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Poor resource quality lowers transmission potential by changing foraging behaviour

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Summary

1. Resource quality can have conflicting effects on the spread of disease. High-quality resources could hinder disease spread by promoting host immune function. Alternatively, high-quality food might enhance the spread of disease through other traits of hosts or parasites. Thus, to assess how resource quality shapes epidemics, we need to delineate mechanisms by which food quality affects key epidemiological traits.

2. Here, we disentangle effects of food quality on 'transmission potential' – a key component of parasite fitness that combines transmission rate and parasite production – using a zooplankton host and fungal parasite. We estimated the components of transmission potential (i.e. parasite encounter rate, susceptibility and yield of parasite propagules) for hosts fed a high-quality green alga and a low-quality cyanobacterium.

3. A focal experiment was designed to disentangle food quality effects on various components of transmission potential. The low-quality resource decreased transmission potential by stunting host growth and altering foraging behaviour. Hosts reared on low-quality food were smaller and had lower size-corrected feeding rates. Due to their slower grazing, they encountered fewer parasite spores in the water. Smaller hosts also had lower risk of an ingested spore causing infection (i.e. lower susceptibility) and yielded fewer parasite propagules. Hosts switched from high- to low-quality food during spore exposure also had low transmission potential – despite their large size – because the poor quality resource strongly depressed foraging.

4. A follow-up experiment investigated traits of the low-quality resource that might have driven those results. Cyanobacterial compounds that can inhibit digestive proteases of a related grazer likely did not cause the observed reductions in transmission potential.

5. Our study highlights the value of using mechanistic models to pinpoint how resource quality can change transmission potential. Overall, our results show that low-quality resources could inhibit the spread of disease through effects on multiple components of transmission potential. They also provide insight into how disease outbreaks in wildlife may respond to shifts in resource quality caused by eutrophication or climate change.

Key-words: *Daphnia*, feeding rate, food quality, parasite production, transmission rate

Introduction

Ecologists increasingly recognize that community-level interactions profoundly influence the spread of disease in natural populations (Ostfeld, Keesing & Eviner 2008; Keesing *et al.* 2010). One particularly important interaction is found between hosts and their resources. Variation in the abundance or quality of resources may shape parasitism in a diversity of systems (Hutchings, Kyriazakis & Gordon

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2001; Dwyer, Firestone & Stevens 2005; Fels 2005; de Roode *et al.* 2008). This variation might enhance or diminish the size of epidemics, depending on how resources affect traits of the host and parasite. For example, more plentiful or higher-quality food might promote the spread of disease by increasing host density (Anderson & May 1992). Resources can also affect other traits that are central to transmission (Hall *et al.* 2007; Beldomenico & Begon 2010) and propagule production (Johnson *et al.* 2007; Seppälä *et al.* 2008; Hall *et al.* 2009c), as described below. Given that key epidemiological traits vary with resources, we need a mechanistic framework to tease apart the various roles of resources in the spread of disease.

As an important step, this framework must delineate effects of food quality on transmission potential – the focus of this paper. Here, transmission potential is the product of transmission rate and yield of parasite propagules from infected hosts. Resource quality could influence components of both parts. Transmission rate is itself the product of host–parasite contact rate (exposure) and the probability of infection upon contact (susceptibility). Resources can alter exposure, particularly for the diverse array of hosts that encounter their parasites while foraging [e.g. mammals–nematodes (Hutchings, Kyriazakis & Gordon 2001), gypsy moths–viruses (Dwyer, Firestone & Stevens 2005), and butterflies–protozoans (de Roode *et al.* 2008)]. For example, better fed hosts may grow larger, which could lead to more parasite encounters if feeding rate increases with surface area (Kooijman 1986, 2010). Food quality can also affect foraging behaviour independent of body size. Hosts may compensate for poor food quality by increasing their rate of consumption (Cruz-Rivera & Hay 2003; Darchambeau & Thys 2005; Fink & von Elert 2006) or by using alternative resources that might increase parasite exposure (Hutchings, Kyriazakis & Gordon 2001; Johnson *et al.* 2009). Alternatively, undernourished animals may conserve energy by foraging less, thereby decreasing their risk of exposure (Wang, Hung & Randall 2006). Resources can also influence whether a given dose of parasites results in infection (i.e. susceptibility). This can occur through effects on host physiology (Ali *et al.* 1998; Hall *et al.* 2007) or immune function (Babin, Biard & Moret 2010; Cotter *et al.* 2011; Venesky *et al.* 2012), or through chemical compounds that directly antagonize or facilitate the parasite (Felton & Duffey 1990; Cory & Hoover 2006). However, even if high-quality food decreases transmission rate, it could still increase parasite production. For instance, better fed hosts may provide more energy and space for parasite replication (Johnson *et al.* 2007; Frost, Ebert & Smith 2008; Hall *et al.* 2009c). Thus, resource quality might pull the components of transmission potential (exposure, susceptibility and propagule yield) in opposing directions. This possibility confounds straightforward connections between resources and transmission potential – and therefore, disease spread.

