Why Are *Daphnia* in Some Lakes Sicker? Disease Ecology, Habitat Structure, and the Plankton

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*Some aspects of habitat seem to enhance the spread of disease whereas others inhibit it. Here, we illustrate and identify mechanisms that connect habitat to epidemiology using a case study of disease in plankton. We see a pronounced relationship between the basin shapes of lakes and fungal (*Metschnikowia bicuspidata*) disease in the zooplankton grazer* Daphnia dentifera. *As we work through seven mechanisms that could explain why* Daphnia *in some lakes are sicker, we can eliminate some hypotheses (i.e., those relating an index of lake productivity to disease through host density, links between resource quality and transmission rate, and variation in host susceptibility) and find support for others involving food-web actors (e.g., selective predation on infected hosts by fishes, "sloppy predation" by an invertebrate, a possible dilution effect in V-shaped lakes). Furthermore, we identify physical mechanisms (gravity currents, turbulence) that could lead to greater transport of fungal spores to habitat occupied by* Daphnia *hosts in U-shaped lakes. These results highlight how habitat structure, through its effects on food-web structure and physical processes, can shape wildlife disease.*

Keywords: gravity currents, dilution effect, host-parasite interaction, productivity, selective predation

Epidemiologists have long known that disease occurs nonrandomly in space (Ostfeld et al. 2005, 2008, McCallum 2008). For instance, human and wildlife diseases are often strongly associated with particular features of habitat structure, such as patch size or landscape composition in tick- and rodent-borne diseases (Langlois et al. 2001, Allan et al. 2003), deforestation in mosquito-borne malaria (Molyneaux 2002), and fragmentation in salamander-virus systems (Greer and Collins 2008). The goal of uncovering general mechanisms that link habitat to these types of disease outbreaks is an exciting challenge. Once these mechanisms are delineated, sharper predictions might then be made concerning the likelihood of epidemics in particular locations (McCallum 2008).

How, then, does habitat foster or depress disease? Habitat variation might mechanistically shape disease through one of three potentially overlapping routes. First, habitat may vary in quality for pathogens or hosts. For example, forest harvest can create temporary pools used by *Anopholes* mosquitoes (Molyneaux 2002), eutrophication can boost host density (or even decrease it; Johnson and Carpenter 2008), and favorable temperature and rainfall patterns can promote survival of tick vectors (Cumming 2002, Brownstein et al. 2003). Second, habitat may create variation in densities of other species that either increase disease presence (e.g., through spillover from other species; Power and Mitchell 2004) or decrease it (e.g., through selective predation on infected hosts, removal of infectious stages through the "dilution effect," and others; Keesing et al. 2006). Third, habitat quality, patch size, or connectivity can aid in transfer of infectious individuals or stages of the parasite (measles in Britain, Grenfell et al. 2001; plague in prairie dogs, Strapp et al. 2004). Thus, links between habitat and disease can arise from the effect of habitat on host or parasite density, on community context, or on the spread of disease in space.

A habitat-disease pattern in lakes

This article examines the ability of habitat-disease links to explain a pronounced spatial pattern of disease in aquatic systems. Our focal host is a crustacean zooplankton grazer, *Daphnia dentifera*. Like most *Daphnia* species, it plays a pivotal role as grazer and prey in freshwater lentic food webs. These zooplankton in lakes and ponds, like most animals, suffer infection from a variety of parasites (Ebert 2005, Wolinska et al. 2007, Johnson et al. 2009, Duffy et al. 2010). Infection by many of these parasites has undergone extensive study (summarized in Ebert 2005); thus, *Daphnia* and their parasites offer an excellent opportunity to link ecology with disease. *Daphnia* species (including *Daphnia magna*, *Daphnia pulicaria*, *Daphnia dentifera*, and the *Daphnia galeata/hyalina/*

BioScience 60: 363–375. ISSN 0006-3568, electronic ISSN 1525-3244. doi:10.1525/bio.2010.60.5.6

cucullata complex) seem particularly vulnerable to a set of bacterial, fungal, and oomycetous parasites. These parasites vary in prevalence across locations and years (from 0% to 50% infection rates, even up to 100% infection rates), and cause highly variable negative effects on host survival and reproduction. These effects can sometimes translate into negative impacts on host populations (Duffy and Hall 2008, Johnson et al. 2009).

Here, we focus on a case study involving a particular host-parasite combination. In a set of small north temperate lakes, we see large epidemics of a common, virulent fungus (*Metschnikowia bicuspidata*, an ascomycetous yeast) in *D. dentifera* host populations in lakes with a certain habitat characteristic (Cáceres et al. 2006). More specifically, steep-sided lakes with more U-shaped basins often have large epidemics, whereas more gently sloped, V-shaped ones typically do not (figure 1; see box 1 for specifics about the basin-shape indices—one could wade out many tens of meters in typical V-shaped lakes but could only walk out a couple of meters at most in very U-shaped lakes). This disease pattern most likely arises from the prominent role that basin morphology plays in shaping key components of the underlying *Daphnia*-fungus epidemiology—factors that influence host or parasite density, the abundance of other species that modulate disease, or movement of parasites. Decades of prior research highlight the intimate relationships between basin shape and primary and ecosystem production (particularly through the release of limiting nutrients from sediments and vertical mixing; Fee 1979, MacIntyre and Jellison 2001, Vadeboncour et al. 2008), the facilitation of vertical refuges from predation resulting in profound impacts on *Daphnia* grazers and their algal prey (Wright and Shapiro 1990, Tessier and Welser 1991, Tessier and Woodruff 2002), and the impacts on internal transport through physical processes (Sturman et al. 1999). We draw on this background to evaluate seven hypotheses that could explain this basin shape–disease pattern (table 1, figure 2).

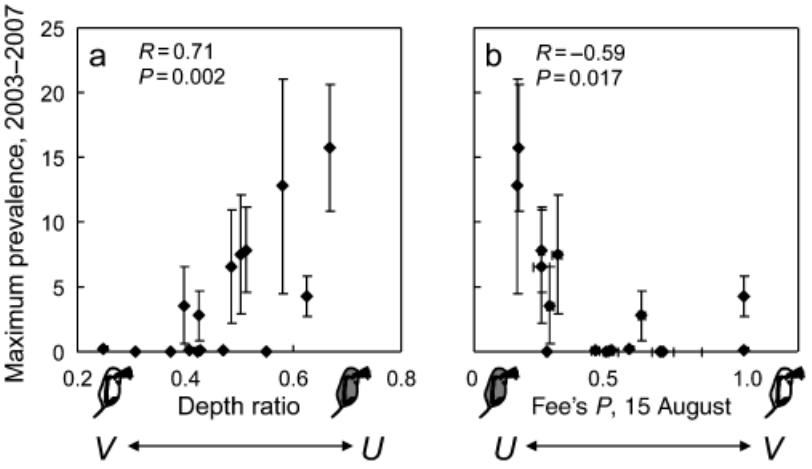

Figure 1. Mean of the maximum prevalence of infection of the fungus* Metschnikowia bicuspidata *in lake populations of* Daphnia dentifera*, 2003–2007, versus two indices of lake morphology. (a) Depth ratio is mean to maximum depth, where high values indicate steeper-sided basins (i.e., U-shaped rather than more shallow-sloped V-shaped). (b) Fee's* P *measures the proportion of lake sediments that are exposed to the epilimnion. Values here correspond to estimates of 15 August values, averaged over 2004–2006 (see box 1 for details). Fee's* P *integrates depth ratio and epilimnetic depth (which themselves are correlated); lower values also correspond to steeper basins, often with shallower epilimnion. All points are means ±1 standard error. Pearson correlation statistic,* R*, with associated* P *values are provided. Gray* Daphnia *drawings here correspond to lakes with more disease (i.e., U-shaped basins), whereas white ones indicate those with less (i.e., V-shaped basins).

