Ecology, 90(10), 2009, pp. 2850–2858

Predator–spreaders: Predation can enhance parasite success in a planktonic host–parasite system

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Abstract. The "healthy herds" hypothesis suggests that selective predators, by acting as parasite sinks, may inhibit the start of epidemics and reduce prevalence of infection. Here, we describe a counter-example using field patterns, experiments, and a model. The predator *Chaoborus* releases infective spores of a fungal parasite and, in doing so, may facilitate epidemics in *Daphnia* populations. In the field, epidemics occur in lakes with higher *Chaoborus* densities. Experiments revealed that nonselective *Chaoborus* release many of the spores contained in their prey. Since these released spores remain infective, this predator can catalyze epidemics when a lake's physical environment might otherwise impede them. Without *Chaoborus*, *Daphnia* dying of infection may sink to the lake bottom before releasing spores. A model tracking hosts and spores in the water column (where hosts contact spores) and in bottom sediments (where they cannot) illustrates this mechanism. Thus, by dispersing spores while feeding, this predator spreads disease. Many invertebrates are parasitized by obligately killing parasites, offering a variety of systems for additional tests of this "predator–spreader" hypothesis. In the meantime, this planktonic disease system prompts a very important, general warning: before we use predators to keep the herds healthy, we need to carefully think about the interface between predator feeding biology and the underlying epidemiology of wildlife disease.

Key words: Chaoborus *spp.;* Daphnia dentifera; *epidemic; fungal spores; host–parasite interactions; lake epilimnion;* Metschnikowia bicuspidata; *midges; planktonic disease system; predators; transmission rate*.

Introduction

What controls the distribution and abundance of disease? The answer to this question demands a broad perspective that places host–parasite interactions within a food web framework. In this context, it has become clear that a host's interaction with competitors, resources, and predators can drastically alter disease dynamics and profoundly affect the ability of parasites to invade and persist with their host (Ostfeld and Keesing 2000*a*, *b*, Johnson and Chase 2004, Hatcher et al. 2006, Keesing et al. 2006, Hall et al. 2007*a*, 2009*a*). Predators in particular may play a central role in controlling disease (Hudson et al. 1992, Packer et al. 2003, Ostfeld and Holt 2004, Duffy et al. 2005, Hall et al. 2005). In the "healthy herds" hypothesis (Packer et al. 2003), selective predation on infected hosts inhibits epidemics in part because predators act as sinks for the parasite that would otherwise spread environmentally or through host–host contact (e.g., bluegill sunfish predation on infected *Daphnia*; Duffy et al. 2005, Hall et al. 2006, Johnson et al. 2006). Despite understandable optimism for this idea, it has become increasingly clear that predators do not always "keep the herds healthy." For instance, models that incorporate immune function of hosts (through a recovered class) suggest that predation (Holt and Roy 2007, Roy and Holt 2008) and harvesting (Choisy and Rohani 2006) can actually enhance disease. In both of these cases, predators and harvesting act as sinks for parasites, but this sink effect is overwhelmed by an indirect one: both harvesting and removal of recovered (immune) hosts elevate production of newborn susceptible hosts that can then become infected. Thus, interactions involving immunity can undermine or even reverse the healthy herds effect.

Here, we present a different mechanism that also challenges the healthy herds hypothesis: some predators may actually spread (disperse) free-living stages of a parasite. To be clear, we do not refer here to trophically transmitted parasites, i.e., those that have evolved to require predation to complete their complex life cycles (Lafferty 1999, Thomas et al. 2005). Instead, we consider a rather different phenomenon. Predators could spread parasites while consuming infected hosts either through sloppy eating (e.g., shredding infected prey, regurgitating partially consumed prey) or by defecating spores that can survive digestion (Duffy 2009). As we argue here, such dispersion by predators may be particularly crucial for parasites such as environmentally transmitted obligate killers (Ebert and Weisser 1997). Obligate killers require

Manuscript received 20 November 2008; accepted 21 January 2009. Corresponding Editor: D. K. Skelly.
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both the death of their host for release of infective propagules and a hospitable environment for successful disease transmission. For this class of parasites, new infections require contact between infective propagules released from dead hosts and new susceptible hosts. Such a life cycle puts transmission of these types of parasites at the mercy of their environment, but perhaps dispersal of infective stages by predators might help these parasites overcome environmental obstacles and facilitate disease invasion.

We illustrate this "predator–spreader" argument by combining evidence from a field survey, laboratory experiments, and a minimal model built around the community ecology and epidemiology of a planktonic host–parasite interaction. *Daphnia dentifera* is an abundant grazer of algae and prey for both vertebrate and invertebrate predators in pelagic (open water) areas of thermally stratified, freshwater lakes. It also hosts numerous, fatal parasites (Green 1974, Ebert 2005), including the fungus *Metschnikowia bicuspidata*. However, obligate killers such as *Metschnikowia* face a dilemma during late summer when epidemics begin (Cáceres et al. 2006, Duffy et al. 2009): although host death is required for release of infective spores, dead hosts housing those spores quickly sink from the epilimnion (an upper, mixed layer) to the lake bottom. Unfortunately for the parasite, spores released on the lake bottom must then be mixed up into the water column to contact susceptible hosts. The likelihood of sufficient mixing seems extremely low during late summer (a period of intense stratification). Hence, this unfavorable physical environment poses a major challenge to the parasite. We argue here using three lines of evidence that the midge *Chaoborus*, an abundant invertebrate predator, may play a key role in short-cutting this environmental trap for the fungus. Our results suggest that predators such as *Chaoborus* can enable invasion and persistence of parasites in otherwise challenging environments, in effect reversing the "healthy herds" phenomenon.