In this study, we quantified links between resource quality and the components of transmission potential. To craft these links, we combined mechanistic models with experiments built around a focal planktonic host–parasite system. This system involves a zooplankton grazer (*Daphnia dentifera*), a fungal parasite (*Metschnikowia bicuspidata*), and phytoplankton resources of varying quality (Fig. 1). In lakes, *Daphnia* are confronted with a wide variety of food quality over space and time (Sterner & Hessen 1994; Tessier & Woodruff 2002; O'Neil *et al.* 2012). This variation may matter for disease because resource quality affects *Daphnia* traits including rates of ingestion, assimilation, and growth (DeMott, Zhang & Carmichael 1991; Urabe, Clasen & Sterner 1997; Ravet, Brett & Müller-Navarra 2003; Martin-Creuzburg, von Elert & Hoffmann 2008). These are key epidemiological traits because the host becomes infected by eating fungal spores (Ebert 2005; Hall *et al.* 2007), and the parasite uses assimilated within-host resources to reproduce (Hall *et al.* 2009c). Larger, faster feeding hosts encounter more parasites (Hall *et al.* 2007; Civitello *et al.* 2013a). Therefore, resource quality could alter exposure rate through effects on host size or foraging behaviour, such as size-corrected feeding rate (Darchambeau & Thys 2005). Food quality might also influence the other component of transmission rate, susceptibility, which varies with body size and other factors (Hall *et al.* 2007; Bertram *et al.* 2013). Additionally, high-quality food could enhance transmission potential by promoting host growth and production of fungal spores (Hall *et al.* 2009b,c; Duffy *et al.* 2011).

We quantified the components of transmission potential of hosts using experimental manipulations of resource

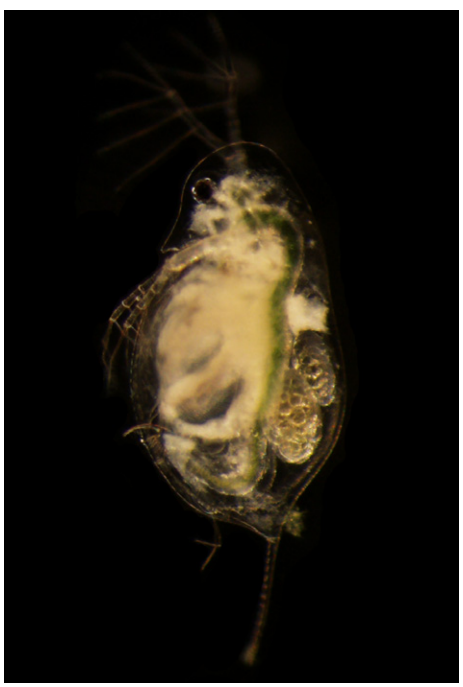

Fig. 1. *Daphnia dentifera* host infected with the fungal parasite *Metschnikowia bicuspidata*. Fungal spores appear as white material throughout the host's body cavity. Photograph credit: Isabella Oleksy.

quality. First, we paired an infection assay with a feeding rate assay to quantify the exposure and susceptibility components of transmission rate. Hosts were fed a high-quality green alga (*Ankistrodesmus falcatus*) or a low-quality cyanobacterium (*Microcystis aeruginosa*); the latter was expected to reduce somatic growth rate (von Elert, Zitt & Schwarzenberger 2012), hence size at exposure. By fitting mechanistic models to our data, we distinguished between effects of food quality on body size at the time of parasite exposure and on traits controlled for size (hereafter, 'size-corrected' traits). Any size differences between food quality treatments would cause variation in exposure. However, other size-corrected traits may depend on effects of food quality before, during, or after exposure. Thus, a third group of hosts was reared on high-quality food, but switched to low-quality food at the time of spore exposure. This 'high- to low-quality' treatment differentiated effects of food quality early in life from effects of food quality from spore exposure onward. For each treatment group, we then multiplied transmission rate and spore yield to calculate transmission potential.

A follow-up experiment tested traits of the cyanobacterium (specifically, protease inhibitors; von Elert, Zitt & Schwarzenberger 2012) that might have rendered it a low-quality food. In this second infection assay, we quantified transmission rate and spore yield for hosts fed the high-quality green alga ('control') or the green alga coated with organic compounds extracted from the cyanobacterium ('extract'). As before, a 'control-to-extract' treatment let us quantify how cyanobacterial compounds modified elements of transmission potential before and during/after parasite exposure. Thus, by designing experiments based on a mechanistic model of parasite transmission, we were able to assess the role of resource quality – and of specific resource traits – in components of transmission potential.

Materials and methods

HOST–PARASITE SYSTEM

Daphnia dentifera is a common planktonic grazer in small, stratified lakes of temperate North America (Tessier & Woodruff 2002). *Daphnia dentifera* incidentally ingests spores of the fungal parasite *Metschnikowia bicuspidata* while filter feeding (Hall *et al*. 2007, 2009a). The parasite pierces the gut wall of its host and proliferates in the haemolymph (Green 1974; Ebert 2005). As the fungus uses host resources to fuel its own reproduction, it reduces host fecundity and survivorship (Hall *et al*. 2009b,c). Upon host death, fungal spores are released that can infect new hosts (Ebert 2005). Epidemics of this parasite occur in *D. dentifera* populations in the Midwestern USA (Hall *et al*. 2011; Civitello *et al*. 2013b).

HIGH- AND LOW-QUALITY RESOURCES

We used the green alga *Ankistrodesmus falcatus* as high-quality food for hosts (Hall *et al*. 2007, 2009b; Duffy *et al*. 2011). The low-quality food was the cyanobacterium *Microcystis aeruginosa* Kützing 1846 (strain NIVA-Cya 43; Norwegian Institute for Water Research Culture Collection, Oslo, Norway; Lürling & van der Grinten 2003; von Elert, Zitt & Schwarzenberger 2012). Both are single-celled and edible to *Daphnia*. We cultured both species in 5-L glass vessels of Cyano medium (von Elert & Jüttner 1997). Cells in stationary phase were harvested by centrifugation and either immediately fed to hosts (first experiment) or frozen at −20 °C followed by lyophilization (second experiment; see Appendix S1 in Supporting information for extraction, fractionation and coating methods).