The basics of *Daphnia*-fungus epidemiology

Before delving into these ideas, we need to review the basics of *Daphnia*-fungus epidemiology. Relative to many wildlife disease systems, this epidemiology is fairly simple (solid arrows, figure 2). *Daphnia dentifera* is a dominant grazer in the open waters of many of the deeper north temperate lakes (Tessier and Woodruff 2002). These hosts come into contact with free-living infective stages (spores) of the fungus while consuming their algal prey (Hall et al. 2007a). If successfully infected, hosts experience virulent effects on their fecundity (Duffy and Hall 2008, Hall et al. 2009b, 2009c) before they eventually die, full of spores. Thus, this "obligate killer" fungus must kill its host to spread (Ebert and Weisser 1997). However, spores exiting dead hosts risk sinking out of the water column before they come into contact with new, uninfected hosts (figure 2).

Seven mechanisms could produce the basin shape–disease pattern

Basin shape could indirectly shape this epidemiology through at least seven different pathways (table 1, figure 2). The ecological factors behind these pathways are not necessarily independent. Instead, several could arise from interrelated links between habitat structure and food-web architecture, especially those factors involving vertebrate fish predation and its subsequent direct impacts on host density and habitat use, community composition of other zooplankton, and algal resource quality, among others (Tessier and Woodruff 2002). However, these plausible correlated factors might conflict with each other (e.g., selective predation and the dilution effect, as outlined below). Regardless, each factor also operates independently on epidemics, through unique mechanisms. For this reason we treat them separately and conclude with a synthesis.

Box 1. Methods for field surveys and experiments testing the seven potential mechanisms.

We evaluated the possible mechanisms using different field surveys and experiments, and describe our findings below.

Field surveys: The basin shape–disease pattern, productivity-density (mechanism 1), fish predation (4), sloppy predation (5), and dilution effect (6).

To evaluate several mechanisms, we used data collected from our ongoing monitoring program in 18 lakes in southwest Michigan (see Cáceres et al. 2006).

Indices of basin shape: During fortnightly visits to each lake, we used a Hydrolab multiprobe to measure temperature-depth profiles. With these data, we calculated the depth of the epilimnion (i.e., the upper, warmer layer of stratified lakes; Wetzel 2001), which was then used to estimate Fee's *P* index (Fee 1979). This metric reflects the proportion of lake sediments exposed to the epilimnion (see Cáceres et al. 2006). Since epilimnetic depth drops during the summer-autumn transition, we estimated Fee's *P* on 15 August of each year as a metric. This 15 August metric is highly correlated among years (Pearson $R > 0.97$), and it correlates strongly with a similar metric calculated for 15 September (Pearson $R > 0.90$). Thus, although Fee's *P* changes temporally, it provides a robust index of lake shape. Depth ratio, another related but static index of lake shape, correlates negatively each year ($R > -0.80$) with the 15 August measurement of Fee's *P*.

Infection prevalence, host density and size, and water chemistry: At each visit, we also collected two samples of zooplankton, each with three bottom-to-surface tows of a Wisconsin net. We visually diagnosed more than 400 live animals from one sample following Green (1974), then preserved the other for estimation of densities of crustacean zooplankton (mechanisms 1, 4, and 6). Additionally, during these surveys in September 2004, we measured the length of uninfected adult *Daphnia dentifera* (mechanism 4; data from Hall et al. 2007a). During July 2007, we estimated densities of *Chaoborus* in 12 lakes (mechanism 5) with samples collected from each lake at night (data from Cáceres et al. 2009). Finally, we used spring-turnover measurements of total phosphorus, an index of lake productivity (data from Cáceres et al. 2006).

Laboratory experiment: Resource quality–transmission experiment (mechanism 2)

During July 2007 we evaluated links between variation in resource quality among lakes and disease transmission rate (mechanism 2). We describe related experiments in detail elsewhere (Hall et al. 2009a). We collected algal seston from eight deep, stratified lakes that spanned a gradient of basin shape and adjusted the concentration to achieve 1.0 milligram (mg) per dry liter (L). We added this seston to beakers containing six, six-day-old animals and 25 spores per milliliter (mL). Following a 24-hour exposure period at 20 degrees Celsius (°C), we placed hosts into fresh water with good food (2.0 mg/L of *Ankistrodesmus*) and allowed them to develop infection. Meanwhile, we measured resource quality with a juvenile growth rate assay following protocols used previously (i.e., with the "Geedey" clone; Tessier and Woodruff 2002; see Hall et al. 2009a).

Field microcosm experiment: Host susceptibility (mechanism 3)

From late June through early July 2005, we assayed hosts from 12 populations for susceptibility to infection (mechanism 3) using *in situ* assays of infectivity. On the day of setup, we filled four 1-gallon plastic jugs with epilimnetic water and suspended them at a depth that achieved approximately 25°C (approximately 2.0–3.5 meters (m), depending on the lake). Then, at night, we returned to each lake to collect and place 30 adults in each jug filled with 100 spores per mL. (Spores originated from an infected animal in one of the study lakes [Baker], raised *in vivo*). On day eight we collected animals from each jug and tested them to determine infection prevalence.

Field deployment: Gravity currents (mechanism 7)

Arrays of temperature loggers were deployed in several lakes in 2004 and 2006 to measure onshore-offshore temperature differences and look for signs of gravity currents (mechanism 7). To create thermistor chains, temperature loggers were attached at regular depth intervals to weighted lines buoyed by floats. Two to three thermistor chains of varying depths were then deployed along an inshore-to-offshore transect along the sloping boundary, and another subsurface thermistor chain was deployed near the deepest area of the basin.

Field survey: Hosts and distance from shore (mechanism 7)

We characterized spatial location of *Daphnia* hosts using nearshore-offshore surveys during August and September of 2005. In eight lakes spreading a gradient of depth ratio, we collected zooplankton along four transects at five locations each from shore to open waters. We then fit a line through density-distance data from each transect and estimated the *x*-intercept from each regression (mean intercept ±1 standard error is shown). The *x*-intercept corresponds to the distance from shore at which we first find *D. dentifera*.

To begin, we can eliminate the possibility that the fungal parasite is simply missing from V-shaped lakes that lack epidemics, as we have found low levels of infection (< 1%) in all but two lakes in this study. We therefore consider three factors that influence host density and parasite transmission. First, we address a "productivity-density" mechanism (mechanism 1; Johnson et al. 2007, Johnson and Carpenter 2008). Basin shape, through its effects on nutrient regeneration (particularly phosphorus, a common index of lake productivity and eutrophication), could increase host density and therefore enhance disease transmission. Additionally, algal resource quality in heavily grazed, U-shaped lakes could enhance transmission relative to V-shaped lakes (mechanism 2, "resource quality transmission"; Hall et al. 2009a, see also Frost et al. 2008a). Alternatively, hosts in V-shaped lakes could be particularly resistant to infection as a result of genetic or genetic-by-environment mechanisms (mechanism 3, "host susceptibility"; Carius et al. 2001, Ebert 2005, Duffy and Sivars-Becker 2007, Duffy et al. 2008).

