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PHYSICAL STRUCTURE OF LAKES CONSTRAINS EPIDEMICS IN *DAPHNIA* POPULATIONS

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Abstract. Parasites are integral parts of most ecosystems, yet attention has only recently focused on how community structure and abiotic factors impact host–parasite interactions. In lakes, both factors are influenced by habitat morphology. To investigate the role of habitat structure in mediating parasitism in the plankton, we quantified timing and prevalence of a common microparasite (*Metschnikowia bicuspidata*) in its host, *Daphnia dentifera*, in 18 lakes that vary in basin size and shape. Over three years, we found substantial spatial and temporal variation in the severity of epidemics. Although infection rates reached as high as 50% in some lakes, they did not occur in most lakes in most years. Host density, often considered to be a key determinant of disease spread, did not explain a significant amount of variation in the occurrence of epidemics. Furthermore, host resistance does not fully explain this parasite's distribution, since we easily infected hosts in the laboratory. Rather, basin shape predicted epidemics well; epidemics occurred only in lakes with steep-sided basins. In these lakes, the magnitude of epidemics varied with year. We suggest that biological (predation) and physical (turbulence) effects of basin shape interact with annual weather patterns to determine the regional distribution of this parasite.

Key words: Daphnia dentifera; *disease epidemics; host–parasite interaction; indirect effects; lake basin shape;* Metschnikowia bicuspidata; *morphometry; turbulence; weather patterns; zooplankton.*

Introduction

In recent decades, ecologists have discovered key mechanisms that govern the spread of infectious disease in nature (Anderson and May 1986, Regoes et al. 2003). Yet it remains difficult to predict why epidemics erupt in some host populations at some times whereas others show little or no infection. One major reason for this uncertainty results from the difficulty of quantifying transmission rates of parasites to hosts (Fenton et al. 2002, Lafferty and Holt 2003). In addition, the spread of disease within a host population often depends upon the structure of the community in which it is embedded (Packer et al. 2003, Ostfeld and Holt 2004, Duffy et al. 2005). Both transmission rate and community structure depend on environmental factors. For example, abiotic factors such as weather and seasonality clearly can affect, even synchronize, transmission of parasites in human disease systems (Rohani et al. 1999, Pascual et al. 2000). The spread of disease in wildlife populations is also influenced by climatic variation and ecosystem level change (Dobson and Foufopoulos 2001, Johnson and Chase 2004).

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Quantifying the relative importance of the various drivers of epidemics, however, often requires large-scale and/or long-term observational studies, which can reveal the variability and context dependency of local host–parasite interactions. The microparasitic infections of lake zooplankton offer an ideal system for such an approach. In lakes, the common planktonic grazer, *Daphnia* (Crustacea: Cladocera) is infected by a diversity of lethal microparasites including bacteria, microsporidia, and fungi (Green 1974, Stirnadel and Ebert 1997). *Daphnia* population dynamics can be rapid, and infection dynamics can easily be monitored in multiple lake populations over time. Moreover, environmental influences on host–parasite interactions may be particularly prominent in aquatic habitats (Koelle and Pascual 2004, McCallum et al. 2004), where most of the microparasites are spread by means of free-living infective stages (Lafferty and Holt 2003), and where hydrodynamics mediate many biological interactions (Wetzel 2001, Tessier and Woodruff 2002).

In lakes, basin size and shape influence both the physics and biology of planktonic systems. From an epidemiological perspective, basin shape may regulate the distribution of epidemics directly through physical processes or indirectly though modification of food web effects. For example, factors such as surface area, transparency, depth, and shape of the basin influence

hydrodynamical processes such as mixing and turbulence (Wetzel 2001). These processes may be important to disease transmission by resuspending infective propagules of parasites and allowing them to remain suspended in the water column, where they can encounter hosts. The importance of abiotic drivers to *Daphnia*–parasite interactions is suggested by seasonal cycles to parasitism; epidemics are common at times when a larger volume of water is turbulent due to cooling events (Doggett and Porter 1996, Bittner et al. 2002). Basin shape and size also mediate the strength of interactions among fish, zooplankton, and phytoplankton (Tessier and Woodruff 2002). Predation on infected hosts by fishes can be severe (Duffy et al. 2005). Hence, occurrence of epidemics could also be mediated through food web effects that differ in lakes of various sizes and shapes.

