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LETTER

Quality matters: resource quality for hosts and the timing of epidemics

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Abstract

Epidemiologists increasingly realize that species interactions (e.g. selective predation) can determine when epidemics start and end. We hypothesize here that resource quality can also strongly influence disease dynamics: epidemics can be inhibited when resource quality for hosts is too poor and too good. In three lakes, resource quality for the zooplankton host (*Daphnia dentifera*) was poor when fungal epidemics (*Metschnikowia bicuspidata*) commenced and increased as epidemics waned. Experiments using variation in algal food showed that resource quality had conflicting effects on underlying epidemiology: high-quality food induced large production of infective propagules (spores) and high birth rate but also reduced transmission. A model then illustrated how these underlying correlations can inhibit the start of epidemics (when spore production/birth rate are too low) but also catalyse their end (when transmission becomes too low). This resource quality mechanism is likely to interface with other ones controlling disease dynamics and warrants closer evaluation.

Keywords

Consumer–resource, *Daphnia–Metschnikowia,* epidemic, host–parasite, resource quality, transmission rate.

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INTRODUCTION

What determines the timing, duration and extent of epidemics in wildlife populations? This question and our answers to it – or lack of satisfactory ones – point to fundamental challenges involved in predicting and controlling wildlife disease. As made evident by the rapidly growing literature, ecologists increasingly understand that other species may play critical roles in either increasing or diminishing epidemics (Hatcher *et al.* 2006; Keesing *et al.* 2006). In particular, predators that selectively prey on infected hosts can inhibit disease (Packer *et al.* 2003; Ostfeld & Holt 2004; Duffy *et al.* 2005; Hall *et al.* 2005a) or actually enhance it (depending on immune response of hosts: Holt & Roy 2007). Furthermore, other hosts can reduce disease through a 'dilution effect' or spread it through 'spillover' (Holt *et al.* 2003; Keesing *et al.* 2006; Hall *et al.* 2009). Interestingly, mechanisms involving predators and other hosts and the mathematical models used to describe them largely ignore resources of the focal host (Hatcher *et al.* 2006; Hall *et al.* 2008). This hole in theoretical disease ecology seems surprising as dynamics of resources have immense implications for interactions and persistence of species in food webs (Grover 1997). Indeed, the 'consumer–resource interaction is arguably the fundamental unit of ecological communities' (Murdoch *et al.* 2003). Thus, models for ecology of disease may remain incomplete without considering the indirect effects of resources. After all, host–resource interactions are likely to modulate interactions of hosts with their predators and other hosts species.

In this study, we push this line of reasoning one step further: resources on their own may play critical roles in shaping the timing and nature of disease dynamics. This argument arises from synthesis from two currently separate bodies of literature involving food web ecology and resource-dependence of epidemiology. First, hosts can often strongly interact with their resources. For instance, insect defoliators can create pronounced fluctuations in their resources (Crawley 1983; Berryman 1987) and can induce plant defences; this induction then reduces the quality of the resource for the host-grazer (Karban & Baldwin 1997). In another example, the zooplankton grazer *Daphnia* can create considerable fluctuations in algal density (McCauley & Murdoch 1987; McCauley *et al.* 1999) and shunt species composition of their algal resources to

increasingly resistant forms of poor quality (Tessier *et al.* 2001; Tessier & Woodruff 2002a). The second, *epidemiological* component connects this food web ecology to interactions between hosts and their parasites. A growing literature, particularly involving invertebrate hosts (such as these insect and microcrustacean grazers), shows how resource quantity and quality directly shapes virulent effects of parasites on survivorship (Brown *et al.* 2000; Hodgson *et al.* 2002; Tseng 2004; Frost *et al.* 2008; Seppälä *et al.* 2008) and directly influences the production of parasites (Ebert *et al.* 2000; Bedhomme *et al.* 2004; Tseng 2004; Johnson *et al.* 2007; Ryder *et al.* 2007; de Roode *et al.* 2008). Typically, elevated quantity and/or quality of resources enable longer survival of infected hosts but also enhance the production of infective propagules. Transmission rate (susceptibility) can increase or decrease with resources. In many terrestrial systems, plant quality diminishes due to defensive compounds against herbivory from hosts, but these compounds can also inhibit transmission rate (Keating *et al.* 1990; Hunter & Schultz 1993; de Roode *et al.* 2008; but see D'Amico *et al.* 1998; Dwyer *et al.* 2005). However, other mechanisms are likely to link transmission to quality more broadly (Cory & Hoover 2006), so generalities relating resource quality to transmission seem harder to conjure. Regardless of directionality of effect, the vital point is that the key aspects of host–parasite interactions often hinge on resources consumed by the host. Therefore, variation in resources, caused by the host-grazer or imposed externally, may have considerable, direct influence on disease dynamics.