MATHEMATICAL MODEL OF TRANSMISSION POTENTIAL

We used mathematical models of the two parts of transmission potential – transmission rate and spore yield – as the framework for testing roles of food quality in disease spread (see also Table 1). The first model captures the transmission process over the short time scale relevant to our infection assay (i.e. there are no birth, death, or spore production terms). This model tracks changes in densities of susceptible (S) and infected (I) hosts, as well as free-living infective stages of the parasite (Z):

$$dS/dt = -\beta SZ \qquad \text{eqn 1.a}$$

$$dI/dt = \beta SZ \qquad \text{eqn 1.b}$$

$$dZ/dt = -f(S+I)Z \qquad \text{eqn 1.c}$$

Susceptible hosts (S) become infected (I) as they contact spores (Z), at rate β (eqn 1a,b). Spores decrease as susceptible and infected hosts consume them at rate f (eqn 1c). Infection risk (transmission rate), β, can be decomposed into its components:

$$\beta = uf = (\hat{u}L_\beta^2)(\hat{f}L_\beta^2) \qquad \text{eqn 2}$$

where u is per spore susceptibility and f is feeding (spore encounter) rate. Both u and f can be further broken down into host size at the time of exposure (length squared, L_β^2, which is proportional to surface area) and size-corrected parameters, $\hat{u}$ and $\hat{f}$. These size-corrected traits influence per spore susceptibility and feeding rate, respectively (eqn 2; Kooijman 1986; Hall *et al*. 2007). The size-dependence of f occurs because *Daphnia* feeding rate increases with surface area (Kooijman 1986). Because β increases with size more steeply than L_β^2, (Hall *et al*. 2007), we assume u also increases with body size. Biologically, an increase in u with L_β^2, may involve gut size; larger hosts have bigger guts that hold more spores and provide a larger surface through which spores can penetrate and infect the host (Hall *et al*. 2007). In the first experiment, we estimated each element of transmission rate ($\hat{u}$, $\hat{f}$, and L_β^2) using data from independent assays of feeding and transmission rate. Then, we multiplied these components to estimate β (eqn 2).

We modelled the relationship between spore yield (σ) and host size at the end of the experiment (L_e) as:

$$\sigma = \sigma_0 + \sigma_1 L_e^3 \qquad \text{eqn 3}$$

which states that σ increases linearly with host volume at the end of the experiment (L_e^3, where length cubed is proportional to volume), with slope σ_1 and intercept σ_0. Then, we defined transmission potential as the product of transmission rate and spore yield, βσ.

FIRST EXPERIMENT: INFECTION ASSAY

We used an infection assay to estimate transmission rate (β) and spore yield (σ) for hosts using three different manipulations of resource quality. We used an isofemale line of *D. dentifera* (host) and a strain of *M. bicuspidata* (parasite) both originally collected from lakes in Barry County, Michigan, USA. To minimize mater-

Table 1. Variables and parameters in the mathematical models of parasite transmission (eqn 1) and spore yield (eqn 3)

Symbol	Units	Meaning
S	Host L^{-1}	Density of susceptible hosts
I	Host L^{-1}	Density of infected hosts
Z	Spore L^{-1}	Density of spores
t	Day	Time
L_β	mm	Length of hosts at exposure to parasites
L_e	mm	Length of hosts at end of experiment
$\hat{u}$	Host spore^{-1} mm^{-2}	Size-corrected per spore susceptibility of hosts
u	Host spore^{-1}	Per spore susceptibility of hosts
$\hat{f}$	L host^{-1} day^{-1} mm^{-2}	Size-corrected feeding (exposure) rate of hosts
f	L host^{-1} day^{-1}	Feeding (exposure) rate of hosts
β	L spore^{-1} day^{-1}	Transmission rate
σ	Spore host^{-1}	Spore yield per infected host at end of experiment
σ_0	Spore host^{-1}	Intercept of spore yield model (eqn 3)
σ_1	Spore host^{-1} mm^{-3}	Slope of spore yield model (eqn 3)
$\beta\sigma$	L day^{-1} host^{-1}	Transmission potential

nal effects, *D. dentifera* were reared in groups of six in 150-mL beakers containing a 100 mL mixture of Artificial *Daphnia* Medium (ADaM; Klüttgen *et al.* 1994) and filtered water from Lake Lanier (Georgia, USA) and fed 0·73 μg C mL^{-1} day^{-1} (hereafter, 'standard' level) of high quality food. Neonate hosts born within a 24-h period were placed in groups of 10 into 150-mL beakers, fed standard levels of either high- or low-quality food, and kept at 20 °C in a 16:8 h light/dark cycle.

Six-day-old hosts were transferred singly to 50-mL beakers containing 40 mL of medium and exposed to 275 parasite spores mL^{-1} for 24 h. Hosts were fed half the standard amount of food during exposure to increase consumption of spores (Hall *et al.* 2007). On the day of exposure, half of the individuals reared on high-quality food were permanently switched to low-quality food. Thus, there were three treatments: 'high' (always fed high-quality food), 'low' (always fed low-quality food), and 'high-to-low' (initially raised on high quality food, and switched to low-quality food at exposure). We created the 'high-to-low' treatment to disentangle pre-exposure effects of food quality from those experienced during and after exposure. Hosts initially reared on high quality resources (i.e. the 'high' and 'high-to-low' groups) had similar early life experience and size at exposure (same mean L_β^2; eqn 2). Therefore, a difference in transmission rate between these two groups stemmed from food quality effects on the other, size-corrected traits during and after parasite exposure. In contrast, a difference in transmission rate between hosts in the 'low' and 'high-to-low' groups would indicate that food quality effects early in life outweighed those during and after exposure.