Empirical Methods and Results

Field patterns

To establish the relationship between predators and epidemics in the field, we quantified *Chaoborus* (*punctipennis* and *flavicans*) densities in 12 lakes in Barry and Kalamazoo counties, Michigan, USA, on 11–12 July 2007. This period preceded the start of epidemics (i.e., when the parasite would "invade"). Sampling included six lakes with and six lakes without epidemics (defined by >2% maximum infection prevalence in 2002–2007; Cáceres et al. 2006). Three bottom-to-surface vertical tows were collected at night from offshore areas of each lake with a 50 cm diameter, 500-μm mesh net and preserved individually in >70% ethanol. PROC TTEST (SAS version 9.1; SAS Institute, Cary, North Carolina, USA) was used to test whether *Metschnikowia* epidemics occurred in lakes with higher *Chaoborus* densities.

Laboratory experiments

We used laboratory experiments to examine three critical components of the infected host–predator interaction. First, to determine whether *Chaoborus* selectively preys on infected *Daphnia dentifera*, we placed individual, starved *Chaoborus punctipennis* (9.38 ± 0.13 mm [mean ± SE]) in 500 mL of filtered lake water at 20°C with 70 adult *Daphnia* (35 each of infected and uninfected, size range 1.44–1.92 mm). Predators were allowed to feed in the dark for 21 h, after which we counted the remaining animals and noted other deaths or animals showing signs of attack. With these data, we determined selectivity on infected animals by calculating Chesson's alpha (Chesson 1983), a metric that compares the proportion of prey items consumed (here, estimated from those animals missing or attacked) vs. the proportion of those items in the environment. With two prey types, alpha values above 0.5 indicated selective predation on infected hosts.

Second, we asked whether *Chaoborus* release spores while feeding and if these released spores remain infective to *Daphnia*. In each of four treatments, we incubated five infected *Daphnia* per 140 mL water in a beaker at 20°C. Each of the four treatments was replicated 10 times. In the first treatment, infected hosts that died from infection were removed daily. This treatment conservatively simulated sinking of dead, infected hosts from the water column to the bottom of the lake. In both the second and third treatments, infected *Daphnia* were eaten by *Chaoborus*. *Chaoborus* swallow prey whole, but then regurgitate the carapace of the *Daphnia* and possibly spores contained within it (Pastorok 1980). In the second treatment, the five regurgitated carapaces were removed daily from the beaker, assuming that spores still trapped in the carapace sink to the lake bottom. In the third treatment, the five regurgitated carapaces were transferred to a microcentrifuge tube, homogenized in 1 mL filtered lake water, and returned to the beaker. This procedure liberated all spores from the regurgitant and offers a more liberal assessment of spore release from *Chaoborus*. In the fourth and final treatment, hosts first died from the disease, then they were homogenized to release spores that were then returned to the beaker. This treatment estimated the average number of spores per host (and maximum potential release of spores). Following the death of the last *Daphnia*, *Chaoborus* were removed. Then, spores in 35 mL of water were stained with cotton blue, filtered onto two Nucleopore filters, and counted by scanning five random fields per slide at 400×. Planned contrasts in PROC GLM (SAS version 9.1) addressed three questions regarding spore release via *Chaoborus* predation: (1) Does predation increase spore release relative to spores that may escape a host dying from infection before it sinks from the water column (treatment 1 vs. treatment 2)? (2) Are a significant number of spores trapped in the regurgitated carapace (2 vs. 3)? (3) Do *Chaoborus* liberate all the

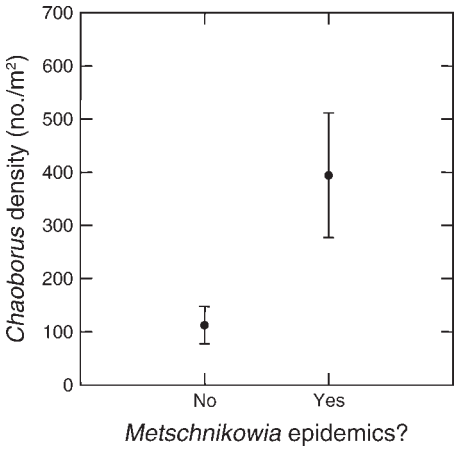

FIG. 1. Lakes in Michigan, USA, with *Metschnikowia* fungal epidemics (>2% averaged from 2002 to 2007) had higher areal abundances of predatory midges *Chaoborus* (*punctipennis* and *flavicans*) in July 2007 than did those lakes without *Metschnikowia* epidemics. Error bars are ±SE.

spores from the host (2 vs. 4)? Spore counts were log-transformed prior to analysis to equalize variances.

