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LETTER

Eating yourself sick: transmission of disease as a function of foraging ecology

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Abstract

Species interactions may profoundly influence disease outbreaks. However, disease ecology has only begun to integrate interactions between hosts and their food resources (foraging ecology) despite that hosts often encounter their parasites while feeding. A zooplankton–fungal system illustrated this central connection between foraging and transmission. Using experiments that varied food density for *Daphnia* hosts, density of fungal spores and body size of *Daphnia*, we produced mechanistic yet general models for disease transmission rate based on broadly applicable components of feeding biology. Best performing models could explain why prevalence of infection declined at high food density and rose sharply as host size increased (a pattern echoed in nature). In comparison, the classic mass-action model for transmission performed quite poorly. These foraging-based models should broadly apply to systems in which hosts encounter parasites while eating, and they will catalyse future integration of the roles of *Daphnia* as grazer and host.

Keywords

Akaike information criterion, *Daphnia*, foraging ecology, host–parasite, maximum likelihood, *Metschnikowia*, model competition, plant–herbivore, transmission.

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INTRODUCTION

Epidemiologists and ecologists increasingly realize that species interactions can shape disease dynamics. Other species, such as competitors and predators, can amplify or diminish the severity of epidemics in host populations (Hudson & Greenman 1998; Holt *et al.* 2003; Duffy *et al.* 2005; Hall *et al.* 2005a, 2006a), and both empirical and theoretical work continue to develop this community-ecology-of-disease perspective (Keesing *et al.* 2006). However, this perspective still has not embraced a fundamental yet critical species interaction – that between hosts and their own resources (Smith & Holt 1996; Hall *et al.* 2006b). Hosts require resources to survive, grow and reproduce, and foraging strategies form the backbone of community ecology (Tilman 1982; Grover 1997). At first, this point mainly seems to highlight the obvious – hosts require food to produce new hosts for parasites to infect. Yet, for a broad class of host–parasite systems (e.g. snails–trematodes; forest insect defoliators–viruses; grazing mammals–worms; hosts of vectors such as ticks), hosts can become infected by encountering free-living parasites or vectors while searching for and/or consuming food (Williams & Barker 2001; Fenton *et al.* 2002; Fels 2005; Wobeser 2006). Therefore, for these types of systems, transmission of disease should fundamentally connect with foraging ecology. Furthermore, resources of hosts often fluctuate (McCauley & Murdoch 1987; Altizer *et al.* 2006; Jolles *et al.* 2006). If transmission depends upon foraging biology, fluctuations in resources may create pronounced – yet predictable – variability in transmission of these types of parasites to their hosts.

However, disease ecology currently offers surprisingly few models to link foraging with transmission of parasites (but see Ferrari *et al.* 2006). Typical models of transmission of free-living parasites assume that hosts become infected at some constant rate proportional to random contact with free-living stages (i.e. the linear mass-action model of McCallum *et al.* 2001) and sometimes include nonlinear, density-dependent terms (Hochberg 1991; reviewed by McCallum *et al.* 2001; Fenton *et al.* 2002). Certainly, much

progress has been made using the mass-action model and its phenomenological variants, and it offers a suitable starting point for epidemiological studies (Anderson & May 1981, 1991). However, in this study, we show the relatively poor ability of the mass-action model to predict infection of a common zooplankton species, *Daphnia dentifera* Forbes, by its fungal parasite, *Metschnikowia bicuspidata* (Metschnikov) Kamenski, given variation in food density and host size. Failures like this one accentuate some missing yet fundamental component of the host–parasite interaction (as seen in similar studies: D'Amico *et al.* 1996; Knell *et al.* 1996; Begon *et al.* 1999; but see Regoes *et al.* 2003). For our system, alternative models that incorporated elementary aspects of foraging ecology performed remarkably better.

The *Daphnia*–phytoplankton parasite system provides an excellent opportunity for building links between epidemiology and foraging ecology. *Daphnia* hosts numerous parasites (such as our focal fungus *Metschnikowia*) that produce free-living stages (spores). Infection by these parasites occurs when the grazer-host inadvertently consumes spores suspended with phytoplankton food (Ebert *et al.* 2000; Ebert 2005; Fels 2005). Additionally, in the laboratory, variation in food density and body size alters transmission of several parasites of *Daphnia* (Pulkkinen & Ebert 2004; Ebert 2005; Fels 2005). As the feeding rate of *Daphnia* also depends upon food density and body size (Kooijman 1993), this previous work suggests strong links between foraging and infection. Furthermore, these potential links to foraging are relevant in nature, where the signature of body size-dependent transmission appears during epidemics of *Metschnikowia* in lake populations of *D. dentifera* (Hall *et al.* 2005b; Cáceres *et al.* 2006; see Appendix S1 in Supplementary material). While it could be caused by multiple factors in nature, this field evidence remains consistent with mechanism-based hypotheses that feeding biology links with transmission rate.

