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Long-term disease dynamics in lakes: causes and consequences of chytrid infections in *Daphnia* populations

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Abstract. Understanding the drivers and consequences of disease epidemics is an important frontier in ecology. However, long-term data on hosts, their parasites, and the corresponding environmental conditions necessary to explore these interactions are often unavailable. We examined the dynamics of *Daphnia pulicaria*, a keystone zooplankter in lake ecosystems, to explore the long-term causes and consequences of infection by a chytridiomycete parasitoid (*Polycaryum laeve*). After quantifying host–pathogen dynamics from vouchered samples collected over 15 years, we used autoregressive models to evaluate (1) hypothesized drivers of infection, including host density, water temperature, dissolved oxygen, host-food availability, and lake mixing; and (2) the effects of epidemics on host populations. Infection was present in most years but varied widely in prevalence, from <1% to 34%, with seasonal peaks in early spring and late fall. Within years, lake stratification strongly inhibited *P. laeve* transmission, such that epidemics occurred primarily during periods of water mixing. Development of the thermocline likely reduced transmission by spatially separating susceptible hosts from infectious zoospores. Among years, ice duration and cumulative snowfall correlated negatively with infection prevalence, likely because of reductions in spring phytoplankton and *D. pulicaria* density in years with extended winters. Epidemics also influenced dynamics of the host population. Infected *D. pulicaria* rarely (<1%) contained eggs, and *P. laeve* prevalence was positively correlated with sexual reproduction in *D. pulicaria*. Analyses of *D. pulicaria* density-dependent population dynamics predicted that, in the absence of *P. laeve* infection, host abundance would be 11–50% higher than what was observed. By underscoring the importance of complex physical processes in controlling host–parasite interactions and of epidemic disease in influencing host populations, our results highlight the value of long-term data for understanding wildlife disease dynamics.

Key words: chytridiomycete; climate change; Daphnia pulicaria; *epizootic; limnology; parasitoid; pathogen;* Polycaryum laeve; *time series; zooplankton.*

Introduction

Parasites and pathogens are ubiquitous members of all ecosystems. Because of their small size and cryptic nature, however, pathogens have historically been sidelined in ecological studies relative to more obvious interactions such as predation and competition. Short-term studies have established that parasites can have significant impacts on host behavior, fecundity, population dynamics, and even community composition. Parasites can also play a powerful, albeit cryptic, role in ecological food webs (Lafferty et al. 2006, Wood et al. 2007). Nevertheless, the significance of these effects over longer time scales, and identification of the environmental factors that may initiate epidemics in wildlife represent important frontiers in ecology (National Research Council 2001). Unfortunately, the data necessary to explore these questions are often lacking; pathogens are difficult to observe and quantify while the corresponding environmental information needed to identify the drivers of infection is frequently unavailable.

Interactions between *Daphnia* and their parasites may offer valuable insights into the long-term causes and consequences of wildlife disease epidemics (see Plate 1). *Daphnia*, which are ubiquitous in pond and lake ecosystems, serve as hosts for numerous endo- and ectoparasites, many of which produce conspicuous infections easily recognized through the transparent carapace of their hosts (e.g., Green 1974, Ebert 2005). Although zooplankton population dynamics are traditionally thought to be controlled by food availability and predation (e.g., Lampert et al. 1986, Carpenter et al. 1987), increasing evidence suggests that parasite infection can have important effects on *Daphnia*, including decreased reproduction, increased mortality, enhanced

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vulnerability to predators, and altered migratory and grazing behavior (Brambilla 1983, Yan and Larsson 1988, Bittner et al. 2002, Decaestecker et al. 2005, Duffy et al. 2005, Ebert 2005). Because *Daphnia* are also considered a keystone component of lake food webs, epidemic diseases of *Daphnia* have the potential to affect community- and ecosystem-level properties of lakes (Duffy 2007).

Despite continued advances in the understanding of *Daphnia*–parasite interactions in the laboratory, the factors governing disease epidemics in natural host populations and their long-term consequences remain poorly understood. Most field studies have been short-term (1–3 years), focused on simplified systems without vertebrate predators (e.g., ponds and rock pools), and accompanied by limited amounts of environmental data (e.g., Green 1974, Brambilla 1983, Bengtsson and Ebert 1998, Ebert et al. 2000, 2001, Decaestecker et al. 2005). Few studies have examined the significance of *Daphnia* pathogens in lake ecosystems with more complex food webs and physical processes (e.g., Duffy et al. 2005, Cáceres et al. 2006). What information is available indicates that infection levels in *Daphnia* populations are often highly variable with pronounced seasonality (e.g., Lass and Ebert 2006). Hypotheses advanced to explain these patterns include fluctuations in host density, temperature limitation, food availability, selective predation of infected animals, size-specific susceptibility to infection, and host genetic composition (Ruttner-Kolisko 1977, Yan and Larsson 1988, Little and Ebert 2000, Duffy et al. 2005). Evidence for these factors in natural *Daphnia* populations is mixed and inconclusive. Although density-dependent transmission is a cornerstone feature of many epidemiological models (e.g., Anderson and May 1981) and has been demonstrated for *Daphnia* parasites in the laboratory (Bittner et al. 2002), changes in host abundance are rarely sufficient to explain the duration or magnitude of epidemics in nature (Brambilla 1983, Yan and Larsson 1988). Observed infection patterns are likely the product of interactions among the host population, the life history of the particular pathogen, and the environmental conditions (Lass and Ebert 2006).

To identify the ecological drivers and consequences of disease epidemics in lake ecosystems, we used a long-term (15-year) data set on a chytrid parasitoid (*Polycaryum laeve*), its host (*Daphnia pulicaria*), and the concomitant environmental conditions. Unlike many parasites of *Daphnia*, *P. laeve* remains easily visible even among preserved hosts, making it ideal for long-term studies on disease. We used time-series analysis to evaluate the individual and combined importance of hypothesized drivers of infection, including host density, food availability, water temperature, dissolved oxygen, winter conditions, and lake stratification. Finally, to evaluate the effects of *P. laeve* epidemics on *Daphnia* host populations, we considered infection as an input variable and characterized its consequences for *Daphnia* fecundity, reproductive mode (sexual vs. asexual), and population dynamics. Given the ecological significance of large-bodied *Daphnia* in lake ecosystems, both as an important determinant of water clarity and as a food resource to fish (Lampert et al. 1986, Carpenter et al. 1987, Dodson and Frey 2001), studies of *Daphnia*–parasite interactions in lakes could provide insights into the direct and indirect effects of disease in aquatic food webs.