We then used 100 mL of the water remaining in each beaker to determine whether the spores liberated from hosts that had been consumed by *Chaoborus* remained viable. Six, 6-d-old healthy *D. dentifera* were added to each beaker. These animals were incubated at 20°C and fed 2 mg C/L *Ankistrodesmus falcatus* every day. After 10 d, animals were visually assayed for infection. We again used planned contrasts using logistic ANOVA in PROC GENMOD (SAS version 9.1) to ask two questions: (1) Do the spores liberated by predation increase infection rate relative to spores released from hosts dying from infection (treatments 1 vs. 2)? (2) Does predation reduce infection rates relative to the maximal rate that could be achieved if all spores from a host dying of infection were instantly liberated (2 vs. 4)?

Finally, to confirm that *Chaoborus* can facilitate disease spread within a host population, we conducted a 31-d experiment in 1-L flasks. To begin, 25 uninfected adult *D. dentifera* were added to each of 10 flasks filled with 1 L of filtered lake water. *Daphnia* were fed high food (2 mg C/L *Ankistrodesmus falcatus* every other day), incubated at 20°C, and allowed to increase in density for two weeks. We then added 30 spores/mL to each flask. The following day, one larval *Chaoborus punctipennis* (7.86 ± 0.09 mm) was added to five of the flasks; the other five remained predator-free. All flasks were incubated at 20°C and fed high food for 31 d, and any *Chaoborus* that emerged or died was replaced. Given that we can diagnose infection after 10 d and that most hosts typically die within 15 d under these conditions, we assume the 31-d run of the experiment represents at least two rounds of infection. At the end of the experiment, we counted all juvenile and adult *Daphnia* in each flask and recorded which individuals were infected. We used PROC GENMOD (SAS version 9.1) to determine whether the proportion of adults infected (number of adults infected/total adults) and the total proportion of the population infected (number of adults and juveniles infected/total population) differed between the two treatments. We analyzed infection in adults separately because it can be harder to diagnose infected juveniles (even though we note infection in some of them).

Results

The field survey provided no evidence that these invertebrate predators "keep the herds healthy"; *Chaoborus* densities were higher in lakes with epidemics than those without ($t = -2.56$, df = 5.9, $P = 0.02$; Fig. 1). Therefore, this parasite tends to invade and persist in lakes with more invertebrate predators. In the laboratory selectivity experiments, the Chesson's alpha for infected *D. dentifera* was 0.5 ± 0.08 (mean ± SE), indicating no preference for either infected or uninfected hosts.

The spore release experiment showed that *Chaoborus* release 40–70% of the spores contained in their infected-host prey (Fig. 2A). The number of spores released in the four treatments ranged from 20 to 830 spores/mL (ANOVA, $F_{3,36} = 81.55$, $P < 0.0001$). It takes several days for spores to escape from dead hosts (Hall et al. 2006), so not surprisingly, few spores were liberated from animals that died from the disease and were removed daily. In contrast, predation by *Chaoborus* increased the number of spores in the water fourfold (contrast 1 vs. 2; $P < 0.0001$). *Chaoborus* released ~40% of the maximum possible number of spores (contrast 2 vs. 4; $P < 0.0001$). Spores must also remain trapped in the regurgitated host's carapace, given that manually releasing spores from regurgitated hosts increased spore numbers (contrast 2 vs. 3; $P < 0.0001$). We assume that the remaining 30% of spores within consumed infected prey (treatment 3 vs. 4) were digested by this predator.

Importantly, the transmission assay established that spores remained infective to *Daphnia* following predation by *Chaoborus* (Fig. 2A). Since spore densities differed per treatment, the resulting infection prevalence also varied (logistic ANOVA; $\chi^2 = 69.10$, df = 3, $P < 0.0001$). Predation by *Chaoborus* resulted in higher infection rates relative to treatment 1, in which the minimum number of spores were released (contrast 1 vs. 2; $\chi^2 = 18.63$, df = 1, $P < 0.0001$). However, infection prevalence did not vary between the lower *Chaoborus* release treatment (2) and the maximal spore release one (4) (contrast 2 vs. 4; $\chi^2 = 2.20$, df = 1, $P = 0.14$).

Finally, the 31-d long experiment confirmed that *Chaoborus* can act as a spreader of disease in this host–parasite system (Fig. 2B). We found significantly higher incidences of disease when *Chaoborus* was present both in the adults (PROC GENMOD, $\chi^2 = 75.37$, df = 1, $P < 0.0001$) and the entire population ($\chi^2 = 43.6$, df = 1, $P < 0.0001$) (population sizes, *Chaoborus* treatment, 176 ± 27 *Daphnia*/L [mean ± SE]; no *Chaoborus* treatment,

161 ± 6 *Daphnia*/L). Although these results likely do not represent equilibrial prevalence of disease, they clearly demonstrate that the presence of this predator facilitates disease spread once epidemics have been initiated.