The resource hypothesis for disease dynamics proposed here arises from the marriage of these food web and resource-mediated, epidemiological components, as illustrated by an example. Using an algal resource-*Daphnia* host–fungal parasite system, we combine field observations, experiments and a simple model to demonstrate how variation in resource conditions could translate into the inhibition of epidemics. We focus on resource quality here because it is a dominant cause of resource limitation for *Daphnia* in lakes (DeMott & Tessier 2002; Tessier & Woodruff 2002a). Intensive sampling of three lakes revealed that epidemics started in late summer when resource quality was poor, and that late-season declines in disease prevalence corresponded to within-lake, temporal increases in resource quality/quantity. In the laboratory, we used field-collected and lab-reared algae to create a gradient of resource quality. Using this gradient, we found a negative correlation between the transmission rate and resource quality; additionally, production of offspring of uninfected hosts and parasite spores elevated with quality. Therefore, resource quality imposed negative relationships between transmission and spore production and also transmission and birth rate. We have seen similar relationships imposed by resource quantity (Hall *et al.* 2007a,b, in review). These quality-mediated relationships matter in the *Daphnia*–fungus system because they could influence the timing of epidemics. Using a mathematical model, we illustrate how the transmission-parasite production–birth rate relationship could explain the pattern in nature: seasonal variation in resource quality, when accounting for its correlation with water temperature, can catalyse the end of fungal epidemics but is likely to also inhibit their start.

EMPIRICAL METHODS

Lake sampling

During 2004, we intensively monitored large epidemics in three lakes (Bassett, Bristol and Warner Lakes) in southwest Michigan (USA). Every 3 days, we estimated the prevalence of fungal infections, densities of susceptible and infected *Daphnia dentifera* hosts, and a coarse yet revealing index of food quality/quantity (egg ratios of uninfected adults). At each visit, we collected four samples; in each sample, we pooled three bottom-to-surface tows of a standard Wisconsin plankton net (13 cm diameter, 153 μm mesh) sampled at locations separated by at least 20 m. From one sample, we estimated the prevalence (no. infected/total hosts) of infection in at least 400 live hosts, visually diagnosing the infection according to Green (1974) using a dissecting microscope. We also examined 50 or more randomly sampled adult females to estimate a coarse index of food quantity/quantity and egg ratio (number of eggs carried per adult; see Figure S1 in Appendix S1 for a comparison of egg ratios in uninfected vs. infected females). We preserved the other three samples in 50–75% ethanol for later estimation of densities.

Experiments: quality, virulence and transmission

We linked the transmission and virulence of the fungal parasite with the variation in algal resources using three experiments conducted during July 2007 at the W. K. Kellogg Biological Station. To create a gradient of resource quality for each of these experiments within a narrow range of time, we capitalized on a previously documented, spatial gradient of resource quality (Tessier & Woodruff 2002a). We selected three deep lakes (Little Mill, Lawrence and Three Lakes II) known to have poor quality algae and three shallow lakes (Douglas, Duck, Three Lakes III) to cover moderate quality (see Tessier & Woodruff 2002a for more details on each study lake; Barry and Kalamazoo Counties, Michigan); lab-reared *Ankistrodesmus falcatus* provided the algal food of high quality. Each morning, we used an integrated tube sampler to collect algae. In the shallow lakes, we sampled from surface to 0.5 m above bottom,

while in the deep lakes, we sampled from surface to 1 m below the thermocline (i.e. the depth at most rapid temperature change). We kept the 3 L of water harvested from each lake dark in coolers. Using this field collected water, we then controlled the quantity for each of the three experiments by first measuring dry mass of 'edible' seston (< 60 μm, strained using Nitex mesh, Wildlife Supply, Saginaw, MI, USA) filtered onto 47 mm glass fibre filters (GF/F, 0.7 μm pore size, Whatman, Florham Park, NJ, USA). Using these measurements, we diluted algae collected the next day from each lake (using GF/F filtrate) to achieve 0.9 mg dry mass L^{-1}. This level was chosen because it was the minimal level observed in pilot collections. This procedure was repeated two to three times weekly, and dilutions were adjusted accordingly. To achieve similar dry mass of the lab-reared algae, we used an absorbance (750 nm)-dry weight regression and appropriate dilution in a 50 : 50 mix of water collected from Gull and Pleasant Lakes (Barry County, MI, USA; herein 'stock water'). Then, we used these seven sources of algal seston in the three sets of experiments.

Experiment 1: growth rate assays

We quantified the variation of quality of these algal resources using a well-established technique, the juvenile growth rate bioassay (g). Previous work has shown that the accrual of dry mass of juvenile *Daphnia* tightly predicts instantaneous rates of increase (r) fuelled by variation in the quality (and quantity) of algal food resources (Desmarais & Tessier 1999). We measured g as the mass accrued by neonates to day 4 of the assay. A standard clone of a *Daphnia pulex–pulicaria* hybrid (the 'Geedey clone') was used to determine the resource quality because this hybrid grows quickly and is sensitive to variation in quality. Furthermore, its growth response correlates very tightly not only with that of this clone of *D. dentifera* but also with that of other species of *Daphnia* (Tessier and Woodruff 2002a,b). To provide initial, day 0 measurements ($\bar{m}_0$), 15 neonates (< 24 h old) were dried at 55 °C, then weights were estimated using a Mettler microbalance (Mettler-Toledo, Columbus, OH, USA) and averaged. Simultaneously, we placed 10–15 neonates in separate 50 mL jars of mass-controlled water containing lake or lab-grown algal seston. For 4 days ($d = 4$), we transferred these animals into freshly collected water (using the collection methods above), then similarly dried and weighed each individual day 4 animal (m_4). Our growth rate/quality measure was then: $g = [\ln(m_4) - \ln(\bar{m}_0)]/d$.