Materials and Methods

Study system

Devil's Lake is a 151-ha mesotrophic seepage lake located in south-central Wisconsin, USA (43°24′54″ N, 89°43′54″ W; see Appendix). In 2002, we observed epidemic infections in *D. pulicaria* by an unknown parasite. Subsequent study identified the pathogen as a chytridiomycete (*Polycaryum laeve*) that has only recently been described in North America (Johnson et al. 2006*a*). In severe infections, *P. laeve* forms large numbers of darkly colored sporangia throughout the hemocoel of infected *Daphnia*, allowing rapid identification of infected individuals among living as well as preserved *Daphnia*. The life history of *P. laeve* and its use of daphniid hosts conform to the classification of a parasitoid (Lafferty and Kuris 2002). Infected individuals experience increased mortality, reduced growth, and a complete cessation of reproduction (Johnson et al. 2006*a*). Following death of the host, which is required for successful transmission, *P. laeve* sporangia complete development and release flagellated zoospores that exit the *Daphnia* carcass. While chytrid parasites of phytoplankton have been studied extensively (see reviews by Van Donk 1989 and Ibelings et al. 2004), we know comparatively little about chytridiomycetes that parasitize zooplankton. The complete life cycle of *P. laeve* is not known, and transmission might be either direct, passing from dead infected hosts to susceptible *Daphnia*, or indirect, requiring additional hosts or free-living stages. Chytrids in a related genus (*Coelomomyces*) alternate between mosquito larvae and cyclopoid copepods. However, for most parasites of *Daphnia* in which transmission is known, it is typically direct (e.g., Green 1974, Ebert 2005). Attempts to transmit this parasite in the laboratory have been unsuccessful, despite trials incorporating a range of host sizes, temperatures, light levels, lake sediments, and possible alternate hosts (Johnson et al. 2006*a*).

Sampling methods

Between 1982 and 2003, the Wisconsin Department of Natural Resources (WDNR) sampled Devil's Lake approximately once every two weeks during ice-free months from mid-April until early November at the deepest point in the lake (14.5 m). On each sampling date, we measured water clarity (Secchi depth, m) and epilimnetic chlorophyll *a* (μg/L) using standard methods (Lathrop and Carpenter 1992; see Appendix). We used a Yellow Springs Instruments (YSI; Yellow Springs, Ohio, USA) meter to measure water temperature (°C) and

dissolved oxygen (mg/L) at 1-m depth intervals and subsequently averaged these values to generate a single value per sampling date. We sampled zooplankton in 1982–1983, 1986–1987, 1989, and 1994–2003 (15 years total) with a vertical tow of a Wisconsin net (30 cm diameter opening, 80-μm mesh netting). To assess infection prevalence for each sample, we examined a minimum of 200 mature (>1.2 mm) *D. pulicaria* with a stereodissecting microscope (see Johnson et al. 2006*a*). We measured all infected animals and a subset (≥50) of randomly selected uninfected *D. pulicaria* (carapace length, mm). We quantified the total number of *D. pulicaria*, the percentage of fecund *D. pulicaria*, the number of eggs per fecund female, the number of male and ephippial *D. pulicaria*, and the abundance of *D. mendotae*, *Holopedium gibberum*, cyclopoid copepods, and calanoid copepods.

Lake mixing

We hypothesized that lake stratification could influence parasite transmission in *Daphnia* by influencing spatial separation of susceptible hosts and infectious parasites. Because many parasites have free-living infectious stages that persist in lake sediments ("spore bank"; Green 1974), the amount of time *Daphnia* spend near the sediments may be a major determinant of infection risk (Decaestecker et al. 2002, 2005). By limiting either the proximity of *Daphnia* to the sediment (owing to oxygen depletion in the hypolimnion) or the circulation of parasite infectious stages throughout the water column, lake stratification may act to inhibit parasite transmission during summer (see also Doggett and Porter 1996).

We used buoyancy frequency (N^2) as a measure of lake stratification. Buoyancy frequency is the local water column stability (s^{-1}) and varies both by depth and by time of year. For 1 m depth intervals on each sampling date, we calculated buoyancy frequency as follows (Hamilton et al. 2004):

$$N^2 = -\left(\frac{g}{\rho_0}\right)\left(\frac{\Delta\rho}{\Delta z}\right)$$

where g is gravitational acceleration (m/s^2), ρ_0 is water density at the lake surface, $\Delta\rho$ is the change in density, and Δz is the change in depth. N^2 conveys two pieces of information: its maximum value offers a measure of stratification strength, and the depth corresponding to the maximum value represents the thermocline. Large values of N^2 indicate that the lake is thermally stratified. We further estimated the proportion of the water column included in the epilimnion (and therefore oxygenated) as the size of the epilimnion divided by maximum depth.

Epidemiology of P. laeve *infection within years*

We considered two approaches to estimating the effects of ecological variables on infection using within-year fluctuations in *D. pulicaria* density and infection prevalence. First, we estimated the population-level transmission rate of infection, β, assuming that *P. laeve* is directly transmitted between infected and susceptible hosts. The advantage of this approach is that it gives a simply structured autoregressive model with the form of common models for disease transmission involving the densities of susceptible and infected individuals. Second, we regressed ecological variables against changes in the prevalence of infection. The advantage of this second approach is that its dependent variable, infection prevalence, is measured directly in our data collection and therefore is the most precise variable quantifying the disease. Because within-year data on *D. pulicaria* abundance and infection were temporally autocorrelated, we used time-series analyses for statistical tests (Harvey 1989). For these analyses, we restricted our examination to the contiguous 10 years between 1994 and 2003 owing to fewer gaps in the data and more consistent sampling methodologies (see Appendix).

In the first approach, we assumed transmission takes the following form:

$$I_t = \boldsymbol{\beta}(w_{1,t}, \ldots, w_{m,t}) S_{t-1}^{b_1} I_{t-1}^{b_2} \quad (1)$$

where I_t is the abundance of infected hosts in sample t, S_t is the abundance of susceptible hosts, $\boldsymbol{\beta}(w_{1,t}, \ldots, w_{m,t})$ is the transmission rate that depends on m ecological drivers $w_{i,t}$, and b_1 and b_2 measure the effect of infected and susceptible host abundance on transmission. If both b_1 and b_2 equal 1, then this model produces "mass action" transmission, in which transmission is directly proportional to the density of both infected and susceptible hosts; this has the general form of a standard "SI model" (McCallum et al. 2001). In contrast, values of b_1 or b_2 equal to zero imply no effect of susceptible or infected host densities on transmission, respectively. To fit this model to data, we assumed that the abundance of susceptible hosts between sample $t - 1$ and sample t equals the total (susceptible and infected) abundance of hosts in sample t. This is justified because the samples are taken two weeks apart, and no adult *D. pulicaria* is likely to survive over the two-week sample period. Thus, the total number of susceptible and infected hosts at sample t represents the total number of individuals that were born (and hence susceptible) between sample $t - 1$ and sample t. Taking the log of each side of the transmission equation, and replacing S_{t-1} with the abundance of *D. pulicaria* in sample t gives

$$y_t = b_0 + b_1 D_t + b_2 y_{t-1} + \sum_{i=3}^{m+3} b_i w_{i,t} + \varepsilon_t \quad (2)$$

where y_t is the log abundance of infected *D. pulicaria,* and D_t is the log total abundance of *D. pulicaria*. Here, we have assumed that the log transmission rate, log $\boldsymbol{\beta}(w_{1,t}, \ldots, w_{m,t})$, can be expressed as a linear combination of the (suitably transformed) ecological drivers w_i:

$$\sum_{i=3}^{m+3} b_i w_{i,t} + \varepsilon_t$$

with the random variable, ε_t, capturing unexplained variation in transmission.