In this study, we document considerable variation in the spatial occurrence of the common yeast pathogen *Metschnikowia bicuspidata* in populations of *Daphnia dentifera* Forbes. As the infective stage of this parasite likely requires vertical mixing for suspension, we expected that physical aspects of basin shape would strongly influence the occurrence of epidemics. More specifically, we hypothesized that lakes with gradually sloping sides should promote epidemics. In such basins, a larger percentage of the sediments are within the epilimnion. The probability would thus be higher that the turbulent eddies that resuspend particles would entrain infective parasite spores. To test the hypothesis that basin shape influences the occurrence of epidemics, we recorded the prevalence of *M. bicuspidata* in populations of *D. dentifera* in 18 lakes that differed in basin geometry. Simultaneously, we tested the alternative hypothesis that higher host density (and its correlate, ecosystem productivity) should encourage disease spread. Surprisingly, our results revealed an association between basin shape and prevalence of this parasite, but in a direction contrary to our original prediction.

Methods

Host, parasite, and habitats

We studied the freshwater crustacean *Daphnia dentifera* (Crustacea: Cladocera) and its ascomycetous yeast parasite *Metschnikowia bicuspidata* (Mendonça-Hagler et al. 1993) in 18 lakes in southern Michigan (Barry and Kalamazoo Counties), USA. All lakes are of glacial origin and reside within a thick till plain of sand and loam. Lakes were chosen to reflect a gradient of surface area, depth, basin shape, and productivity (Appendix A). While we sought to include a broad range of basin morphology, we constrained our choices to lakes containing *D. dentifera* as a common member of the zooplankton assemblage. We also incorporated an index of ecosystem productivity (total phosphorus).

Transmission of *Metschnikowia* among *Daphnia* hosts is horizontal. *Daphnia* become infected by ingesting fungal asci, which are similar in size and shape to many algal cells. Because they are not motile, the asci likely depend on turbulence to keep them suspended in the water column. Once in the gut, the parasite propels its needle-shaped ascospores through the host's gut wall and proliferates in the host's body (Lachance et al. 1976). Infections are fatal, and the host does not become infective to other *Daphnia* until death, at which time asci are liberated. Hosts disappear from the water column in winter but remain in the sediment as diapausing eggs.

Field methods and analysis

Host density, thermal structure, and parasite prevalence were monitored every two weeks from July–November during 2002–2004. Water temperatures were recorded at 0.5–1 m intervals using a Hydrolab Minisonde 4a attached to a Surveyor 4a datalogger (Hach Environmental, Loveland, Colorado, USA). In 2004, time series temperature measurements were also obtained using self-contained temperature loggers (Brancker TR1050s [Richard Brancker Research, Ottawa, Ontario, Canada] in Wintergreen and Lawrence Lakes and Onset Stowaways [Onset Computer, Bourne, Massachusetts, USA] in other lakes). We also measured the two horizontal components and one vertical component of velocity at 8 Hz over a 24-hour period in Lawrence Lake, using a Nortek acoustic Doppler velocimeter (ADV; NortekUSA, Annapolis, Maryland, USA).