Experiments 2 and 3: estimation of virulence and transmission

We also quantified how the virulence and transmission of the fungal parasite depended on the variation in resource quality among these seven sources. We used a life table experiment to estimate virulent effects of the parasite on host survival, fecundity, size at death and spores produced. Using 6-day-old juveniles of a single clone the host *D. dentifera* (raised on 2.0 mg L^{-1} of *Ankistrodesmus*), we exposed 'infection' treatment animals (10 per resource quality level) to a high spore dose (1500 fungal spores mL^{-1} for 24 h in stock water, 0.5 mg dry mass L^{-1} of *Ankistrodesmus*, room temperature) to ensure infection. Uninfected animals received similar treatment – except they did not receive spores. Then, when starting the experiment, we placed individual unexposed ($n = 6$) and infected *Daphnia* in 150-mL beakers of the various mass-adjusted lake waters – without spores. This spore-free water was changed daily; during these changes, we noted the number of offspring produced, date of death and size reached upon death (measured at 50× using a micrometre, Leica Microsystems, Wetzlar, Germany). After measuring the dead *Daphnia,* we placed infected animals into 0.25 mL of lake water in plastic centrifuge tubes, gently smashed corpses using tweezers and counted spores in the resulting slurry using a haemocytometre (Fisher Scientific, Pittsburgh, PA) and a compound microscope (200×), Leica, Microsystems, Wetzlar, Germany.

We used a simple infection assay to estimate the transmission rate. We placed five 6-day-old *D. dentifera* into 100 mL of the seven lake-lab waters, all adjusted to have 0.9 mg dry mass of algal seston. To these seven combinations, we added one of two levels of a spore addition treatment (25 spores mL^{-1} as shown in the text, 75 spores mL^{-1} treatment is shown in Appendix S3; replicated eight times) and incubated the hosts for *c.* 24 h (20 °C, 16 : 8 day night cycle). Afterwards, we transferred animals from each beaker into spore-free 'stock' water filled with 2.0 mg dry mass L^{-1} of *Ankistrodesmus*. Animals in each beaker were fed daily for 10 days until infection diagnosis could be made visually (using a dissecting microscope, following Green 1974). Animals were scored as infected or uninfected to calculate the prevalence of infection.

EMPIRICAL RESULTS

Lake sampling

In each of the three intensively sampled epidemics in 2004, we saw consistent correlations between the index of resource quality (egg ratios) and infection dynamics. First, epidemics start when resource quality was poor. Then, after the peak of infection, decreases in infection prevalence and/or density of infected hosts correlated negatively with egg ratios: the *Daphnia* populations became less sick as the resource quality increased (Fig. 1, Table 1). Simultaneously, in two of the populations (Bassett and Bristol), the elevation of resource quality correlated with increases in the density of uninfected hosts (Table 1).

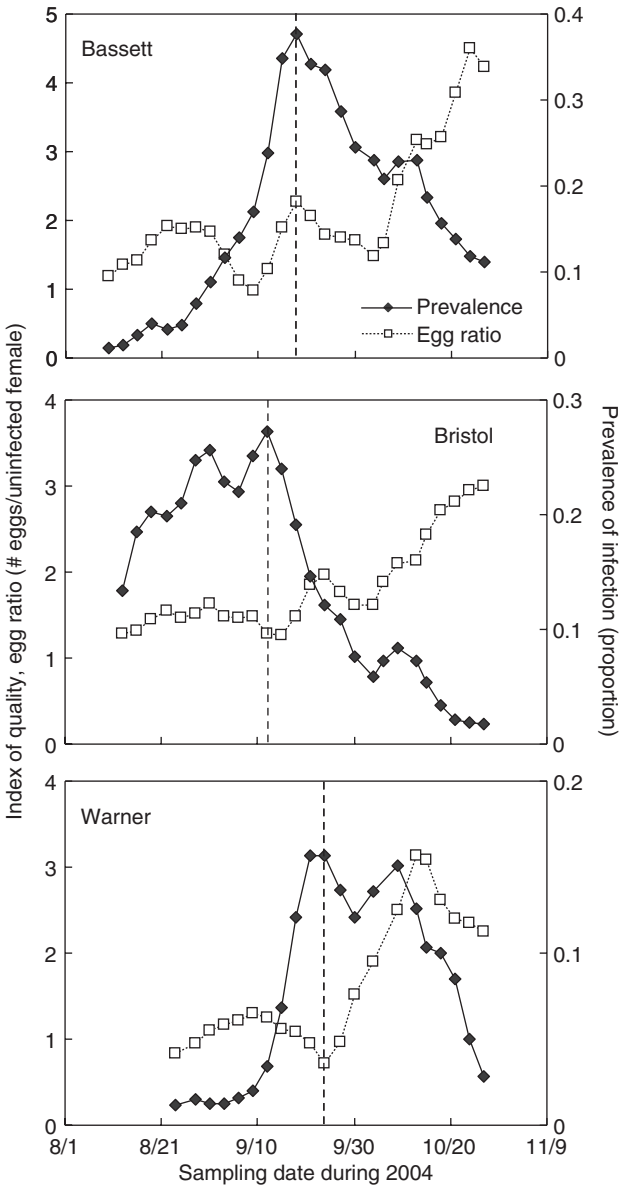

Figure 1 Dynamics of resource quality, as indexed by egg ratio of uninfected females, during the course of three epidemics of the virulent fungus *Metschnikowia bicuspidata*, sampled intensively during 2004. Epidemics are represented by prevalence (proportion) of infection in the planktonic host *Daphnia dentifera*. Dashed lines indicate peak prevalence of infection (as used in Table 1). Data were smoothed before plotting with a three-point moving average (with weights 0.25, 0.5, 0.25).