Our second approach was to estimate the effect of ecological variables on changes in infection prevalence. Specifically, we used the following model:

$$z_t - z_{t-1} = b_0 + b_1 D_t + b_2 z_{t-1} + \sum_{i=3}^{m+3} b_i w_{i,t} + \varepsilon_t \qquad (3)$$

where z_t is the arcsine-square-root transformed prevalence of infection, and D_t is the log total abundance of *D. pulicaria*. We analyzed this model in the same way as the model of transmission (Eq. 1).

For both approaches, we used the following covariates w_i: lake stratification (N^2), mean water temperature (Temp), mean dissolved oxygen levels (DO), the ratio of the epilimnion size to the size of the total water column (epilimnion ratio), epilimnetic chlorophyll *a* concentration (chl *a*), the abundance of potential alternate hosts (cyclopoid copepods [$\log_{10} + 1$-transformed], which have tested PCR-positive for *P. laeve* infection [Johnson et al. 2006*a*]), and year as a categorical variable. We estimated model coefficients using conditional least squares (Ives et al. 2003), computed bootstrapped 95% confidence intervals for coefficients, and obtained the statistical significance of the year categorical variable using a likelihood-ratio test. We also determined the best-fitting model according to Akaike's information criterion (AIC).

Epidemiology of P. laeve *infection among years*

To investigate the role of environmental factors in explaining infection levels at the annual scale, we used all years with *P. laeve* infection (1986–1987, 1989, 1994–2003), excluding only the first two years of data (1982–1983) during which no infection was observed. Because infection was highly seasonal, we used the average infection prevalence (arcsine-square-root transformed) and average *Daphnia* density ($\log_{10}$-transformed) during the month of May as an estimate of annual infection, as this period fell after ice-off in all years and generally corresponded with peak infection. We used simple correlations and least-squares stepwise linear regression to explore the importance of annual-scale variables. Given the large interval between responses (one year), we assumed samples were independent and verified this assumption with autocorrelation function (ACF) and partial autocorrelation function (PACF) plots of the residuals. We included the following input variables: recorded density of *D. pulicaria* following ice-off, the number of brown trout stocked annually, and climate indices related to winter severity (ice-off date, mean snow depth during ice cover, and cumulative snowfall between September and June). We obtained ice data from Devil's Lake State Park and snow data from the National Climatic Data Center, Baraboo, Wisconsin station (*available online*).[6]

[6] ⟨http://www.ncdc.noaa.gov/oa/ncdc.html⟩

Consequences of infection on host reproduction

We analyzed the effect of *P. laeve* infection prevalence on *Daphnia* fecundity (proportion of females gravid), the average number of eggs per fecund females, and reproductive mode (sexual and asexual); we defined reproductive mode as the proportion of the population involved in sexual reproduction (males plus females with ephippia). In these regressions, we also included total *Daphnia* density ($\log_{10}$-transformed), temperature, N^2, DO, epilimnion ratio, chl *a*, and year (as a categorical variable), and we accounted for possible autocorrelation between samples taken through time using autoregressive models.

Impact of infection on host populations

The consequences of infection for *Daphnia* population density depend not just on the prevalence of infection, but also on the density-dependent response of *Daphnia* population growth. For example, if the decrease in population density caused by infection leads to compensatory increases in the population growth rate, infection will have relatively small effects on the population density, whereas absence of a compensatory response will mean a much greater long-term impact on *Daphnia* populations. Therefore, to estimate the impact that *P. laeve* had on *Daphnia* populations, we fit dynamical models to the data. Specifically, we used the Gompertz and logistic models, because they can produce distinctly different dynamics. In the Gompertz model, population growth rate is a monotonically increasing function of density (Reddingius 1971). Therefore, the dynamics cannot show "overcompensation" in which densities exhibit alternating peaks and troughs at consecutive observations. In contrast, the discrete-logistic model, or Ricker model (Ricker 1954), is hump-shaped, such that population growth rate declines as densities reach very high levels. Therefore, it can show overcompensation leading to perpetual oscillations and even chaos (May and Oster 1976). These two models potentially show contrasting fits to density-dependent data (Dennis and Taper 1994, Dennis et al. 2006). We used the following Gompertz model:

$$D_t = (D_{t-1} - I_{t-1})\exp[r(w_{1,t}, w_{2,t}, \ldots, w_{m,t}) + b_d \log D_{t-1} + \varepsilon_t]. \qquad (4)$$

In this equation, the exponential term gives the per capita population growth rate, with the intrinsic rate of increase,

$$r(w_{1,t}, w_{2,t}, \ldots, w_{m,t}) = b_0 + \sum_{i=3}^{m+3} b_i w_{i,t}$$

depending on possible environmental drivers $w_{i,t}$. The per capita population growth rate also depends on log-transformed density, $\log D_t$, with the autoregression coefficient b_d giving the strength of density dependence; b_d is negative, and the lower its value, the stronger the