Table 1. Summary of the biological and physical mechanisms pursued to determine why **Daphnia** *in some lakes are sicker.*

Mechanism	Rationale	Result/finding	Conclusion
0. Absence of parasites	Epidemics cannot occur without parasites	At least small levels of infection in all but 2 of 18 lakes	Reject
1. Productivity–host density	Higher total phosphorus (TP) supports higher host densities, hence more disease	Small gradient of TP, weak negative relationship between Fee's *P* and TP, no link between TP and host density; however, a trend toward positive TP-prevalence relationship	Reject
2. Resource quality transmission	Resource quality can influence transmission rate	Resource quality was poor in both U- and V-shaped lakes, transmission rate was high in both types of lakes	Reject
3. Host susceptibility	Host genotypes are resistant to infection in V-shaped lakes	Hosts are equally susceptible in V-shaped lakes as in U-shaped lakes, if not more so	Reject
4a. Fish predation: "healthy herds"	Highly selective predation can inhibit epidemics	Two indices of vertebrate predation (host size and plankton community composition) indicate higher vertebrate predation in V-shaped lakes than in U-shaped ones	Support
4b. Fish predation: body size	Larger body size enhances disease transmission directly	Hosts were larger in U-shaped lakes than V-shaped lakes	Support
5. Sloppy predator	This predator may spread spores and facilitate epidemics	*Chaoborus* densities were higher in U-shaped lakes than in V-shaped lakes	Support
6. Dilution effect	High relative densities of other hosts may inhibit disease through a "dilution effect"	Epidemics occurred in lakes with higher densities of the diluter *Daphnia pulicaria*, but not in lakes with other diluters	Mixed
7. Gravity currents	Horizontal water movement could transport spores from nearshore to offshore waters	Evidence for gravity currents in U-shaped lakes but not V-shaped lakes in paired observations of temperature-depth profiles; hosts appear closer to shore in U-shaped lakes	Support

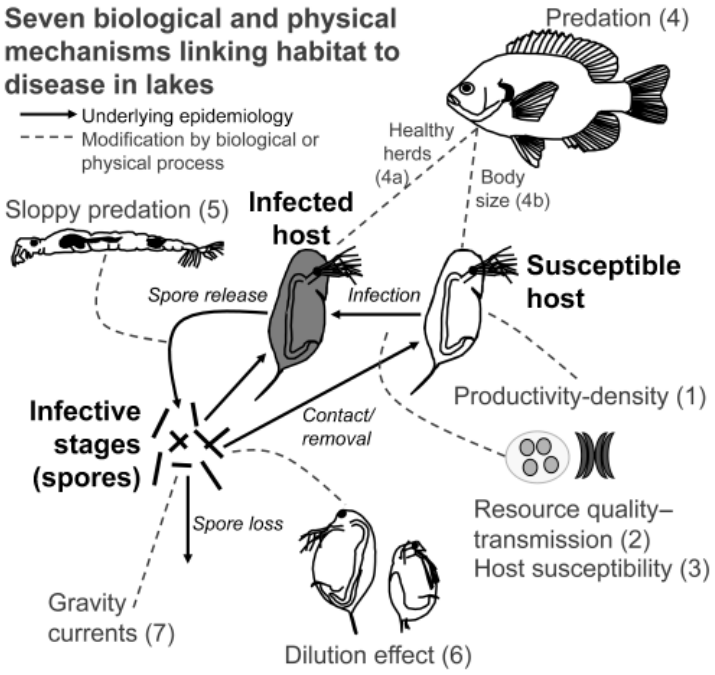

Figure 2. Graphical diagram of the seven mechanisms that basin shape could mediate interactions between* Daphnia *hosts and fungal parasites. Each of the potentially interrelated mechanisms (indicated by a number) influences some component of the underlying epidemiology of this system (solid arrows). In this type of "obligate killer" epidemiology, infection occurs as susceptible hosts contact and consume free-living (floating) infective stages (spores) of the parasite. Once successfully infected, sick hosts accumulate spores within hemolymph (blood) and experience reduced fecundity until they ultimately die. Following death, spores are released. Basin shape could influence this epidemiology by (dashed lines): increasing host density through nutrient regeneration (productivity-density, mechanism 1), and altering transmission through resource quality (2) or host susceptibility (3); boosting density of highly selective vertebrate predators (particularly bluegill sunfish, through two pathways, "healthy herds" and body size (4), or sloppy predators which facilitate spore release from infected hosts (5); enhancing or diminishing densities of hosts which consume spores without becoming infected (i.e., a dilution effect, number 6); or enabling a physical process (gravity currents) that helps transport nonmotile spores to open water areas of the lake inhabited by* Daphnia *hosts (7).

The next three mechanisms involve the influence of basin shape on other species that enhance or disrupt key aspects of the host-parasite interaction. Higher levels of vertebrate predation (indexed by body size of hosts and zooplankton community composition; see box 1) in V-shaped lakes could reduce disease in two interrelated ways. In the "fish predation: healthy herds" mechanism (4a), highly selective predation on infected hosts could inhibit epidemics—if, for example, bluegill sunfish (*Lepomis macrochirus*) ultimately serve as net sinks for parasite propagules in infected hosts (Duffy et al. 2005, Hall et al. 2005, Duffy and Hall 2008, Cáceres et al. 2009, Duffy 2009). Additionally, higher vertebrate predation should reduce the mean size of *Daphnia* hosts; this size reduction directly depresses disease transmission, all else being equal ("fish predation: body size" mechanism [4b]). Alternatively, a high abundance of another type of predator, invertebrate species of *Chaoborus*, could spread disease through a completely different pathway. Elsewhere, we detail how "sloppy predation" (mechanism 5) works: In short, *Chaoborus* that eat infected hosts could spread disease if they disperse spores into areas where spores could remain suspended and able to infect hosts (Cáceres et al. 2009). While suspended, those spores could also be eaten by other unselective grazers that are not as readily infected (e.g., *D. pulicaria*). These species could thus reduce disease transmission through a "dilution effect" (mechanism 6; Keesing et al. 2006, Hall et al. 2009a) in V-shaped lakes if those lakes are proportionally dominated by unsuitable hosts.

The final mechanism involves a basin shape–induced difference in the physical processes that resuspend and transport settled spores into open waters where they can encounter hosts. Since these infective propagules cannot swim, they remain at the mercy of water motion for transport: Some kind of physical process must connect spores resting in lake-bottom or nearshore habitats to offshore hosts. We have already rejected the hypothesis that epidemics are more common in V-shaped lakes because they have a greater surface area of sediments in contact with turbulent epilimnetic water (Cáceres et al. 2006). Epidemics begin after cooling events in late summer and early autumn, after many weeks of high host abundance (Cáceres et al. 2006). Density-driven gravity currents are a physical mechanism consistent with both the basin-shape pattern and the weather when epidemics begin. Gravity currents arise from differential cooling of nearshore versus offshore waters; this cooling could then drive a flow capable of transporting spores deposited nearshore to offshore waters (James and Barko 1991). Once offshore, those spores could infect *Daphnia* hosts. Transport by gravity currents is more likely in U-shaped lakes, as steeper-sided slopes should promote faster and larger flows (Sturman et al. 1999). Furthermore, transported spores might need to be carried shorter horizontal distances before encountering hosts in U-shaped lakes; in steep-sided basins, the deep refuge required for vertically migrating hosts like *D. dentifera* may occur closer to shore.

