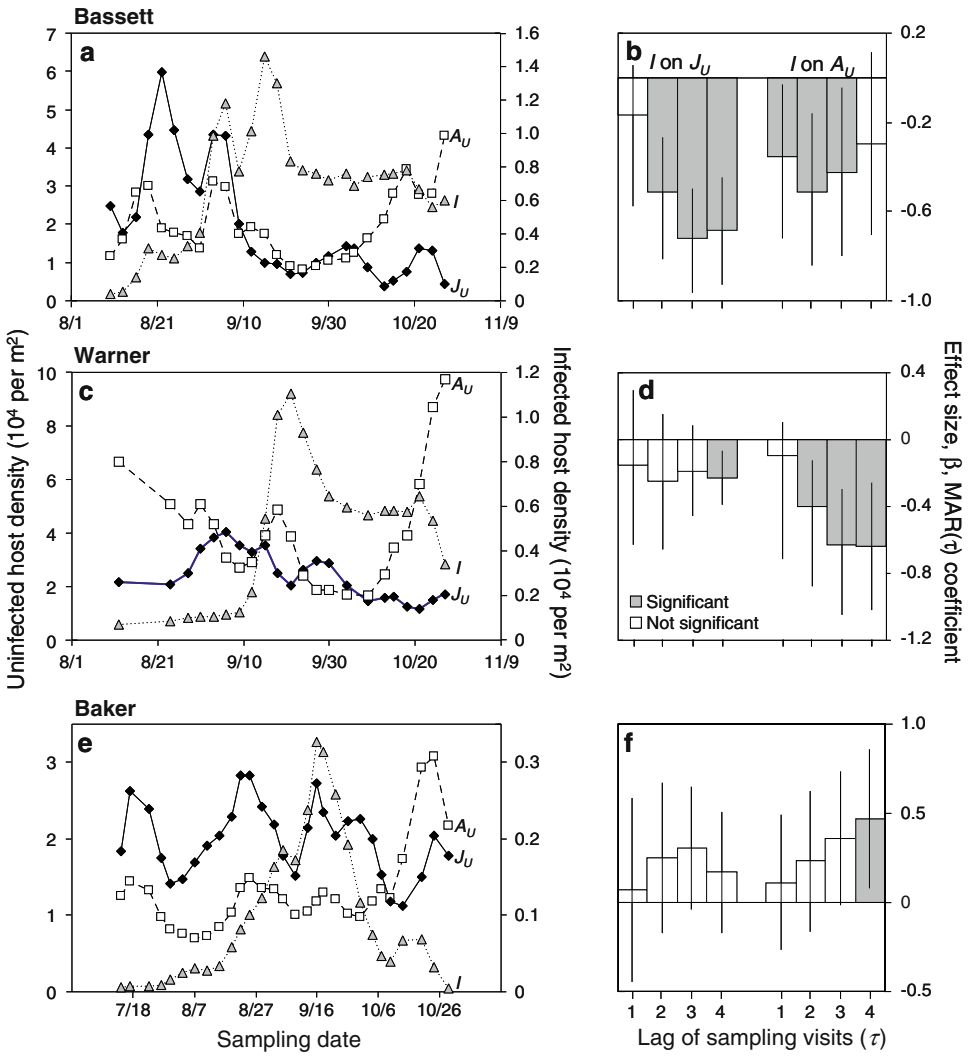

Fig. 3 Time series of densities of uninfected juvenile (J_U) and adult (A_U) stage class and infected (I) class of *D. dentifera* hosts for Bassett (**a**), Warner (**c**), and Baker (**e**) Lakes during epidemics of a virulent fungus (*M. bicuspidata*). Effect sizes are expressed as multivariate autoregressive [*MAR*(τ)] coefficients (β) fit at different lags (τ) for each lake (**b** Bassett, **d** Warner, **f** Baker), with bootstrapped 95% confidence intervals. These β coefficients represent interaction strengths of infected hosts on the densities of one of the uninfected stage classes at a sampling visit in the future ($\tau = 1$–4). Data were smoothed prior to analysis. *Shaded bars* indicate significance at $\alpha = 0.05$ (i.e., 95% confidence intervals did not overlap with zero)

declining from a high of 2.3 eggs/adult around day 10 and ended at a low of <0.6 egg/adult (Fig. 4e; time $F_{7,42} = 22.5$, $P < 0.0001$). Uninfected hosts in spore addition mesocosms also had higher egg ratios (marginally significant treatment effect: $F_{1,7} = 4.8$, $P = 0.0709$). Finally, chlorophyll *a* levels declined rapidly beginning on day 10 (Fig. 4f; time $F_{7,42} = 54$, $P < 0.0001$). By the final sampling date, chlorophyll *a* concentrations in the enclosures dropped to 25% of the levels seen in the lake itself.