After spore exposure, we transferred hosts to fresh medium and resumed the standard food level. Hosts were transferred to fresh medium again 4 days later. At 10 days post-exposure to the parasite, we visually examined each individual for infection at 25–50× magnification (Duffy & Sivars-Becker 2007). Host length (L_e) was measured from the middle of the eye to the base of the tail at 40× magnification using DP2-BSW software (Olympus America, Center Valley, PA, USA). To quantify spore yield (σ), we transferred infected animals to microcentrifuge tubes, gently smashed each individual using a pestle, and counted the released spores using a haemocytometer at 200× magnification. We started the first experiment with 64 individuals (replicates) per treatment, and 36–38 individuals per treatment survived to the end (see Appendix S1 for more details).

FIRST EXPERIMENT: FEEDING RATE ASSAY

An independent assay of feeding rate enabled us to estimate the contributions of body size (L_β^4) and foraging behaviour (i.e. size-corrected feeding rate, $\hat{f}$) to transmission rate (β). On the day of spore exposure, we measured feeding rates of a subset of hosts from each food treatment (i.e. hosts reared on either high- or low-quality food). These individuals were not used in the infection assay. Hosts were placed singly in 15-mL centrifuge tubes and fed either high-quality food (n = 20 hosts from the 'high' group) or low-quality food (n = 20 hosts from the 'high' group, n = 20 hosts from the 'low' group) at concentrations used at spore exposure (0·365 μg C mL^{-1}). For both food species, we also set up ungrazed controls (n = 10), following Sarnelle & Wilson (2008). During the 3-h grazing period, tubes were inverted every 15–20 min and briefly uncapped after 1·5 h to allow air exchange. Host size (length, L_β) was measured at the end of the grazing period. We used a Trilogy fluorometer (*in vivo* module, Turner Designs, Sunnyvale, CA, USA) to quantify the food remaining in each tube.

SECOND EXPERIMENT: INFECTION ASSAY

In a follow-up experiment, we tested whether effects of food quality were caused by protease inhibitors in the low-quality cyanobacterium. To do this, we performed an infection assay similar to that in the first experiment. Prior to parasite exposure, hosts were fed the high-quality green alga coated with solvent only ('control'), or with solvent plus organic compounds extracted from the cyanobacterium ('extract'; see Appendix S1 for details). On the day of spore exposure, half of the hosts from the control group were permanently switched to food coated with extract. This 'control-to-extract' treatment allowed us to test whether effects of cyanobacterial compounds on transmission potential occurred before spore exposure, or during and after exposure. The experiment began with 68 individuals per treatment, and an average of 43 (range: 39–50) individuals per treatment survived to the end (see Appendix S1 for more details).

STATISTICAL ANALYSIS

Statistical analyses were performed in R (R Core Team 2011) and Matlab (Matlab v.7.8 R2009a; MathWorks, Natick, MA, USA). Body size of hosts on the day of parasite exposure and at the end of the experiment were analysed with one- and two-way ANOVAS, respectively. We used a generalized linear model (GLM) with binomial error distribution to analyse proportion infected among beakers. Parasite spore load per infected host was analysed using a GLM with quasipoisson error distribution (for overdispersed count data). When there were significant effects in omnibus tests, we performed *post hoc* pairwise comparisons using Tukey's honestly significant difference (HSD) tests.

Details of parameter estimation for the transmission model are provided in Appendix S1. Briefly, in the first experiment, we estimated the size-corrected components ($\hat{u}$ and $\hat{f}$) of transmission rate (β) by simultaneously fitting the transmission model (eqn 1) to data from the infection assay (i.e. body size at exposure and infection status at 10 days post-exposure), and a foraging model (eqn S4) to data from the feeding rate assay (i.e. body size and initial and final concentrations of food; Sarnelle & Wilson 2008; Bertram *et al.* 2013). Best-fit estimates of $\hat{u}$ and $\hat{f}$ were obtained by minimizing the sum of the negative log-likelihood values produced from fitting the

transmission and foraging models. In the second experiment, we did not perform a feeding rate assay, so we estimated β by fitting just the transmission model to the infection data. We estimated 95% confidence intervals around these point estimates using 10 000 bootstraps, and we bootstrapped over the infection and spore yield data to generate confidence intervals for transmission potential (βσ). In Tables S1 and S2 (Supporting information), we present the results of randomization tests for comparisons between treatments (with Holm–Bonferroni adjusted significance levels) in the first and second experiments, respectively (Gotelli & Ellison 2004).

Results

Infection risk depended on food quality (proportion infected: $\chi^2 = 33{\cdot}75$, d.f. = 2, $P < 0{\cdot}0001$, Fig. 2A; transmission rate, β: Fig. 2B). Hosts fed lower quality food during parasite exposure had lower infection risk (comparison of 'high-to-low' and 'low' groups vs. the 'high' group; Fig. 2A, B). We can understand this result with pairwise comparisons between groups. First, consider the 'high' versus 'low' groups. Hosts fed exclusively high-quality food ('high') had higher overall infection risk than those fed only low-quality food ('low') due to differences in body size (L_β; $F_{2,57} = 92{\cdot}34$, $P < 0{\cdot}0001$; Fig. 2C) and foraging behaviour (i.e. size-corrected exposure rate, $\hat{f}$, Fig. 3A). Therefore, 'high' group hosts had greater exposure to spores than 'low' group hosts (since $f = \hat{f}L_\beta^2$, Fig. 3B). Food quality did not affect size-corrected per spore susceptibility ($\hat{u}$, Fig. 3C). However, larger 'high' group hosts had greater overall per spore susceptibility (u, Fig. 3D) than 'low' group hosts due to size effects on u (i.e. since $u = \hat{u}L_\beta^2$). Now, consider the 'high-to-low' group. Hosts that were switched from high- to low-quality food at exposure had lower infection risk than those in the 'high' group (Fig. 2A,B). This result arose despite similar body size between the groups (Fig. 2C) because poor food quality depressed size-corrected exposure rate ($\hat{f}$, Fig. 3A). The negative effect of poor food quality on $\hat{f}$ was so strong that hosts in the 'high-to-low' group had the lowest spore exposure overall (Fig. 3B), despite being large. Per spore susceptibility of the 'high-to-low' group was similar to that in the other two treatments, however (Fig. 3D). Thus, low infection risk in the 'low' group was due to effects of poor quality food on growth and foraging behaviour (size-corrected feeding rate), while low infection risk in the 'high-to-low' group stemmed entirely from an effect on foraging behaviour.