To determine host density and parasite prevalence, we used an 80-μm mesh (2002) or 153-μm mesh (2003–2004) Wisconsin bucket net to collect 6–8 bottom-to-surface samples of zooplankton. The sampling sites varied among dates but were always in the deepest part of the basin. Nets with smaller mesh sizes are known to be less efficient at capturing large zooplankton (Edmondson and Winberg 1971); hence, we corrected the 2002 data by applying a 70% net efficiency. On each date, half of the tows were pooled and preserved in >70% ethanol, and the other half were pooled but kept alive. Host densities were estimated from the preserved samples. From the live samples, we quantified the magnitude of infection in each lake by scanning each sample until at least 400 *D. dentifera* were scored or the entire sample had been searched. *Metschnikowia* infections are easily detected in the host's transparent body by a buildup of asci around the host's heart, and these spores eventually fill the majority of the body cavity. Prevalence of the parasite on each date was measured as the number of hosts that were infected relative to the total number of hosts scored.

We used standard morphometric parameters (Wetzel 2001) to compare basin shape with the prevalence of infections. Surface area, maximum depth, and mean depth were calculated from bathymetric maps. Most maps were available through the Michigan Recreational Boater Information System (*available online*).[7] For the other lakes, we generated maps with a Trimble GPS

(Trimble Navigation, Sunnyvale, California, USA; accuracy <0.7 m) and an Eagle Z-6100 depth finder (Lowrance Electronics, Tulsa, Oklahoma, USA). We took 165–429 points per lake and estimated depth contours using a distance-weighted least-squares algorithm in SYSTAT 10.0 (SPSS 2000).

A classic limnological metric that defines basin shape is depth ratio (mean depth/maximum depth). In general, as depth ratio increases, so does the overall steepness to the sides of the basin. Morphometric classifications by depth ratio best approximate basin shape when the lake has a single central depression (Carpenter 1983); however, most of our lakes are not simple basins. Hence, we calculated an additional morphometric parameter (Fee 1979). This parameter, which we refer to as "Fee's probability," is the probability that a sedimenting particle will fall on epilimnetic sediments as opposed to sinking through the thermocline and into deeper water. Although not as widely used as depth ratio, Fee's probability is more likely to accurately reflect the biology of this host–parasite interaction. Fee's probability is calculated as $1 - A_t/A_0$, where A_t is the surface area at the bottom of the epilimnion and A_0 is the surface area of the lake. Lower values of Fee's probability imply a more steeply sided basin, with less sediment exposed to the mixed epilimnion.

We used the regression tree routine available in SYSTAT 10.0 to explore the effects of productivity (spring total phosphorus), basin size (surface area) and shape (maximum depth, mean depth, depth ratio, Fee's probability), host density, and year of sampling on the maximum occurrence of *Metschnikowia* epidemics in these populations. Regression trees explain variation in a response variable (infection prevalence) by repeatedly splitting the data into groups that are as homogenous as possible (Breinam et al. 1984, De'ath and Fabricius 2000). We used the average value of our August and September samples for both Fee's probability (which changes seasonally with thermal structure) and host density (which changes over time). Because some of our metrics vary among years (infection prevalence, host density, Fee's probability) whereas others (phosphorus, surface area, maximum depth, mean depth, depth ratio) are constant among years, we fit two models. The first included each lake once with the three-year average for density and prevalence of infection, and the second considered each annual epidemic to be a unique event. We used the least squares loss function with a stopping rule of four cases per terminal node.

Finally, to investigate the possibility of spatial autocorrelation among our epidemics, we calculated the Mantel statistic, a common test for spatial relationships among variables (Legendre and Legendre 1998). Using a statistic closely related to Pearson's correlation, the Mantel test measures the extent to which variation in dissimilarity in measured variables (e.g., prevalence of infection) corresponds to spatial distances among lakes. Typically, comparisons between such distance matrices produce positive Mantel correlations, so we computed a single-tailed test using 49 999 randomizations (Legendre and Legendre 1998).

[7] ⟨http://www.mcgi.state.mi.us/MRBIS/⟩

Results

Metschnikowia epidemics displayed a similar seasonal phenology among lakes. Outbreaks in *Daphnia dentifera* do not develop before mid- to late August (Fig. 1), despite the fact that the host becomes abundant (>16 000 animals/m^2) in the water column of these lakes by June (C. E. Cáceres and A. J. Tessier, *unpublished data*). Infection prevalence often peaked in September, but infections were recorded in some lakes throughout October. Mean prevalence of infection among lakes is not strongly spatially autocorrelated (Mantel statistic, $r_M = 0.1395$, $P = 0.10$).