Evaluation of the seven hypotheses

On the basis of data from a combination of field surveys, lab and field experiments, and deployments of instruments monitoring water temperature (see box 1 for details), we can now separate the more probable hypotheses from those less likely to explain the basin shape–disease pattern.

1. Productivity-density mechanism: Tentatively reject

The weak relationship between basin shape and total phosphorus (TP) probably does not explain the basin shape–disease pattern, at least not by the mechanism that we hypothesized, because TP and host density seem disconnected (Johnson et al. 2007, Johnson and Carpenter 2008). Total phosphorus declined somewhat with basin shape; V-shaped lakes (high Fee's *P*, a shallow slope, and a high proportion of sediment exposed to the epilimnion) had lower TP (figure 3a; TP data from Cáceres et al. 2006). However, TP did positively correlate with infection prevalence (figure 3b), but it appears that host density was not involved here because neither TP (figure 3c) nor disease prevalence (figure 3d) showed any relationship to host density (averaged during August and September of 2004–2006). That said, the positive TP-prevalence correlation suggests some connection that does not involve host density. One simple possibility might be a link between light extinction and TP; perhaps higher TP correlates with more algal biomass, which shades the water column and provides a darker refuge for hosts from visually oriented predators (fish). Although TP and light extinction were related (all lakes: $R = 0.80$, $P < 0.001$; excluding eutrophic Wintergreen: $R = 0.60$, $P = 0.014$), light extinction and infection prevalence were quite uncorrelated ($R = 0.02$, $P = 0.98$; excluding Wintergreen: $R = 0.2$, $P = 0.46$). So, although we can reject the TP-induced light-extinction explanation, other mechanisms meriting future exploration may be involved.

2. Resource quality transmission: Reject

We can eliminate preepidemic resource quality as a driver of transmission rate along a U- to V-shaped lake gradient (mechanism 2). Previous work showed that transmission rate is strongly shaped by quality of algal resources available to *Daphnia* hosts (as indexed by juvenile growth rate assays; Hall et al. 2009a). Although algal resource quality shifts dramatically (a) along a basin-shape gradient from shallow ponds to deep lakes and (b) within a season in particular lakes (Tessier and Woodruff 2002, Hall et al. 2009a), overall resource quality was rather poor along the gradient in deeper lakes from U- to V-shape during our experiment (figure 4a). This result suggests that all of these lakes support sufficient grazing pressure to drive algal communities toward more indigestible and less nutritious forms (DeMott and Tessier 2002). Nonetheless, no resource quality transmission signal emerged from the experiment (figure 4b). Furthermore, we detected higher resource-dependent transmission using seston collected from V-shaped lakes, where epidemics were lower, than from U-shaped lakes (marginally significant; figure 4c). Resources do not seem to impede transmission of parasites in V-shaped lakes.

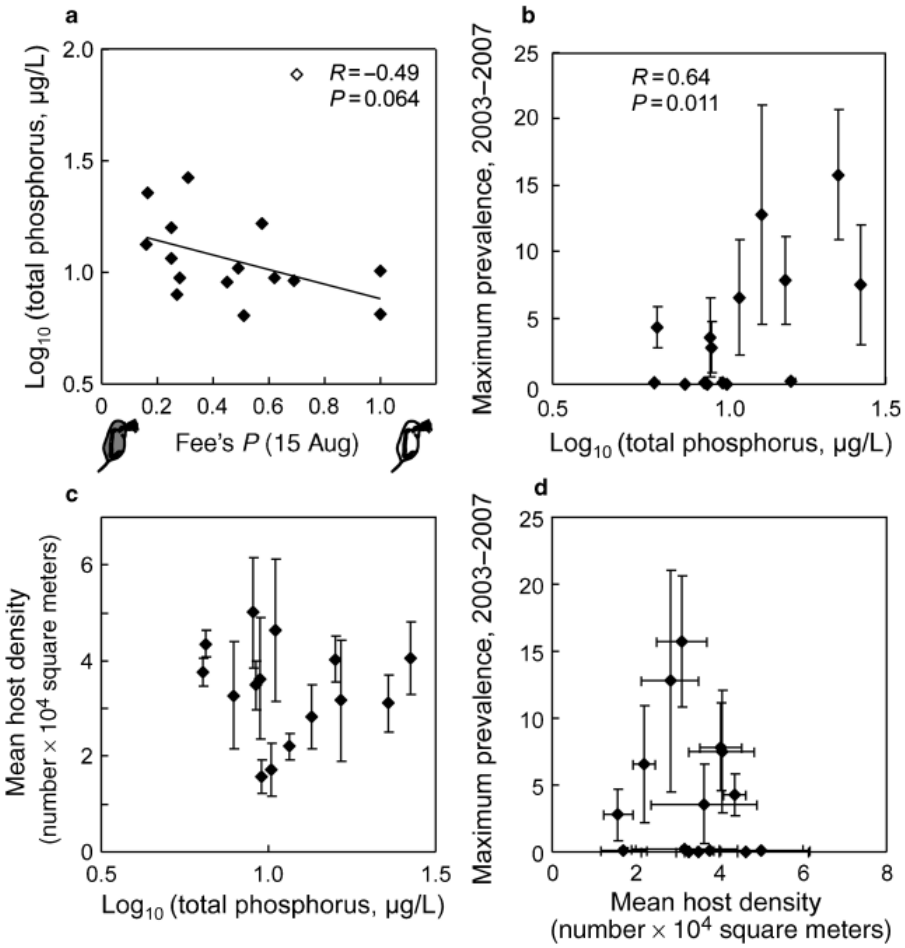

Figure 3. Phosphorus, basin shape, host density, and disease prevalence in the* Daphnia *host-fungal parasite system (mechanism 1). (a) Although total phosphorus (TP, an index of system productivity) at spring turnover does not correlate with the depth ratio–based index of basin shape (R = 0.12, P = 0.68; not shown), TP does decline with the 15 August average of Fee's P if we exclude the obvious outlier (eutrophic Wintergreen Lake, the open diamond point). (b) Since mean prevalence of infection also declines with Fee's P, not surprisingly we do find a positive correlation between maximum prevalence of infection and TP (in micrograms per liter, µg per L). However, this TP-prevalence link does not seem to involve host density, since (c) TP (µg per L) and host density are not correlated, nor for that matter are (d) host density and maximum prevalence. Pearson correlation statistic, R, with associated P-values provided.