The quality of food eaten early in life (i.e. before spore exposure) determined body size at the end of the experiment (L_e). Regardless of infection status, hosts in the 'high-to-low' group were just as large as those from the 'high' group; hosts in the 'low' group were significantly smaller (Food: $F_{2,106} = 291{\cdot}95$, $P < 0{\cdot}0001$; Infection: $F_{1,106} = 0{\cdot}98$, $P = 0{\cdot}33$; Food×Infection: $F_{2,106} = 0{\cdot}53$, $P = 0{\cdot}59$; Fig. 4A). Despite their larger sizes, infected animals from the 'high' and 'high-to-low' groups did not yield significantly more spores (σ) than hosts from the 'low' group [though this test had low sample size in the 'low' group ($n = 3$) due to low infection risk; $F_{2,29} = 1{\cdot}53$, $P = 0{\cdot}23$; Fig. 4B]. However, those three

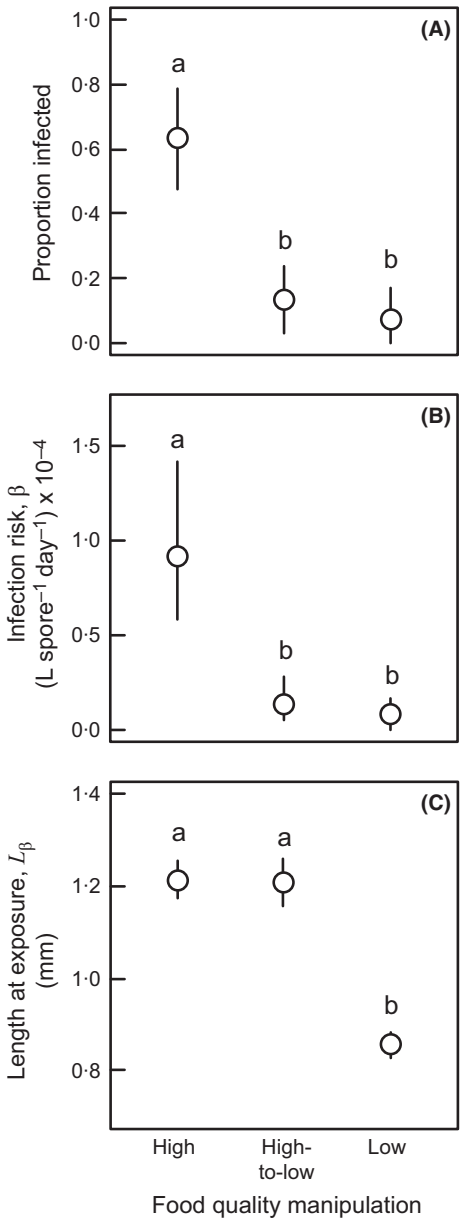

Fig. 2. Infection risk and body size at spore exposure in the first experiment. Compared to hosts fed 'high' quality food, those in the 'low' or 'high-to-low' quality treatment groups had lower infection risk, quantified as either (A) infection prevalence or (B) transmission rate (β). (C) Hosts reared on high-quality food (the 'high' and 'high-to-low' groups) were larger at exposure (length, L_β) than those reared on low quality food (the 'low' group). Error bars in all panels are bootstrapped 95% confidence intervals. Lowercase letters denote significant differences between treatments after correcting for multiple comparisons.

infected hosts from the 'low' group fell along a significant positive relationship between body size (L_e^3, proportional to volume) and spore load (σ) across food treatments ($R^2 = 0{\cdot}26$, $P = 0{\cdot}002$; Fig. 4C).

When we pulled the components together, we found that transmission potential (βσ) was greatest for hosts in the 'high' group and did not differ significantly between the other two groups (Fig. 4D). Thus, for hosts in the