Fee's probability was the best predictor of the magnitude of epidemics in the first regression tree model. When single, three-year average values for host density and epidemics were used for each lake, basin shape (as Fee's probability) explained 49.8% of the variation in the magnitude of epidemics (Fig. 2A), and no other explanatory variable was useful in further partitioning the remaining variation. Contrary to our original hypothesis that lakes with gently sloping sides (high Fee's probability) would contain the majority of epidemics, outbreaks were only common in the lakes with the steepest sides (regression tree cut value: Fee's probability < 0.33, Appendix B). Baker Lake was the only lake with an average Fee's probability >0.33 in which we observed an epidemic (Fee's probability = 0.37). Fee's probability was a better predictor of epidemics than was depth ratio, although a similar relationship is also evident for this metric of basin shape (Fig. 2B). There were two lakes (Deep and Shaw) with large depth ratios that did not have epidemics. Shaw Lake is shallow and rarely thermally stratified, which changes the nature of trophic interactions. Although Deep Lake did not have infections in 2002–2004, it has had sizeable epidemics in the past (A. J. Tessier, *personal observation*).

Epidemics did not occur in all steep-sided lakes in every year (Fig. 1). When each annual epidemic in a lake is treated as a unique event in the second regression tree model, 19.5% of the variance in the magnitude of *Metschnikowia* was explained by basin shape (Fee's probability), which was the first of two splits in the regression tree (cut value, 0.45). An additional 22.7% of infection variation was explained in the second split due to year (Appendix B). This split reflects large interannual variation. In 2002, we only observed a *Metschnikowia* outbreak in Baker Lake, with the maximum prevalence reaching almost 17% of the population. In 2003, three lakes (Baker, Bristol, and Bassett) showed epidemics, but the maximum prevalence in any lake was

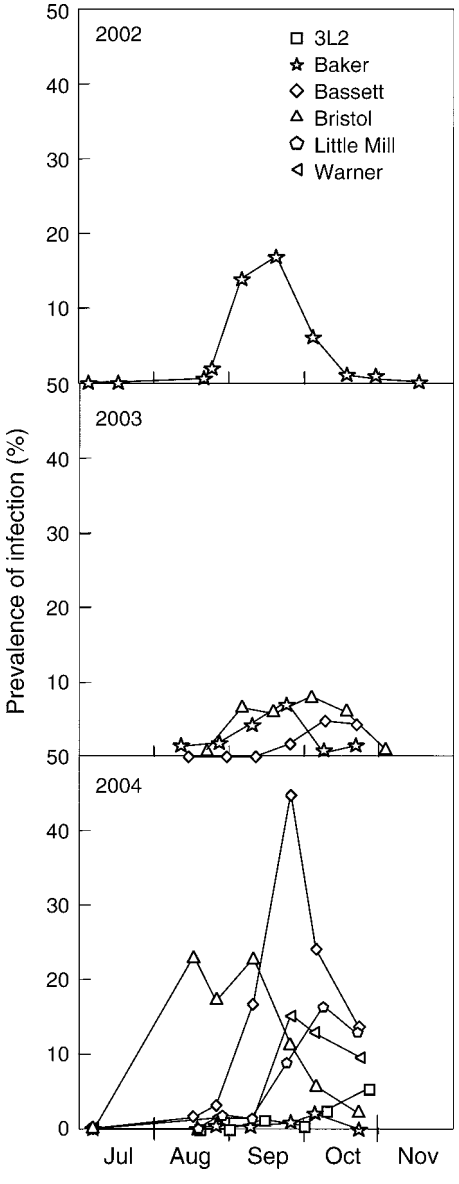

FIG. 1. Seasonal change in infection prevalence in *Metschnikowia* epidemics in six host populations of *Daphnia dentifera*, 2002–2004. For clarity, only populations that showed >5% incidence of infection in at least one of the years are plotted.

just over 8%. By 2004, six lakes had a maximum prevalence of *Metschnikowia* of at least 2%. However, this influence of year is only observed in the lakes having a Fee's probability <0.45. In short, interannual variation interacts with basin shape. Lakes with gradually sloping sides were devoid of epidemics, while those with a steep-sloped basin showed wide interannual fluctuation in parasite prevalence.

