Infectious diseases and habitat alteration are changing ecosystems worldwide (Daszak et al. 2000; Foley et al. 2005). Furthermore, these two factors may interact: habitat alteration may catalyze further spread of epidemics (Patz et al. 2004; Ostfeld et al. 2005). But how does habitat structure drive disease mechanistically? That is, through which direct and indirect pathways does habitat structure influence epidemics? Direct effects of habitat on disease arise through several mechanisms: the size, shape, and connectedness of habitat patches can determine host densities and dispersal rates, contact rates between hosts and free-living parasite stages, and disease transmission at habitat edges (Patz et al. 2004; Ostfeld et al. 2005). Habitat structure may also indirectly alter density of other species that catalyze or inhibit disease spread (Hall et al. 2010). For example, habitat might increase or suppress density of “diluting host” species that remove free-living parasites without becoming infected (Keesing et al. 2006). Furthermore, habitat structure may favor or disfavor predators that selectively cull infected hosts (Duffy et al. 2005). Given the range of possibilities, the challenge becomes delineating mechanistic connections between habitat and drivers of disease spread.

Freshwater ecosystems offer ideal environments in which to connect habitat to disease. Major drivers of habitat structure vary among lakes, including basin size and shape, light penetration, thermal stratification, and dissolved oxygen concentration. In a given lake, some of these factors (e.g., light attenuation, stratification, and hypoxic zones) vary within seasons (Tessier and Welser 1991; Johnson et al. 2009) and among years (De Stasio et al. 1996; Fee et al. 1996). Human activities, such as those that cause eutrophication, can also alter habitat structure, e.g., by decreasing light penetration and increasing the extent of hypoxia (Marcogliese 2001; Mazumder et al. 1990). This variation in habitat can determine densities and distributions of many aquatic organisms (Thralkeld 1979; Kitchen and Kitchell 1980), including parasites (Marcogliese 2001; Johnson et al. 2009; Hall et al. 2010).

Here, we illustrate how habitat can connect to disease via multiple pathways in thermally stratified lakes in the midwestern U.S.A. In these lakes, a yeast parasite (Metschnikowia bicuspidata) infects its host Daphnia dentifera, a dominant zooplankton grazer (Tessier and Welser 1991). Yeast epidemics start in late summer and extend until early winter (Cáceres et al. 2006; Hall et al. 2011; Overholt et al. 2012). During epidemics, the yeast kills its infected host, thereafter releasing infectious propagules (spores) into the environment to infect new hosts (Ebert 2005). All mechanisms described here ultimately involve this life stage of the parasite. Spore density, not host density, is a sensitive driver of epidemic size in this system (Cáceres et al. 2006; Hall et al. 2009a, 2010). Using correlative evidence from an extensive field survey conducted in 2009, we focused on indirect relationships between habitat and disease involving two nonhost species (Pathways 1 and 2; Fig. 1). We also argue for a potential direct connection from habitat to disease involving a driver of stratification (Pathway 2; Fig. 1).
The two indirect pathways involve nonhost species that we mechanistically connected to yeast epidemics in previous work (Cáceres et al. 2009; Hall et al. 2009a, 2010). Here, we link these species to large-scale habitat structure. Both indirect pathways begin with lake size (indexed as surface area) as an ultimate driver of habitat structure (Fig. 1). In Pathway 1, bigger lakes were deeper, and greater depth permitted a larger refuge from vertebrate (fish) predation for large-bodied zooplankton (as defined below; Threlkeld 1979; Tessier and Welser 1991). This refuge provided essential habitat for *Daphnia pulicaria*, a zooplankton grazer that consumes yeast spores and removes them from the environment but does not become infected (i.e., it functions as a completely resistant “diluter” in disease ecology; Keesing et al. 2006; Hall et al. 2009a). Higher density of this diluter species, in turn, delayed the start date of epidemics. This delay mattered because epidemics that started earlier grew larger (Hall et al. 2011; Overholt et al. 2012), likely due to thermal mechanisms such as increases in host birth rate, parasite transmission rate, and parasite production with water temperature (Hall et al. 2006). Pathway 2 also begins with lake size, but then moves along a different, uncorrelated path involving stratification and a predator. Solar radiation (indexed by extinction of photosynthetically active radiation [PAR]) penetrated less deeply into smaller lakes. Because more heat was absorbed in shallower waters, lakes with higher light extinction became more strongly stratified (Kling 1988). These more strongly stratified lakes, in turn, supported higher density of a “sloppy predator,” *Chaoborus punctipennis*, which correlates positively with epidemic size. Direct pathways may also connect solar radiation to epidemic metrics (Fig. 5). Positive (+) and negative (−) symbols denote sign of the relationships involved.