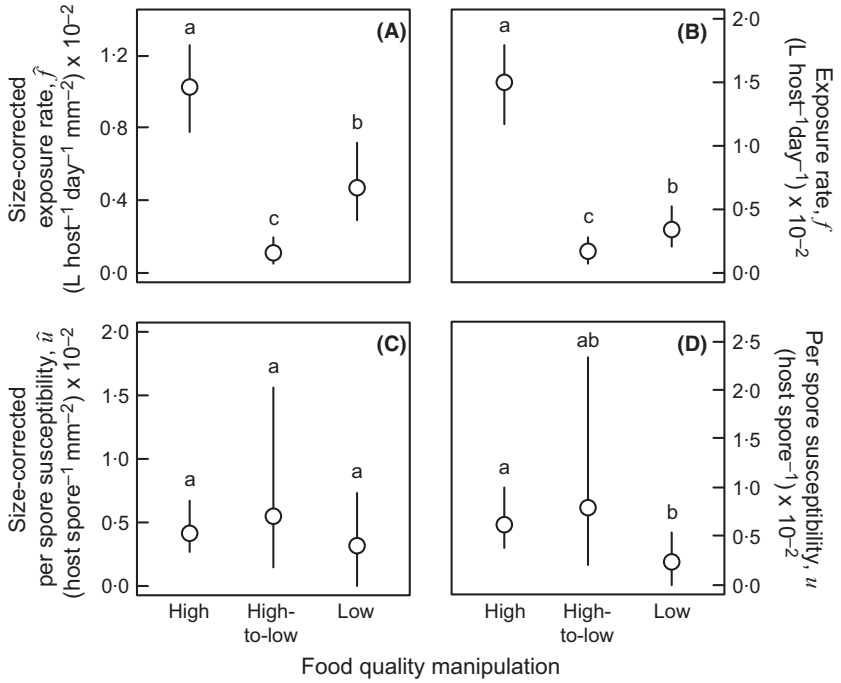

Fig. 3. Components of transmission rate in the first experiment (see eqn 2). Both (a) size-corrected feeding rate ($\hat{f}$) and (B) feeding rate (f) were highest for hosts in the 'high' group, lowest in the 'high-to-low' group, and at an intermediate level in the 'low'-quality group. (C) Food quality did not affect size-corrected per spore susceptibility ($\hat{u}$). However, (D) due to the influence of body size on susceptibility (u), hosts in the 'high' group had greater u than those in the 'low' group, while the 'high-to-low' quality group had highly variable susceptibility. Lowercase letters denote significant differences between treatments after correcting for multiple comparisons.

'high-to-low' group, the steep drop in transmission rate (β; Fig. 2B) due to foraging behaviour outweighed positive effects of their size on spore production (σ; Fig. 4C). As a result, these hosts had low overall transmission potential.

The second experiment revealed that protease inhibitors in the cyanobacterium were likely not responsible for the effects of this low quality resource. High-quality green algal cells coated with cyanobacterial extract (containing protease inhibitors; see Appendix S1 for details) did not decrease infection risk relative to the green algal 'control', regardless of whether hosts were fed the extract-coated food from exposure onward ('control-to-extract'), or throughout the experiment ('extract'; proportion infected: $\chi^2 = 2{\cdot}25$, d.f. = 2, $P = 0{\cdot}33$, Fig. 5A; transmission rate, β: Fig. 5B). Body size at exposure (L_β) did not differ between hosts from the two initial food treatments (i.e. fed control vs. extract from birth until exposure; $F_{1,23} = 1{\cdot}10$, $P = 0{\cdot}30$; Fig. 5C). Neither diet nor infection status affected size at the end of the experiment (L_e; Food: $F_{2,123} = 1{\cdot}59$, $P = 0{\cdot}21$; Infection: $F_{1,123} = 0{\cdot}48$, $P = 0{\cdot}49$; Food × Infection: $F_{2,123} = 0{\cdot}74$, $P = 0{\cdot}48$; Fig. 5D). At 10 days post-exposure, spore yield from infected hosts (σ) was similar across the three food treatments ($F_{2,44} = 0{\cdot}39$, $P = 0{\cdot}68$; Fig. 5E). Thus, overall, the cyanobacterial extract did not significantly influence transmission potential (βσ; Fig. 5F).

Discussion

Our study illustrates mechanistic connections between resource quality and components of transmission potential. In the first experiment, hosts in the 'high' group (i.e. those that always ate high quality food) had the highest rate of spore exposure because they were large and foraged quickly for their size. In addition, their large body size boosted per spore susceptibility. Thus, exposure rate and susceptibility worked together to enhance transmission rate in the 'high' group. Furthermore, larger hosts yielded more parasite propagules, as commonly seen in this system (Hall *et al.* 2009c). This combination of high transmission rate and spore yield meant that hosts in the 'high' group had the greatest transmission potential. By contrast, the 'low' group (i.e. hosts that always ate low quality food) were small and foraged slowly for their size. As a result, low exposure rate and susceptibility yielded low transmission rate for this group. Because these small hosts also yielded few spores when infected, their transmission potential was depressed further. Interestingly, transmission potential was similarly low for hosts in the 'high-to-low' group (i.e. those that were switched from high- to low-quality food at exposure). This result arose despite these hosts being as large as hosts in the 'high' group. In the 'high-to-low' group, the positive influence of body size on exposure, susceptibility and spore yield was overwhelmed by effects of poor food quality on foraging behaviour (i.e. lower size-corrected feeding/exposure rate). This quality–foraging rate effect might have diminished some as hosts acclimated to the shift in resource quality. Our results, then, probably captured the maximal effects of quality on foraging rate. Still, quality-mediated plasticity in exposure was pronounced.