3. Host susceptibility: Reject

Additionally, we found no relationship between basin shape and host susceptibility in an *in situ* microcosm experiment in which hosts from each lake were incubated with spores (figure 4d). Thus, hosts were not dramatically less susceptible to this virulent fungal parasite in V-shaped lakes where epidemics are uncommon. This result echoes those from more controlled lab experiments with genotypes from fewer lakes (Duffy and Sivars-Becker 2007). We continued to find no relationship between host susceptibility and basin shape even when we controlled for slight variation of temperature between lakes (since temperature elevates transmission; Hall et al. 2006) and mean body size of adults among lakes (since size also elevates transmission; Hall et al. 2007a). If anything, hosts collected in summer 2005 from lakes that had epidemics in the previous year (2004; gray symbols in figure 4d) were marginally less susceptible to infection than hosts collected from lakes without epidemics in 2004 (*t*-test, $t = 1.84$, $df = 10$, $P = 0.095$), a result consistent with previously observed rapid evolution of hosts in response to epidemics (Duffy and Sivars-Becker 2007, Duffy et al. 2008). This assay indicates that epidemics were not inhibited by resistance of hosts in V-shaped lakes on a regional, across-lake scale.

4. Vertebrate predation: Healthy herds (a) and body size (b): Support

A suite of food-web factors that responded strongly to basin shape also correlated with disease prevalence. First, the size index of vertebrate predation intensity (body size of uninfected adult hosts) diminished with a measure of basin shape, Fee's *P* (figure 5a; it also increased significantly with our second measure of basin shape, depth ratio, not shown: $R = 0.54$, $P = 0.026$). In other words, hosts were smaller in V-shaped basins. Furthermore, the density of large-bodied species (e.g., *D. pulicaria*) was higher in U-shaped basins (figure 5e), but smaller cladocerans (*Daphnia retrocurva*, *Bosmina*, *Ceriodaphnia*, *Diaphanosoma*) became more abundant in V-shaped basins (figure 5g). These patterns reflect a classic signal arising from decades of research in freshwater ecological research: Higher vertebrate predation pressure correlates with smaller plankton (Brooks and Dodson 1965, Mills et al. 1987, Carpenter and Kitchell 1993). Therefore, vertebrate predation pressure in V-shaped lakes (without epidemics) almost certainly exceeds that in U-shaped lakes. This result further extends the well-known role of basin geomorphology in determining predation pressure in freshwater systems: Shallower lakes that lack deep refuges for large-bodied zooplankton experience higher predation pressure and are

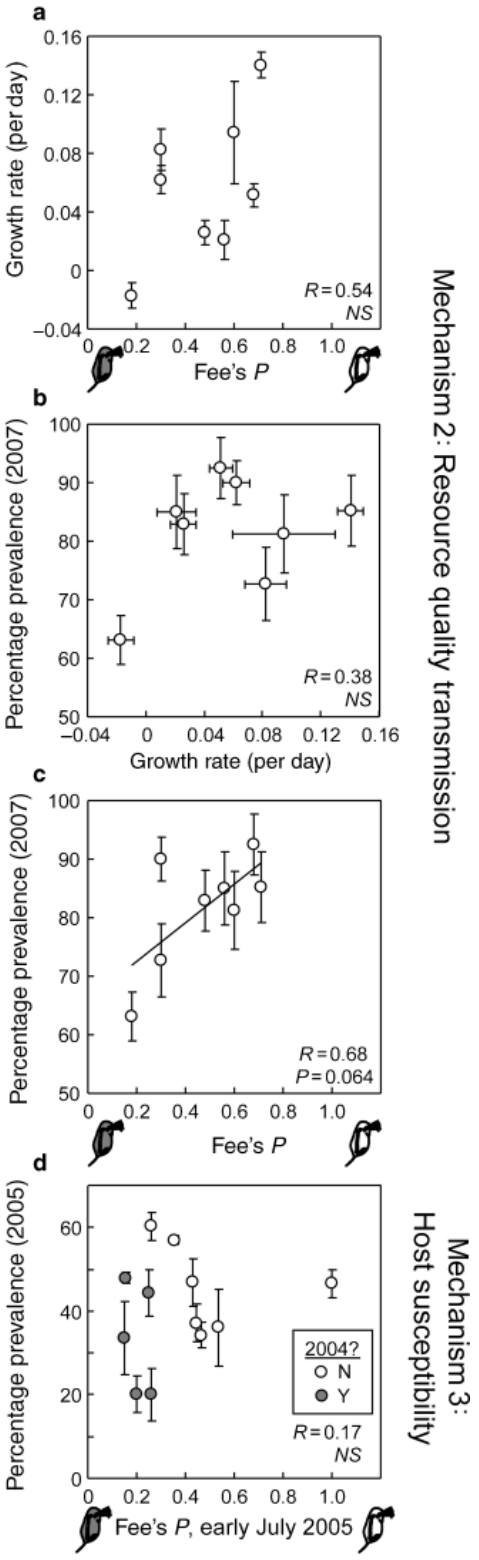

Figure 4. Two factors that likely do not explain the basin shape–disease relationship. Mechanism 2: Resource quality–transmission rate. (a) Resource quality (indexed as juvenile growth rate per day) correlates positively but insignificantly with Fee's* P*—but resource quality overall is rather poor. (b) Resource quality does not correlate with the index of transmission rate, but (c) basin shape does positively correlate with transmission. Source: Experimental data from 2007. (d) Mechanism 3: Host susceptibility. Hosts in the field microcosm experiment were not overall more or less susceptible along the basin-shape gradient. Source: Experimental data from 2005. Pearson correlation statistic,* R*, with associated* P*-values provided. Gray* Daphnia *correspond to lake shapes with more disease; white* Daphnia *indicate those with less.

dominated by smaller plankton (Tessier and Welser 1991, Tessier and Woodruff 2002). Therefore, both depth (demonstrated in previous work) and lake shape (demonstrated here) modulate the influence of predation on community structure of plankton.

These vertebrate predation–body size results have major implications for disease in *Daphnia* in deeper lakes for two reasons. First, bluegill sunfish prey selectively on infected hosts (mechanism 4a; Duffy et al. 2005, Johnson et al. 2006, Duffy and Hall 2008). If predators act as net sinks for parasites contained in infected hosts and do not ultimately disperse many spores, a high intensity of selective predation could inhibit overall epidemics—that is, predators "keep the herds healthy" by culling sick hosts (Packer et al. 2003, Ostfeld and Holt 2004, Duffy et al. 2005, Hall et al. 2005, 2006, Cáceres et al. 2009). Of course, fish do not act as complete sinks for fungal spores; we know that at least some spores remain viable post defecation (Duffy 2009). However, the preponderance of spores trapped in egested fecal pellets most likely sink out of the water column before they are freed (Threlkeld 1979). Second, all else being equal, larger-sized hosts are more easily infected (mechanism 4b; Ebert 2005, Hall et al. 2007a). This size-infection link arises because hosts come into contact with spores while feeding, and feeding rate increases with host size (Mourelatos and Lacroix 1990; see Hall et al. 2007a for further discussion of this issue as it relates to the gut size of hosts). Additionally, larger hosts might be older; prevalence might reach higher levels in older hosts that are in contact with parasites' spores for longer periods of time. For both reasons, then, higher vertebrate planktivory in V-shaped lakes should reduce the success of the parasite through size-dependent transmission rate, all else being equal. The lake-shape pattern here indicates that variation in vertebrate planktivory may strongly shape the prevalence of infection through multiple mechanisms. However, the net effect of higher fish predation—given its intensity and selectivity, attendant changes in host size, egestion of some spores, and so on—inhibits epidemics.