Depth ratio, surface area, maximum depth, mean depth, total phosphorus, or host density did not explain sufficient variance to result in further splits in either regression tree analysis. Host densities ranged from 300 to 378 000 animals/m^2, but all epidemics occurred when host densities were between 16 000 and 39 000 animals/m^2 (Appendix C). Because areal abundances do not take into account the potential for host aggregation, we also fit regression tree models with an estimated "maximum" host density. Since daphniids often spend much of the day concentrated in the area below the thermocline but above the zone of anoxia, we converted the areal abundances in each lake to an estimate of number of host animals per liter, assuming that all hosts were concentrated in this restricted area of the water column. This estimate of host density had no effect on the outcome of the regression tree analysis.

DISCUSSION

Our comparative study revealed that occurrence of disease in a common zooplankter is strongly related to the physical environment of lakes. Host density did not explain the spatial or temporal occurrence of epidemics, since outbreaks began months after the host *Daphnia dentifera* became abundant, and prevalence of infection did not correspond to variation in host density or lake productivity among systems. Instead, basin shape was a good predictor of epidemics. More specifically, epidemics only occurred in lakes with steep-sided basins. Several recent studies have documented the influence of environmental factors on host–parasite dynamics. For example, there is evidence linking climate variability to outbreaks of cholera (Pascual et al. 2000, Koelle et al. 2005). Johnson and Chase (2004) hypothesize that recent increases in parasitism rates in amphibians may be linked to cultural eutrophication. In the arctic, rising temperatures have altered the development time of a nematode parasite and consequently altered transmission dynamics (Kutz et al. 2005). These examples, together with our results, indicate that environmental factors, including physical structure of the habitat, are often important drivers of parasite epidemics.

Originally, our hypothesis regarding basin shape emphasized the direct physical processes of mixing and turbulence. Our predictions stemmed in part from the fact that *Metschnikowia* epidemics are seasonal and correlated with the onset of late-summer cooling and increasing turbulence. There is growing evidence connecting the role of turbulence to the seasonal occurrence of parasite epidemics in plankton. Doggett and Porter (1996) suggested that epidemics of chytrids on phytoplankton were correlated with periods of increased lake turbulence. Bittner et al. (2002) found that infections by the protist *Caullerya* in *Daphnia* increased in autumn, a time when a large volume of water should be turbulent due to cooling events. Our time series temperature measurements, obtained in lakes both with and without infections in 2004, indicated that epidemics were initiated following the passage of cold fronts (S. MacIntyre, *unpublished data*). Cold fronts increase turbulence within the epilimnion. Moreover, in Lawrence Lake, we recorded two turbulent mixing events,