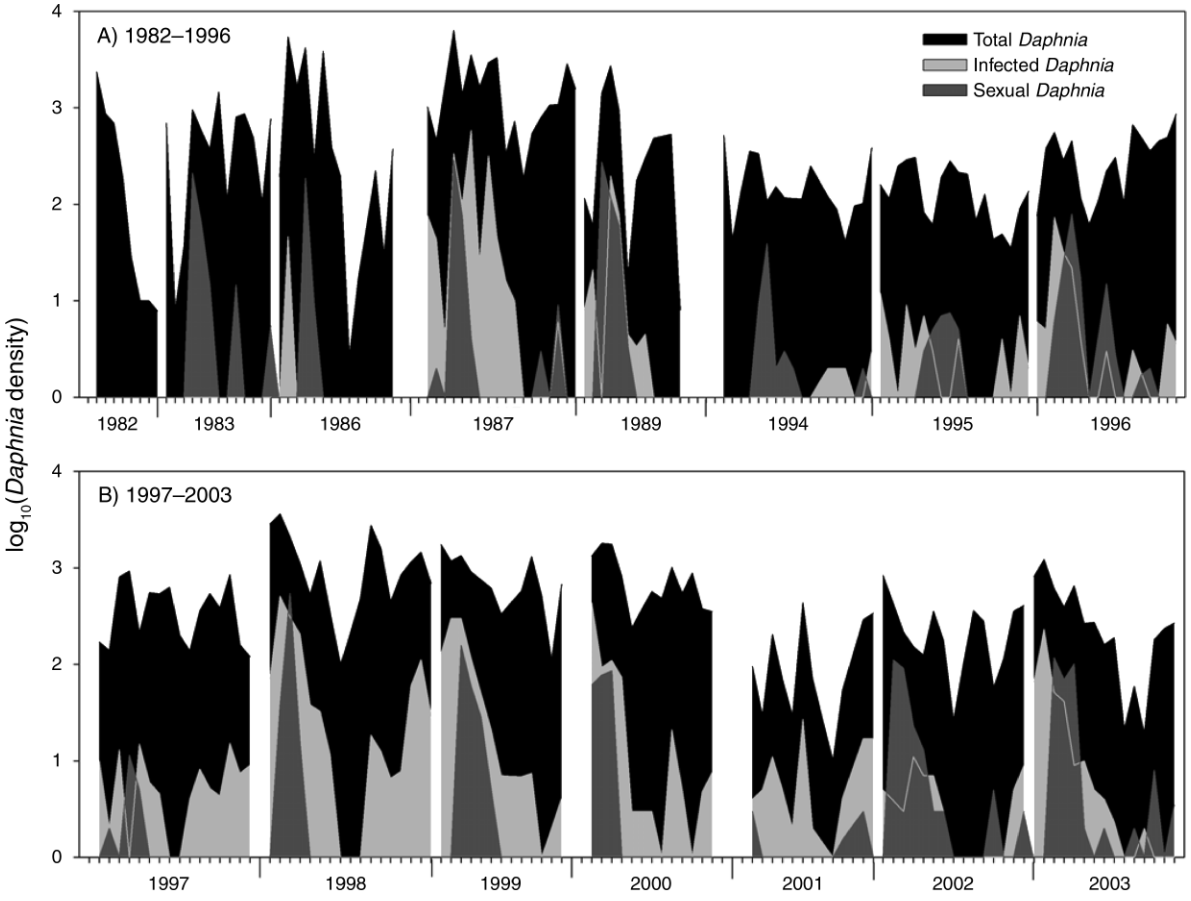

FIG. 1. Log-transformed density (measured as number per liter) of all *Daphnia pulicaria* (black shaded area), *D. pulicaria* infected with *Polycaryum laeve* (light gray area), and sexual *D. pulcaria*, defined here as the sum of male and ephippia-bearing female *D. pulicaria* (dark gray area), in Devil's Lake, Wisconsin, USA, between 1982 and 2003. Sampled years include 1982–1983, 1986–1987, 1989, and 1994–2003.

density dependence. Unexplained environmental variability is captured in the term ε_t which is treated as an independently and identically distributed random variable. Finally, we assumed infected individuals, I_t, did not reproduce and therefore contributed nothing to the population at the next time sample. The discrete-logistic is similar, having the form

$$D_t = (D_{t-1} - I_{t-1})\exp[r(w_{1,t}, w_{2,t}, \ldots, w_{m,t}) + b_d D_{t-1} + \varepsilon_t] \quad (5)$$

in which log D_{t-1} in the per capita population growth rate of Eq. 4 is replaced by D_{t-1}.

We fit both models to the data using two methods, the first assuming no measurement error (conditional least squares, CLS) and the second including measurement error (state–space model, SS; see Appendix). If not explicitly accounted for, measurement error will often inflate the strength of density dependence estimated from time series data (Dennis and Taper 1994, Ives et al. 2003). This, in turn, will likely lead to underestimates of the long-term impact of disease. After fitting the models, we assessed the impact of *P. laeve* by simulating the models after removing the estimated mortality caused by infection. To make the simulated time series without *P. laeve* mortality directly comparable to the observed time series, we used the residuals from the fitted model as the random environmental variation ε_t in the simulated time series.

RESULTS

We found *Daphnia pulicaria* in all collected samples but their abundance varied considerably, from 1 to 7782 individuals/m^3 (n = 221 samples; Fig. 1). Infection prevalence ranged from <1% to 34% and was strongly seasonal, exhibiting the highest levels in spring with a secondary peak in fall (Fig. 1). *Polycaryum laeve* was absent or at very low levels in late summer, even though *D. pulicaria* generally persisted at moderate densities. We did not detect infection in samples from 1982–1983, or within *D. mendotae* or other cladocerans.

Epidemiology of P. laeve *infection within years*

From Eq. 1, the log transmission rate β depended significantly on both the densities of infected and

TABLE 1. Autoregression of factors affecting parasite transmission based on the density of infected hosts (Eq. 1) and the prevalence of *Polycaryum laeve* infection (Eq. 2) for years 1994–2003.

Dependent variable	Independent variable	Coefficient	P
log(infected [I_t])	log(infected [I_{t-1}])	0.44	0.001
	log(susceptible [S_{t-1}])	0.57	0.001
	N^2	−0.12	0.001
	log(copepod density)	0.30	0.01
	log(chl *a*)	−0.27	0.1
Prediction R^2		0.43	
Infection prevalence	N^2	−0.007	0.001
	log(copepod density)	0.016	0.02
	log(chl *a*)	−0.020	0.07
	autocorrelation	0.46	0.001
Prediction R^2		0.35	

Notes: $I(t)$ = the abundance of infected *Daphnia* in sample t; $I(t-1)$ = the abundance of infected *Daphnia* in sample $t-1$; $S(t)$ = the abundance of uninfected *Daphnia* in sample t. Independent variables not reported here included log(*Daphnia pulicaria* density), mean temperature, buoyancy frequency (N^2), dissolved oxygen, chl *a*, and year (treated as a categorical variable). The models reported contain only those variables included in the AIC best-fitting model. Parameters were estimated using conditional least squares (CLS), with P values obtained by bootstrapping 5000 simulated data sets. Infection prevalence was arcsine-square-root transformed.

susceptible *Daphnia* (Table 1). For both variables, the coefficients b_1 and b_2 were less than 1, implying that transmission increased less than linearly with the densities of infected and susceptible *Daphnia*. Furthermore, the log transmission rate $\boldsymbol{\beta}$ was negatively affected by buoyancy frequency (N^2), positively affected by cyclopoid copepod density, and negatively affected by chl *a* concentrations (Table 1). The model for infection prevalence (Eq. 2) also supported the strong statistical effects of N^2 on transmission, along with weak effects of cyclopoid copepod density and chlorophyll *a* concentrations (Table 1). Inclusion of an alternative metric of stratification (e.g., Schmidt's stability) provided very similar results. While mean temperature correlated negatively with infection, measures of stratification explained more of the variance in transmission and thereby excluded temperature from the best-fitting models.

Epidemiology of P. laeve *infection among years*

Among years, the duration and severity of winter related negatively with measures of *P. laeve* infection the following spring (Fig. 2). Infection prevalence (arcsine-square-root transformed) and infected host density (log[density + 1]) correlated negatively with mean snow depth (prevalence, $r = -0.57$, $P = 0.042$; infected density, $r = -0.58$, $P = 0.038$), cumulative snowfall (prevalence, $r = -0.65$, $P = 0.016$; infected density, $r = -0.618$, $P = 0.02$), and ice-off date (prevalence, $r = -0.30$, $P > 0.05$; infected density, $r = -0.56$, $P = 0.048$). Total host density (infected and uninfected, log-transformed) after ice-off correlated positively with both measures of infection (prevalence, $r = 0.59$, $P = 0.034$; infected density, $r = 0.78$, $P = 0.002$). However, if we included host density and winter severity in stepwise regressions to predict annual infection, only host density was selected, possibly suggesting that the effects of winter on infection patterns are mediated, in part, through effects on host density. Simply stated, winter conditions determine the abundance of *Daphnia* hosts, which then control patterns of infection by *P. laeve*. Indeed, a mediation test of the effects of ice-off date on infected density with host density as the mediator was significant (Sobel test statistic = −2.10, $P = 0.03$ [Preacher and Hayes 2004]), suggesting that winter affected infection indirectly through changes in host density. Given the limited number of years in our analysis ($n = 13$ with infection), however, we suggest these results be interpreted with caution, as more data are needed to formally evaluate the proposed causal pathways.

