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Selective predators and their parasitized prey: Are epidemics in zooplankton under top-down control?

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Abstract

Seasonal change in the intensity of fish predation affects succession in lake zooplankton communities. Predation affects not only the zooplankton prey, but also their parasites. Because the ability of a parasite to spread depends in part on the death rate of the hosts, seasonal reductions in the intensity of predation on zooplankton could lead to parasite epidemics. We examined seasonal population dynamics, mortality rate, and incidence of parasitism in lake populations of *Daphnia* to determine whether parasitism displayed seasonality and synchrony among lake populations and whether any such patterns are consistent with seasonal changes in predation rates. Infections of a bacterial parasite (*Spirobacillus cienkowskii*) in *Daphnia dentifera* populations were seasonal with epidemics in many lakes occurring synchronously in autumn. In situ foraging behavior of the dominant fish planktivores, bluegill sunfish, is highly selective on infected *Daphnia.* Mortality rates on the *Daphnia* drop just prior to the initiation of epidemics. An epidemiological model shows that this magnitude of decrease in mortality rate, if driven largely by a reduction in predation, can account for the seasonal occurrence of epidemics in our *Daphnia* populations. Together, these results suggest that parasitism in *Daphnia* populations may be seasonally restricted by fish predation.

Historically, ecologists studying food webs regarded parasites as add-ons (Marcogliese and Cone 1997), while ecologists studying parasitism focused on the isolated interactions of hosts and parasites. Yet the ability of a parasite to spread and persist in a host population depends critically on the other members of the community (e.g., competitors and predators; Holt 2003; Packer et al. 2003). For example, predators, when consuming their prey, also consume their prey's parasites. An increase in predation mortality not only directly harms the parasite, it also decreases the length of time an infected animal is in the population, making it less likely infected animals will contact and infect others. Hence, predation should decrease the likelihood of a parasite spreading through or persisting in a prey population (Grenfell and Dobson 1995). In addition, many predators preferentially kill diseased prey (Poulin 1994; Packer et al. 2003). When this occurs, the predation rate on the parasite will exceed that on the prey population at large, further restricting the conditions that allow a parasite to persist in a host population (Packer et al. 2003).

In the plankton of lakes and oceans, selective predation on infected zooplankters is likely to be common. Fish are highly selective predators, and zooplankton species that coexist with fish frequently rely on transparency to minimize predation risk (Zaret 1980). Because most parasites increase the opacity of the host, infected animals should be more easily detected by fish (Bittner et al. 2002). By selectively culling infected animals, fish might therefore reduce the prevalence of infection in zooplankton populations. Willey and colleagues (Willey et al. 1990, 1993) have suggested that this increase in visibility is at least partially responsible for decreases in the prevalence of epibionts on *Daphnia* in the presence of planktivorous fish. However, the intensity of selective predation by planktivores changes seasonally due to changing temperature, behavior, and ontogeny. Temperature mediates bioenergetic demands of the predators (Hewett and Johnson 1992), leading to the highest rates of fish for-

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aging in summer (Threlkeld 1979). In addition, the amount of time spent by fish foraging in the littoral versus limnetic regions shifts seasonally, with the most intense feeding in the limnetic zone occurring in summer (Werner 1969; Hall and Werner 1977; Threlkeld 1979). Finally, larval fish are important planktivores, and their recruitment in spring and summer can increase zooplankton mortality at these times (Rettig 2003).

Thus, we expect seasonal variation in the intensity of selective predation on zooplankton populations to be a factor in determining the occurrence of parasite epidemics. During periods of high predation in summer, fish should cull infected animals from zooplankton populations, thereby inhibiting the spread of parasites at this time. In autumn, when rates of planktivory should decrease, we expect that the incidence of parasite infections in zooplankton will increase.

In this study, we examine multiple lake populations of *Daphnia dentifera* facing predation by bluegill sunfish and parasitism by a bacterial pathogen. We quantify the in situ foraging selectivity of the fish predator for infected versus uninfected *D. dentifera* and follow the seasonal population dynamics of the *D. dentifera* and the prevalence of infection. These empirical findings are incorporated into a general host–parasite model, which we use to predict levels of fish predation that allow parasite invasion into these *D. dentifera* populations. By looking for correspondence between model predictions and field observations, we seek to determine whether selective fish predation may play a role in seasonally restricting parasitism in *D. dentifera* populations.

Methods

Study system—We studied *D. dentifera* Forbes populations in five lakes (Baker, Bassett, Cloverdale, and Pine Lakes, Barry County, and Three Lakes Two, Kalamazoo County) in southwestern Michigan. This *Daphnia* species is a common grazer in the planktonic food web of lakes throughout much of temperate North America (Hebert 1995; Duffy et al. 2004) and is the dominant zooplankter during summer in our study lakes. In these populations, *D. dentifera* hatch out of resting eggs in spring and are common throughout summer and autumn (Threlkeld 1979; Cáceres and Tessier 2004). Resting egg production occurs in mid-October through November, and there is little overwintering in the water column (Cáceres and Tessier 2004).

Since *Daphnia* are transparent, it is easy to detect the presence of internal parasites without dissection (Green 1974). We previously had observed that several microparasites become prevalent in *D. dentifera* populations during late summer and autumn. Here, we focus on a bacterial pathogen, *Spirobacillus cienkowskii* Metchnikoff. This microparasite is present in most local lakes, and we can easily detect infected animals since they are red. *S. cienkowskii* cells range in size from 0.5 to 2.5 μm. When placed in laboratory cultures with *S. cienkowskii*–infected *Daphnia*, uninfected *D. dentifera* become infected within several days. Once *Daphnia* show signs of infection (i.e., hemolymph becomes cloudy and red with bacterial cells), they generally die within ~2 days at 25°C and ~4 days at 15°C (M. A. Duffy unpubl. data). Further, this pathogen greatly reduces reproduction of infected *D. dentifera*, since infected animals are only very rarely observed with eggs (M. A. Duffy unpubl. data).