EXPERIMENTS

The laboratory experiments showed that the variation in resource quality can have pronounced effects on the key components of host–parasite dynamics. The various food resources indeed produced a very broad quality gradient ranging from very poor (deep lakes) to very good (lab-reared *Ankistrodesmus*; Fig. 2, *x*-axes). This large variation in quality did not significantly influence the time until death of infected hosts, as the mean time until death did not vary much between quality treatments (between 8 and 10 days; $R = 0.47$, $P = 0.24$; Fig. 2a). However, resource quality had pronounced effects on spore production: infected hosts that ate higher quality food grew to a larger size and produced more spores (Fig. 2b,c). Additionally, birth rate of both uninfected and infected hosts increased with quality (Fig. 2d), but, as expected, infected hosts produced fewer offspring (only *c.* 30% of those produced by uninfected hosts). Meanwhile, infection prevalence at 25 spores mL^{-1} (an index of transmission rate) declined with the resource quality (Fig. 3a). Thus, resource quality (an external, ecological factor) imposed a strong, negative relationship between spore production and transmission (Fig. 3b) and between the birth rate of uninfected hosts and transmission rate (Fig. 3c). When resource quality is poor, transmission rate is high but spore production remains poor and birth rate stays low; in contrast, with high resource quality, infected hosts produce many spores while uninfected hosts produce many babies but are harder to infect. These negative relationships provide insights in the theory developed below.

Table 1 Correlations between changes in resource quality, as indexed by egg ratios of uninfected adults, and prevalence of infection or densities of either infected or uninfected hosts.

		Post-peak		
Category	Statistic	Bassett (n = 14)	Bristol (n = 16)	Warner (n = 12)
Prevalence of infection	R	−0.73	−0.82	−0.46
	P	0.002	< 0.001	0.13
Infected host density	R	−0.48	−0.75	−0.73
	P	0.082	< 0.001	0.008
Uninfected host density	R	0.83	0.51	0
	P	< 0.001	0.007	n.s.

In all three lakes, these correlations involve portions of the epidemics following peak infection prevalence (see dotted lines in Fig. 1). Significance of Pearson correlation statistics were determined using 9999 randomizations. n.s., non-significance.

THEORY

A simple model helped to synthesize our observations of the three epidemics and the assays of resource quality, parasite virulence and transmission rate. This model tracks the change in densities of susceptible hosts (S), infected hosts (I) and free-living infective stages (spores) of the parasite (Z; see Hall *et al.* 2006 for a formal stability analysis of a version of this model and Appendix S2 for presentation and analysis of more complex variants):

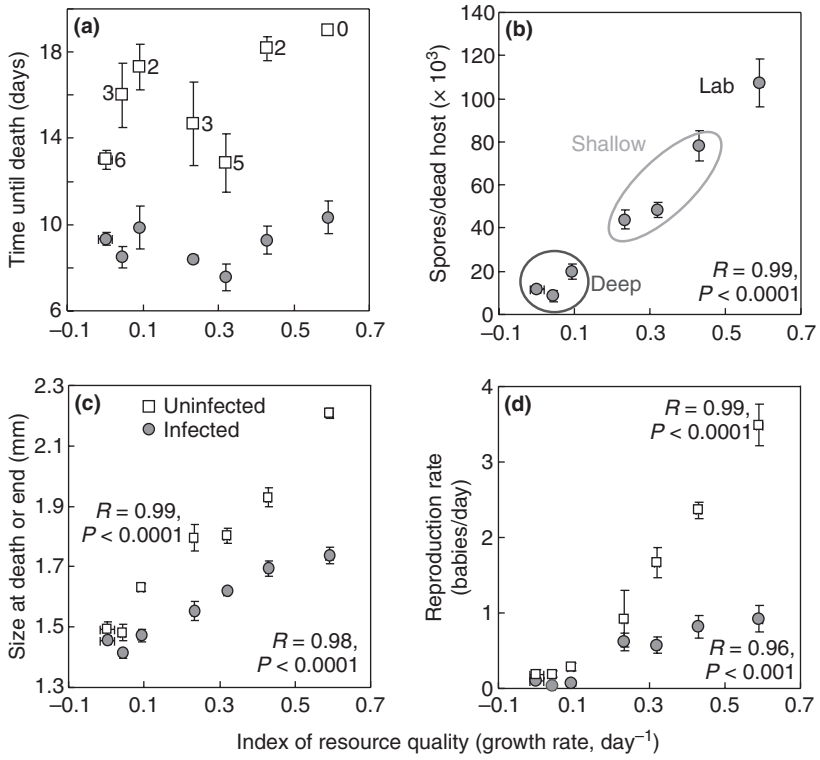

Figure 2 Results from a life table experiment in which *Daphnia* hosts were either uninfected or infected with a fungal parasite (*Metschnikowia*) and fed 0.9 mg L^{-1} of food spanning a wide gradient of quality. Resource quality (*x*-axis) was estimated using separate juvenile growth assays, and the gradient was created by using water from three deep lakes (poor quality), three shallow lakes (moderate quality) and lab-reared algae (*Ankistrodesmus*, high quality). Response variables include: (a) time until death for infected hosts and time of death or end of experiment for uninfected hosts (numbers next to points for uninfected hosts indicate number of six replicates that actually died); (b) spores contained in hosts upon death; (c) size at death or end of the experiment and (d) average production of babies per day in the two host classes. Points are means ± 1 SE.