5. Sloppy predation: Support

Density of the sloppy-feeding invertebrate predator *Chaoborus* responded oppositely to our indices of vertebrate predation. *Chaoborus* declined with Fee's *P* (as estimated when *Chaoborus* was sampled; figure 5c; *Chaoborus* increased with depth ratio, not shown: $R = 0.62$, $P = 0.032$). Thus, hosts in U-shaped basins experienced higher

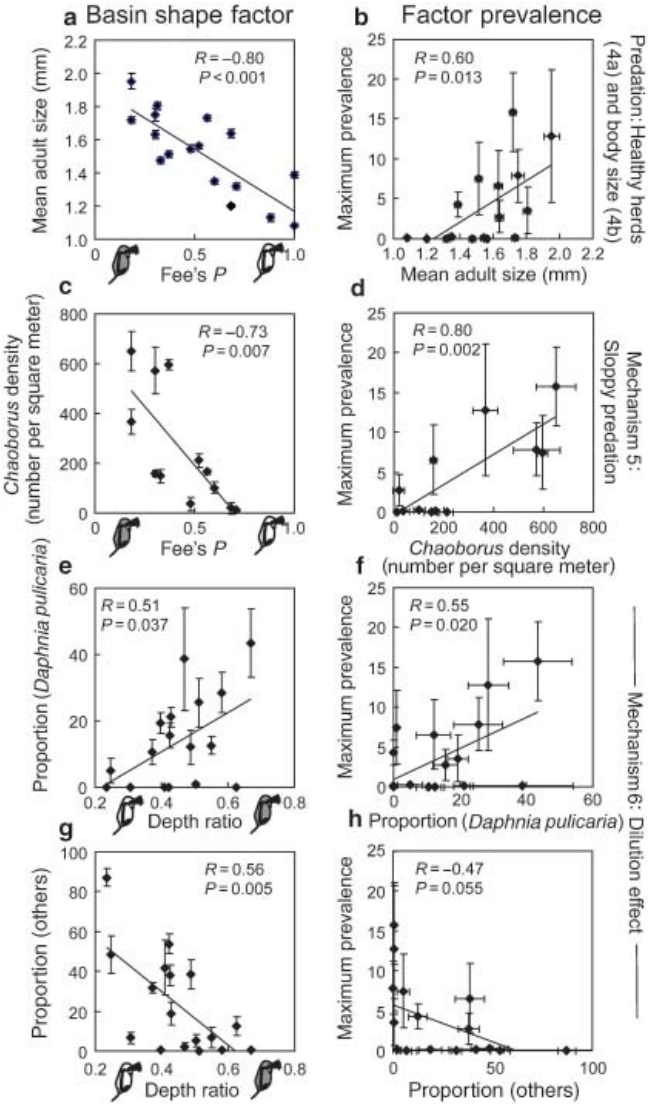

Figure 5. A correlated suite of factors implicating vertebrate and invertebrate predation in the basin shape–disease pattern. Mechanism 4: Fish predation. (a) Vertebrate predation often scales negatively with body size (in millimeters) of adult* Daphnia. *Thus, declining body size with Fee's* P *indicated higher vertebrate predation intensity in V-shaped basins. (b) Lakes with larger hosts have more disease. Mechanism 5: Sloppy predation. (c) The invertebrate predator* Chaoborus *(sampled July 2007) reaches higher density in U-shaped lakes (low Fee's* P*), and (d)* Chaoborus *density and disease prevalence correlate positively. Mechanism 6: Dilution effect. (e) Relative density of the larger-bodied* Daphnia pulicaria *increases in U-shaped basins (higher depth ratio), and (f) lakes with proportionately more* D. pulicaria *have more disease. (g) Conversely, V-shaped lakes (lower depth ratio) have higher proportion of other, smaller-bodied cladocerans (especially* Daphnia retrocurva, Ceriodaphnia, Diaphanosoma, Bosmina*), and (h) a higher proportion of other species correlates with less disease. Pearson correlation statistic,* R*, with associated* P*-values provided. Gray* Daphnia *correspond to lake shapes with more disease; white* Daphnia *indicate those with less.

invertebrate predation pressure, yet disease prevalence increased with this index of *Chaoborus* density (figure 5d). At first glance, this pattern throws a figurative curveball at the "healthy herds" predation mechanism: How can a predator enhance disease? One recently proposed idea centers on interactions between predation and host immunity: In theory, lower levels of selective predation can reduce recruitment of immune (recovered) hosts and fuel higher production of susceptible hosts, which can then become infected (Holt and Roy 2007). We eliminated this possibility (even though it's interesting) because diseased *Daphnia* do not recover (Ebert 2005) from infection. Instead, two other factors seem more likely. First, *Chaoborus* are gape-limited predators that preferentially eat smaller prey (Pastorok 1981). In response to higher invertebrate predation, *Daphnia* size structure can shift (through predation and indirect, life-history response) toward larger-sized animals (Spitze 1992, Riessen 1999) that can then be more readily infected (Hall et al. 2007a). Second, and perhaps more important, when preying on infected *D. dentifera* at night (Tjossem 1990, McQueen et al. 1999), *Chaoborus* releases a considerable proportion of spores consumed into the epilimnion, where they can remain suspended, and these released spores retain high infectivity (Cáceres et al. 2009). Therefore, rather than acting as a net sink, this predator may spread the parasite through sloppy feeding and regurgitation of spores in the epilimnion. This sloppy feeding behavior may help initiate epidemics during a time of year that is otherwise unfavorable to the parasite (i.e., when strong stratification would ordinarily cause infected hosts laden with spores to sink out of the water column upon death, before releasing spores; Cáceres et al. 2009). Thus, vertebrate and invertebrate predators responded differently along the basin-shape gradient, and these different predators shaped fungal epidemics through different mechanisms.

6. Dilution effect: Mixed at this large spatial scale

We found one apparently dissonant aspect of food-web structure in this basin shape–disease story. Large epidemics most commonly erupt in systems with a proportionally higher density of the congeneric species *D. pulicaria*. More specifically, as we noted above in the vertebrate predation discussion, larger-bodied *D. pulicaria* achieved higher relative abundance in U-shaped lakes (higher depth ratio, figure 5e), and other smaller cladocerans were relatively more common in V-shaped lakes (lower depth ratio, figure 5g; analogous correlations were not significant with average 15 August Fee's *P*). Furthermore, we found elevated