Discussion

The results reported here demonstrate that the virulent fungal parasite (*M. bicuspidata*) can depress the density of host zooplankton (*D. dentifera*) populations when epidemics become sufficiently large (greater than approx. 8% maximum prevalence). This depression effect at the population level stems from the net effects of the virulent parasite on the survival and fecundity of host individuals (Duffy and Hall 2008; Hall et al. 2009a, b). Epidemiological models predict that such parasites should reduce and/or regulate population size of their hosts (Anderson and May 1979), a result seen in laboratory and field experiments (Ebert et al. 2000; Duffy 2007; this study). However, despite this virulence, our prevalence–severity results show that *Metschnikowia* did not always negatively affect host densities [Duffy and Hall 2008; see Johnson et al. (2009) for a similar result in a single lake and for a different *Daphnia* sp.–parasite system]. This insight matters because many examples of population-level consequences of disease come from case studies of newly introduced parasite species or strains (e.g., Mutze et al. 1998; Hochachka and Dhondt 2000; LaDeau et al. 2007; see Lafferty et al. 2008 for other examples). The endemic fungus in our study exerted less dramatic—but still considerable—effects that were manifested in a spatio-temporally variable manner. Future research may show that

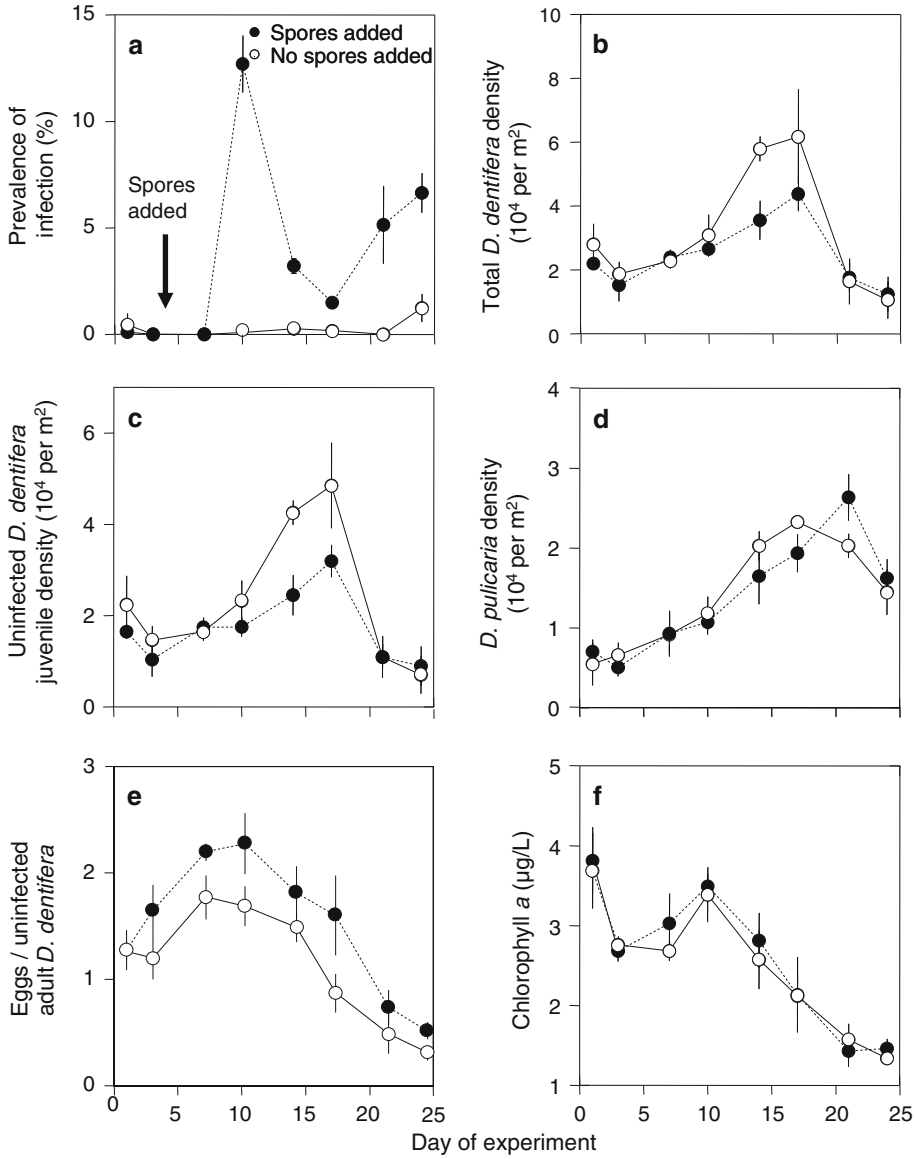

Fig. 4 Results from the mesocosm experiment. **a** Epidemics were created in the bags to which spores were added. **b** Host densities began diverging in the treatment bags in the days following peak infection. **c** Difference in density was most pronounced in the juvenile stage class. **d** Abundance of the *Metschnikowia*-resistant congener *D. pulicaria* increased in all enclosures. **e** Egg ratios of uninfected *D. dentifera* declined from day 10 until the end of the experiment. **f** Chlorophyll *a* levels also began declining on day 10. First sampling date was 11 July 2006. *Error bars* Standard errors (of $n = 4$ replicates per treatment)

similar spatio-temporal variation may more adequately characterize the norm for endemic wildlife disease.

In our study, large epidemics were more severe, but during small epidemics infection prevalence typically correlated positively with host densities. This positive relationship may be a common feature of fungal epidemics in *Daphnia* spp. It was observed at the beginning of even large epidemics (ESM Fig. A5) and may reflect a shared relationship with a seasonal, physical driver. Smaller epidemics peaked later in autumn, a period when lake waters mix more thoroughly (Smyth 2010). This mixing could simultaneously stimulate growth of *Daphnia* populations by enriching their algal resources and (re)suspend non-motile fungal spores that rely on