Methods

**Study system**—We studied two *Daphnia* species that are common planktonic grazers in small, thermally stratified lakes in temperate North America (Tessier and Welser 1991). *D. dentifera* and *D. pulicaria* encounter and ingest spores of the yeast parasite *Metschnikowia bicuspidata* (hereafter: yeast) while nonselectively foraging on small algae (Ebert 2005; Hall et al. 2009b). The parasite penetrates the gut wall of its focal host (*D. dentifera*) and multiplies in its hemolymph (Ebert 2005). As it uses host resources to fuel its own reproduction, this parasite reduces host growth, fecundity, and survivorship (Hall et al. 2009b). Parasite spores, once released from the carcasses of dead hosts, can then infect new hosts (Ebert 2005). Yeast epidemics occur in lake populations of *D. dentifera* in the upper midwestern U.S.A. (Hall et al. 2011). However, in these lakes, the diluter species (*D. pulicaria*) resists infection by this parasite (Hall et al. 2009a).

**Lake survey**—We sampled 18 lakes in southern Indiana (Greene and Sullivan Counties) weekly from August until the first week of December 2009. On every sampling visit, we collected two replicate plankton samples that each contained three pooled tows of a Wisconsin net (13 cm diameter, 153 μm mesh, towed bottom to surface). From one of the plankton samples, we diagnosed infection status of at least 400 live *D. dentifera* under a dissecting scope at

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**Table 1.** Pathways connecting habitat to epidemic metrics. Lake size is the ultimate habitat driver of disease. However, it acts through two physical drivers that influence proximate habitat factors (Figs. 2, 3) that relate to key community players that shape epidemics (Fig. 4). Pathway 1: Larger, deeper lakes have bigger refuges from vertebrate predation that bolster density of a species that can dilute disease, *Daphnia pulicaria*. Higher density of the “diluter” delays the start date of epidemics, and a later start date can constrain the size of epidemics through thermal physiology. Pathway 2: Light penetrates less deeply in smaller lakes, intensifying stratification. More strongly stratified lakes support higher density of a “sloppy predator,” *Chaoborus punctipennis*, which correlates positively with epidemic size. Direct pathways may also connect solar radiation to epidemic metrics (Fig. 5). Positive (+) and negative (−) symbols denote sign of the relationships involved.

<table>
<thead>
<tr>
<th>Ultimate driver</th>
<th>Physical driver</th>
<th>Proximate habitat</th>
<th>Community player</th>
<th>Epidemic metric</th>
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<tbody>
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<td>Lake size</td>
<td>Maximum depth</td>
<td>Refuge size</td>
<td>Diluter</td>
<td>Epidemic start date</td>
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<td></td>
<td>Solar radiation</td>
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<td>Pathway 1</td>
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<td>Fewer nutrients</td>
<td>Light penetration</td>
<td>Stratification strength</td>
<td>Sloppy predator</td>
<td>Pathway 2</td>
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20–50× magnification, following Ebert (2005). Body length of a subset of uninfected adult Daphnia dentifera hosts was also measured as an index of fish predation: smaller mean length indicates stronger predation pressure (Kitchell and Kitchell 1980). The other sample was preserved in 60–75% ethanol and counted under a dissecting scope to estimate areal densities of D. dentifera (the focal host), D. pulicaria (the diluter), and Chaoborus punctipennis (the sloppy invertebrate predator). We only present data on Chaoborus large enough to eat Daphnia hosts (instars 3+) (Moore 1988).

We calculated two metrics of yeast outbreaks, start date and epidemic size. We defined start date of epidemics as the sampling date on which infection prevalence first exceeded 1%. Based on this definition, 15 of the 18 lakes experienced outbreaks, but two of them started before we began sampling. For those two lakes, we assigned the day of first sampling as the start date. We estimated the size of epidemics by integrating infection prevalence (proportion infected) through time using the trapezoid rule. This metric (integrated prevalence, with units of proportion × days) correlated strongly with maximal prevalence of infection (Pearson correlation, \( r = 0.93, p < 0.0001 \)).