Bluegill sunfish (*Lepomis macrochirus* Rafinesque) are the most common planktivorous fish in our study lakes (Werner et al. 1977; Tessier and Woodruff 2002). As discussed above, the intensity of foraging by bluegill on *Daphnia* declines from summer to autumn because the fish, especially juveniles, spend more time in the littoral zone towards the end of the summer (Werner 1969; Hall and Werner 1977). In addition, bioenergetic demands and activity of fish decline with the drop in water temperature in the autumn (Collins and Hinch 1993; McDermot and Rose 2000).

In summer, *D. dentifera* minimize the risk of visual predation by migrating to deeper waters during the day, residing below the thermocline and at low (<10%) light levels (Leibold and Tessier 1997; M. A. Duffy and G. A. Gerrish unpubl. data). However, *D. dentifera* migrate to the warm surface waters at night in order to accelerate development time, especially of eggs (Leibold and Tessier 1997; Lampert et al. 2003). Consequently, most foraging by fish on *D. dentifera* occurs at dawn and at dusk as *D. dentifera* migrate between habitats.

Field data—We measured parasite prevalence and population dynamics in these five lake populations between July and November 2003. In previous years, infected animals were found throughout the period when *D. dentifera* is in the water column (i.e., late spring through autumn). However, infected animals remain rare until late August (M. A. Duffy and A. J. Tessier pers. obs.). We began sampling in July to collect preepidemic population dynamic data. The frequency of sampling varied based on the presence of infections and water temperature, with the average interval between samples being 6 days (generation time ranged from ~1 week in July to ~3 weeks in November). On each sampling date, we used a 153-μm mesh Wisconsin bucket net to collect four samples of the zooplankton. Each of these four samples constituted a pooling of whole water column, vertical net tows taken from four different sites within the deep basin of each lake. Three of the samples were preserved in 60%–90% ethanol and later counted for *D. dentifera* density. The remaining sample was analyzed immediately to determine infection prevalence (No. of infected *D. dentifera*/total No. of *D. dentifera*) and average fecundity (No. of eggs per individual). We determined the prevalence of infection by *S. cienkowskii* by examining a random subsample of at least 400 live *D. dentifera* under a stereomicroscope at 25–50× magnification.

We compared the seasonal change in *Daphnia* death rate from midsummer to immediately prior to the start of epidemics. To calculate death rate, we needed to determine population birth rate and the instantaneous population growth rate. We determined birth rate in each population by the egg ratio method (Paloheimo 1974). We recorded the number and developmental stage of eggs carried by 50–100 adult *D. dentifera* and the percentage of adult females in the sample. This information, along with the temperature-dependant egg development time, was used to calculate the per capita birth rate (Rigler and Downing 1984). Since these lakes are strat-

ified, the temperature experienced by the *D. dentifera* depends on the depth at which they live. We collected samples with a 20-liter Schindler trap at 1–2-m intervals during the day and at night to determine the diel vertical movement of the *D. dentifera* within each lake. In Bassett, Cloverdale, and Pine Lakes, these samples were collected at the beginning of the epidemic in August, and again in September in Pine Lake. A diel Schindler series was done in Baker Lake in July and in Three Lakes Two in September. In previous years, we have found that the diel vertical distributions of *D. dentifera* are relatively constant throughout this time period (M. A. Duffy unpubl. data). The vertical distributions were used to calculate a time-weighted temperature, and egg development times were determined according to Bottrell et al. (1976).

Calculation of death rate requires instantaneous population growth rate (r), which for the time interval $t = i$ to $t = j$ was calculated as

$$r_{ij} = \frac{\ln N_j - \ln N_i}{j - i} \quad (1)$$

where $\ln N_j$ and $\ln N_i$ are the natural log of the densities on sequential sampling days i and j. The instantaneous per capita death rate (d_i) of the population was calculated by subtracting the population growth rate (r_{ij}) from the per capita birth rate (b_i). Birth rates and natural log of densities were smoothed using proc loess in SAS prior to calculations (SAS Version 8, SAS Institute 1999).

Epidemics were arbitrarily defined as beginning when the prevalence of infection in the population surpassed 1%. This threshold was chosen since once the prevalence of infection surpassed 1%, the epidemic quickly rose to its peak value. Therefore, this provides us with a good metric for when the parasite has established itself in a population. We chose to use a relatively low threshold to minimize the effect of the parasite itself on the population death rate, allowing us to better compare death rates at the beginning of an epidemic with those before. Therefore, we averaged death rates on the day the epidemic surpassed 1% and the previous and subsequent sampling dates to characterize a mean death rate for the beginning of the epidemic. A preepidemic death rate was determined by averaging death rates from three sequential sampling dates 1 month before the start of the epidemic. We were unable to characterize the preepidemic death rate in Bassett Lake, since it occurred before we began sampling. Baker Lake did not have an *S. cienkowskii* epidemic, but is still of interest for comparative purposes. If death rate changes in lakes that have epidemics, but not in Baker Lake, that would provide additional support for our hypothesis. We used the average date when the other lakes were beginning their epidemics as the date for the start of the ''epidemic'' for Baker Lake. For the three lakes that had epidemics and for which we could characterize the preepidemic and start of epidemic death rates, we used a paired *t*-test to compare the death rates from the two periods. To explore whether changes in other population parameters are correlated with the onset of infections, we also used paired *t*-tests to compare the egg ratio of adult females (No. eggs/adult female), population birth rate, and *D. dentifera* density in these three lakes from the two periods.

We quantified the selectivity of bluegill sunfish foraging on infected *Daphnia* by comparing the percentage of infected adult *D. dentifera* in bluegill stomachs with the composition of the adult *D. dentifera* population in the bluegill's foraging environment. Since bluegill feed only in the epilimnion of these lakes (McDermot and Rose 2000), we compared the proportion of infected *D. dentifera* in the fish guts with the proportion in the epilimnion. In this case, we know that the vertical distribution of infected *D. dentifera* does not differ from that of uninfected *D. dentifera* based on diel Schindler trap studies taken in our study lakes (S. R. Hall and M. A. Duffy unpubl. data). Hence, the estimated selectivity from the epilimnion is equivalent to the selectivity of bluegill on infected *D. dentifera* in the population as a whole.