hydrodynamics to connect them to *Daphnia* hosts (Lampert and Sommer 2007; Smyth et al., in review); both factors could spread disease (but see Hall et al. 2009b). In addition, compensatory reproduction may shield populations from the negative effects of parasitism during sufficiently small epidemics; this factor could then potentially produce this positive relationship between epidemic size and prevalence. Increased mortality inflicted on infected hosts could indirectly enhance the per capita reproduction of food-limited, uninfected hosts (as seen in the mesocosm experiment; ESM Fig. A6). This elevated fecundity might buffer populations when epidemics remain small. However, once epidemics became sufficiently large (i.e., in the mesocosms or at greater than approx. 8% maximum prevalence in the field), any compensatory effects became overpowered by fecundity reduction and enhanced mortality imposed on infected hosts (see also Johnson et al. 2009). In these cases, larger epidemics would exact more negative effects on population density [as seen

in, for example, pond *Daphnia* (Decaestecker et al. 2005) and crabs (Lafferty and Kuris 2009)].

We observed that larger epidemics depressed host populations, but their deleterious effects became most apparent with time lags. Infection prevalence–density signals arose most strongly at lags of 9 days (three sampling visits). These time lags likely reflected stage-structured feedbacks central to the epidemics. Strong effects of disease on adults manifested themselves at shorter lags, while negative consequences for the juvenile stage class arose more potently at later lags. This delayed impact on the juvenile stage class likely originated from the fecundity and survival reduction that infection imposed on the adults that produced them (Duffy and Hall 2008; Hall et al. 2009a, b) rather than from the direct effects of infection on the juvenile stage class per se. Individuals in the juvenile stage class are less vulnerable to infection because they contact fewer spores due to their smaller size and shorter exposure time (Hall et al. 2007). Such stage-dependent results might provide epidemiological models for hosts with stage-structured interactions. Stage-structured features have been built in models for disease carried by vectors, such as ticks (Van Buskirk and Ostfeld 1995; Caraco et al. 2002). However, in species such as *Daphnia*, interactions between adult and juvenile stage classes are important drivers of variation in host population dynamics. Such species would experience different epidemiology than ticks, so different models may be needed for them. These new models could address how the differential effects of disease on stage classes of adults and juveniles—with subsequent impacts on resource availability—influence the population dynamics of hosts with stage-structured interactions.