To investigate links between lake morphometry and habitat structure, we obtained data on lake surface area and maximum depth from the Indiana Clean Lakes Program (W. W. Jones unpubl.). Fetch was measured as the greatest uninterrupted distance across a lake in the direction of the average prevailing winds. Several key habitat indices stemmed from temperature- and oxygen-based calculations. On each sampling visit, we measured vertical profiles of temperature and dissolved oxygen at 1 m intervals using a Hydrolab multiprobe (Hach Environmental). We vertically interpolated the temperature data to a 0.1 m depth interval (using a piecewise cubic hermite interpolating polynomial, “pchip”; Matlab version 7.8 R2009a, MathWorks). Then we identified the bottom of the epilimnion (\( Z_E \)) as the depth at which temperature decreased by > 1°C m⁻¹. Refuge size (Pathway 1) was calculated as the distance between \( Z_E \) and a deeper, low-oxygen (1 mg L⁻¹) threshold (\( Z_{LO} \)) also found with splines (Tessier and Welser 1991). Additionally, we calculated buoyancy frequency at the thermocline—an index of the strength of stratification (Pathway 2)—based on a density criterion. To calculate it, we converted water temperature at each depth \( j \) into a density, \( \rho_j \) (following Chen and Millero 1977). Buoyancy frequency (\( N_j \)) was then calculated as

\[
N_j = \sqrt{\frac{g}{\rho} \left( \frac{dp_j}{dz} \right)}
\]

(1)

where \( \rho \) is the mean density of the water column, \( dp_j/\text{dz} \) is the vertical density gradient at depth \( j \), and \( g \) is gravitational acceleration (9.81 m s⁻²). The thermocline occurs at the depth of maximum strength of stratification (\( N_{max} \), cycles per hour [cph]). We used August \( N_{max} \) at the start of epidemic season, as our stratification index. All lakes were strongly stratified (\( N_{max} > 48 \) cph) during this period (MacIntyre and Melack 1995).

We estimated penetration of PAR using irradiance data collected at 1 m intervals (0–4 m, duplicate profiles) with a LI-250A light meter (LI-COR). Then, we regressed natural log-transformed irradiance \( I(z) \) against depth (\( z \)):

\[
\ln(I(z)) = a - kz + \epsilon
\]

(2)

with intercept \( a \) and residual errors \( \epsilon \). The slope is the coefficient of light extinction, \( k \) (μmol quanta cm⁻² s⁻¹ m⁻¹). Values of \(-k\) that are closer to zero indicate deeper light penetration while more negative values of \(-k\) indicate shallower light penetration.

We also measured epilimnetic concentrations of total phosphorus (TP) and chlorophyll \( a \). TP samples were analyzed on a UV-1700 spectrophotometer (Shimadzu Scientific Instruments) using the ascorbic acid method following persulfate digestion (APHA 1995). We measured chlorophyll \( a \) using narrow-band filters on a Trilogy fluorometer (Turner Designs) following chilled ethanol extraction (Welschmeyer 1994).

Statistical analysis—Statistical analyses were performed in R (R Development Core Team 2012) and Matlab. Linear and nonlinear relationships were assessed using correlations and nonlinear regressions, respectively. To assess the linear relationship between refuge size and epidemic start date including an outlier point, we used the least absolute residual (LAR) method, which is robust to outliers (Neter et al. 1996). In all other cases, we estimated parameters by minimizing sums of squares. We log-transformed surface area, zooplankton density, and chlorophyll \( a \) data to meet assumptions of normality and homoscedasticity. For variables that did not meet assumptions of normality after transformation (according to the Shapiro–Wilk test), we computed significance of correlations using permutation tests (9999 randomizations; Bishara and Hittner 2012). We also used permutation tests to compute significance of the nonlinear (exponential: \( Y = a \times \exp[bX] + e \) and LAR regressions. Confidence intervals around parameters were estimated using 10,000 bootstraps.