Consequences of infection on host reproduction

Infected *Daphnia* rarely contained eggs (<1%) and infection prevalence had a significant negative effect on the average number of eggs per fecund female (Table 2). Among uninfected hosts, the percentage of gravid individuals ranged from 0% to 45% (10.2% ± 0.012%, mean ± SE). Alongside infection, host density, buoyancy frequency, and dissolved oxygen levels were also negatively related to *Daphnia* fecundity (Table 2). However, the effect of infection on the proportion of *Daphnia* with eggs was not significant ($P < 0.28$), because much of the variation in fecundity was explained by *Daphnia* density, N^2, DO, and temporal

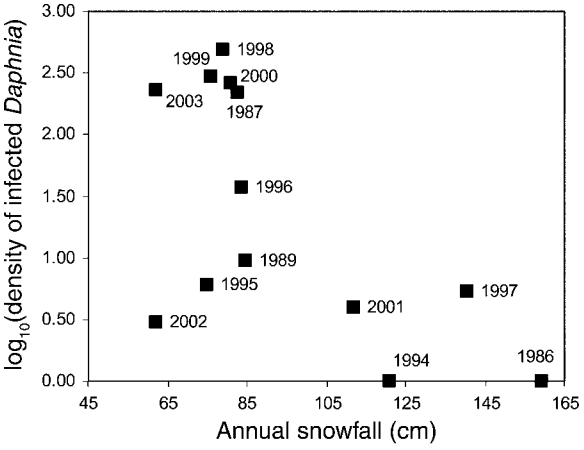

FIG. 2. Relationship between cumulative snowfall and spring infection in *D. pulicaria* for all years in which *P. laeve* was detected (1982 and 1983 were omitted owing to a lack of infection). Annual snowfall is presented as the total snow recorded between September and June of each year, whereas spring infection is the average density ($\log_{10}$-transformed) of *P. laeve*-infected *D. pulicaria* during the month of May, as most epidemics occurred following ice-off and May was the most consistently sampled spring month. The year of each observation is presented next to each data point.

TABLE 2. Regression of host fecundity (percentage of egg-bearing females), average number of eggs per fecund female, and mode of reproduction (combined percentage of ephippial females and males) on infection prevalence for years 1994–2003.

Dependent variable	Independent variable	Coefficient	P
Mean eggs	infection prevalence	−1.25	0.02
	log(*Daphnia* density)	−0.054	0.02
	N^2	−0.024	0.01
	dissolved oxygen	−0.033	0.02
	autocorrelation	0.18	0.04
Fecundity	infection prevalence	−0.35	0.27
	log(*Daphnia* density)	−0.45	0.001
	dissolved oxygen	−0.014	0.02
	N^2	−0.030	0.001
	autocorrelation	0.28	0.001
Reproductive mode	infection prevalence	0.72	0.001
	autocorrelation	0.41	0.001

Notes: Independent variables included in the regressions were: log *Daphnia pulicaria* density, mean temperature, N^2, dissolved oxygen, epilimnion ratio, chlorophyll *a*, and year (treated as a categorical variable). Temporal autocorrelation was included by treating dependent variables as autoregression, AR(1), processes. The models reported contain only those variables (other than infection prevalence) included in the AIC best-fitting model. Parameters were estimated using CLS, with P values obtained by bootstrapping 5000 simulated data sets. Fecundity (proportion of females gravid), mode of reproduction (proportion of individuals either males or containing ephippia), and infection prevalence were arcsine-square-root transformed. The mean number of eggs per female was transformed to the ¼ power.

autocorrelation (Table 2). Excluding *Daphnia* density, N^2, and DO, the best-fitting model contained year (as a categorical variable) and autocorrelation, with infection prevalence as marginally significant ($P < 0.051$).

Infection prevalence was positively related to the degree of sexual reproduction in *Daphnia* ($P = 0.001$; Table 2). Although infection was never observed in *Daphnia* carrying ephippia, their presence correlated positively with *P. laeve* epidemics. Males were infected with *P. laeve* at a similar frequency to females collected in the same sample. In many years between 1995 and 2003, both the timing and magnitude of sexual reproduction coincided closely with infection prevalence (Fig. 1). No other population or environmental drivers explained significant additional variation, including chlorophyll *a* and mean temperature. However, inclusion of a categorical "season" variable into the model eliminated the effect of infection prevalence on reproductive mode ($P = 0.11$), possibly suggesting another unmeasured intrinsic or extrinsic driver was influencing the timing of sexual reproduction. Correspondingly, even while no infection was observed in 1983, the timing and magnitude of sexual reproduction by *Daphnia* was similar to years with infection.

Impact of infection on host populations

The fit of the Gompertz model (prediction $R^2 = 0.44$ [CLS] and 0.43 [SS]) was better than the discrete-logistic (prediction $R^2 = 0.21$ [CLS] and 0.23 [SS]) (Table 3, Fig. 3A). In both models, mean DO was the only environmental variable included in the AIC best-fitting model, whereas in the Gompertz model, year as a categorical variable was also included. Incorporation of measurement error led to a lower estimate of the strength of density dependence in the Gompertz model (−0.58 [SS] vs. −0.68 [CLS]), although this was not the case for the logistic model.

TABLE 3. Gompertz and logistic models (Eqs. 4 and 5) fit to *Daphnia pulicaria* population dynamics to estimate the consequences of *Polycaryum laeve* infection on host abundance.

Model results	Input variables	CLS estimates	SS estimates
Gompertz			
	D_{t-1}	–0.68**	–0.58**
	dissolved oxygen	0.090**	0.089**
	year	†*	‡**
Prediction R^2		0.44	0.43
Mean percentage increase		9.4	11
Maximum percentage increase		49	55
Logistic			
	D_{t-1}	–0.00044**	–0.00046**
	dissolved oxygen	0.099**	0.095**
Prediction R^2		0.21	0.23
Mean percentage increase		11	12
Maximum percentage increase		49	49

Notes: The listed variables were included in the best-fitting AIC models out of the variables host density in the preceding sample (D_{t-1}), dissolved oxygen, mean temperature, chl *a*, and year (treated as a categorical variable). Each model was fit using both CLS and placing the equations into state–space form and assuming that the measurement error in log density is normally distributed with mean zero and variance 0.16. Mean and maximum proportional increases are the estimated changes in host population density in the absence of infection.

* $P < 0.05$; ** $P < 0.01$.

† Categorical variable tested with a likelihood ratio test ($\chi^2 = 35.58$, df = 9).