The results of the mesocosm experiment confirm that the fungus itself exerted negative effects on the population density of hosts, but it also hinted at a deleterious effect of another key species on host densities. *D. pulicaria* density enlarged beyond that seen in the lake. This increase correlated with a decline in *D. dentifera*, with or without disease, prompting a potentially important lesson for the management of epidemics (Keesing et al. 2006). Since *D. pulicaria* eats and removes fungal spores without becoming infected, this species might benefit *D. dentifera* during epidemics through a "dilution effect" (Keesing et al. 2006; Hall et al. 2009c). However, this diluter also competes with the host (Leibold 1991). Negative competitive interactions might outweigh the positive dilution effect on the focal host species, leading to diluter-competitors inflicting net negative effects on host populations. However, more work is needed on this competition–dilution aspect, as our study was not designed to delineate between them.

If large epidemics depress host populations, what factors drive variation in epidemic size? In our *Daphnia*–fungus system, larger epidemics started and peaked earlier than small epidemics. We hypothesize that variation in the start date of epidemics involves interplay between physical processes and species interactions. An increased intensity of mixing of lake waters, induced by storms and convection driven by cooling, can overcome physical barriers imposed by stratification to connect fungal spores to hosts (Bittner et al. 2002; Johnson et al. 2009; Hall et al. 2010; Smyth 2010). Once potentially initiated by physical processes, food web structure then influences how epidemics proceed. For example, selective predation and poor resource quality may inhibit the start date of epidemics while invertebrate predators may catalyze their spread (Duffy et al. 2005; Hall et al. 2005, 2006, 2009a, b, 2010; Cáceres et al. 2009). An earlier start then enables epidemics to reach higher maximal prevalence/severity because the fungus spreads for a longer time before cooler temperatures, improving resource quality, increased host resistance through rapid evolution, and the elevated density of unsuitable hosts catalyze their end (Hall et al. 2006, 2009a, c; Duffy and Hall 2008; Duffy et al. 2009). The links between the timing of epidemics, their size, and their severity highlight why variation in all of these bio-physical (environmental) factors matter from the host's perspective.

Finally, our results illustrate that endemic parasites can be an important but variable factor shaping host populations (Hudson et al. 2006; Lafferty et al. 2006, 2008; Hall et al. 2010). In our case, this endemic fungal parasite altered host densities during some lake-years—once epidemics became large enough. These larger epidemics (approx. $\geq$8% maximum prevalence) tended to start earlier during the season than the smaller ones. Yet, on occasion, the presence of the fungus seemed to be fairly inconsequential in terms of the ecological dynamics of the host (Duffy and Hall 2008)—although even small epidemics may strongly shape the genetic composition of hosts during epidemics through parasite-mediated selection (Duffy and Sivars-Becker 2007; Duffy et al. 2008). Therefore, disease should become viewed as a potentially important but spatio-temporal variable factor shaping plankton (specifically *Daphnia*) populations and communities (see also Bittner et al. 2002; Decaestecker et al. 2005; Johnson et al. 2009). While factors such as predation and resource availability receive much more attention in studies of population dynamics in planktonic (Tessier and Woodruff 2002; Lampert and Sommer 2007) and other ecosystems, future research may show that parasitism can sometimes rival these factors in importance.

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References

Albon SD, Stien A, Irvine RJ, Langvatn R, Ropstad E, Halvorsen O (2002) The role of parasites in the dynamics of a reindeer population. Proc Roy Soc Lond B 269:1625–1632

Anderson RM, May RM (1979) Regulation and stability of host–parasite population interactions 1—regulatory processes. J Anim Ecol 47:219–247

Bittner K, Rothhaupt K-O, Ebert D (2002) Ecological interactions of the microparasite *Caullerya mesnili* and its host *Daphnia galeata*. Limnol Oceanogr 47:300–305

Cáceres CE, Hall SR, Duffy MA, Tessier AJ, Helmle C, MacIntyre S (2006) Physical structure of lakes constrains epidemics in *Daphnia* populations. Ecology 87:1438–1444

Cáceres CE, Knight CJ, Hall SR (2009) Predator spreaders: predation can enhance parasite success in a planktonic host–parasite system. Ecology 90:2850–2858

Caraco T, Glavanakov S, Chen G, Flaherty JE, Ohsumi TK, Szymanski BK (2002) Stage-structured infection transmission and a spatial epidemic: a model for Lyme disease. Am Nat 160:348–359