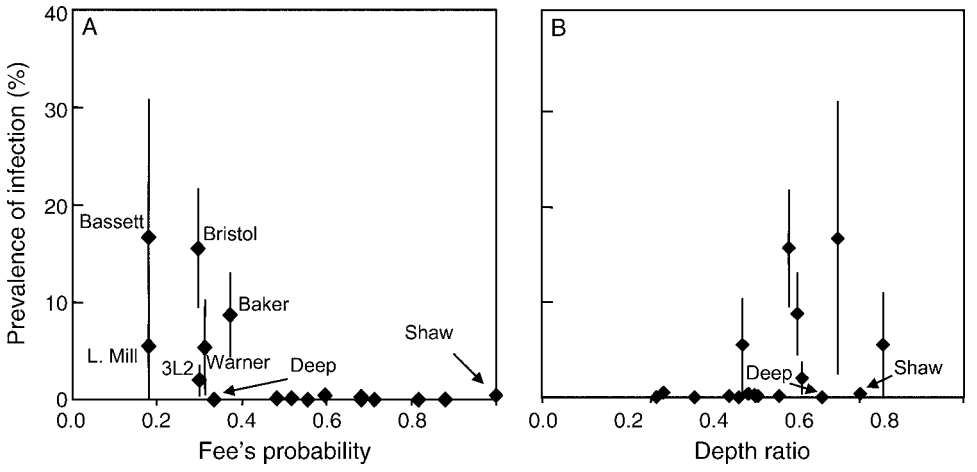

FIG. 2. Relationship between basin shape and magnitude of *Metschnikowia* epidemics in 18 populations of *Daphnia dentifera*. Infection rates are plotted as the three-year average of maximum prevalence in each population (mean ± SE). (A) Fee's probability is the probability that a sedimenting particle will fall on epilimnion sediments as opposed to falling through the thermocline and into deeper water. Lower values imply a more steeply sided basin. Values for Fee's probability are the three-year average. (B) Depth ratio is calculated as mean depth/maximum depth. In general, as depth ratio increases, so does the overall steepness to the sides of the basin. Unlike Fee's probability, depth ratio does not consider the depth of the thermocline.

which both indicate the potential for spore resuspension and horizontal transport (S. MacIntyre, *unpublished data*). Hence, our studies confirm earlier work indicating that turbulence induced by cold fronts or autumn cooling leads to onset of infections. Despite this correlation in timing, our original hypothesis regarding the role of basin shape, mixing, and epidemics was incorrect; epidemics were not more common in lakes with gently sloping sides. Although basin shape clearly influences the distribution of epidemics, our results suggest that interactions between the physical and biological effects of basin shape may also be important determinants of epidemics.

We propose two separate but related mechanisms to explain the prevalence of infections in deep, steep-sided lakes. The first is an indirect explanation: extrinsic mortality rate on the host may be lower in lakes where epidemics occur. Predation by fish is a major mortality source on *Daphnia*, and fish are highly selective on parasitized individuals (Duffy et al. 2005). By selectively culling infected hosts, predators can dramatically alter the dynamics of host–parasite interactions (Packer et al. 2003, Ostfeld and Holt 2004, Hall et al. 2005). In some lakes, *Daphnia* can reduce mortality rates due to visual predators by vertically migrating into a deepwater refuge (Wright and Shapiro 1990, Tessier and Welser 1991). However, some of our lakes (e.g., Shaw, Hall) are too shallow to stratify thermally. Without stratification, these lakes contain no deepwater refuge in which the hosts can escape predation. Thus in these shallow lakes, selective predation may prevent epidemics from occurring (Ebert et al. 1997, Duffy et al. 2005). In addition, although species richness of planktivorous fishes is similar in all of these lakes, variation in absolute and relative abundance of individual fish species among lakes of different basin shape may influence the amount of planktivory experienced by the hosts (Tessier and Woodruff 2002).

Our second explanation considers alternative mechanisms for how basin shape may directly influence the encounter rate of host and parasite. First, the hydrodynamic processes that induce resuspension may vary along gradients of basin shape in manners different from our original predictions. For instance, the effectiveness of physical processes in entraining and spreading spores may depend upon the presence or absence of rooted aquatic plants (macrophytes). High plant biomass reduces sediment (and likely spore) resuspension (James et al. 2004). Since macrophyte prevalence is often greater in lakes with sloping sides than steep sides (Kalff 2002), a biological factor (macrophytes) might influence physical transport of spores and explain the basin shape result. Additionally, the macrophytes may retard the horizontal dispersion of spores by wind-induced currents, gravity currents (James and Barko 1991), or internal wave motions (Lemmin and Imboden 1987). In addition, the encounter rate of spores and *Daphnia* may be influenced by host behavior, which is thought to play an important role in disease transmission in *Daphnia* (Ebert et al. 1997, Decaestecker et al. 2002, Pulkkinen and Ebert 2004). In our study lakes, *Daphnia dentifera* may increase their exposure to disease by swimming closer to shore in some lakes but not others. In lakes with steep sides, the pelagic zone (which includes the deepwater refuge) closely contacts nearshore sediment along the basin. Thus *Daphnia* in these lakes may inhabit regions with higher resuspension of spores. However, in lakes with more gently sloping sides, *Daphnia* tend to avoid the nearshore areas and remain offshore in deeper water (Gliwicz and Rykowska 1992). Avoidance of