$$\frac{dS}{dt} = b(S + \rho I)(1 - c(S + I)) - dS - \beta SZ \quad (1a)$$

$$\frac{dI}{dt} = \beta SZ - (d + v)I \quad (1b)$$

$$\frac{dZ}{dt} = \sigma(d + v)I - mZ \quad (1c)$$

Susceptible hosts (eqn 1a) increase from density-dependent births, where b is the maximal birth rate, ρ is the proportional decrease of this maximum in infected hosts, I (due to virulent effects on fecundity; $0 < \rho < 1$), and c is the strength of density-dependence. This host class dies at a background rate of d and is converted into infected hosts after contact with spores (Z) at horizontal transmission rate β (There is no vertical transmission for this parasite.). Infected hosts increase (eqn 1b) following infection of susceptible hosts but die at a rate elevated from the background mortality rate (d) due to infection (v). Once dead, each infected host releases σ spores (in eqn 1c), which are then lost at rate m (due to sinking, etc.). In this present variation of the model, we assume that the host classes do not remove spores (but see Hall *et al.* 2007a; we relax this assumption in Appendix S2).

Following a standard approach to analysing these types of epidemiological models (Anderson & May 1986), we solve for a key, component parameter, reproductive ratio, R_0. This criterion governs when the parasite can successfully invade (and persist with) a host population. Importantly, the R_0 criterion exceeds 1 (meaning that parasites can invade) when:

$$R_0 = \frac{S^*_{bnd}}{S^*_{int}} > 1, \quad (2)$$

where S^*_{bnd} is the equilibrial density of susceptible hosts without parasites (i.e. the 'boundary' equilibrium), S^*_{int} is that when persisting with parasites (the 'interior' equilibrium) and:

$$S^*_{bnd} = \frac{b - d}{bc}, \quad S^*_{int} = \frac{m}{\sigma\beta}. \quad (3)$$

Criterion R_0 increases, and consequentially the parasite becomes fitter, when the susceptible host has higher maximum birth rate (b), lower background mortality rate (d) and weaker density dependence (lower c); when the parasite more easily infects susceptible hosts (higher β), infected hosts produce more spores (higher σ), and spore loss decreases (lower m). Notice that neither virulent effects on birth rate (measured with ρ) nor death rate (via v) enter this calculation of R_0.

Based on the results from the life table and transmission assays, we see that resource quality (Q) imposes tension in this epidemiological system via its effects on transmission rate vs. other factors. We can readily illustrate this tension with a tiny bit of calculus. First, we assume that the

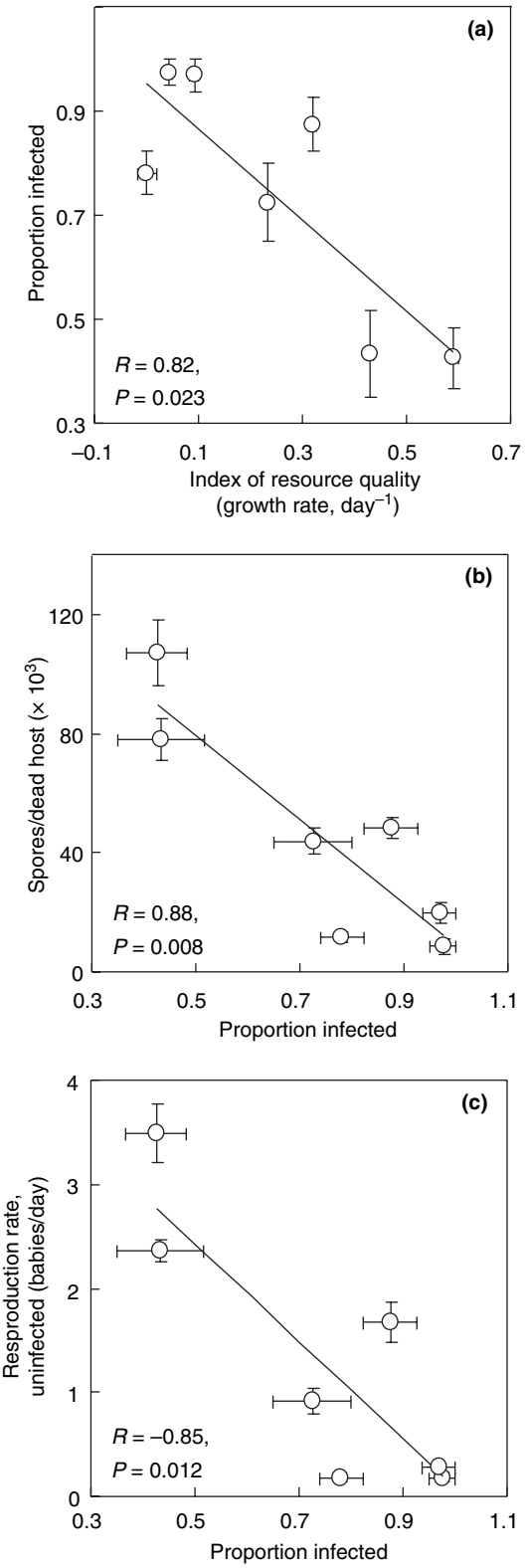

Figure 3 Relationships between transmission rate (as indexed by infection prevalence when exposed to 25 fungal spores mL^{-1}) and (a) juvenile growth rate, and (b) spore production of infected hosts and (C) birth rate of uninfected hosts in the life table experiment (Fig. 2). Variation in resource quality creates a negative relationship between transmission rate and spore production and between transmission rate and birth rate. Pearson R correlation statistics are accompanied by P-values. Points are means ± 1 SE.