Therefore, integration of basic foraging ecology with epidemiology should produce powerful models for transmission rate in relevant systems. We illustrate this potential with the *Daphnia*–fungus–phytoplankton system. Given suboptimal performance of the mass-action model, we considered fundamental aspects of foraging ecology: *feeding rate*; *clearance rate* or the amount of habitat per unit time from which food – and spores – are removed by foraging; *gut residence time*, or time that food spends inside a gut; and *gut size* (Kooijman 1993; Grover 1997). Using various combinations of these foraging components, we then constructed several different *a priori* hypotheses (models) for transmission rate and compared their performance statistically (Hilborn & Mangel 1997; Burnham & Anderson 2002) with data from two experiments. These experiments used variation in body size of the host and densities of food and spores of the parasite as treatments. From this model–data interface, we confirmed an intimate connection between foraging and transmission that should generally apply to other systems in which hosts inadvertently eat their parasites.

METHODS

Laboratory experiments

We conducted Spore Density and Food Density experiments which followed the same general procedures. Each one used a single clone of the host, *D. dentifera*, a strain of fungal spores isolated from Baker Lake (Barry County, MI, USA) during September 2003 and farmed *in vivo*, and chemostat-grown algae (*Ankistrodesmus falcatus*). Prior to the start of each experiment, neonate *Daphnia* were collected over a 24-h period on different days. This procedure produced different age-size classes of hosts. Five (Spore Density experiment) or six (Food Density experiment) randomly selected *Daphnia* within a size class were placed in 150-mL beakers filled with 100-mL filtered (1 μm) lake water. Filtering removed any spores present; tiny amounts of picoplankton and bacteria (< 1 μm) were distributed evenly among treatments. Initiation of the experiment followed creation of food or spore treatments. Animals were exposed to fungal spores (and variation in food levels in the Food Density experiment) for 20–24 h while incubated at 20 °C. After this incubation period, hosts from both experiments were moved to fresh water containing no spores and daily addition of high levels of algal food (1.5 mg dry weight L^{-1}). *Daphnia* were maintained in these conditions (with one change of water) for 10 days, at which point each host was diagnosed for infection. Infection becomes apparent sooner at 20 °C, but the 10-day interval yielded unequivocal results as opaque spores packed ordinarily translucent bodies of *Daphnia*. We discounted animals that died before diagnosis (which never exceeded one per beaker). With remaining animals, we calculated prevalence as the number of infected animals divided by infected plus uninfected animals. Thus, prevalence varied between zero and one.

The main differences between the two experiments involved variation in food or spore densities during the 20- to 24-h incubation period. In the Food Density experiment, animals were exposed to moderate levels of fungus (2×10^5 spores L^{-1}) but were initially fed four different food levels (0.25, 0.5, 1.0 and 2.0 mg dry L^{-1}, determined spectrophotometrically using absorbance–dry mass regressions). These food levels span gradients seen in our study lakes (Tessier & Woodruff 2002) but still promoted reproduction of the host. Body size of animals at the time of exposure to parasites ranged over four size classes, mean (standard deviation): small: 0.98 mm (0.083 mm); medium-small: 1.24 (0.085); medium-large:

1.46 (0.092); and large: 1.64 (0.066). In the Spore Density experiment, animals received the same density of algae (1.5 mg dry L^{-1}) but were exposed to four levels of spores (0.875, 1.75, 3.5 and 7×10^5 spores L^{-1}). (This second experiment resembles the design of Regoes *et al.* 2003, except here we considered variation in body size but had fewer levels of the spore treatment.) The four size classes differed slightly from the other experiment, mean (standard deviation): small: 1.09 mm (0.083 mm); medium-small: 1.20 (0.037); medium-large: 1.33 (0.077); and large: 1.57 (0.095). Both experiments contained 16 treatments replicated eight times, yielding 128 experimental units.