Model

The striking field pattern between disease prevalence and *Chaoborus* densities strongly suggests that this predator could enhance invasion of this fungal parasite. The laboratory experiments revealed mechanisms to explain why: although not particularly selective, *Chaoborus* release the majority of spores that they consume (both directly to the water and in the regurgitated carapace), and these spores remain highly infective. Furthermore, natural history tells us that *Chaoborus* largely feed on *Daphnia* at night in well-mixed, epilimnetic waters (Pastorok 1980); therefore, spores are likely released in a place where they can then infect new *Daphnia*. Armed with empirical information and natural history, we turn to a minimal model to show how a predator such as *Chaoborus* could enhance invasion and persistence of the parasite. In this model, we assume that infection converts susceptible hosts (S) into infected hosts (I) after contact with spores in the water column (Z_w). However, once hosts die from infection, spores are released in a bottom pool (Z_b) in the lake sediments. Spores in this pool must then mix into the water column before they can contact hosts. Predators, such as *Chaoborus* (C), consume both host classes but can release spores into the water column pool. Given this biology, the model becomes (Fig. 3, Table 1):

$$dS/dt = bS + \rho bI - d_S S - uf_S S Z_w - f_C C S \quad (1a)$$

$$dI/dt = uf_S S Z_w - (d_S + v)I - \theta f_C C I \quad (1b)$$

$$dZ_b/dt = \sigma(d_S + v)I - mZ_b - d_Z Z_b + \lambda Z_w \quad (1c)$$

$$dZ_w/dt = \sigma_C \theta f_C C I + mZ_b - f_S(S + I)Z_w - \lambda Z_w. \quad (1d)$$

Susceptible hosts (S, Eq. 1a) increase due to births from both host classes (albeit at a reduced rate from infected hosts; $0 < \rho < 1$ captures the virulent effect of the parasite on births). There is no density dependence of births, an assumption that offers us the only hope of analytical tractability here. Susceptible hosts then die at the background rate d_S; they become infected after contact (at rate f_S) with spores in the water column that themselves have per-spore infectivity u; and they are eaten by predators, following a linear predation term (governed by feeding rate f_C). Infected hosts (I, Eq. 1b) increase following infection, die at a rate enhanced by the parasite ($d_S + v$), and are preyed upon by predators. Since in nature *Daphnia* can be consumed by both predatory invertebrates and planktivorous fish, the model assumes that predators either choose their prey at random in the case of *Chaoborus* ($\theta = 1$) or selectively in the case of fish ($\theta = 9$). If infected hosts die directly due to virulence of the parasite, spores are released in the bottom pool (Z_b, Eq. 1c), assuming that each dead host produced σ spores. Spores in this bottom pool are then mixed into the water column (at rate m) or buried and hence lost from the system entirely (at rate d_Z). This pool also increases as spores in the water column sink (at rate λ). Finally, spores in the water column (Z_w, Eq. 1d) increase due to release

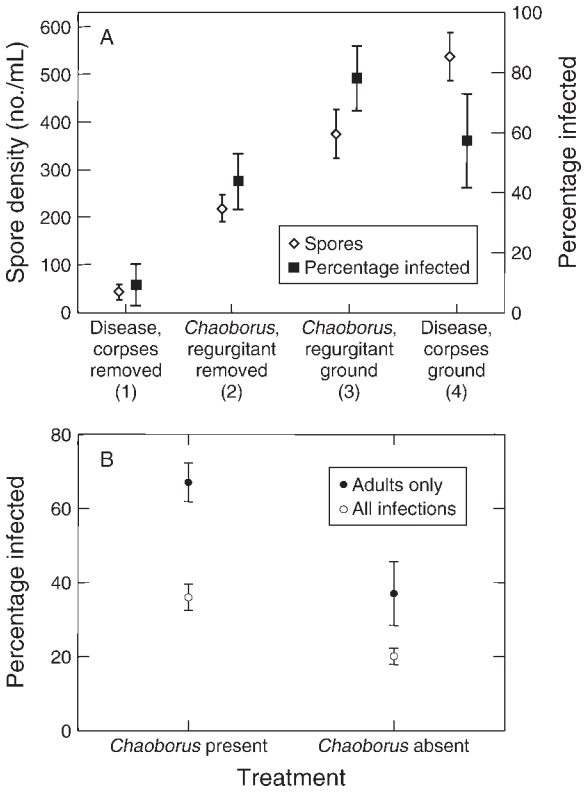

FIG. 2. Results (mean ± SE) of laboratory experiments. (A) The experiment in which infected hosts (*Daphnia dentifera*) either died from disease (treatments 1 and 4) or were eaten by *Chaoborus* (treatments 2 and 3). Treatments also differed in whether dead animals were removed from the beaker (treatments 1 and 2) or ground up and then returned (treatments 3 and 4; see *Empirical methods and results: Laboratory experiments*). Open symbols indicate that death by *Chaoborus* resulted in an increase in *Metschnikowia* spores released from infected hosts relative to those hosts that died from the disease (contrast 1 vs. 2). The increase in spore number in treatment 3 vs. 2 indicates that some spores remained trapped in the regurgitated host's carapace. Treatment 4 suggests that *Chaoborus* was not 100% effective in releasing spores; more spores were liberated when we manually ground infected individuals (contrast 2 vs. 4). Solid symbols indicate that, by increasing the number of spores released from dead hosts, the presence of *Chaoborus* increased infection prevalence relative to the treatment in which the hosts died from the disease and were removed from the beaker (right-hand y-axis). (B) The longer-term dynamics of this host–parasite–predator system. After 31 days, the presence of the predator resulted in higher infection rates. This was the case when we focused the analysis only on adults (solid symbols) or on adults and juveniles combined (open symbols).