Results

We first established links between the ultimate habitat driver (lake size), two proximate habitat features (refuge size and stratification strength), and two epidemic metrics (start date and size) along the two pathways (Fig. 1). Despite sharing an ultimate driver (lake size), the proximate habitat features in each pathway were uncorrelated (\( r = -0.07, p = 0.79 \)). Several correlations significantly supported the mechanisms of Pathway 1. Larger lakes, i.e., those with greater surface area, had greater maximum depth (\( r = 0.59, p = 0.003 \); Fig. 2A). This physical driver, lake depth, created room for larger habitat refuges from fish predation in August (\( r = 0.91, p < 0.0001 \); Fig. 2B). Lakes with larger refuges, in turn, had later epidemic start dates (LAR regression: \( p = 0.046 \); correlation when excluding the outlier denoted with an arrow: \( r = 0.67, p = 0.009 \); Fig. 2C). Thus, epidemics started later in bigger lakes with larger refuges. Pathway 2 was also supported by several significant correlations. Larger lakes had deeper light penetration in August (\( r = 0.51, p = 0.018 \); Fig. 2D).
Deeper light penetration then correlated with weaker strength of stratification in August ($r = -0.55, p = 0.013$; Fig. 2E). More weakly stratified lakes had smaller epidemics, as quantified by integrated prevalence ($r = 0.63, p = 0.004$; Fig. 2F). Thus, lake size drove variation in light penetration, which was a physical driver of stratification strength. Stratification strength, in turn, correlated positively with the size of yeast outbreaks.

Lake size connected to the two forms of proximate habitat structure through physical drivers (Fig. 1). In Pathway 1, lake size correlated with thickness of the predation refuge due to differential response of epilimnetic depth ($Z_E$, top of refuge) and the 1 mg L$^{-1}$ dissolved oxygen threshold ($Z_O$, bottom of refuge). The epilimnion was deeper in lakes with larger surface area, $SA$ ($r = 0.55, p = 0.017$; Fig. 3A), longer fetch, $F$ ($r = 0.74, p = 0.0005$; $\ln[F]$ and $Z_E$: $r = 0.66, p = 0.003$), and deeper light penetration ($r = 0.59, p = 0.002$). However, depth to the hypoxic zone ($Z_O$) increased with maximum depth, $Z_{max}$ ($r = 0.88, p < 0.0001$; Fig. 3B), more steeply than did $Z_E$ ($r = 0.64, p = 0.003$; Fig. 3B). Since refuge size is $Z_O - Z_E$, larger lakes had bigger refuges (Figs. 2B, 3B). By contrast, lake size did not correlate with August stratification strength, $N_{max}$ ($r = 0.12, p = 0.32$; $N_{max}$ and $\ln[SA]$: $r = 0.04, p = 0.89$). In Pathway 2, the positive correlation between lake size and light penetration (Fig. 2D) related to nutrient loading and phytoplankton biomass. Smaller lakes had greater TP concentrations ($\ln[SA]$: $r = -0.50, p = 0.035$; $R^2 = 0.55, p = 0.006$; Fig. 4C). Thus, epidemics were smaller overall in lakes with more diluters at the beginning of...
epidemic season \((r = -0.54, p = 0.037; \text{Fig. 4D})\). However, density of diluters was not significantly correlated with strength of stratification in August, the other proximate habitat feature \((r = -0.36, p = 0.08)\). Instead, in Pathway 2, stratification strength correlated positively with density of the sloppy predator, *Chaoborus* \((r = 0.58, p = 0.012; \text{Fig. 4E})\). Lakes with more sloppy predators, in turn, had larger epidemics \((r = 0.68, p = 0.002; \text{Fig. 4F})\). However, this predator did not increase with refuge size \((r = -0.17, p = 0.51)\) and only weakly correlated with start date of epidemics \((r = -0.51, p = 0.055)\). Densities of the diluter and sloppy predator were also uncorrelated \((r = -0.08, p = 0.39)\). Thus, based on insights from these relationships, the two pathways involved different players: the refuge pathway (Pathway 1) involved the diluter, whereas the stratification pathway (Pathway 2) involved the sloppy predator.

The field data also suggest a direct effect of a physical driver of habitat structure, light, on epidemics in Pathway 2 (Fig. 1). Light can damage parasite spores. In lakes with deeper penetration of light, epidemics started later \((r = -0.55, p = 0.010; \text{Fig. 5A})\) and were smaller \((r = -0.64, p = 0.012; \text{Fig. 5B})\).