Fish were collected at dawn by angling in Baker, Bassett, Cloverdale, and Pine Lakes in 2002 and 2003. The gut contents of 3–10 bluegill were analyzed per lake, with an average of over 300 infected *D. dentifera* per gut. The proportion of infected *D. dentifera* in the environment was determined by collecting replicate net tows in the epilimnion immediately preceding angling and examining all *D. dentifera* in each sample using a stereomicroscope.

Selectivity was calculated using Chesson's alpha (Chesson 1983):

$$\alpha_I = \frac{\frac{g_I}{e_I}}{\left(\frac{g_I}{e_I} + \frac{g_S}{e_S}\right)} \quad (2)$$

where α_I is the selectivity of bluegill on infected *D. dentifera*, g_I and g_S are, respectively, the proportions of infected and uninfected (and presumably susceptible) adult *D. dentifera* in the fish gut, and e_I and e_S are the proportions of infected and uninfected adult *D. dentifera* in the environment, respectively. An alpha of 0.5 indicates neutral selectivity, and values greater than 0.5 indicate preferential feeding on a prey type. Since there are only two prey types, α_S, the selectivity on uninfected *D. dentifera*, is $(1 - \alpha_I)$.

Modeling—We developed a compartment model, based on a susceptible–infected (*SI*) model (Heesterbeek and Roberts 1995), to explore the range of death rates over which *S. cienkowskii* can invade populations of *D. dentifera*. The dynamics of our model can be described using the following two equations:

$$\frac{dS}{dt} = bS\left(1 - \frac{S + I}{K}\right) - nS - \beta SI - m_S S \quad (3)$$

$$\frac{dI}{dt} = \beta SI - (n + \nu)I - m_I I \quad (4)$$

(*see Table 1* for a summary of symbols used in the model). In this model, growth of the *D. dentifera* population is negatively density dependent, and infected *D. dentifera* (I) do not contribute to reproduction, as infected animals are only very rarely observed with eggs (M. A. Duffy unpubl. data). Infected *D. dentifera* transmit *S. cienkowskii* to susceptible

Table 1. Model parameters and variables.

Par./var.	Units	Definition	Value/range
α_I	—	Chesson's alpha; selectivity on infected *D. dentifera*	0.5–1*
b	d^{-1}	Maximum birth rate	0.4†
β	$(No./L)^{-1}\ d^{-1}$	Transmission rate	0.05, 0.1‡
I	No. L^{-1}	Density of infecteds	—
K	No. L^{-1}	Carrying capacity	10, 20§
m_I	d^{-1}	Death rate due to fish predation, infecteds	—
m_S	d^{-1}	Death rate due to fish predation, susceptibles	0–0.4‖
n	d^{-1}	Nonselective predation death rate	0.05¶
S	No. L^{-1}	Density of susceptibles	—
t	d	Time unit	—
ν	d^{-1}	Death rate due to parasite (virulence)	0.05, 0.15#

* This covers the range from unselective on infecteds ($\alpha_I = 0.5$) to the theoretical maximum ($\alpha_I = 1.0$).
† Maximum birth rate for *D. dentifera* at summer temperatures was based on laboratory life table studies using food collected from local lakes (Tessier and Woodruff 2002).
‡ Based on Ebert et al. (2000).
§ This range is based on the densities at which *Daphnia* equilibrate in lake enclosure experiments in which predators are excluded (Tessier et al. 2000, 2001, A. J. Tessier unpubl. data).
‖ This covers the range of death rates observed in these populations over the study period (M. A. Duffy unpubl. data).
¶ Death rate from sources other than parasite virulence and selective fish predation; our conclusions do not vary qualitatively with changes in this parameter (explored over the range $n = 0.01$–$0.15\ d^{-1}$) and, therefore, only results for $n = 0.05\ d^{-1}$ are shown. These values are typical for these lake populations, based on sediment trap measurements (M. A. Duffy, unpubl. data).
# Based on life table studies of infected animals collected from local lakes (M. A. Duffy, A. J. Tessier, and S. R. Hall, unpubl. data).

hosts at rate β according to a density-dependent, pseudo–mass action (de Jong et al. 1995) interaction (βSI). Death of *D. dentifera* is split into several terms: m_S and m_I, which are the per capita death rates due to fish predation on susceptible and infected *D. dentifera*, respectively; ν, which is virulence, i.e., the per capita death rate on infected *D. dentifera* resulting directly from the infection; and n, which is the per capita death rate from other sources.

To incorporate selective predation into the model, we converted the fish selectivity measured in the lakes (using Chesson's alpha) into one that is dependent on the per capita rates of mortality from fish predation. We assume the ratio of the proportion of a prey type in the fish gut to its proportion in the environment (i.e., the numerator of Eq. 2) is proportional to the per capita predation mortality rate (m) for that prey type. Therefore,

$$\alpha_I = \frac{m_I}{m_I + m_S} \tag{5}$$

Since α_I and α_S sum to one when there are only two prey types, we can rearrange Eq. 5 by solving for m_I,

$$m_I = \left(\frac{\alpha_I}{1 - \alpha_I}\right) m_S \tag{6}$$

Thus, for cases where the predator is not selective on infected prey ($\alpha_I = 0.5$), m_I equals m_S and fish feed on both susceptibles and infecteds at the same rate.