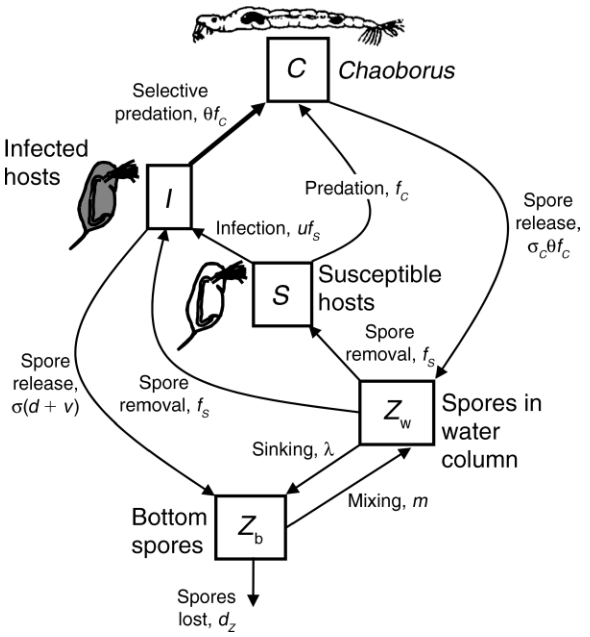

FIG. 3. Diagram of the model for the *Daphnia* host–fungal parasite–*Chaoborus* predator system. Susceptible *Daphnia* hosts (S) become infected (I) after they contact spores of the parasite *Metschnikowia* that are dispersed in the water column (Z_w). Infected hosts then either die due to infection or from selective predation (i.e., at a higher rate than experienced by susceptible hosts if $\theta > 1$). Spores contained in hosts that die due to infection are released into a bottom pool of spores (Z_b), while spores within consumed, infected hosts are released directly into the water column (Z_w). Spores in the bottom pool can mix into the water column pool or become lost from the system (due to burial, etc.). Spores in the water column can sink to the bottom pool or be cleared by both classes of hosts; however, only removal by susceptible hosts promotes infection. See Table 1 for an explanation of variables and their abbreviations.

from selective predation on infected hosts (where σ_C spores are released per eaten host; $\sigma_C \leq \sigma$) and mixing from the bottom pool. They are lost, in turn, following removal by both host classes (both contacting and removing spores at rate f_S) and from sinking to the bottom sediments (at rate λ).

We can use this model to derive a key threshold delineating conditions in which the parasite can successfully invade ($R_0 > 1$) and persist ($I^* > 0$) with the host. Since the host would increase exponentially without parasitism, this condition minimally requires that the parasite is sufficiently virulent to regulate its host (see also Appendix). Furthermore, predator density cannot be so high that susceptible hosts cannot persist (i.e., $C < (b - d)/f_C$). Assuming these conditions are met, we can derive this threshold in terms of mixing rate (m), or the rate at which spores leave the bottom pool and enter the water column pool. Without predation, invasion and persistence of the parasite require that mixing must be sufficiently strong to move spores between bottom and water column pools. So, the key invasion/persistence threshold becomes

$$\hat{m} = d_Z\left[\frac{d_{I,C}(r_{S,C} - r_{I,C}) + r_{I,C}\varphi_C}{-r_{I,C}(\varphi + \varphi_C) - d_{I,C}(r_{S,C} - r_{I,C})}\right] \quad (2)$$

which is composed of several compound parameters that summarize per capita vital rates of infected and uninfected hosts (see also Appendix for further development of this model):

$$d_{I,C} = d_S + v + \theta f_C C \quad (3a)$$

$$r_{S,C} = b - d_{S,C} > 0, \quad \text{where } d_{S,C} = d_S + f_C C \quad (3b)$$

$$r_{I,C} = \rho b - d_{I,C} < 0 \quad (3c)$$

$$\varphi_C = u\sigma_C(\theta f_C C) \text{ and } \varphi = u\sigma(d_S + v). \quad (3d)$$

The composite mortality term (Eq. 3a) represents per capita death rates of susceptible hosts ($d_{S,C}$), death rates of infected hosts ($d_{I,C}$). The $r_{i,C}$ terms (Eqs. 3b, c) then are the net difference of per capita birth minus composite mortality terms for susceptible ($r_{S,C}$) and infected ($r_{I,C}$) hosts (where we note a death term for susceptible hosts without infection; notice that $r_{S,C}$ is positive but $r_{I,C}$ is negative [see Appendix]). Finally, the φ_C and φ terms (Eq. 3d) represent the total infectivity of spores released into the water column from predation by *Chaoborus* and directly into the benthic pool from virulence of the parasite, respectively.

The model reveals that predators can indeed enhance invasion and persistence of the parasite (which occurs in mixing predator density parameter space above the lines; Fig. 4). However, spore release from predators (σ_C) matters greatly. In fact, spore release from a predator such as *Chaoborus* that releases spores directly into the water could effectively shortcut the bottom pool-mixing route otherwise required for parasites to invade and persist. Without release from predators, spores produced by infected hosts that died due to infection are deposited on the bottom of the lake (Z_b); the spores need to mix into the water column to infect new hosts. With sufficiently high release from predators (high σ_C), no mixing is required from the bottom pool to sustain epidemics; the disease system can become disconnected from the bottom pool of spores entirely (Fig. 4A). Furthermore, predators can enhance the parasite's persistence even if they do not release spores (seen in the $\sigma_C = 0$ case; Fig. 4A). This effect arises because predators cull infected hosts that otherwise would remove spores in the water column (Z_w) that could infect susceptible hosts. Simply put, while alive, infected hosts are sinks for spores (see Appendix for further explanation).