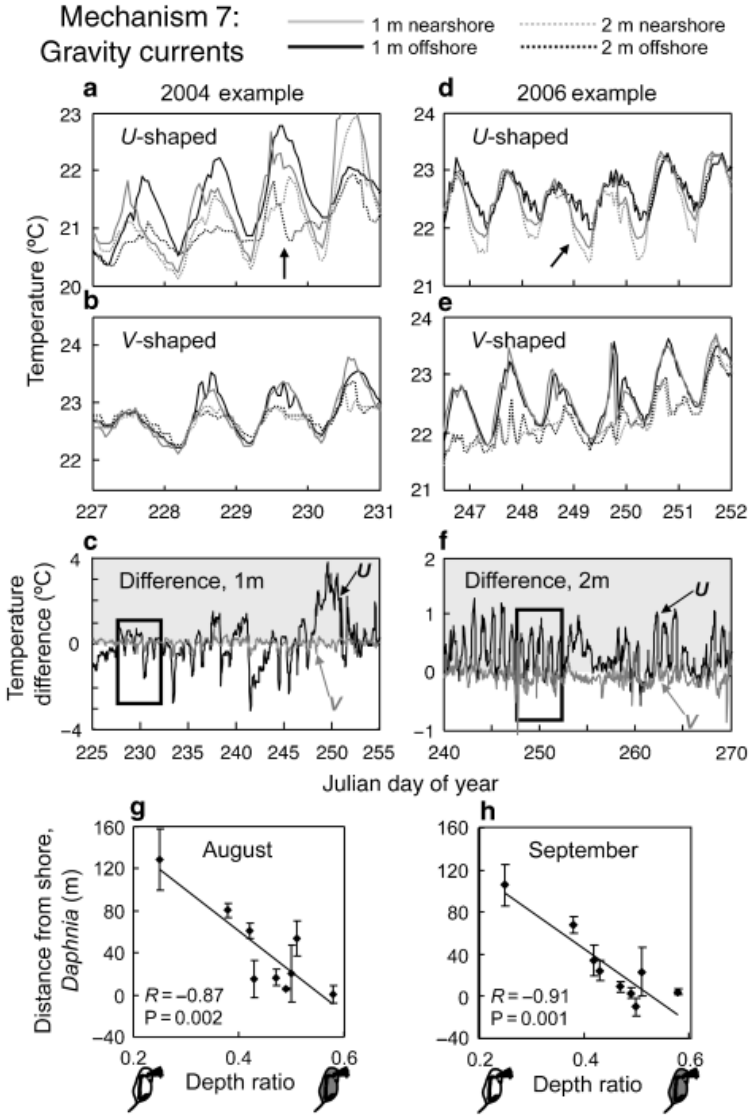

Figure 6. Mechanism 7: Gravity currents. Time series of temperature measurements from approximately 1 meter (m; solid) and 2 m (dashed) at a nearshore (gray) and offshore (black) location in U- and V-shaped lakes. (a) U-shaped Bassett Lake (depth ratio [DR]: 0.58, 15 August mean Fee's* P *[FP]: 0.16) and (b) V-shaped Pine Lake (DR: 0.43, FP: 0.51). (c) Differences in temperature (°C) between offshore and nearshore 1-m depth. Positive differences (gray region) indicate colder nearshore temperatures and the potential for subsurface gravity current flow offshore. Negative differences (white region) indicate warmer nearshore waters and the potential for offshore flow at the surface. Boxed area corresponds to examples shown in (a) and (b). (d) U-shaped Bristol Lake (DR: 0.49, FP: 0.25), (e) V-shaped Pleasant Lake (DR: 0.39, FP: 1.0), and (f) differences at 2-m depth (same interpretation as [c]). Host location: This lateral movement of water could carry spores offshore to hosts; host locations were estimated in (g) August 2005 and (h) September 2005 surveys. In U-shaped lakes (high depth ratio), we see hosts appearing in the water column closer to shore than in V-shaped lakes. Distance from shore values were estimated from nearshore-offshore transects.

parasite prevalence in lakes with high dominance of *D. pulicaria* and less disease in lakes with greater numbers of smaller-bodied cladocerans (figures 5f and 5h, respectively; the latter correlation was marginally significant). The result involving *D. pulicaria* seems surprising at first. After all, we expected that this unsuitable host should dilute disease (Keesing et al. 2006, Hall et al. 2009b). Furthermore, we previously found that higher relative and absolute abundance of *D. pulicaria* corresponds with smaller epidemics on two time scales: Between lake-years and within a season, higher relative and absolute density of *D. pulicaria* correlated with decreasing infection prevalence (Hall et al. 2009b). However, these findings do not create internal inconsistencies. Temporally, epidemics are indeed larger in the U-shaped lakes during years with lower abundance of *D. pulicaria*, a result signaling the expected dilution effect (Hall et al. 2009b). However, the summaries of community composition, here focused on variation in space, are too coarse to catch that temporal signal. Additionally, it is likely that other grazers in V-shaped lakes (e.g., *Ceriodaphnia*) also consume spores but are more resistant to infection. Therefore, a dilution effect may operate in this case, too. When integrating research on vertebrate predation (mechanism 4) and dilution-effect processes (mechanism 6), we can say that lower vertebrate predation most likely permits epidemics to start in U-shaped lakes at the across-basin scale—even though lower vertebrate predation also encourages higher abundance of *D. pulicaria*, an important diluter.

7. Gravity currents: Initial support for a future direction

Finally, we detected evidence pointing to a key physical mechanism (gravity currents) operating in U-shaped lakes but not V-shaped lakes. This mechanism could trigger the start of epidemics by transporting fungal spores from nearshore areas, where they do not come into contact with hosts, to offshore areas, where they can. We see conditions conducive to the formation of gravity currents in the data collected from deployment of nearshore-offshore temperature loggers at different depths. For example, in 2004 we found that temperatures inshore were approximately 0.5 degrees Celsius lower than at the same depth offshore in Bassett Lake (figure 6a) for several days. On day 229

(16 August), we observed evidence of a "lateral intrusion" of colder water (figure 6a arrow), first at the 2-meter (m) logger inshore (figure 6a, gray dotted line), then at the 2-m logger offshore (figure 6a, black dotted line); the temperature at this depth decreased while the temperature at the logger directly above it at the 1-m depth continued warming in response to solar heating. More generally, if we look at differences in temperature between offshore and nearshore areas as an indicator of conditions favorable for horizontal transport, we find such conditions occurring more often in U-shaped lakes (figure 6c). Colder water nearshore (positive values, gray box; figure 6c) is denser than warmer water at the same depth offshore. This sets up the potential for subsurface gravity current flow offshore, which could transport resting spores to hosts. In another example from 2006, we see pronounced drops in temperature nearshore, at both 1-m and 2-m depths, in U-shaped Bristol Lake (figure 6d), but not in the V-shaped Pleasant Lake (figure 6e). On day 249 (6 September), we found evidence of horizontal water flow in the U-shaped lake (figure 6d, arrow); the rate of cooling increased overnight in the nearshore water, suggesting a lateral movement of colder water along the nearshore slope. When plotting temperature differences for these lakes, we again see more opportunities for horizontal transport in the U-shaped basin (figure 6f).

Gravity currents are slow flows (< 1 centimeter per second), but they can facilitate nearshore-offshore exchange. This exchange might be particularly effective in environments sheltered from wind, such as our study lakes (James and Barko 1991, Sturman et al. 1999). Furthermore, basin shape (especially length of the vegetated nearshore zone and angle of the bottom slope) influences the formation and subsequent propagation of flows (Sturman et al. 1999, Wells and Sherman 2001): We find evidence for gravity currents in U-shaped lakes where the shorter littoral zones and steeper bottom slopes favor the formation of faster gravity currents with greater discharge from the nearshore littoral zone (Sturman et al. 1999). Furthermore, the vegetated nearshore area (littoral zone) extends much farther in the gradually sloping V-shaped lakes. As a result, spores must be transported longer distances in V-shaped lakes to reach offshore habitat of daphniid hosts (figure 6e). This horizontal distance between the shore and the appearance of hosts ranges from between 5 and 10 m for a small, U-shaped lake to more than 100 m for a large, V-shaped lake (figure 6g, 6h)—a very significant difference given that gravity currents are episodic, low-velocity flows. Although the gravity current–basin shape pattern is consistent with the disease–basin shape pattern, this idea needs further empirical and theoretical development. In particular, we intend to explore the effect of nearshore aquatic vegetation on these flows (since drag from emergent vegetation greatly slows currents; Oldham and Sturman 2001). The effects of submerged vegetation on gravity current flows have yet to be explored empirically or theoretically.