We use our model to derive conditions under which the parasite is expected to invade the *D. dentifera* population (*see Web Appendix 1* for details; http://www.aslo.org/lo/toc/vol_50/issue_2/0412al.pdf). Values for model parameters were based on the *D. dentifera* population dynamics we measured in these study lakes or on prior laboratory and lake experiments (*see Table 1* for details). For key parameters, a realistic range of values was explored. Specifically, *Daphnia* carrying capacity is known to vary with lake productivity, so we used a range representative of the productivity of our study lakes, as determined by enclosure experiments that excluded predators (Tessier et al. 2000, 2001; A. J. Tessier unpubl. data). Similarly, transmission rates are difficult to measure in nature and poorly known for any *Daphnia* pathogen. Therefore, we considered a range based on published studies on several *Daphnia* microparasites (Ebert et al. 2000) and supported by our laboratory observations of infection probability. Variation in some parameters (maximum birth rate, b, and background mortality, n) over ranges common in lake populations had no qualitative effect on our conclusions, so we present only a single, representative value. We measured the virulence of this and other pathogens common in these lakes in laboratory experiments, conducted over a range of temperatures (15–25°C). We determined virulence, measured as the mortality rate of infected animals, using both infected animals collected from the field and animals infected in the lab. We used values that bracket the variation in virulence: $0.05\ d^{-1}$ at 15°C to $0.15\ d^{-1}$ at 25°C (M. A. Duffy, S. R. Hall, and A. J. Tessier unpubl. data).

Our primary use of this model is to explore whether differences in the rate of selective predation on *D. dentifera* populations between lakes and seasons can explain differences in prevalence of parasitism. The ability of the parasite to invade the *D. dentifera* population depends on the total mortality rate from selective predation. In our model this term is determined by the rate of fish predation on susceptibles (m_S) and the selectivity of fish on infecteds (α_I). We vary m_S over the range in population death rates we observed in the lakes during the study (0–$0.4\ d^{-1}$). We consider predator selectivity on infecteds to range from nonselective ($\alpha_I = 0.5$) to the theoretical maximum ($\alpha_I = 1.0$). Model predictions are compared with observed differences in mortality rates and parasite prevalence, both of which vary between seasons and lakes.

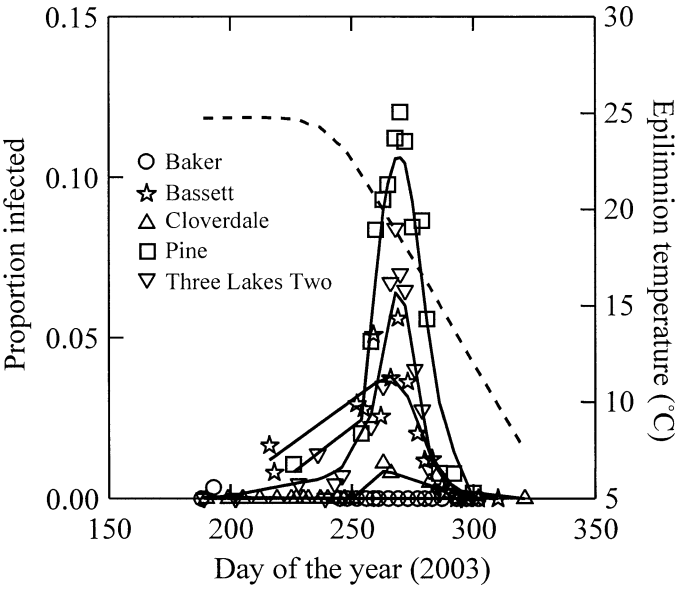

Fig. 1. Seasonal change in infection prevalence (solid lines) and surface water temperature (dashed line). Locally weighted (LOESS) smoothing lines are shown.

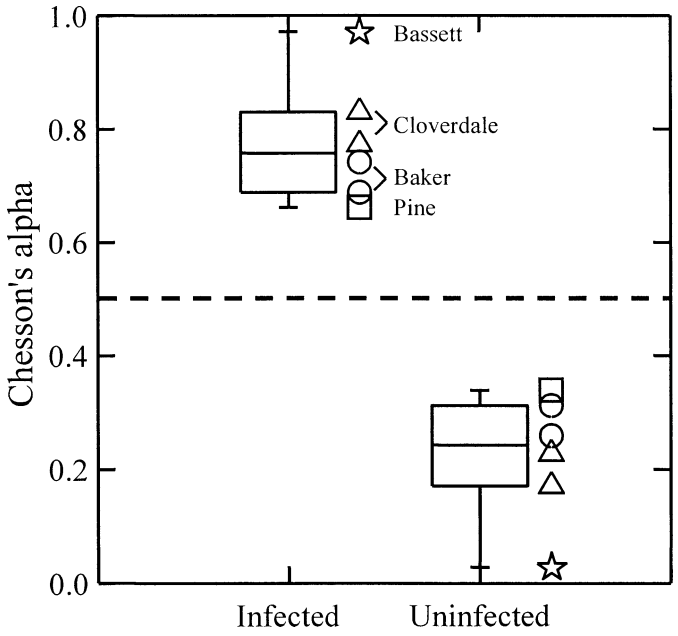

Fig. 2. Fish selectivity on *S. cienkowskii*–infected and uninfected *D. dentifera*. Symbols indicate mean Chesson's alpha for each lake day. The line at $\alpha = 0.50$ indicates neutral selectivity. Fish were collected from Cloverdale Lake at the start of the epidemic that occurred in the fall of 2002, from Baker Lake during the short period of time when *S. cienkowskii* infections were present on one date in 2002 and one in July 2003, and from Bassett and Pine lakes at the start of their epidemics in 2003.

Results

We observed *D. dentifera* infected with *S. cienkowskii* in all lakes, with incidence of infection very low prior to August. The prevalence of infection increased sharply in September, concurrent with a decrease in surface water temperature (Fig. 1). Three of the five study populations (Bassett Lake, Pine Lake, and Three Lakes Two) had large *S. cienkowskii* epidemics. The epidemics (defined as the point when the prevalence of infection exceeded 1%) in Pine Lake and Three Lakes Two began at approximately the same time, while the epidemic in Bassett started slightly earlier. In Cloverdale Lake the infection peaked at only 1% infected *D. dentifera*. Broadly speaking, the epidemics in these four, isolated lake populations were synchronous, all peaking at the same time (Fig. 1). The prevalence of infection in Baker Lake never exceeded 0.3%; therefore, we did not consider this population to have experienced an epidemic.