Second, we find that predator selectivity (θ) can alter the shapes of these curves. In the case of a nonselective predator such as *Chaoborus*, all curves (as parameterized) decrease with predator density (in the parameter space shown). That is, with increasing predator density,

TABLE 1. Summary of symbols for variables and parameters, their units and interpretation, values/ranges of parameters, and sources used to generate Fig. 4 and Appendix: Fig. A1.

Symbol	Units	Interpretation	Value	Source of parameter
I	no./L	infected hosts (density)	...	
S	no./L	susceptible hosts (density)	...	
Z_b	spore/L	spores in the bottom sediment pool	...	
Z_w	spore/L	spores in the water column pool	...	
t	d	time	...	
b	d^{-1}	birth rate (density-independent), susceptible hosts	0.2	Hall et al. (2009*b*)†
C	no./L	predator density	0–1.5	Hall et al. (2009*b*)†
d_S	d^{-1}	background mortality rate, susceptible hosts	0.03	S. R. Hall, C. Becker, and C. E. Cáceres (*unpublished data*)
d_Z	d^{-1}	loss rate of spores from the bottom	0.05	educated guess
f	no. spore·L^{-1}·d^{-1}	contact rate of hosts with spores	0.006	Mourelatos and Lacroix (1990)
f_C	L·no.$^{-1}$·d^{-1}	feeding rate of predator	0.05	this study
m	d^{-1}	mixing rate of spores from bottom to water column	0–0.05	educated guess
u	no./spore	per spore infectivity	0.002	Hall et al. (2009*b*)†
v	d^{-1}	mortality due to infection (virulence on survivorship)	0.08	Hall et al. (2009*b*)†
θ	...	predator selectivity ($0 < \theta$), estimated as $\alpha/(1 - \alpha)$	1, 9	this study, Duffy and Hall (2008)
λ	d^{-1}	sinking rate of spores from the water column to bottom	0.05	educated guess
ρ	...	fecundity reduction due to virulence ($0 \leq \rho < 1$)	0.3	Hall et al. (2009*b*)†
σ	no. spores	spores produced per infected host dying of infection	15×10^3	Hall et al. (2009*b*)†
σ_C	no. spores	spores released per depredated infected host ($\sigma_C \leq \sigma$)	$0–15 \times 10^3$	possible range

† Assuming values for deep, stratified lakes in late summer (when epidemics begin).

invasion and persistence of the parasite becomes more likely. However, with a highly selective predator, such as a bluegill sunfish (*Lepomis macrochirus*; $\theta = 9$; Duffy and Hall 2008; Fig. 4B), spore release matters greatly for the parasite. If the predator releases few or no spores (low σ_C), the benefit of predators for the parasite diminishes as predator density increases. In fact, with enough predators, invasion and persistence actually become more difficult for the parasite. In this case, the negative consequences of the predator (killing infected and susceptible hosts, releasing few or no spores) outweigh the positive ones (spreading spores, reducing the infected-spore removal phenomenon). However, if highly selective predators release most spores, they can enhance persistence of the parasite by lowering the mixing levels needed to sustain epidemics. Thus, highly selective predators can either enhance or diminish invasion success of parasites, depending on predator density and spore release from predators to the water column.

Of course, the model also describes the effect of selective predators on infection prevalence or proportion of total hosts infected. Here we find some tension between predator effects on disease invasion/persistence vs. equilibrial prevalence (since the two are not one and the same). Assuming that the parasite can indeed invade (which is the big question considered here), equilibrial infection prevalence declines with predator density (C) and selectivity of predation on infected hosts (θ; see Appendix for equations). Spore release from predators (σ_C) does not enter into the equilibrial prevalence equation. Thus, highly selective predators that release many spores may enhance invasion of the parasite in unfavorable environments but still depress infection prevalence in environments more favorable to the parasite (e.g, when mixing rates are higher). Conversely, nonselective predators ($\theta = 1$), such as *Chaoborus*, permit higher infection prevalence, again assuming that the parasite can invade.

DISCUSSION

This *Chaoborus–Daphnia*–fungus system provides a striking counter-example to the "healthy herds" hypothesis. Michigan lakes with higher densities of this invertebrate predator tend to have large epidemics; those with low densities do not. Clearly this pattern contradicts expectations from the healthy herds idea. The experiments and model suggest this pattern is not merely correlative: *Chaoborus* could actually enhance invasion and persistence of this obligately killing, environmentally transmitted fungal parasite. Indeed, the laboratory experiments paint a very different picture than the "healthy herds" view of predators as sinks for parasites, since these predators release a high proportion of infective propagules from their infected prey. In fact, *Chaoborus* likely release 40–70% of the spores contained in their infected prey, indicating that these predators are not necessarily sinks for parasites. Furthermore, those spores remained highly infective, as evidenced both by the short-term infection assay and the longer-term experiment. The model shows that such high rates of spore release from predators matter greatly when invasion and persistence of the parasite is otherwise limited by densities of infective propagules in the environment, i.e., when the environment sufficiently inhibits transmission of this parasite.