‡ Categorical variable tested with a likelihood ratio test ($\chi^2 = 39.34$, df = 9).

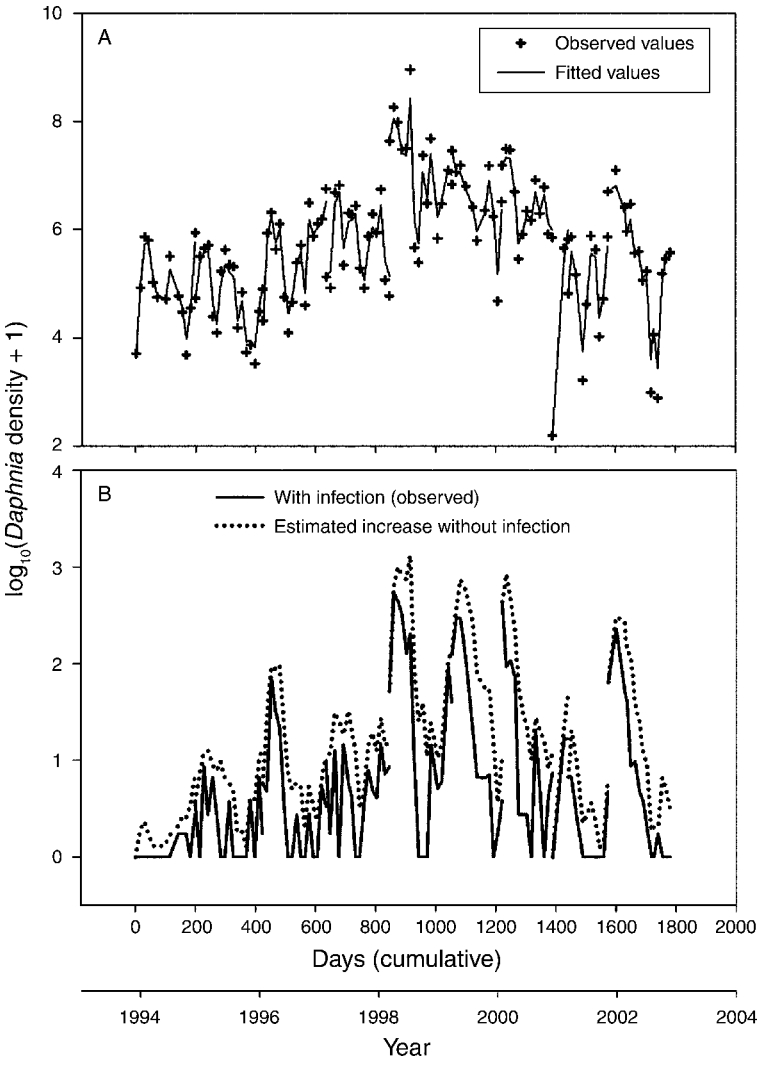

FIG. 3. (A) Comparison of the fit of the Gompertz state–space model (solid line) to the observed data on *D. pulicaria* population density (+ symbols). On the *x*-axis, "Days" represents the cumulative number of days since the first sampling date in 1994 (winter days omitted). (B) Impact of infection on *D. pulicaria* populations. The solid line reflects the number of infected *Daphnia* from the data, whereas the dashed line presents the difference between the fitted and simulated densities of *Daphnia* using parameters fit in the SS model. This indicates the estimated increase in *Daphnia* in the absence of infection.

The estimated impact of disease on the *Daphnia* population was similar for both models and both fitting procedures, with projected mean increases in host density of roughly 11% and projected maximum increases of roughly 50% in the absence of infection (Fig. 3B). These values are considerably higher than the mean and maximum infection levels of 3.1% and 34%, respectively. This occurs because those individuals killed or sterilized by infection in one generation do not produce offspring that could reproduce in the following generation, causing a carry-on effect of disease. If density dependence were extremely strong (e.g., −1 in the Gompertz model), the reductions in density caused by infection would be compensated for rapidly. However, density dependence is only moderate (in the Gompertz model, −0.58 [SS]), so that population recovery is not immediate and the long-term impact of disease on population density is greater.

DISCUSSION

Long-term data on disease dynamics in wildlife are uncommon owing to inadequate preservation of the etiological agent, an inability to identify pathogens in preserved samples, and the unreliability of historical records (e.g., surveillance bias [Persing et al. 1990, Johnson et al. 2003]). By combining long-term data on the dynamics of a chytridiomycete pathogen, its *Daphnia* host, and the environmental conditions surrounding their interactions, our analyses reveal the importance of physical conditions in driving infection epidemics and of

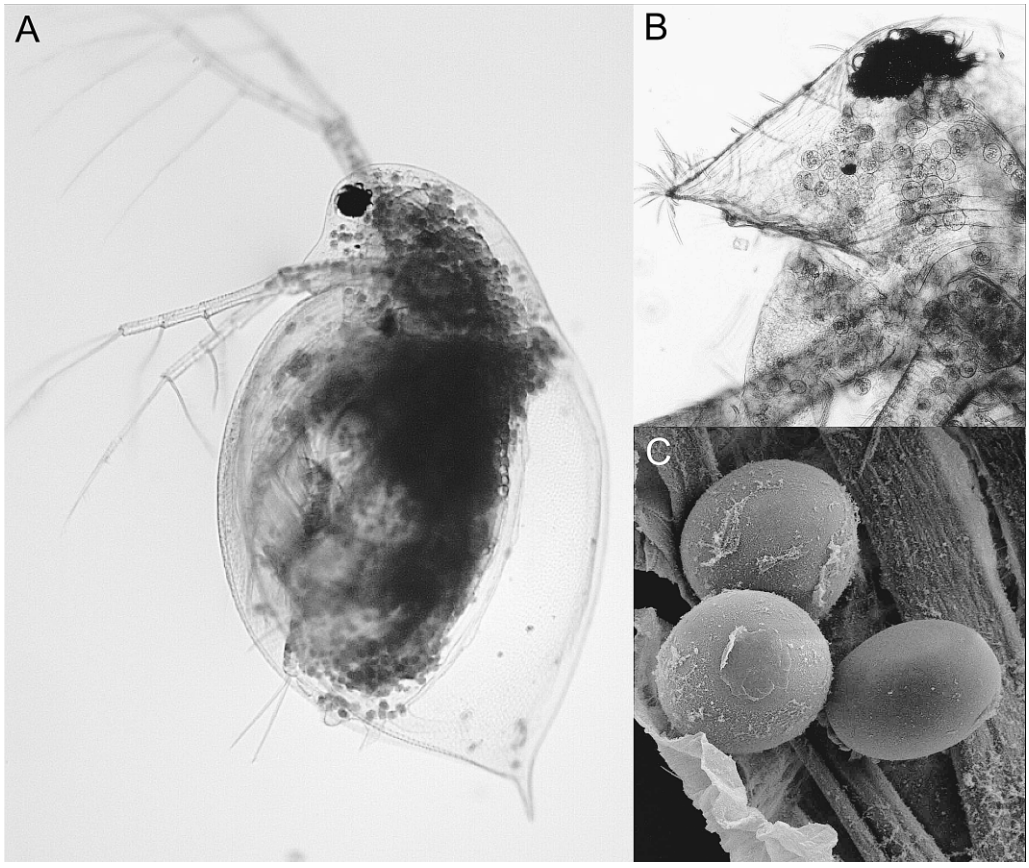

PLATE 1. Images of *Daphnia pulicaria* infected with chytridiomycete pathogen, *Polycaryum laeve*. (A) Whole-body image of adult *D. pulicaria* with large numbers of chytrid sporangia throughout the hemocoel; sporangia appear as darkly colored circles. (B) Magnified view of *Daphnia* head and clusters of sporangia (note: this specimen also has rod-shaped epibionts on the outside of the carapace). (C) Scanning electron microscope image of three sporangia and associated host tissue. Photo credits: P. T. J. Johnson.