Bluegill foraging in each lake were highly selective on *D. dentifera* infected with *S. cienkowskii* (Fig. 2). The mean Chesson's alpha for infected *D. dentifera* was 0.76 (95% confidence interval: 0.67–0.85).

For these values of fish selectivity ($\alpha_I \approx 0.7$–0.8) our model predicts a range of realistic mortality rates that allow parasite invasion. Invasion of the parasite requires

$$\hat{m}_S < \frac{b(\beta K - n - v) - n\beta K}{\beta K + \left(\frac{\alpha_I}{1 - \alpha_I}\right) b} \qquad (7)$$

(*see Web Appendix 1* for details). The isoclines on Fig. 3 depict those values where on average each infected individual infects one additional individual. In the area below the isocline each new infection results in more than one additional infection, and the parasite can spread through the population. At high carrying capacity, the isoclines fall between mortality rates of approximately 0.15 and 0.20 d^{-1} (Fig. 3). At low carrying capacity, they are between approximately 0.05 and 0.15 d^{-1} (Fig. 3). Overall mortality rates in these lake populations ranged from approximately 0.10 to 0.25 (Fig. 3). Therefore, if mortality rates in these populations are primarily driven by selective fish predation, our model predicts that even small decreases in the rates of selective predation on these *D. dentifera* populations may allow parasite epidemics.

There was a decrease in population death rate at the start of the epidemics in Three Lakes Two, Cloverdale, and Pine Lakes compared with rates earlier in summer (Fig. 3; paired $t_2 = 5.438$, $p = 0.03$). There was a trend toward decreased adult female egg ratio in these lakes over this same time period, but it was not significant (paired $t_2 = 2.066$, $p = 0.18$); population birth rate showed the same trend, but was also not significant (paired $t_2 = 1.452$, $p = 0.28$). *D. dentifera* density increased in one of these lakes (Three Lakes Two) during this time span but decreased in the other two. Therefore, change in *D. dentifera* density in these lakes over this time period was not significant (paired $t_2 = 0.012$, $p = 0.99$).

The observed decline in population death rate occurs in a range where our model predicts that, for reasonable combinations of carrying capacity, transmission rate, and parasite virulence, a decrease in fish predation can allow parasite spread. We do not have preepidemic death rates for Bassett Lake, but death rates measured immediately prior to the ma-

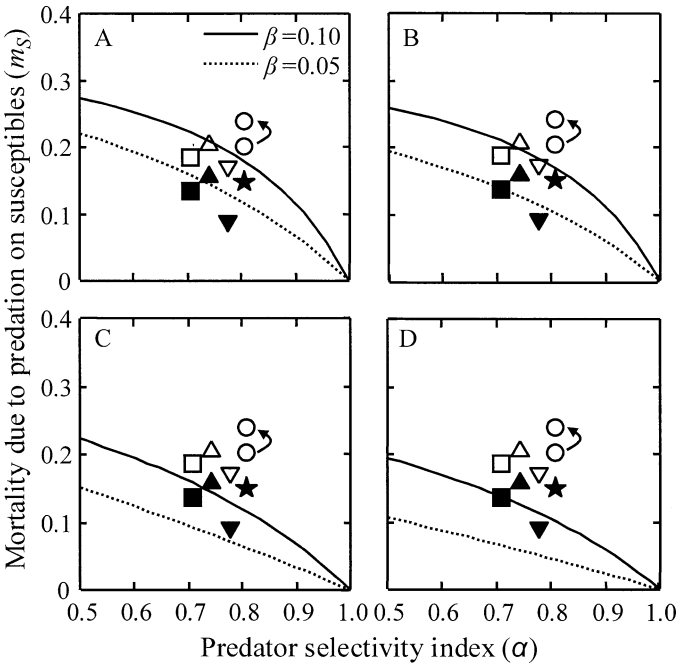

Fig. 3. Parasite invasion thresholds over a range of selectivity and predation mortality. Invasion thresholds are shown for two carrying capacities (A and B, $K = 20$ L^{-1}; C and D, $K = 10$ L^{-1}), two virulences (A and C, $v = 0.05$ d^{-1}; B and D, $v = 0.15$ d^{-1}), and two transmission rates (β). For a given parameter set, the parasite can invade at combinations below the lines. The symbols (as in Fig. 1) show overall death rate for individual populations before (open symbols) and at the start of (filled) an epidemic. This death rate (d^{-1}) is plotted at the average fish selectivity (0.76) with values offset along the *x*-axis for visibility. Note that points refer to measured death rates, while the *x*-axis refers to modeled death rates due to fish predation. Baker Lake did not have an epidemic, and the points depicted show the mean death rate for the same dates as in Pine Lake and Three Lakes Two (the upper symbol is the later date). There is no preepidemic death rate data for Bassett Lake; therefore, only one value is plotted for this lake.

jor increase in parasite prevalence also fall in the region of parameter space allowing parasite invasion. Interestingly, the Baker Lake population maintained a high death rate throughout this same period and did not have an epidemic.

Discussion

Our results illustrate that parasitism increases synchronously in multiple lake *Daphnia* populations concurrent with decreasing temperature in autumn. While not shown here, we have noted that other species of *Daphnia* and their parasites also display increased incidence of infection at this time of the year. This suggests that zooplankton–parasite dynamics are strongly influenced by external factors (Ranta et al. 1995), i.e., seasonal change in temperature and photoperiod. By combining studies of population dynamics, fish selectivity, and parameterization of a general epidemiological model, we illustrate the plausibility that these epidemics occur in response to a seasonal decrease in selective fish predation. Specifically, we hypothesize that parasite epidemics are prevented during midsummer by high mortality from fish predation. However, the intensity of fish foraging is reduced in autumn due to both decreased bioenergetic demands (Collins and Hinch 1993; McDermot and Rose 2000) and movement of fish to their winter habitats (Werner 1969; Hall and Werner 1977). In support of this hypothesis, we observed that *D. dentifera* populations that experienced parasite epidemics displayed significantly lowered mortality rates immediately prior to the start of the epidemic. In addition, the one lake (Baker Lake) that maintained a high mortality rate throughout the study period did not have an epidemic.