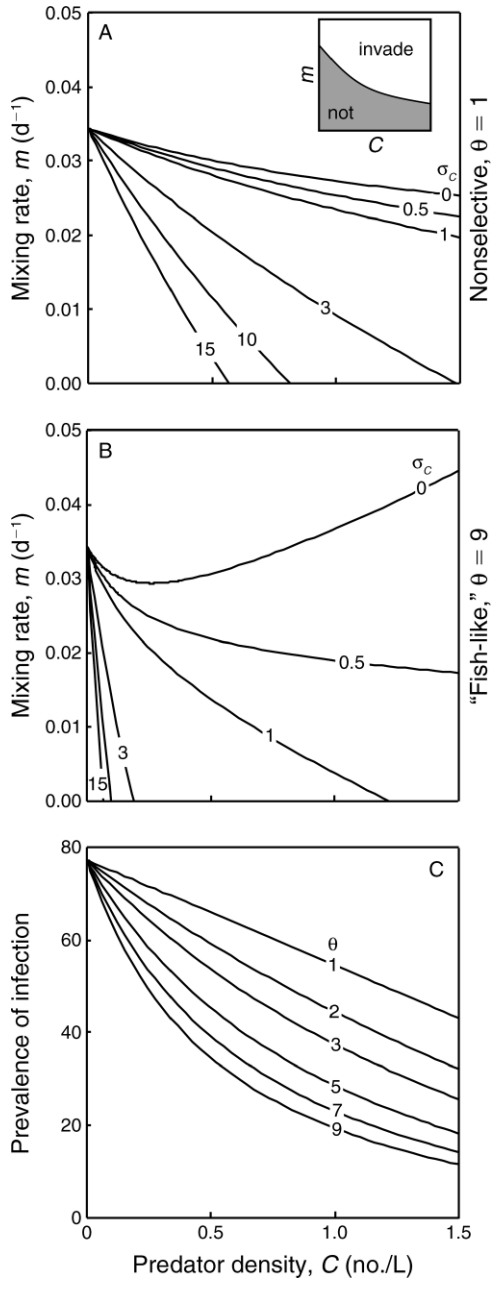

FIG. 4. Invasion and persistence thresholds for the model (Eq. 1) diagrammed in Fig. 3. Lines delineate mixing rates of spores (m, y-axis) from the bottom sediment pool to the water column that are required at a given predator density (C, x-axis) to allow parasites to invade and persist with the host (above the line) or not. Each line denotes a level of spores released per infected host eaten by a predator (σ_C, spores/host; reported as thousands of spores); these levels increase from zero to 15 000, the number produced by hosts dying from infection (σ). (A) Nonselective predators ($\theta = 1$) such as *Chaoborus*. (B) A predator that preys as selectively as a bluegill sunfish (*Lepomis macrochirus*; $\theta = 9$) but feeds at the same rate (for comparison purposes). In both cases, increasing predator density can enhance invasion/persistence of the parasite (i.e., require lower rates of mixing), even if predators do not release any spores from infected hosts. Furthermore, if predators release enough spores into the water column, they can sustain epidemics even without mixing from the bottom pool at all (i.e., when $m = 0$). This effect can be particularly pronounced for highly selective predators, assuming that they release enough spores. If they do not release spores, increasing densities of these highly selective predators can make it increasingly difficult for the parasite to invade. (C) Equilibrial infection prevalence (Appendix: Eq. A.3) over a gradient of predator densities (C, x-axis) and predator selectivities (contours), ranging from nonselective ($\theta = 1$, such as *Chaoborus* here) to highly selective ($\theta = 9$, such as bluegill sunfish). Prevalence declines with higher predator density and higher selectivity, assuming that the parasite can invade (i.e., mixing is sufficiently high) over this entire predation gradient.

Why does the environment pose such a hurdle for the fungal parasite, and how does the predator enable the parasite to overcome it? Fungal epidemics begin in late summer (Cáceres et al. 2006, Duffy and Hall 2008; R. L. Smyth et al., *unpublished manuscript*), when spore limitation almost certainly impinges on fungal epidemics. Late-summer thermal stratification essentially seals the deep-water sediment (benthic spore pool) from the rest of the lake that the host inhabits. This environment is quite unlike that in ponds, where hosts can contact parasite spores while foraging in the sediment (e.g., the *Daphnia–Pasteuria* system; Ebert 1995, Decaestecker et al. 2002). Instead, in lakes, infected dead hosts and the spores contained within them sink out of the water column. Spores released on the lake bottom from these hosts then cannot contact their pelagic hosts, and this feature poses a problem for such an environmentally transmitted parasite. The model does suggest that epidemics can be sustained without predators (since we delineated a critical mixing rate required for invasion/persistence of the parasite), but the required mixing rate may be too high during late summer in these small, stratified lakes. In contrast, the model shows that predators such as *Chaoborus* can sustain epidemics without any input of spores from the benthic pool. *Chaoborus* largely forage on *Daphnia* at night in the epilimnion (Pastorok 1981, Moore 1988). Because they regurgitate prey soon after consumption, they release spores into a portion of the water column where spores can contact hosts and likely remain suspended. In essence, this "predator–spreader" shortcuts the bottom-mixing route otherwise required for disease transmission. In doing so, *Chaoborus* can alleviate this major environmental constraint on the fungus.