**Discussion**

The fusion of limnology with community ecology of disease can powerfully link habitat structure to epidemics. Here, variation in the start date and size of epidemics correlated with two features of proximate habitat structure (Fig. 1). The pathways connecting habitat to disease originated from physical factors related to lake size. Ultimately, both pathways potentially influenced disease by governing the fate of yeast spores, not host density. In the first pathway, epidemics started earlier in lakes with smaller hypolimnetic refuges and lower density of a diluter (*Daphnia pulicaria*; Hall et al. 2009a). In the second pathway, epidemics became larger in lakes with stronger thermal stratification and higher density of a sloppy predator (*Chaoborus punctipennis*) that can spread disease (Cáceres et al. 2009). Below, we summarize the limnological links between lake size and the proximate habitat factors. Then, we describe each pathway in more detail. Finally, we describe how a complementary mechanism, related to light penetration (Pathway 2), may also directly affect disease.

Connections between habitat and disease involve some well-studied limnological phenomena. Both refuge size and stratification strength stem from physical drivers correlated with lake size, specifically surface area. In Pathway 1, bigger lakes had longer fetches and deeper epilimnia, as seen in other studies (Gorham and Boyce 1989; Fee et al. 1996). All else equal, greater epilimnetic depth could compress hypolimnetic refuges. However, depth to the zone of hypoxia increased more steeply with lake size than did epilimnion depth. As a result, bigger lakes had larger refuges, despite their deeper epilimnia. In Pathway 2,
smaller lakes had shallower light penetration, which was a likely physical driver of stratification strength (Mazumder et al. 1990; Fee et al. 1996). The light gradient among lakes reflected variation in nutrients and primary producers. Smaller lakes had higher TP, therefore higher algal biomass. Higher algal biomass, in turn, absorbed more solar radiation in shallower waters. This effect yielded sharper density gradients between warmer, shallower and colder, deeper layers (Kling 1988). Thus, through depth and light drivers, lake size ultimately set up the two habitat–disease pathways.

Fig. 4. (A–D) Pathway 1: An indirect mechanism for the refuge size–start date relationship. (A) Lakes with larger refuges had higher density of the diluter species, *Daphnia pulicaria*. (B) Higher density of this diluter correlated with delayed start of epidemics. (C) Delayed start matters because epidemics that started earlier grew larger. (D) Density of the diluter at the start of epidemics correlated less strongly with the overall size of epidemics. (E, F) Pathway 2: An indirect mechanism for the stratification–epidemic size relationship. (E) More strongly stratified lakes had higher densities of the sloppy predator, *Chaoborus*, and (F) epidemics grew larger with greater density of this sloppy predator.
Before proceeding, we note that density of the focal host (*D. dentifera*) had little role in these two habitat pathways. Standard epidemiological models predict increasing disease prevalence (i.e., larger epidemics) with increasing host density (Anderson and May 1986). However, August host density did not correlate with refuge size. Density did correlate with stratification strength, but not in a way that could explain patterns here: host density was greater in weakly stratified lakes, where epidemics were smaller. Furthermore, August host density was not correlated with start date of epidemics or overall epidemic size (see also: Cáceres et al. 2006; Hall et al. 2010). Thus, we focused on other mechanisms that indirectly or directly influenced the fate of yeast spores.

In the first pathway, refuge size correlated with start date and density of a diluter species. Epidemics started later in lakes with larger refuges from fish predation. Considered alone, this pattern seems surprising. Since fish selectively cull infected hosts (Duffy et al. 2005; Johnson et al. 2006), larger refuges might have protected infected hosts and therefore bolstered epidemics. However, fish predation did not correlate with either epidemic metric. Instead, larger refuges supported higher density of a diluter, *D. pulicaria*. This large-bodied species depends on the refuge to persist with fish predators (Threlkeld 1979; Tessier and Welser 1991). Higher density of this species likely inhibited the start of epidemics via consumption of spores (Hall et al. 2009a). Since *D. pulicaria* does not become infected, it acts as a dead end for the parasite, thereby potentially reducing disease in the more competent host (*D. dentifera*) through a dilution effect (Keesing et al. 2006).