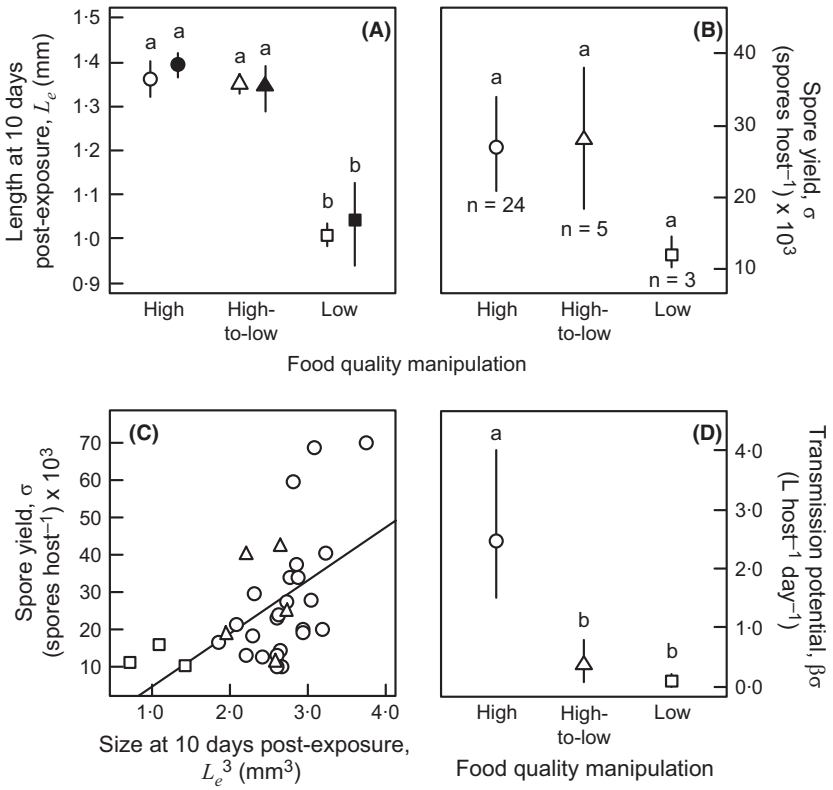

Fig. 4. Host size and spore load at the end of the first experiment (i.e. at 10 days post-exposure to spores). (A) Hosts in the 'low' group (squares) were smaller (length, L_e) than those in the 'high-to-low' (triangles) or 'high' (circles)-quality groups. Within food treatments, there was no difference in size between infected (filled symbols) and uninfected (open symbols) hosts. (B) Food quality did not significantly affect spore load within infected hosts (σ). Sample size, *n*, is indicated for each treatment. (C) Across all three treatments, infected hosts with larger bodies (volume, proportional to L_e^3) yielded more spores at 10 days post-exposure. (D) Transmission potential (βσ) was greater for hosts in the 'high' compared with 'high-to-low' or 'low'-quality groups. Lowercase letters denote significant differences between treatments after correcting for multiple comparisons.

Though our experiments did not reveal which traits of the cyanobacterium decreased host growth and feeding rate, we can rule out some features of this low-quality resource. We can dismiss inedible morphology as a driver because both food species had single, small cells. We can also eliminate phosphorus (P) deficiency, because both food species contained nonlimiting ratios of P to carbon (Sterner & Hessen 1994; Urabe, Clasen & Sterner 1997; see Appendix S1 and Table S3, Supporting information). In addition, the low-quality food lacked a common class of cyanobacterial compounds – microcystins – that can be toxic to *Daphnia* (DeMott, Zhang & Carmichael 1991; Lürling & van der Grinten 2003; Wilson, Sarnelle & Tillmanns 2006). It did contain the compounds nostopeptin BN920 (Ploutno & Carmeli 2002) and cyanopeptolin CP954 (von Elert *et al.* 2005), which can inhibit digestive proteases and stunt somatic growth of a different *Daphnia* species (von Elert, Zitt & Schwarzenberger 2012). However, our second experiment showed that these compounds likely did not underlie the results of the first experiment. Green algal cells coated with cyanobacterial extract containing these two compounds at realistic concentrations did not reduce either transmission rate or spore yield. Thus, food quality effects shown here must involve some other, unmeasured factor. Compared with green algae, cyanobacteria tend to be deficient in sterols and polyunsaturated fatty acids required for *Daphnia* growth and development (DeMott & Müller-Navarra 1997; Ravet, Brett & Müller-Navarra 2003; Martin-Creuzburg, von Elert & Hoffmann 2008). Such lipid deficiency could explain the small size of hosts in the 'low' quality treatment. By contrast, hosts in the 'high-to-low' group were not smaller than those in the 'high' group at the end of the experiment. This could indicate that a critical period of somatic growth was complete before the switch to nutritionally poor food. Hosts in the 'high-to-low' group may also have assimilated the low-quality resource more efficiently because food spent more time in their longer guts (DeMott, McKinney & Tessier 2010). However, nutritional inadequacy probably did not drive reductions in size-corrected feeding rate (Lürling & van der Grinten 2003). Thus, future studies should test other traits (e.g. surface chemicals) of this cyanobacterium that could deter or inhibit grazing by *Daphnia* (Rohrlack, Henning & Kohl 1999; Lürling & van der Grinten 2003).

How general are these effects of resources on the components of transmission potential? The positive relationship between host size and spore yield is consistent with other