Synthesis: Why epidemics in U-shaped lakes?

Putting these various pieces together, we can compose a working verbal model to explain the start of epidemics in U-shaped basins rather than V-shaped ones (figure 7). Epidemics begin in late summer/early autumn in lakes with sufficient host density to support disease spread (Cáceres et al. 2006, Duffy and Hall 2008). Spores deposited in

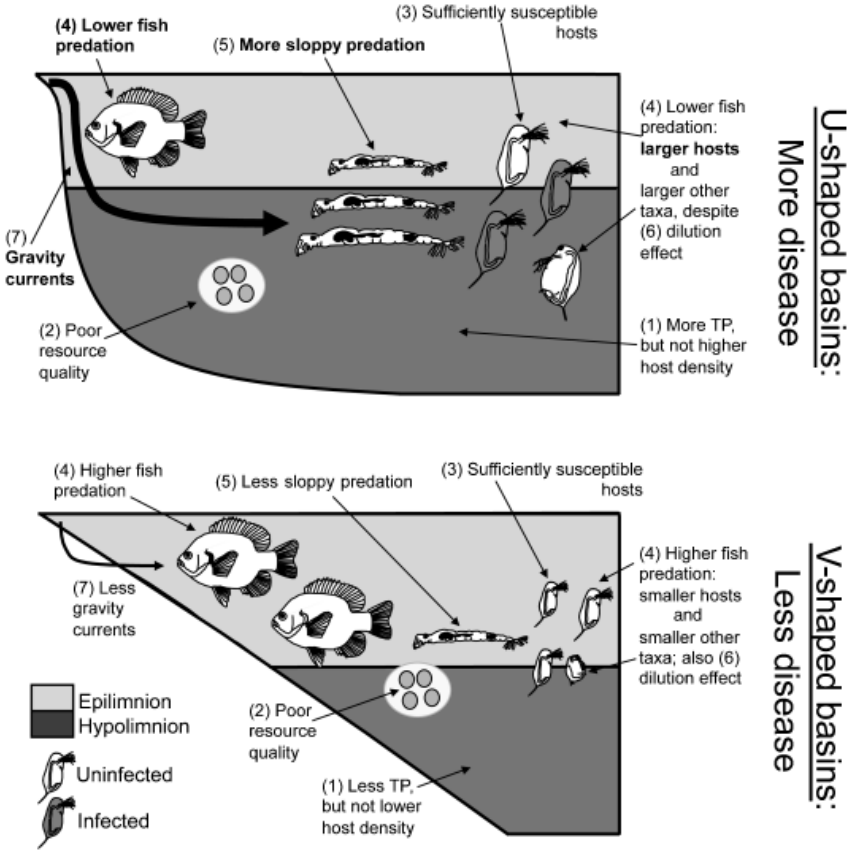

Figure 7. Summary of the key mechanisms most likely driving fungal epidemics in* Daphnia *host populations in lakes with U-shaped and V-shaped basins. Numbers refer to the mechanism numbers in text (and table 1). Key differences uncovered between the two basin types are bolded. This drawing imagines lakes at the initiation of epidemics (late summer) when the lakes are still stratified. During this time, a warmer, upper epilimnion sits on top of a colder, deeper hypolimnion. TP, total phosphorus.

nearshore areas as a result of spring turnover or perhaps by fish fecal pellets (i.e., those spores that remained viable but trapped in pellets before eventual release; Duffy 2009) are transported by gravity currents with the aid of convective turbulence for resuspension during periods of late summer cooling. Those spores are then consumed by at least moderately susceptible *Daphnia* hosts (no host assemblages were completely resistant) with large body size (i.e., sizes that increased probability of infection). In the U-shaped basins, these larger hosts occupy habitat close enough to shore to permit gravity currents laden with spores to reach them (as deduced from the within-basin spatial surveys). Then, fairly poor algal resource quality helps to ensure more efficient disease transmission (a condition that would apply to either U- or V-shaped lakes).

Assuming that resource quality and quantity are not too low (Hall et al. 2007b, 2009a, 2009c; not addressed here), and that vertebrate predation is not too high (Duffy et al. 2005, Duffy and Hall 2008, Cáceres et al. 2009), infected hosts produce spores. Predation on these infected hosts by *Chaoborus* in particular releases spores into the epilimnion where they can remain entrained in strongly stratified waters as epidemics start in late summer (Cáceres et al. 2009). Once epidemics start, their magnitude and duration are then determined by a suite of interacting factors (including rapid host evolution, thermal physiology, vertebrate predation, changes in resource quality, increases in "diluting hosts," etc.; Hall et al. 2005, 2006, 2009a, 2009b, Duffy and Sivars-Becker 2007, Duffy and Hall 2008, Duffy et al. 2009, Cáceres et al. 2009). However, the start of epidemics clearly involves a suite of food-web and physical characteristics more likely found in U-shaped basins (figure 7). At the very least, this suite of factors should influence disease in lakes with similar food-web architectures.

More generally, this case study provides a clear example of how habitat indirectly shapes epidemiology. Certainly habitat can directly influence disease by acting on densities of hosts or parasites themselves. In some disease systems, abiotic constraints imposed on hosts or parasites by habitat structure might critically inhibit the spread of disease. Nonetheless, here—as perhaps in many host-parasite systems—habitat acts most prominently on disease species by determining abundance of other species that then can inhibit or enhance epidemics through a variety of possible processes (dilution effect, healthy herds, the sloppy-predation mechanism). Many of these mechanisms involving other species that we considered are quite general and should apply broadly to other wildlife-disease systems. This finding tells us that assessment of disease risk could be greatly enhanced by taking a more community ecology–oriented approach. However, in our system, we cannot yet quantitatively assess the relative importance of physical habitat connectivity through parasite-dispersal mechanisms (gravity currents) versus the food-web effects described. In fact, we cannot yet say which of the food-web factors is most important for triggering or inhibiting epidemics, although we hope to do so in the future (perhaps aided by some targeted comparisons of structural-equation models fit to expanded data sets similar to that shown here). This study offers a first step toward that goal. Still, the basin shape–disease story provides an example for future, broader applications for work in the community ecology of disease, and should provoke further exploration of biophysical coupling as a driver of epidemics in lakes and other systems. With improved understanding of links between habitat structure, species interactions, physical processes, and parasitism, perhaps disease ecologists can better anticipate outbreaks in wildlife populations.

Acknowledgments

We are grateful for the help of Robert Duffy, Allyson Morgan, Darice Shumway, Pam Woodruff, and Avery Parsons-Field with field collection and sample processing. The Albert, Brehm, Burdick, Champion, and Gregg families graciously permitted access to private lakes. The National Science Foundation (NSF) generously supported this research (OCE 02-35119, 02-35039, 02-35238; DEB 06-13510, 06-14316, 06-40953, and a Research Experience for Undergraduates supplement). The findings and conclusions expressed herein are those of the authors and do not necessarily reflect the views of the NSF. This is contribution #1555 from the W. K. Kellogg Biological Station.

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