Decaestecker E, Declerck S, De Meester L, Ebert D (2005) Ecological implications of parasites in natural *Daphnia* populations. Oecologia 144:382–390

Dobson AP, Hudson PJ (1992) Regulation and stability of a free-living host–parasite system, *Trichostrongylus tenuis* in red grouse. II: population models. J Anim Ecol 61:487–498

Duffy MA (2007) Selective predation, parasitism, and trophic cascades in a bluegill–*Daphnia*–parasite system. Oecologia 153:453–460

Duffy MA, Hall SR (2008) Selective predation and rapid evolution can jointly dampen effects of virulent parasites on *Daphnia* populations. Am Nat 171:499–510

Duffy MA, Sivars-Becker L (2007) Rapid evolution and ecological host–parasite dynamics. Ecol Lett 10:44–53

Duffy MA, Hall SR, Tessier AJ, Huebner M (2005) Selective predators and their parasitized prey: are epidemics in zooplankton under top-down control? Limnol Oceanogr 50:412–420

Duffy MA, Brassil CE, Hall SR, Tessier AJ, Cáceres CE, Conner JK (2008) Parasite-mediated disruptive selection in a natural *Daphnia* population. BMC Evol Biol 8:80

Duffy MA, Hall SR, Cáceres CE, Ives AR (2009) Rapid evolution, seasonality, and the termination of epidemics. Ecology 90:1441–1448

Duffy MA, Cáceres CE, Hall SR, Tessier AJ, Ives AR (2010) Temporal, spatial and between-host comparisons of patterns of parasitism in lake zooplankton. Ecology 91:3322–3331

Ebert D (2005) Ecology, epidemiology, and evolution of parasitism in *Daphnia*. National Library of Medicine (USA), National Center for Biotechnology Information, Bethesda. Available at: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=Books

Ebert D, Lipsitch M, Mangin KL (2000) The effect of parasites on host population density and extinction: experimental epidemiology with *Daphnia* and six microparasites. Am Nat 156:459–477

Grundman AW, Warnock RG, Wassom DL (1976) Some mechanisms of natural regulation of parasite helminth populations. Am Mid Nat 95:347–360

Gulland FMD (1995) Impact of infectious diseases on wild animal populations: a review. In: Grenfell BT, Dobson AP (eds) Ecology of infectious diseases in natural populations. Cambridge University Press, Cambridge, pp 20–51

Hall SR, Duffy MA, Cáceres CE (2005) Selective predation and productivity jointly drive complex behavior in host–parasite systems. Am Nat 165:70–81

Hall SR, Tessier AJ, Duffy MA, Huebner M, Cáceres CE (2006) Warmer does not have to mean sicker: temperature and predators can jointly drive timing of epidemics. Ecology 87:1684–1695

Hall SR, Sivars-Becker L, Becker C, Duffy MA, Tessier AJ, Cáceres CE (2007) Eating yourself sick: transmission of disease as a function of feeding biology of hosts. Ecol Lett 10:207–218

Hall SR, Simonis JL, Nisbet RM, Tessier AJ, Cáceres CE (2009a) Resource ecology of virulence in a planktonic host–parasite system: an explanation using dynamic energy budgets. Am Nat 174:149–162

Hall SR, Knight CM, Becker CR, Duffy MA, Tessier AJ, Cáceres CE (2009b) Quality matters: food quality and the course of epidemics in a planktonic host–parasite system. Ecol Lett 12:118–128

Hall SR, Becker CR, Simonis JL, Duffy MA, Tessier AJ, Cáceres CE (2009c) Friendly competition: evidence for a dilution effect among competitors in a planktonic host–parasite system. Ecology 90:791–801

Hall SR, Smyth R, Becker CR, Duffy MA, Knight CJ, MacIntyre S, Tessier AJ, Cáceres CE (2010) Why are *Daphnia* in some lakes sicker? Disease ecology, habitat structure, and the plankton. Bioscience 60:363–375

Hochachka WM, Dhondt AA (2000) Density-dependent decline of host abundance resulting from a new infectious disease. Proc Natl Acad Sci USA 97:303–530

Holmes JC (1995) Population regulation—a dynamic complex of interactions. Wildl Res 22:11–19

Hudson PJ, Dobson AP, Newborn D (1998) Prevention of population cycles by parasite removal. Science 282:2256–2258