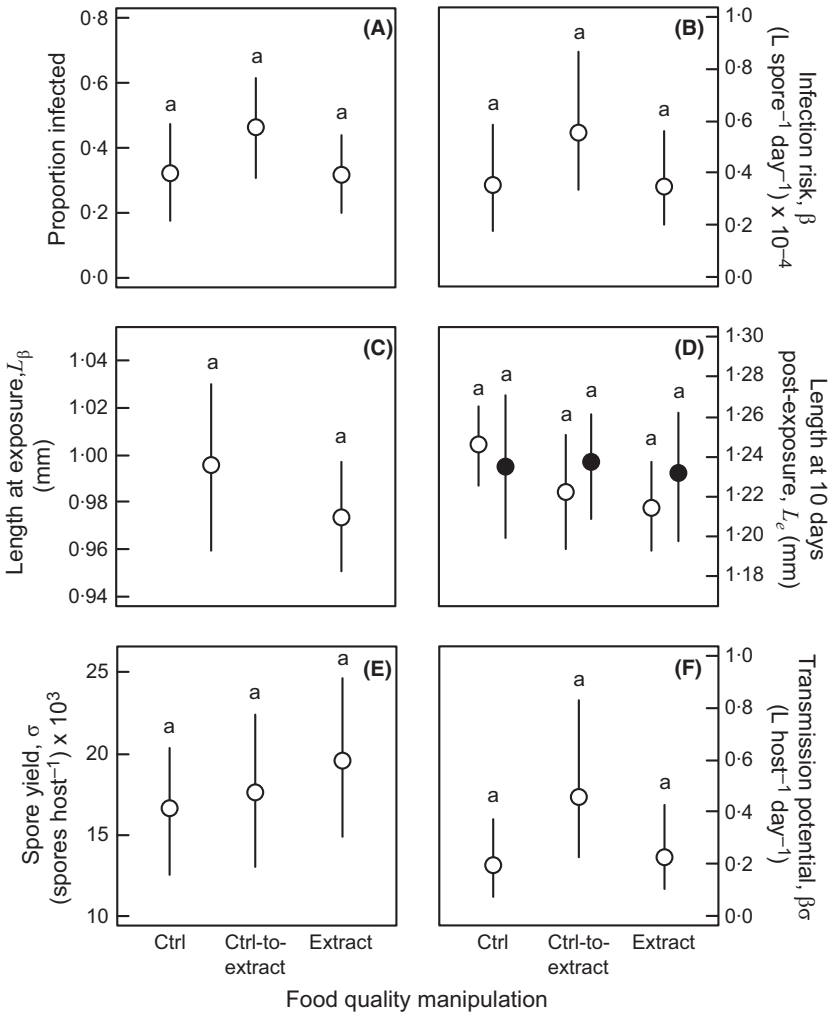

Fig. 5. Infection risk, body size and components of transmission potential in the second experiment. Whether quantified as (A) proportion infected or (B) transmission rate (β), infection risk did not differ among hosts fed the green alga coated with plain solvent [control ('ctrl')] or solvent plus cyanobacterial compounds ['extract' and control-to-extract ('ctrl-to-extract') treatments]. Food treatment did not significantly affect either (C) body size at exposure (length, L_β) or (D) size at 10 days post-exposure (length, L_e) of either infected (filled circles) or uninfected (open circles) hosts. There were also no significant differences across treatments in (E) spore yield per infected host (σ) or (F) overall transmission potential (βσ). Same lowercase letters denote no significant difference between treatments after correcting for multiple comparisons.

studies in this host–parasite system, including experimental manipulations of food quantity (Hall *et al.* 2009c) or quality (Hall *et al.* 2009b), nutrient availability (Civitello *et al.* 2013b), chemical contaminants (Civitello *et al.* 2012), or predator cues (Duffy *et al.* 2011; Bertram *et al.* 2013). The increase in parasite reproduction with host size is also consistent with studies of many other invertebrate hosts (Johnson *et al.* 2007; Seppälä *et al.* 2008; Daniels *et al.* 2013). Relationships between resources and transmission rate are more idiosyncratic, even in this *Daphnia*–fungus system. For example, transmission rate can change with food density (Hall *et al.* 2007), can increase with poor quality resources from lakes (Hall *et al.* 2009b) or certain pollutants (Civitello *et al.* 2012), and may not change in response to other nutrients (Civitello *et al.* 2013b). Thus, manipulations of resources could pull transmission rate and spore yield in opposite directions. However, in this study, low food quality depressed both parts of transmission potential.

All else being equal, these results suggest that poor resource quality could dampen epidemics in natural systems. However, to assess implications of these results for epidemics in nature, we need to consider additional factors including drivers of food quality in lakes, variation among host genotypes in use of poor quality food, and the potential for food quality to determine host density. Nutrient enrichment is a major driver of resource quality in lakes, and eutrophication may promote growth of cyanobacteria over higher quality phytoplankton (Schindler *et al.* 2008; Schindler & Vallentyne 2008; O'Neil *et al.* 2012). At the same time, nutrient enrichment may correlate with other factors that shape disease

spread, such as fish predation (Duffy & Hall 2008) or chemical contamination (Lafferty & Holt 2003; Coors & De Meester 2011; Civitello *et al*. 2012). Thus, correlated factors may influence whether poor food quality suppresses epidemics in eutrophic lakes. Additionally, host genotypes in a natural population will vary in their ability to ingest and assimilate poor quality resources (Hall *et al*. 2010, 2012). Therefore, future studies should test whether the observed effects of food quality on growth and foraging behaviour depend on host genotype. If some genotypes respond less sensitively, resource quality could have variable effects on transmission potential among lakes, or within populations over time. Finally, disease spread may also depend on how resources affect host birth rates. Poor food quality tends to reduce *Daphnia* fecundity (Lürling & van der Grinten 2003; Ravet, Brett & Müller-Navarra 2003; Hall *et al*. 2009b), and a resulting decrease in host density could work with low transmission potential to quell epidemics. Further investigation of these factors will advance our understanding of how resources shape epidemics in nature.

This study offers an approach for delineating mechanisms by which resource quality affects the spread of disease. Such an approach is valuable for two general reasons. First, it can help us anticipate how infectious disease epidemics in a key grazer will respond to climate change and eutrophication, which typically favour cyanobacteria over other phytoplankton (Schindler *et al*. 2008; Carey *et al*. 2012; O'Neil *et al*. 2012). Our results suggest that shifts to cyanobacterial dominance may inhibit transmission potential of some aquatic pathogens. Second, this mechanistic approach could disentangle roles of resource quality in disease spread in other systems. Climate and other human-driven changes are altering resource quality in aquatic and terrestrial ecosystems worldwide (Millennium Ecosystem Assessment 2005; McKenzie & Townsend 2007; Schindler *et al*. 2008; Elser *et al*. 2010). Will these changes alter disease outbreaks in other wildlife populations? To answer this question, we need to understand how resource quality influences key epidemiological traits of hosts and parasites.

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Data accessibility

Data deposited in the Dryad repository: http://doi.org/10.5061/dryad.p7507 (Penczykowski *et al*. 2014).

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Supporting Information

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

Appendix S1. Additional information on nutrient analysis, preparation of food treatments, parameter estimation for the transmission model and host survivorship.

Table S1. Treatment contrasts for components of transmission potential in the first experiment.

Table S2. Treatment contrasts for components of transmission potential in the second experiment.

Table S3. Ratios of nutrients in the two resource species.