infection in influencing host population dynamics. Specifically, we found that lake mixing and turnover enhanced *P. laeve* transmission, with lesser effects for chl *a* levels and copepod densities (potential alternate hosts). Infection also correlated negatively with asexual reproduction in *Daphnia*, likely reflecting the parasite's castrating effects on its host (parasitoid life history). Our models further suggest that chytrid epidemics reduced the population density of *Daphnia* by an average of roughly 10%, with reductions of 50% during peak infection levels. These results agree well with laboratory studies (Johnson et al. 2006*a*), which found that infection decreased the growth, reproduction, and survival of individual *Daphnia*.

Drivers of infection

Hypotheses advanced to explain infection dynamics in cladocerans have included density dependence, temperature limitation, size-dependent susceptibility, genetic variation, selective predation, and food stress (Ruttner-Kolikso 1977, Brambilla 1983, Yan and Larsson 1988, Bittner et al. 2002, Duffy et al. 2005). However, the inadequacy of available data has often precluded rigorous evaluations of the individual and combined roles of such factors in explaining epidemics in natural *Daphnia* populations. In our analyses, lake stratification negatively affected the abundance and prevalence of *Daphnia* infected with *P. laeve*, accounting for significantly more variance in infection levels than alternative drivers (e.g., temperature, dissolved oxygen, host size, and food availability). We suggest that lake stratification influences infection through two related mechanisms. First, increases in stratification strength correlate positively with hypoxia in the hypolimnion, ultimately causing *Daphnia* to spend less time below the thermocline in oxygen-poor waters. Considering that maturation of *P. laeve* requires death of its host ("parasitoid" [Lafferty and Kuris 2002]), zoospore production is probably maximized near the sediments where host carcasses accumulate (Decaestecker et al. 2002). We further suspect that resting stages of *P. laeve* occur in lake sediments, as found for many other parasites of *Daphnia* (e.g., Green 1974, Decaestecker et al. 2002) and other chytrids (Van Donk 1989, Ibelings et al. 2004),

which would enhance the risk of infection associated with proximity to sediment. Thus, reduced contact between susceptible *Daphnia* and lake sediments with increasing stratification probably limits transmission during late summer. Correspondingly, while infection often disappeared with increases in stratification, *D. pulicaria* persisted at moderate levels, suggesting that changes in host density alone cannot explain the observed infection pattern.

Second, because lake stratification limits exchange of water between the hypolimnion and epilimnion, we expect that increased water column stability reduces the hydrodynamic transport of *P. laeve* infectious stages from lake sediments upwards above the thermocline where they would encounter susceptible *Daphnia*. Thus, as carcasses of dead or dying infected *Daphnia* continue to pass through the thermocline into the hypolimnion, a unidirectional downward transport of parasite material establishes. Lake stratification similarly restricts the movement of nutrients across the thermocline, limiting epilimnetic phytoplankton production in summer (Huisman et al. 2004, Ryan et al. 2006), and water turbulence is hypothesized to function similarly in controlling chytrid epidemics in phytoplankton (e.g., Doggett and Porter 1996, Ibelings et al. 2004). Evidence supporting an inhibitory role for stratification on *P. laeve* transmission can also be inferred from the reappearance of infection following fall mixis. Fall epidemics were often lower than those observed in spring, possibly owing to corresponding reductions in host density. These results suggest that shallow or weakly stratified lakes might support larger or more extended *P. laeve* epidemics than deep systems with a smaller fetch and stronger stratification. Although we have assumed direct transmission for *P. laeve* in this model, we expect that stratification will similarly influence the parasite's spread to *Daphnia* if an alternate host (e.g., cyclopoid copepods) is involved.

The importance of physical limnology in driving patterns of disease is supported by other studies of host-parasite interaction in freshwater environments. Cáceres et al. (2006), for example, found that basin shape was the best predictor of *Metschnikowia bicuspidata* epidemics in *Daphnia dentifera* among 18 Michigan lakes. Among years, steep-sided lakes supported higher infection levels in their *Daphnia* populations, which the authors attributed to differences both in hydrodynamic processes and in the abundance of selective predators in such ecosystems. Similarly, in a long-term study of lake eutrophication and parasitism, Marcogliese et al. (1990) found that nutrient-driven hypoxia increased the spatial overlap between sphaeriid clams and burrowing mayflies, enhancing transmission of a trematode parasite (*Crepidostomum cooperi*) that utilizes these species as intermediate hosts. Subsequent improvements in water quality reduced hypolimnetic hypoxia, allowing the oxygen-sensitive mayflies to move into deeper water and leading to a sharp reduction in their infection prevalence (Marcogliese et al. 1990).

Analyses at the annual scale emphasized the importance of host density and winter conditions in driving *P. laeve* infection patterns. Years with lower cumulative snowfall, earlier ice-off, and shallower snow depth exhibited greater infection maxima the following spring. Initially, this result is somewhat paradoxical; *P. laeve* develops optimally at low temperatures (i.e., 5–10°C; D. Stanton, *unpublished data*) and might therefore be expected to fare poorly in warmer years (see also Pounds et al. 2006). We suggest this pattern has less to do with temperature and more to do with the levels of light penetration and spring phytoplankton blooms. Snow is highly reflective and limits penetration of light through the ice (Ragotzkie 1978), thereby limiting primary production and zooplankton population growth (Gerten and Adrian 2002). Years with lower snowfall and/or earlier ice-off likely exhibit increases in phytoplankton blooms that help to increase the overall density of *D. pulicaria*, leading subsequently to an increase in *P. laeve* transmission. As host density correlated positively with *P. laeve* infection (and negatively with winter severity) in our among-year analyses, reductions in *Daphnia* abundance associated with winter conditions could limit spread of the parasite prior to and immediately following ice-off. Support for this hypothesis can be inferred from the negative correlations between *Daphnia* density and ice duration and ice-off date (see also Schindler et al. 2005). Changes in light penetration can also affect chytrid parasites directly by stimulating zoospore release, further contributing to potential links between climate and infection dynamics (Van Donk 1989, Ibelings et al. 2004). Considering the widely observed declines in ice-cover duration for northern hemisphere lakes (e.g., Magnuson et al. 2000), increasing global temperatures could heighten the magnitude of *P. laeve* spring epidemics, but more years of data are needed to rigorously explore this hypothesis.