This dilution effect may have delayed the start of outbreaks, but diluter density did not correlate as strongly with the eventual size of epidemics. That is, diluter density more weakly related to epidemic size (Fig. 4D) than to start date of epidemics (Fig. 4B). This pattern makes sense based on temporal patterns of diluter density. Because its density diminished through autumn, the diluter should have mitigated epidemic size less effectively. Still, links between habitat, the diluter, and start date of epidemics (Pathway 1) mattered for the ultimate size of epidemics, likely through a thermal mechanism. Outbreaks that started earlier began in warmer waters, and higher temperatures enhance transmission rate and other factors involved in disease spread (Hall et al. 2006). Conversely, epidemics that started later began in colder waters, and colder temperatures inhibit disease spread. Thus, due to thermal physiology and declining water temperatures in autumn, any mechanism (like dilution) that inhibits the start of epidemics can indirectly constrain their size (Hall et al. 2011).

Once epidemics began, a different proximate habitat factor correlated with epidemic size via another community player (Pathway 2). Epidemics grew larger in lakes that started the outbreak season more strongly stratified. More strongly stratified lakes also had higher densities of a sloppy predator (*Chaoborus*) known to spread disease (Cáceres et al. 2009; Duffy et al. 2011). The spreading mechanism here is important for the link to habitat: *Chaoborus* can disperse yeast spores into the epilimnion where both the host (Threlkeld 1979) and *Chaoborus* (von Ende 1979) migrate at night. These spores can remain suspended and contact new hosts; otherwise, hosts dying from infection likely would sink to the lake bottom before spores escaped (Cáceres et al. 2009; Johnson et al. 2009; Kirillin et al. 2012). But why did lakes with stronger stratification have greater density of *Chaoborus*? We cannot determine causation from our data. Perhaps shallower penetration of solar radiation (i.e., the driver of stronger stratification) protected *Chaoborus* from visual predators and UV damage (von Ende 1979; Persaud and Yan 2003). Future studies will hopefully address this stratification–*Chaoborus* relationship.

In Pathway 2, light penetration may have also affected the fate of yeast spores through a direct route. As argued above, light penetration can influence habitat structure by shaping the distribution of heat in the water column. Additionally, solar radiation (both UV and PAR) can directly exert deleterious effects on yeast spores (shown experimentally in the lab and field: Overholt et al. 2012). The sensitivity of yeast spores to radiation may at least partly explain why deeper light penetration correlated with later start and smaller size of epidemics.

Habitat–disease patterns arise commonly in aquatic systems, and combinations of indirect and direct mechanisms

![Fig. 5. Potential direct connections between light environment and epidemic metrics. Deeper light penetration (values closer to zero) correlated with (A) later start date of (B) smaller epidemics.](image-url)
may operate in these other examples as well. For instance, *Daphnia* that use deeper pond habitat to avoid predators have greater risk of exposure to spores of a bacterial parasite in sediments (Decaestecker et al. 2002). Similarly, whitefish (*Coregonus lavaretus*) ecotypes that use habitats of different depth host different classes of flatworm parasites (Karvonen et al. 2013). Furthermore, thermal stratification can influence chytrid parasitism in diatoms (Gsell et al. 2013). Habitat structure can also drive variation in host–parasite coevolution, e.g., between snails and their trematode parasites along depth gradients in lakes (King et al. 2009). Even in these examples, spatial distribution of hosts (and thus, infection risk) may ultimately reflect relationships between habitat and other species that drive disease. We hope future studies will continue to unravel interactions between habitat, community context, and disease in an array of aquatic systems.

Our field study connects habitat to disease via indirect community players and through potential direct effects on the parasite. In general, it remains vital to uncover these kinds of mechanisms as humans alter habitats worldwide (Patz et al. 2004; Foley et al. 2005). The intersection of limnology and community ecology of disease can illustrate general principles and also create a predictive framework for lakes themselves. In lakes, climate change and eutrophication alter habitat structure, potentially affecting host–parasite interactions involving diverse taxa (Marcogliese 2001; Ibelings et al. 2011). For example, climate change may alter the timing and strength of thermal stratification, as well as epilimnion depth (De Stasio et al. 1996; Fee et al. 1996). Furthermore, anthropogenic eutrophication can affect stratification and the size of hypolimnetic refuges, through mechanisms involving light penetration, epilimnetic depth, and extent of hypoxic zones (Mazumder et al. 1990; Marcogliese 2001). These and other modifications to aquatic habitats will likely alter disease prevalence through direct and indirect mechanisms. To understand and predict those changes, we must continue to uncover mechanistic links between habitat, ecology, and disease.

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