transmission rate declines with quality ($\partial\beta/\partial Q < 0$; Fig. 3a) but both spore production increases with quality ($\partial\sigma/\partial Q > 0$; Fig. 2b) and maximal birth rate of uninfected hosts also elevates with quality ($\partial b/\partial Q > 0$; Fig. 2c). As background mortality (d) seems to have no relationship to quality ($\partial d/\partial Q \approx 0$; Fig. 2d), we use these pieces of experiment-derived information to delineate changes in reproductive ratio (R_0) with quality (Q). First, we see that change in R_0 with quality depends on the sum of two components:

$$\frac{\partial R_0}{\partial Q} = R_0\left[\underbrace{\frac{1}{\beta}\frac{\partial\beta}{\partial Q}}_{A} + \underbrace{\frac{1}{\sigma}\frac{\partial\sigma}{\partial Q} + \left(\frac{d}{b-d}\right)\frac{1}{b}\frac{\partial b}{\partial Q}}_{B}\right]. \quad (4)$$

Component A involves the dependence of transmission rate (β) on quality and the reciprocal of β itself. As the transmission rate drops with quality, this component is negative, and it becomes more negative as transmission rate becomes smaller (due to higher quality, etc.). Component B centres on the two factors that both increase with quality, spore production (σ) and maximal birth rate (b). This component B also declines as either σ or b increase. So, given 'competition' between these components, does R_0 elevate or decline with increases in quality? The answer depends on which component is larger. If the absolute value of component A exceeds that of B, the parasite has a more difficult time invading and persisting with the host population (because, in that case, R_0 drops with increasing Q). Therefore, increasing resource quality enjoyed by hosts during the course of epidemics (Fig. 1) could actually cause decline of epidemics – if the transmission component outweighed the spore production plus birth rate component (Fig. 4a). Furthermore, poor resource quality could inhibit the start of epidemics earlier in the season if such low quality sufficiently depressed spore production and births of susceptible hosts to counter the transmission rate elevation (i.e. the B component exceeds the A component; Fig. 4a). In this situation, an epidemic might only increase rapidly once resource quality improved sufficiently to support it (Fig. 4a). One can show that these results do not necessarily change if we more realistically allow spore removal by both host classes (Appendix S2).

Now that we have outlined in very general terms what could happen with R_0 with resource quality, we apply it to this plankton system. The details on how we converted data

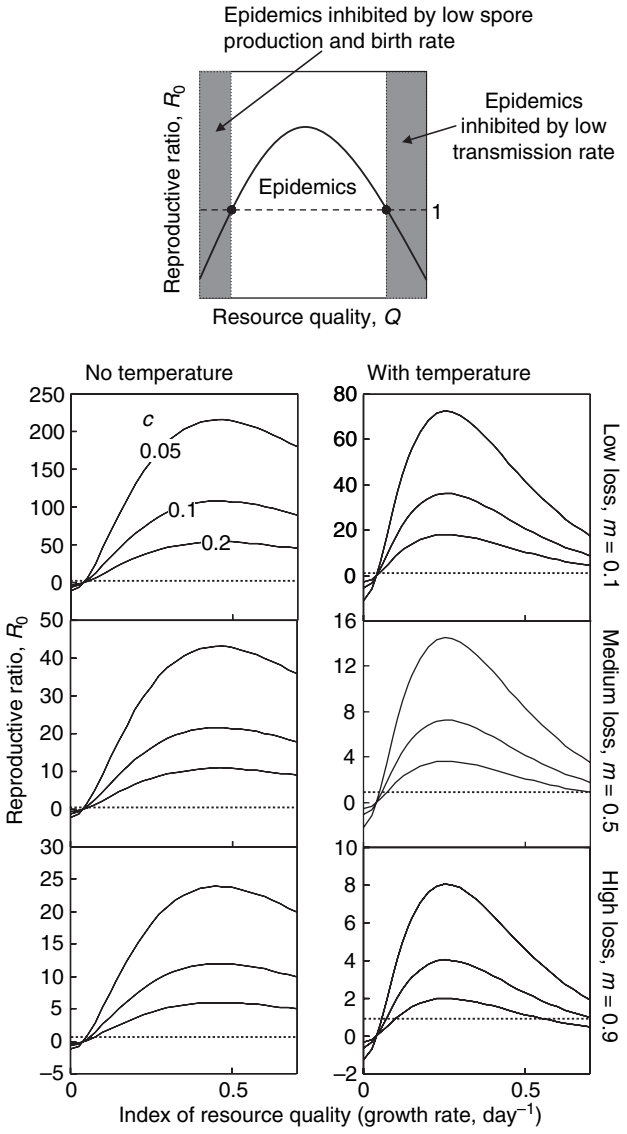

Figure 4 (a) Conceptual diagram synthesizing the hypothesized relationship between net reproductive ratio, R_0, and resource quality of hosts, Q, in the *Daphnia*–fungus–algae system. Epidemics occur when R_0 exceeds 1 (white area) and inhibited when $R_0 < 1$ (grey area). Low resource quality can inhibit epidemics because infected hosts produce few spores (low σ) and uninfected hosts produce few offspring than can become infected later (low b). These two factors outweigh the elevated transmission rate at poor quality. High resource quality, in turn, inhibits epidemics because transmission rate (β) declines. If this factor outweighs the positive link between higher quality and elevated spore production and birth rate, epidemics will be inhibited. Thus, epidemics could be constrained to intermediate quality. (b) Actual values of R_0 as parameterized by the life table experiment (for maximal birth rate, b, spore production, σ, and transmission rate, β) and using reasonable estimates or ranges for others (background mortality rate, $d = 0.08$; values of strength of density dependence, c, and loss rate of spores, m, as shown). In the left column ('No temperature'), we use data from the life table alone. In the right column, ('With temperature'), we assume a simultaneous drop in temperature with increasing quality, as we see in the field sampling data. See text for details on how temperature was added to the R_0 calculations. The horizontal dashed lines indicate where $R_0 = 1$.