The observed patterns of infection were not correlated with several other factors measured during this study. Since new infections depend on both the transmission rate and the number of individuals (Eqs. 3, 4), changes in population density might influence epidemics. However, we saw no clear relationship between changes in population density and the onset of epidemics: population density increased in two lakes and decreased in a third during the period immediately preceding the epidemic. There was a trend toward decreased egg ratio (and, therefore, decreased birth rates), which might indicate lowered food quality and therefore food stress on the host. However, increased food stress would actually make it more difficult for a parasite to invade a population, since parasite growth is much higher in well-fed hosts (Pulkkinen and Ebert 2004). Thus, of the population parameters measured in this study, population death rate is the best correlate of infection.

We acknowledge that we do not have direct measures of the intensity of mortality from fish and that a strong test of this hypothesis will require manipulation of fish predation. The plausibility of our hypothesis rests on known fish ecology (Hall and Werner 1977; Werner et al. 1977; McDermot and Rose 2000) and a striking correspondence of model predictions, observed changes in *Daphnia* mortality rate, and infection incidence. Additional factors undoubtedly influence parasite epidemics in zooplankton, but any alternative explanation for the pattern of the observed epidemics must account for the clear seasonality.

Of course, a direct effect of temperature is an obvious alternative mechanism for the seasonal synchrony in epidemics. Temperature may directly impact the parasite's growth or modify its rate of transmission. However, declining temperature reduces the growth rate of bacteria, which is not a reasonable explanation for the initiation of epidemics in autumn. Temperature would be a sufficient explanation if the thermal tolerance for this parasite is such that it is unable to grow at the higher temperatures in summer. Summer epilimnion temperatures in our lakes are typically around 25°C, and the epidemics largely occurred as temperatures dropped to ~20°C. However, in laboratory studies, *S. cienkowskii* is able to infect *D. dentifera* at 25°C (M. A. Duffy unpubl. data). Further, infections were observed in these lakes at very low levels throughout the summer. Hence, thermal tolerance per se is unlikely to be driving the synchronous onset of epidemics.

Temperature may also influence parasite virulence, which is why we considered a range of values in our modeling, reflecting laboratory data on death rate of infected animals

at temperatures from 15 to 25°C. The virulence effect of this temperature range on parasite thresholds for spread was subtle compared to changes in mortality. Finally, temperature might influence parasite transmission, but declining temperature should inhibit rather than facilitate parasite spread. The rate at which *Daphnia* filter particles decreases at low temperatures (Mourelatos and Lacroix 1990). While the mode of infection for this parasite is unknown, many common parasites infect *Daphnia* through the gut wall after being ingested (Capaul and Ebert 2003). Therefore, seasonal lake cooling will cause a decrease in *Daphnia* ingestion rates that could decrease transmission rate. Hence, while low temperatures may explain the synchronous termination of the epidemics through a decrease in transmission success and growth rate of the parasite, it does not appear to explain epidemic initiation in autumn.

The selectivity of fish foraging has the potential to strongly influence zooplankton–parasite dynamics. Bluegill sunfish (*L. macrochirus*) were consistently selective among lakes on *D. dentifera* infected with *S. cienkowskii,* with the selectivity values in most lakes around 0.75. This means that, for equal densities of uninfected and infected *D. dentifera,* fish consume three infected *D. dentifera* for every uninfected animal. Since our measure of selectivity is based on multiple fish in multiple lakes, dates, and years, this result is broadly relevant for this *Daphnia*–parasite system.

Selective foraging by fish on infected zooplankton, such as we report for bluegill, is likely to be a general situation for plankton communities. Essentially all zooplankton species that coexist with fish rely on transparency to reduce predation risk (Zaret 1980), and most parasites will increase the opacity of their zooplankton hosts. Thus, we expect infection prevalence to be low at times when fish predation is highest (i.e., in summer) and to increase at times of lower fish predation (e.g., in spring and autumn). An earlier study (Willey et al. 1993) found that the prevalence of pigmented epibionts on zooplankton in a pond was significantly reduced after the addition of planktivorous fish. Additional support for our hypothesis that parasite epidemics should be seasonally restricted by fish predation comes from a study on Lake Constance, in which infections of *Daphnia* by a protozoan parasite were only detected in autumn and winter (Bittner et al. 2002). Therefore, our hypothesis that selective predation by fish predators can prevent parasite epidemics is likely to be broadly applicable to other aquatic systems.

Although *S. cienkowskii* turns the *Daphnia* red, it is unclear what role the coloration per se plays in the interaction with fish predators. Red wavelengths attenuate quickly in low-light aquatic environments. Hence from the bluegill's perspective, the dominant feature of *S. cienkowskii*–infected *Daphnia* is likely their opacity, not their color. We have observed that the selectivity of bluegill for *Daphnia* infected with other (nonred) parasites is as high or higher than for those infected with *S. cienkowskii* (M. A. Duffy unpubl. data).

In theory, predators could selectively avoid infected prey (i.e., $\alpha < 0.5$), which should facilitate parasite spread even under moderately high predation (*see Web Appendix 1*). However, avoidance of diseased prey is not typical of predators and is especially hard to imagine for fish foraging in the plankton. Infected animals might become spatially segregated from predators, and this heterogeneity also could mediate parasite spread by providing a refuge from predation. Spatial segregation is not, however, the same as selective foraging behavior. Although we observed no vertical segregation of infected and uninfected hosts in our lakes (S. R. Hall and M. A. Duffy unpubl. data), it is plausible that morbidity of hosts infected by other parasites could restrict those hosts to deeper waters that are inaccessible to fish predators. Alternatively, genetic variation in vertical habitat use and susceptibility could also spatially segregate infected hosts from predators (Decaestecker et al. 2002). In both of these scenarios, infected animals may experience lower per capita predation rates on average than uninfected animals. However, spatial structure of the host population would affect more than just predation, likely impacting other model parameters such as transmission, birth rates, and death rates. Therefore, it is difficult to say whether such segregation would facilitate parasite invasion without analysis of a more appropriate spatially structured model.