Model building and fitting

The null model and general approach

Before constructing foraging-dependent models for transmission rate, we must describe the null model (H0). It assumed that transmission rate (TR) of the parasite occurred with a constant infectivity rate (β) as susceptible hosts (S) randomly contacted spores of the parasite (Z) and most closely resembled McCallum *et al.*'s (2001) 'mass action'. Following this null model, we represented decrease in density of susceptible hosts (and subsequent increase in density of infected hosts, I) through time due to infection using differential equations:

$$dS/dt = -\mathrm{TR} \times S \times Z \quad (1a)$$

$$dI/dt = \mathrm{TR} \times S \times Z \quad (1b)$$

assuming that density of spores did not decrease through time (i.e. $dZ/dt = 0$). This latter assumption could be changed by adding loss terms to a dynamic spore equation (making it more analogous to Anderson & May's (1981) model G), but such a change would not alter our conclusions. One then estimates infectivity parameter β by statistically comparing data on prevalence of infection to that predicted by the model, $p_I(t)$, where

$$p_I(t) = \frac{I(t)}{S(t) + I(t)} \quad (2)$$

and where $I(t)$ and $S(t)$ are densities of the two classes of hosts after exposure to the parasite for t units of time. For simple models of transmission (e.g. eqn 1), one can calculate $p_I(t)$ by analytically solving the differential equations (Fenton *et al.* 2002). For more complicated ones (e.g. those below), one predicts $p_I(t)$ by numerically integrating the differential equations for the duration of experimental exposure to spores.

Construction of models for transmission based on foraging

To provide alternatives to the mass-action model, we developed hypotheses (models) to express transmission rate (TR) as functions of food consumption and body size. We did not necessarily know *a priori* how to combine – and exclude – various aspects of foraging biology, so we compared seemingly logical variants. The first key component of foraging is feeding rate. As in many species, feeding rate of *Daphnia* increases with food density but then slows as density becomes high (Fig. 1a, Table 1, eqn 3). Yet, uncertainty still surrounds which functional response best applies to *Daphnia*. Some data and many models assume that this host feeds according to the classic Type II functional response (Kooijman 1993; Scheffer *et al.* 2000), but much evidence also supports a Type I (Jeschke *et al.* 2004) and even a Type III formulation (Sarnelle 2003). However, maximal feeding rate increases with surface area (length^2) of animals such as *Daphnia* (Fig. 1b, Table 1, eqn 4; Kooijman 1993, his Fig. 3.11 for *Daphnia*). Thus, feeding rate varies with food density and body size of the host.

It might seem that contact rate of hosts with fungal spores, and hence transmission rate, should increase with feeding rate of *Daphnia*. This assumption would forecast higher prevalence of infection at higher food densities (results not shown), a prediction that our data clearly contradicted (see Results). Yet, two feeding-related processes could explain decreasing infection with food density. First, *Daphnia* likely contact spores relative to the rate at which they clear habitat of food, not at which they eat food. Clearance rate equals feeding rate divided by food density (Grover 1997; Table 1, eqn 5) and decreases as food levels rise (Fig. 1c). This assumption implies that spores occur too sparsely to affect feeding rate of the host (which is very reasonable in natural settings). Additionally, food (and spores) spend more time in *Daphnia* guts when food density is low (Fig. 1d). Following Kooijman (1993), we assume that gut residence time equals gut volume divided by feeding rate (Table 1, eqn 6; see Kooijman's Fig. 3.10 for support with *Daphnia*). (Note that this gut residence time model can be modified for species that empty their gut at low food, e.g. copepods, mammals and carnivorous fish; furthermore, gut residence time can remain independent of body volume for species with fixed diameters, e.g. worms; Kooijman 1993). For daphniids at least, longer 'gut residence time' could elevate success of ingested spores that must pierce through the host gut. Furthermore, gut size (length, surface area and volume) increases as body size increases (Fig. 1e, Table 1, eqn 5; again, see Kooijman's Fig. 3.10 for isomorphic *Daphnia*; a similar phenomenon occurs in mammals). Larger guts should hold more spores, and hence increase probability of infection.