Hudson PJ, Dobson AP, Lafferty KD (2006) Is a healthy ecosystem one that is rich in parasites? Trends Ecol Evol 21:381–385

Ives AR, Dennis B, Cottingham KL, Carpenter SR (2003) Estimating community stability and ecological interactions from time-series data. Ecol Monogr 73:301–330

Johnson PTJ, Stanton DE, Preu ER, Forshay KJ, Carpenter SR (2006) Dining on disease: how interactions between infection and environment affect predation risk. Ecology 87:1973–1980

Johnson PTJ, Ives AR, Lathrop RC, Carpenter SR (2009) Long-term disease dynamics in lakes: causes and consequences of chytrid infections in *Daphnia* populations. Ecology 90:132–144

Keesing F, Holt RD, Ostfeld RS (2006) Effects of species diversity on disease risk. Ecol Lett 9:485–498

LaDeau SL, Kilpatrick AM, Marra PP (2007) West Nile virus emergence and large-scale declines of North American bird populations. Nature 447:710–714

Lafferty KD, Kuris AM (2009) Parasitic castration: the evolution and ecology of body snatchers. Trends Ecol Evol 25:564–572

Lafferty KD, Dobson AP, Kuris AD (2006) Parasites dominate food web links. Proc Natl Acad Sci USA 103:11211–11216

Lafferty KD, Dobson AP, Kuris AM (2008) Parasites in food webs: the ultimate missing links. Ecol Lett 11:533–546

Lampert W, Sommer U (2007) Limnoecology: the ecology of lakes and streams. Oxford University Press, Oxford

Leibold MA (1991) Trophic interactions and habitat segregation between competing species of *Daphnia*. Oecologia 86:510–520

Littell RC, Stroup WW, Freund RJ (2002) SAS for linear models. 4th edn. Wiley, New York

May RM, Anderson RM (1978) Regulation and stability of host–parasite population interactions—2 destabilizing processes. J Anim Ecol 47:249–267

McCauley E, Nelson W, Nisbet R (2008) Small-amplitude cycles emerge from stage-structured interactions in *Daphnia*–algal systems. Nature 455:1240–1243

Mills EL, Forney JL, Wagner KJ (1987) Fish predation and its cascading effect on the Oneida Lake food chain. In: Kerfoot WC, Sih A (eds) Predation: direct and indirect impacts on aquatic ecosystems. The University Press of New England, Hanover, pp 118–131

Minchella DJ, Scott ME (1991) Parasitism—a cryptic determinant of animal community structure. Trends Ecol Evol 6:250–254

Mutze G, Cooke B, Alexander P (1998) The initial impact of rabbit hemorrhagic disease on European rabbit populations in South Australia. J Wildl Dis 34:221–227

Price PW (1980) Evolutionary biology of parasites. Princeton University Press, Princeton

Redpath SM, Mougeot F, Leckie FM, Elston DA, Hudson PJ (2006) Testing the role of parasites in driving the cyclic population dynamics of a gamebird. Ecol Lett 9:410–418

Scott ME (1988) The impact of infection and disease on animal populations: implications for conservation biology. Conserv Biol 2:40–56

Scott ME, Dobson A (1989) The role of parasites in regulating host abundance. Parasitol Today 5:176–183

Scott ME, Lewis JW (1987) Population dynamics of helminth parasites in wild and laboratory rodents. Mamm Rev 17:95–103

Singleton GR, Chambers LK, Spratt DM (1995) An experimental field-study to examine whether *Capillaria hepatica* (Nematoda) can limit house mouse populations in Eastern Australia. Wildl Res. 22:31–53

Smyth RL (2010) Stratification and turbulent mixing in small strongly stratified lakes with implications for planktonic disease dynamics. PhD thesis. University of California, Santa Barbara

Tessier AJ, Woodruff P (2002) Cryptic trophic cascade along a gradient of lake size. Ecology 83:1263–1270

Tompkins DM, Begon M (1999) Parasites can regulate wildlife populations. Parasitol Today 15:311–313

Van Buskirk J, Ostfeld RS (1995) Controlling Lyme disease by modifying density and species composition of tick hosts. Ecol Appl 5:1133–1140

Welschmeyer NA (1994) Fluorometric analysis of chlorophyll *a* in the presence of chlorophyll *b* and pheopigments. Limnol Oceanogr 39:1985–1992