The communities in which this host and parasite occur are more complex than we have portrayed in the model. While *S. cienkowskii* is primarily found in *D. dentifera,* it also infects other daphniids whose abundance varies among lakes and seasonally within lakes. In addition, these lakes also contain other predators such as the invertebrates *Chaoborus* and *Leptodora.* While these are tactile predators and unlikely to prey selectively upon infected *Daphnia,* they can impose a substantial mortality on the *D. dentifera* population (González and Tessier 1997). In cases where invertebrate mortality is high, it would make it more difficult for the parasite to persist in the system (Anderson and May 1981). One of our lakes, Baker Lake, did not show a decrease in *Daphnia* mortality that we expected from seasonal changes in fish predation. This lake is similar to other lakes in seasonal pattern in temperature (M. A. Duffy unpubl. data) and in its fish community (M. A. Duffy and A. J. Tessier pers. obs.). However, this lake is the most productive of our study lakes, with high dissolved organic carbon (A. J. Tessier unpubl. data) and numerous invertebrate predators; therefore, invertebrate predation may be more important than fish predation in this lake (Wissel et al. 2003). Further work is required to determine whether the absence of an epidemic in Baker Lake is related to fish predation being a smaller proportion of the overall mortality.

Our model allowed us to quantitatively evaluate the plausibility of our hypothesis that selective fish predation seasonally restricts parasitism in *Daphnia* populations. As predicted by other models (Packer et al. 2003), selective predation greatly decreases the ability of a parasite to invade a host population. More interestingly, for this *Daphnia*–parasite system, our model predicts that if the mortality rates in the field are largely driven by fish predation even small decreases in this predation can allow parasite epidemics. However, the ability of a parasite to invade a population depends on numerous factors. While we have attempted to include major biotic drivers of host–parasite interactions, the model is general and fairly simplistic with respect to other possible influences. For example, it does not predict synchronous termination of the epidemics, possibly because, as already dis-

cussed, we have not included the effect of declining temperature on bacteria growth and transmission. Additionally, we have ignored heterogeneity among *D. dentifera* clones in resistance and seasonal change in clonal composition of these populations. Hence, while our results provide the first evidence for seasonal synchrony of zooplankton–parasite epidemics and support the plausibility of their top-down control, testing this and alternative hypotheses will require manipulative experiments.

The idea that it is important to consider the community context in which host–parasite interactions occur is not new. Early theoretical work demonstrated that the outcomes of host–parasite interactions can change when studies of parasitism include competitors and predators (e.g., Anderson and May 1981; Price et al. 1986). Growing evidence indicates that parasite spread and persistence in a host population depends on the food web in which the host and parasite occur (Holt 2003; Packer et al. 2003; Ostfeld and Holt 2004). However, with few exceptions (e.g., Ives and Murray 1997; Hudson et al. 1998), empirical studies on the community ecology of parasitism have lagged behind theory. We suggest that it is not only important to consider other community members, but also to realize that community interactions are dynamic. Taking advantage of temporal changes will improve our understanding of the unique ways in which the joint interactions of predators, pathogens, and prey–host populations shape ecological and evolutionary dynamics.

References

ANDERSON, R. M., AND R. M. MAY. 1981. The population dynamics of microparasites and their invertebrate hosts. Philos. Trans. R. Soc. Lond., B **291:** 451–524.

BITTNER, K., K.-O. ROTHHAUPT, AND D. EBERT. 2002. Ecological interactions of the microparasite *Caullerya mesnili* and its host *Daphnia galeata.* Limnol. Oceanogr. **47:** 300–305.

BOTTRELL, H. H., AND OTHERS. 1976. A review of some problems in zooplankton production studies. Nor. J. Zool. **24:** 419–456.

CÁCERES, C. E., AND A. J. TESSIER. 2004. To sink or swim: Variable diapause strategies among *Daphnia* species. Limnol. Oceanogr. **49:** 1333–1340.

CAPAUL, M., AND D. EBERT. 2003. Parasite-mediated selection in experimental *Daphnia magna* populations. Evolution **57:** 249–260.

CHESSON, J. 1983. The estimation and analysis of preference and its relationship to foraging models. Ecology **64:** 1297–1304.

COLLINS, N. C., AND S. G. HINCH. 1993. Diel and seasonal-variation in foraging activities of pumpkinseeds in an Ontario pond. Trans. Am. Fish. Soc. **122:** 357–365.

DE JONG, M. C. M., O. DIEKMANN, AND H. HEESTERBEEK. 1995. How does transmission of infection depend on population size?, pp. 84–94. *In* D. Mollison [ed.], Epidemic models: Their structure and relation to data. Cambridge University Press.

DECAESTECKER, E., L. DE MEESTER, AND D. EBERT. 2002. In deep trouble: Habitat selection constrained by multiple enemies in zooplankton. Proc. Natl. Acad. Sci. USA **99:** 5481–5485.

DUFFY, M. A., A. J. TESSIER, AND M. KOSNIK. 2004. Testing the ecological relevance of *Daphnia* species designations. Freshw. Biol. **49:** 55–64.

EBERT, D., M. LIPSITCH, AND K. L. MANGIN. 2000. The effect of parasites on host population density and extinction: Experimental epidemiology with *Daphnia* and six microparasites. Am. Nat. **156:** 459–477.

GONZÁLEZ, M. J., AND A. J. TESSIER. 1997. Habitat segregation and interactive effects of multiple predators on a prey assemblage. Freshw. Biol. **38:** 179–191.

GREEN, J. 1974. Parasites and epibionts of Cladocera. Trans. Zool. Soc. Lond. **32:** 417–515.

GRENFELL, B. T., AND A. P. DOBSON. 1995. Ecology of infectious diseases in natural populations. Cambridge University Press.

HALL, D. J., AND E. E. WERNER. 1977. Seasonal distribution and abundance of fishes in the littoral zone of a Michigan lake. Trans. Am. Fish. Soc. **106:** 545–555.