Moreover, predation by *Chaoborus* may have several indirect effects on the host that may also translate to increased disease. These features could be added to a more sophisticated model in the future. The gape-limited *Chaoborus*, like many predatory invertebrates, can shift the size structure of host populations toward larger animals. The mechanism behind this shift is twofold: these predators selectively prey on intermediate-sized *Daphnia* but also can induce life-history shifts toward growth rather than reproduction, e.g., larger size at

maturity and later reproduction (Pastorok 1980, Neill 1981, Spitze 1991, Riessen 1999). Regardless of the mechanism, any shift toward larger-sized hosts matters for the fungus because larger hosts are more easily infected and produce more spores, all else being equal (Ebert 2005, Hall et al. 2007*a*, 2009*c*). Clearly, more accurate predictions linking predators with disease must consider direct effect of consumption of infected prey as well as multiple indirect effects such as these (Preisser et al. 2005, Keesing et al. 2006).

The model also permits a more nuanced reevaluation of a highly selective predator in lakes. Like many predators, bluegill sunfish prey very selectively on infected hosts (Johnson et al. 2006, Duffy and Hall 2008). In past modeling efforts, we have assumed that this predator acts as a complete sink for parasites (Duffy et al. 2005, Hall et al. 2005, 2006); as a result, such highly selective predation can strongly impinge on invasion and persistence of parasites. A recent experiment indicates that at least some proportion of spores survive digestion and remain infective (Duffy 2009); however, these spores are likely defecated nearshore rather than directly into the epilimnion. Does this revelation change interpretation of this very selective predator? The model suggests that the answer depends critically on predator density and its effective spore release. If many/most spores from these highly selective predators can contact hosts, the "predator–spreader" idea becomes accentuated. However, if no or few spores can actually contact hosts, the "healthy herds" result is largely retained, although low densities of highly selective predation can enhance parasite invasion somewhat. Once the parasite can invade/persist, infection prevalence declines with predator density and degree of selectivity of the parasite. In this sense, then, the "healthy herds" idea still applies: higher densities of selective predators should depress infection prevalence, again assuming that the parasite can indeed invade.

One surprising result emerged from the model; when spore loss limits invasion/persistence of the parasite, predators can enhance parasite success by culling infected individuals that themselves remove spores from the water column. This idea forces us to rethink the various functions of the infected host class. Infected hosts of course produce new infective stages, but they are rarely thought of as sinks for the parasite. However, for environmentally transmitted, obligately killing diseases, infected hosts may produce a within-species dilution effect (Hatcher et al. 2006, Keesing et al. 2006, Hall et al. 2009*a*), i.e., infective spores are removed by a host that cannot produce additional infections. Actually, in *Daphnia* disease systems, consumption of additional spores may even lower spore production from infected hosts (Ebert et al. 2000, Hall et al. 2007*b*). Although this spore dose–production effect was not modeled here, it would likely only accentuate the positive "benefits" of predator culling of infected hosts.

We are not the first to suggest that predators can enhance invasion of disease. Many parasites are trophically transmitted and clearly those systems require predators for the persistence of the parasite (Lafferty 1999, Thomas et al. 2005). In other cases, predators can increase parasitism via effects on the immune class of hosts, a situation that more often applies to diseases of vertebrates than invertebrates (Choisy and Rohani 2006, Holt and Roy 2007, Roy and Holt 2008). Here we uncovered a new mechanism (see also Duffy 2009): in environmentally transmitted disease systems, predators may enhance or inhibit epidemics depending (in part) on whether they act as sources or sinks for infective propagules. Since many terrestrial and aquatic invertebrates are parasitized by obligately killing fungi, bacteria, viruses, and nematodes (Ebert and Weisser 1997), a variety of systems may offer additional tests of this "predator–spreader" hypothesis. In the meantime, this planktonic disease system prompts a very important, general warning: before we use predators to keep the herds healthy, we need to carefully think about the interface between predator feeding biology and the underlying epidemiology of wildlife disease. What really are the various roles of predators in disease transmission in light of other environmental constraints on parasites? How could they facilitate disease spread?

Acknowledgments

Many people enabled the timely completion of our laboratory and fieldwork including C. Becker, C. Bertram, N. Consolatti, E. Litchman, A. Morgan, R. Park, J. Van Dyke, and P. Woodruff. Thanks also to M. Duffy, R. Smyth, S. MacIntyre, and A. Tessier, our collaborators on the *Daphnia* disease project, for endless discussion. The Champion, Brehm, and Albert families provided access to private lakes. This study was supported by NSF grants DEB 06-13510 and DEB 06-14316. C. J. Knight was supported by an REU supplement. This is contribution number 1530 of the W. K. Kellogg Biological Station.

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APPENDIX

Analysis of the "predator–spreader" model and its variants (*Ecological Archives* E090-201-A1).