nearshore areas by hosts in gently sloped lakes may reduce the encounter rate between hosts and spores. Our continued monitoring of these epidemics now includes an explicit consideration of the spatial distribution of the hosts and the infective stage of the parasite.

While basin shape restricted the occurrence of epidemics to particular lakes, interannual variation in disease prevalence was pronounced. We suspect that interannual variation in regional weather patterns contributed to the observed variance in disease prevalence. Cooler weather with an increased frequency of thunderstorms results in increased mixing and turbulence. If physical processes influence this host–parasite interaction, then we expect more epidemics in years with more turbulence. With only three years of data, we cannot yet test for a relationship between onset of annual cooling and regional prevalence in disease. However, the trend is in the right direction. Our first year of study (2002) was relatively warm, with temperatures well above average in August and September (National Oceanic and Atmospheric Administration, Washington, D.C., USA; *available online*);[8] only one lake had a sizeable epidemic. In contrast, 2004 was relatively cool, with cold fronts beginning by late July. It had the greatest number of epidemics, and they began earlier in the year.

There are, of course, other potential explanations for the occurrence of *Metschnikowia* epidemics in *Daphnia* hosts. For example, several authors have argued that host resistance is important in explaining the dynamics of host–parasite interactions (Thrall and Burdon 2000, Little 2002). In our study lakes, individual clones differ in their susceptibility to this parasite (Duffy et al., *unpublished manuscript*), but host resistance alone cannot explain the absence of infections in either space or time. We have conducted laboratory and field enclosure experiments with 16 of our 18 host populations, and we have successfully created epidemics in these 16 populations by introducing spores at moderate concentrations. Moreover, we have observed *Daphnia* infected with *Metschnikowia* at low levels in 15 of the 18 study lakes, and we do not find a strong signal of spatial autocorrelation in disease prevalence. This result means that dispersal of parasites among systems does not seem to limit occurrence of this parasite. Consequently, since most populations can be infected, and the parasite is present in most lakes, factors other than host resistance or interlake dispersal of the parasite must constrain regional occurrence of this disease.

It is becoming increasingly apparent that disease ecology must move beyond the study of host–parasite interactions in isolation. Instead, ecologists sorely need a broader framework that jointly considers interactions among biotic drivers, such as selective predation, and abiotic/physical drivers of epidemics (Lafferty and Holt 2003). Our study system provides an opportunity to begin teasing apart these interactions using basin shape as the relevant environmental gradient. Clearly, basin shape of lakes plays a key role in determining occurrence of *Metschnikowia* outbreaks in *Daphnia*. A priori explanations based purely on the direct effect of mixing on transmission rate, and their links to basin shape, remain incomplete. Therefore, to fully understand planktonic host–parasite interactions, we must simultaneously couple studies of physical mixing processes with exploration of the community ecology of the host and parasite.

[8] ⟨http://www.noaa.gov/climate.html⟩

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APPENDIX A

Habitat characteristics for the 18 lakes included in this study (*Ecological Archives* E087-081-A1).

APPENDIX B

Results from the regression tree analyses (*Ecological Archives* E087-081-A2).

APPENDIX C

Seasonal dynamics of host populations (*Ecological Archives* E087-081-A3).