HEBERT, P. D. N. 1995. The *Daphnia* of North America: An illustrated fauna. CyberNatural Software.

HEESTERBEEK, J. A. P., AND M. G. ROBERTS. 1995. Mathematical models for microparasites of wildlife, pp. 90–122. *In* B. T. Grenfell and A. P. Dobson [eds.], Ecology of infectious diseases in natural populations. Cambridge University Press.

HEWETT, S. H., AND B. L. JOHNSON. 1992. Fish bioenergetics 2: A generalized model of fish growth for microcomputers. University of Wisconsin Sea Grant Technical Report Number WIS-SG-92-250.

HOLT, R. D. 2003. Parasite establishment in host communities. Ecol. Lett. **6:** 837–842.

HUDSON, P. J., A. P. DOBSON, AND D. NEWBORN. 1998. Prevention of population cycles by parasite removal. Science **282:** 2256–2258.

IVES, A. R., AND D. L. MURRAY. 1997. Can sublethal parasitism destabilize predator-prey population dynamics? A model of snowshoe hares, predators and parasites. J. Anim. Ecol. **66:** 265–278.

LAMPERT, W., E. MCCAULEY, AND B. F. J. MANLY. 2003. Trade-offs in the vertical distribution of zooplankton: ideal free distribution with costs? Proc. R. Soc. Lond., B **270:** 765–773.

LEIBOLD, M. A., AND A. J. TESSIER. 1997. Habitat partitioning by zooplankton and the structure of lake ecosystems, pp. 3–30. *In* B. Streit, T. Städler, and C. M. Lively [eds.], Evolutionary ecology of freshwater animals. Birkhäuser Verlag.

MARCOGLIESE, D. J., AND D. K. CONE. 1997. Food webs: A plea for parasites. Trends Ecol. Evol. **12:** 320–325.

MCDERMOT, D., AND K. A. ROSE. 2000. An individual-based model of lake fish communities: Application to piscivore stocking in Lake Mendota. Ecol. Model. **125:** 67–102.

MOURELATOS, S., AND G. LACROIX. 1990. In situ filtering rates of Cladocera: Effect of body length, temperature and food concentration. Limnol. Oceanogr. **35:** 1101–1111.

OSTFELD, R. S., AND R. D. HOLT. 2004. Are predators good for your health? Evaluating evidence for top-down regulation of zoonotic disease reservoirs. Front. Ecol. Environ. **2:** 13–20.

PACKER, C., R. D. HOLT, P. J. HUDSON, K. D. LAFFERTY, AND A. P. DOBSON. 2003. Keeping the herds healthy and alert: Implications of predator control for infectious disease. Ecol. Lett. **6:** 797–802.

PALOHEIMO, J. E. 1974. Calculation of instantaneous birth rate. Limnol. Oceanogr. **19:** 692–694.

POULIN, R. 1994. Meta-analysis of parasite-induced behavioural changes. Anim. Behav. **48:** 137–146.

PRICE, P. W., M. WESTOBY, B. RICE, P. R. ATSATT, R. S. FRITZ, J. N. THOMPSON, AND K. MOBLEY. 1986. Parasite mediation in ecological interactions. Annu. Rev. Ecol. Syst. **17:** 487–505.

PULKKINEN, K., AND D. EBERT. 2004. Host-starvation decreases parasite load and mean host size in experimental populations. Ecology **85:** 823–833.

RANTA, E., V. KAITALA, J. LINDSTRÖM, AND H. LINDÉN. 1995. Synchrony in population dynamics. Proc. R. Soc. Lond., B **262:** 113–118.

RETTIG, J. E. 2003. Zooplankton responses to predation by larval bluegill: An enclosure experiment. Freshw. Biol. **48:** 636–648.

RIGLER, F. H., AND J. A. DOWNING. 1984. The calculation of secondary productivity, pp. 19–58. *In* J. A. Downing and F. H. Rigler [eds.], A manual on methods for the assessment of secondary productivity in freshwater. International Biological Program Handbook 17. Blackwell Scientific.

SAS INSTITUTE. 1999. SAS/STAT user's guide, version 8. SAS Institute Inc., Cary, North Carolina, USA.

TESSIER, A. J., E. V. BIZINA, AND C. K. GEEDEY. 2001. Grazer-resource interactions in the plankton: Are all daphniids alike? Limnol. Oceanogr. **46:** 1585–1595.

———, M. A. LEIBOLD, AND J. TSAO. 2000. A fundamental trade-off in resource exploitation by *Daphnia* and consequences to plankton communities. Ecology **81:** 826–841.

———, AND P. WOODRUFF. 2002. Cryptic trophic cascade along a gradient of lake size. Ecology **83:** 1263–1270.

THRELKELD, S. T. 1979. The midsummer dynamics of two *Daphnia* species in Wintergreen Lake, Michigan. Ecology **60:** 165–179.

WERNER, E. E., D. J. HALL, D. R. LAUGHLIN, D. J. WAGNER, L. A. WILSMANN, AND F. C. FUNK. 1977. Habitat partitioning in a freshwater fish community. J. Fish. Res. Board Can. **34:** 360–370.

WERNER, R. G. 1969. Ecology of limnetic bluegill (*Lepomis macrochirus*) fry in Crane Lake, Indiana. Am. Midl. Nat. **81:** 164–181.

WILLEY, R. L., P. A. CANTRELL, AND S. T. THRELKELD. 1990. Epibiotic euglenoid flagellates increase the susceptibility of some zooplankton to fish predation. Limnol. Oceanogr. **35:** 952–959.

———, R. B. WILLEY, AND S. T. THRELKELD. 1993. Planktivore effects on zooplankton epibiont communities: Epibiont pigmentation effects. Limnol. Oceanogr. **38:** 1818–1822.

WISSEL, B., W. J. BOEING, AND C. W. RAMCHARAN. 2003. Effects of water color on predation regimes and zooplankton assemblages in freshwater lakes. Limnol. Oceanogr. **48:** 1965–1976.

ZARET, T. M. 1980. Predation and freshwater communities. Yale University Press.

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