Alternative drivers of infection

Although infection also correlated negatively with temperature, we suggest that changes in temperature alone are unlikely to explain the observed infection patterns. In laboratory studies, *P. laeve* zoospore release occurred adequately in temperatures found in hypolimnion throughout the year (6–11°C; D. Stanton, *unpublished data*). In both sets of models, however, we did find weak support for contributing roles of cyclopoid copepods and chl *a* availability in determining host infection. Related groups of chytrids (e.g., *Coelomomyces*) utilize copepods as alternate hosts, and these results could reflect involvement of cyclopoid copepods in the life cycle of *P. laeve* (see Johnson et al. 2006*a*). Pooled samples of cyclopoid copepods have tested PCR positive for *P. laeve* in previous studies (Johnson et al. 2006*a*), but attempts to transmit the parasite in the laboratory

(with or without copepods) have thus far proven unsuccessful. Negative relationships between chl *a* and infection may suggest that, under food-limited conditions, *Daphnia* hosts are more susceptible to infection, as found in previous laboratory experiments (Hall et al. 2007). Other studies (e.g., Pulkkinen and Ebert 2004), however, have reported the opposite pattern, and the low statistical significance of current result suggests they should be interpreted with caution.

Duffy et al. (2005) suggested that selective predation of parasitized *Daphnia* may limit disease epidemics in lake systems with complex food webs. Consistent with this hypothesis, *P. laeve*-infected *Daphnia* are preferentially consumed by fish predators because of enhanced conspicuousness (Johnson et al. 2006*b*). In autoregressive models, however, neither fish abundance (as estimated by the number of brown trout stocked annually) nor mean *Daphnia* size, an indicator of planktivory (e.g., Brooks and Dodson 1965), was included in the best final models. Nevertheless, finer-resolution fish data on more species (especially planktivorous species) are needed to definitively evaluate the importance of selective predation in driving *P. laeve* infection dynamics. It is worth noting that planktivory in Devil's Lake is not expected to be very intense, as evidenced by the predominantly piscivorous fish community and the persistence of large-bodied *D. pulicaria* at moderate densities throughout the summer (e.g., Brooks and Dodson 1965).

Changes in the genetic structure of *Daphnia* populations have also been suggested to explain patterns of parasite infection, including the rise and fall of epidemics (e.g., Duffy and Sivars-Becker 2007). Experimental studies have demonstrated interactions between *Daphnia* host population genetic structure and parasite infection levels (Little and Ebert 2000, Decaestecker et al. 2002, Duffy and Sivars-Becker 2007). We have no data to evaluate the importance of seasonal changes in host resistance to *P. laeve* infection by *D. pulicaria*, and it is plausible that changes in environmental conditions shifted the genetic composition of the *D. pulicaria* population in favor of strains more resistant to *P. laeve* infection. We are currently using microsatellites to compare between *P. laeve*-infected and uninfected *D. pulicaria* as well as between spring and summer samples.

Consequences of infection

Polycaryum laeve infection influenced *Daphnia* fecundity and reproductive mode. Of the nearly 3000 infected *Daphnia* inspected, less than 1% contained eggs, even when comparably sized uninfected individuals exhibited high fecundity. Infection prevalence negatively predicted the average number of eggs per female, possibly reflecting population-level effects of parasitism on host fecundity. The lack of a significant relationship between infection prevalence and the percentage of fecund hosts may indicate that infection occurs primarily during periods of low overall *Daphnia* fecundity, such as in times of food stress, that *Daphnia* populations exhibit a strong compensatory response to disease epidemics, or it may reflect a sampling artifact. Preserved *Daphnia* can eject some or all eggs and embryos during the preservation process, potentially biasing estimates of egg prevalence by lowering fecundity among uninfected hosts.

In many years, infection prevalence correlated positively with the amount of sexual reproduction in *D. pulicaria*, as indicated by the percentage of males and ephippia-bearing females in the population. Although ephippial production typically occurs during periods of food or temperature stress (Epp 1996, Dodson and Frey 2001), neither water temperature nor chlorophyll *a* levels explained significant variation in sexual reproduction levels. In contrast, infection prevalence correlated with *Daphnia* sexual reproduction in both timing and magnitude (Fig. 1). These results are consistent with the hypothesis that sexual reproduction helps combat parasitism by enhancing genetic recombination (Hamilton et al. 1990). Parthenogenic organisms such as *Daphnia*, which can alternate between sexual and asexual reproduction, offer a unique opportunity to investigate questions regarding the evolution of sexual reproduction (see Ebert and Hamilton 1996, Lively 2001). Because ephippia-bearing *Daphnia* were not themselves infected, this hypothesis requires that uninfected *Daphnia* can detect increases in infection risk and alter their reproductive mode accordingly. However, it is also possible that the correlation between infection and sexual reproduction was not causal, and other, unmeasured elements of seasonality or host genetics may control the timing of ephippial production. It is unlikely that *P. laeve* infection is the primary determinant of sexual reproduction in *Daphnia* hosts, as even years without infection (e.g., 1983 and 1994) witnessed similar levels of male and ephippial *Daphnia* (Fig. 1). Experimental work is needed to evaluate whether infection contributes to the magnitude or timing of sexual reproduction.

Results of the dynamical models suggest that elimination of infection from the host population would cause average and maximal increases in *Daphnia* density of approximately 11% and 50%, respectively. Forecasted increases in density considerably exceed the observed average and maximal values for infection prevalence (3% and 34%, respectively) because the models identified only moderate potential for host populations to compensate for losses due to epidemics. These results are consistent with laboratory studies on *P. laeve*, in which infected hosts survived half as long as their uninfected counterparts and exhibited a complete cessation of egg production (Johnson et al. 2006*a*). Based on our analyses from Devil's Lake, it appears that increased mortality, rather than decreased reproduction, may be primarily responsible for the projected decreases in host density. However, we cannot evaluate whether

mortality was direct, due to infection, or indirect, due to heightened predation (Johnson et al. 2006*b*).

Collectively, results of this study illustrate the importance of long-term data and physical processes in understanding the environmental drivers of pathogen transmission in lake zooplankton. Host density, while influencing *P. laeve* infection in *Daphnia*, was only part of the story; climate and the spatiotemporal separation of host and parasite contributed significantly to observed patterns. We suggest that lake stratification, through its effects on host habitat use and water circulation patterns, effectively isolated susceptible hosts from parasite source populations (e.g., lake sediments). *Polycaryum* infection also had significant population-level effects on *Daphnia* fecundity, reproductive mode, and host population growth. Considering the importance of *Daphnia* as a food source for young-of-the-year and planktivorous fishes (Lampert et al. 1986, Carpenter et al. 1987, 2001), parasitic diseases of *Daphnia* may have broadscale consequences in lake food webs.

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APPENDIX

A description of sample collection and processing, modeling parasite transmission, modeling host population dynamics, and associated references (*Ecological Archives* E090-008-A1).