from the life table and transmission assays into model parameters are provided in Appendix S3. If we assume that birth rate (b), spore production (σ) and transmission rate (β) scale with resource quality as in the experiments, we then assume a constant background mortality rate ($d = 0.08$), and we consider a range for parameters for which we do not have good estimates, i.e. density-dependence ($c = 0.05 - 0.2$) and spore loss terms ($m = 0.1$–0.9), we can parameterize our derivation of R_0 (eqns 2 and 3). With that parameterization, we see that R_0 should indeed form a hump-shaped curve with our index of resource quality (juvenile growth rate). However, the negative effects of increasing the quality on transmission alone are not strong enough to counter the positive effects of the quality on birth rate and spore production to pull R_0 back to below 1. If we take these results at face value (i.e. we strictly believe the model), we have to conclude that resource quality alone cannot terminate epidemics in this system (Fig. 4b, left column). However, there are many biological realities that are not as easily captured by our simple model. First, this conclusion assumes no stage-structure in the host population, i.e. all hosts could potentially reproduce at the maximal rate. In reality, *Daphnia* populations contain juveniles (e.g. developmental delays in reproduction), so this simplification here in the model favours the parasite by bolstering R_0 in these calculations (but fixing this problem would require a much more complicated, stage-structured model). These changes in resources also co-vary with temperature, a factor that simultaneously influences both resources and grazers. In the field data, our index of resource quality (egg ratio) correlates negatively and strongly to temperature (Appendix S1). Using that information, let us assume here that temperature declines linearly (from 20 °C to 10 °C) as quality increases (from 0 to 0.7 day^{-1}; Figs S2 and S3). Elsewhere, we have described how temperature influences birth rate (b), host mortality rate (d) and transmission rate (β) using the Arrhenius function (Hall *et al.* 2006):

$$k(T) = k_{\mathrm{R}} \exp\left[\gamma T_{\mathrm{A}}\left(\frac{1}{T_{\mathrm{R}}} - \frac{1}{T}\right)\right], \qquad (5)$$

which makes a generic physiological rate k a function of temperature T, $k(T)$. This function changes the physiological rate at a reference temperature (k_{R}, $T_{\mathrm{R}} = 20$ °C) with T as governed by the Arrhenius constant (T_{A}, which is estimated as 6400; Hall *et al.* 2006), and a proportionality

constant for the parasite (γ, estimated as 3 in Hall *et al.* 2006 for transmission rate). The $\gamma = 3$ estimate means that the transmission rate declines more steeply with temperature than does birth and death rates of the host (Hall *et al.* 2006). If we adopt the quality–temperature correlation as seen in the field and assume that temperature and quality operate independently (multiplicatively) on vital rates, these factors combined can bend R_0 to 1 or close to 1 (Fig. 4b, right column). This hump becomes especially strong if the strength of density-dependence (c) and the rate of spore loss (m) are both quite high (Fig. 4B, bottom right panel).

DISCUSSION

Our first key finding established that fungal epidemics in host *Daphnia* populations declined as algal resource quality improved. This boost in algal quality (and perhaps also quantity) likely reflected two general phenomena. The first is related to the seasonal shifts in thermal structure of lakes. During autumn, thermally stratified lakes (such as our focal study lakes) begin to cool. This cooling elicits turbulence that ultimately mixes waters and promotes the growth of more nutritious and easily digestible algae (Sommer *et al.* 1986). The second almost certainly relates to enhanced death rates of *Daphnia* grazer-hosts by this virulent parasite. *Metschnikowia* reduces fecundity and enhances mortality of its host (Ebert 2005; Hall *et al.* 2006; Duffy & Hall 2008); factors that enhance the mortality of grazer-hosts (e.g. predation or parasitism) should favour the development of less-defended, higher quality producers (Grover 1997; Tessier & Woodruff 2002a,b). This internal, mortality-resource effect seems particularly potent for algae–*Daphnia* interactions. In this system, tight feedbacks between these producers and their grazers can produce fluctuations and spatiotemporal variation that have long fascinated ecologists (McCauley & Murdoch 1987; McCauley *et al.* 1999; Tessier & Woodruff 2002a).

The experiment and model then highlight how increasing food quality later in the season connects to – and possibly even helps to cause – the waning of epidemics. The mechanism involves a quality-imposed, negative correlation between the transmission rate and two other key factors for the parasite, spore production and birth rate of hosts. In the experiment, we found that transmission rate (as indexed by infection prevalence) dropped with increasing resource quality. Similar results emerged when hosts enjoy higher quantities of high-quality algae (Ebert 2005; Hall *et al.* 2007a; but infection can drop with lower phosphorus content of algae: Frost *et al.* 2008). At the same time, birth rate of uninfected hosts and production of fungal spores both increased with enhanced quality (see also Frost *et al.* 2008). The birth rate response to high-quality resources should benefit the parasite because high birth rates provide new susceptible hosts to infect. Meanwhile, the positive quality–spore production relationship commonly arises in a variety of invertebrate host–parasite systems (see Introduction). It is likely to reflect an energy-theft mechanism – hosts enjoying higher quality resources make more energy and nutrient available for parasites to steal within the host (Hall *et al.* 2007b, in review). Resource quality/quantity, then, imposes a conflict between key tasks for the parasite. Higher quality late in the season ensures higher production of spores and susceptible hosts to infect (i.e. higher than would be expected, all else being equal, as temperatures declined: see Appendix S1), but quality itself decreases the transmission probability. This decrease in infectivity occurs in nature at a time when the transmission rate also declines steeply with cooling temperature due to thermal physiology of the parasite (Hall *et al.* 2006). We used this tight temperature–quality correlation in calculations of R_0. The net result: autumn imposes a figurative double whammy on the parasite's transmission rate. If the joint negative effect of quality and temperature on transmission rate overwhelms the positive effects of quality on birth rate (as moderated by cooling) and spore production, high resource quality can catalyse the end of epidemics.

The experiment–model combination also suggests another interesting possibility: poor resource quality could also inhibit the start of epidemics. Certainly, resource quality was low when epidemics commenced in 2004 (During this time of year and beyond, quantity is usually sufficiently high to satiate feeding rates, so quality is the major driver of growth performance: DeMott & Tessier 2002.). Unfortunately, our survey started too late to characterize the quality index before the epidemics started, so admittedly the following idea remains theoretical. If resource quality was even worse before epidemics started, the experiment and model indicated that the negative effects of poor quality on spore production and birth rate could overwhelm the positive effects of poor quality on transmission rate – and therefore very poor quality could have also inhibited epidemics. From the parasite's perspective, high infection rates during summer when resource quality remained poor and water temperatures elevated (Hall *et al.* 2006) may matter little if infected hosts produced too few spores and susceptible hosts yielded too few offspring to then continue the epidemic (especially when rates of selective predation stayed high: Duffy *et al.* 2005; Hall *et al.* 2006; Duffy & Hall 2008). Therefore, epidemics in this system may start later rather than earlier in the season because poor resource quality inhibits an earlier start. In other systems, quality-induced inhibition of disease might become even more prominent. Especially in systems involving terrestrial plants and insect herbivores, transmission of parasites may actually decline as plant quality decreases (due to interactions between defensive compounds of plants and propagules

of the parasite consumed by hosts; Keating *et al.* 1990; Hunter & Schultz 1993; Cory & Hoover 2006; de Roode *et al.* 2008). Unfortunately, quality-inhibition mechanisms might undermine the control of forest defoliators using parasites (Cory & Hoover 2006).

More broadly, links between resource quality (and quantity) and disease dynamics characterized here may offer exciting new opportunities to re-examine and expand the role of selective predators and incompetent hosts (as mentioned in the Introduction) in controlling or enhancing epidemics. For instance, selective predators that preferentially cull infected hosts can directly reduce disease; therefore, variation in predation pressure could translate into variation in disease incidence through time and space (Packer *et al.* 2003; Ostfeld and Holt 2004; Hall *et al.* 2005a). However, another pathway may emerge from this interaction. Changes in predation pressure (via host-grazer mortality rate) can indirectly alter resource abundance and quality (Tessier & Woodruff 2002a). When resources can strongly modulate host–parasite interactions, the indirect effects of predators on the host-grazer's resources could either accentuate or undermine the ability of the predator to control epidemics (depending on how changes in resource quality directly shape success of the parasite). Furthermore, hosts that remove infective propagules of a parasite from a system (like other species of *Daphnia*) could inhibit epidemics via a 'dilution effect' (Keesing *et al.* 2006; Hall *et al.* 2009). However, if these incompetent hosts also compete with hosts for resources, pathways involving resource competition could potentially undermine the dilution effect (if, for instance, high grazing pressure from diluting hosts drove resource quality poorer, thereby enhancing infectivity of parasite propagules). Clearly, the resource dimension adds complexity to selective predation, the dilution effect or other types of interactions in community ecology of disease. We hope that disease ecologists will embrace these complexities to pursue a richer and more predictive understanding of disease dynamics in wildlife populations.

This intersection between resources, hosts and parasites remains a promising yet fairly untapped frontier for disease ecology (Smith 2007). This three-partner interaction in systems such as the planktonic one illustrated here seems particularly intimate. This intimacy stems from the ability of each component of the three-part system to rapidly respond to the other components – after all, parasites, hosts and resource reproduce on fairly similar timescales. This tight interaction contrasts particularly with predator–host–parasite interactions. In those cases, predator densities are likely to respond much more slowly to host(prey)–parasite dynamics. Could rapid response of resources during epidemics produce fundamentally different infection dynamics than seen in other types of interactions in the community ecology of disease? The answer to this question remains unknown because fully dynamic models of these kinds of situations are underdeveloped (but see Lively 2006). It is hoped that this planktonic example presented here will catalyse increased empirical and theoretical evaluation of 'quality matters' mechanisms in this and other disease systems.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Appendix S1 Additional field results.

Appendix S2 Additional theoretical results.

Appendix S3 Moving from the experiments to the R_0 calculations.

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