A priori, general feeding biology suggested that several factors – clearance rate, gut residence time and gut size – might influence transmission of the fungal parasite. All of these factors depend upon body size of the host, and the first two decrease with higher food density. We constructed

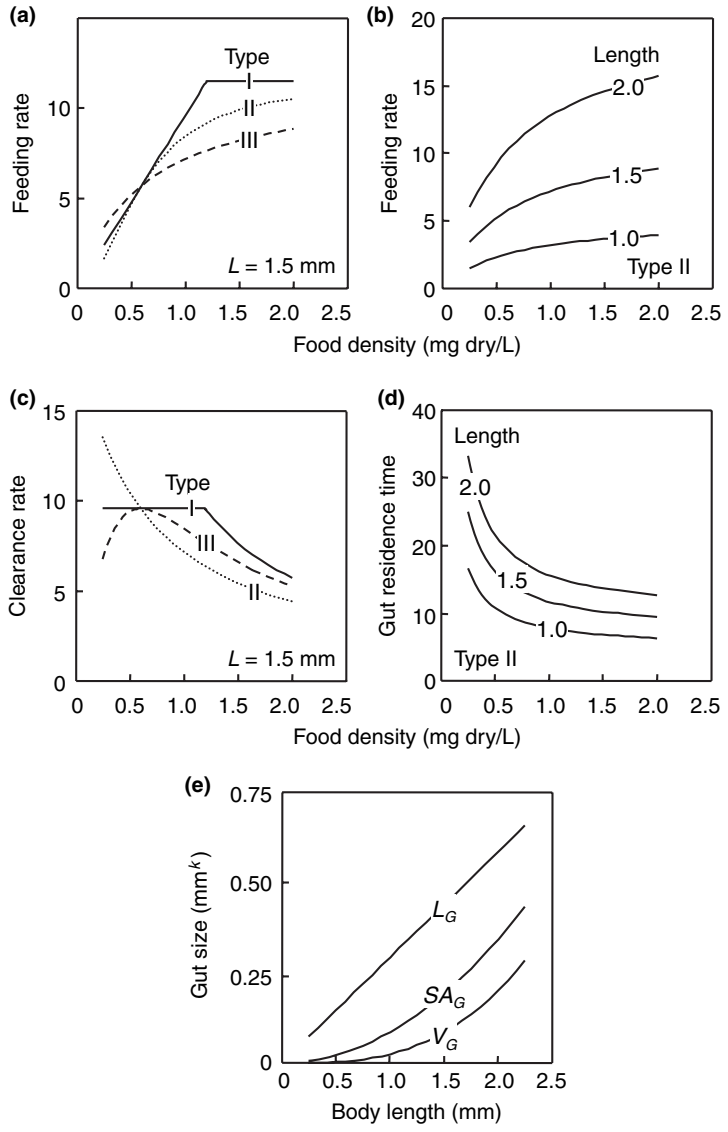

Figure 1 Components of the competing hypotheses for food- and size-dependent transmission of a fungal parasite (*Metschnikowia bicuspidata*) to a zooplankton host (*Daphnia dentifera*). (a) Classic Type I, II and III functional responses parameterized for *Daphnia* show various ways in which feeding rate (FR) increases but slows (gradually or abruptly) as food density, A, rises. (b) These functional responses are size-dependent because maximal feeding increases proportionately to surface area (L^2) of the host. (c) Clearance rate, CR, or the amount of habitat with which the animal interacts while feeding, equals feeding rate divided by food volume (FR/A) and decreases with food density. We assume that *Daphnia* contact fungal spores proportionately to CR. (d) Similarly, gut residence time, which equals gut volume divided by feeding rate (V_G/FR) also decreases with food density. (e) The size of the gut of the animal increases proportionally with the length (L) raised to a power: $k = 1$, 2 or 3 for length (L_G), surface area (SA_G), and volume (V_G) respectively.

several reasonable hypotheses combining these factors (Table 1b). All hypotheses included clearance rate, as hosts must clear spores to contact them. The first (H1) assumed that transmission is only proportional to clearance rate, which itself has three forms (a–c) based on the three functional responses (I–III). The second through fourth hypotheses (H2–H4) assumed that transmission increases with the product of clearance rate and gut size (length, surface area and volume respectively). These hypotheses accentuated a role for body size beyond that contained in H1 due to the capacity of the gut to contain spores. Each of these hypotheses can also be derived for three functional responses, so each has three versions (a–c). The fifth hypothesis incorporated gut residence time. However, this hypothesis simplified to gut volume divided by food density (as the feeding rate components of clearance rate and gut residence time cancel each other; see Table 1a, eqn 5 and 6). Similarly, the sixth through eighth hypotheses, which assume transmission increases with the product of clearance rate, gut residence time, and gut size, simplified to gut volume divided by food density times gut size (see Table 1b). We parameterized these functions with reasonable literature-based values and our own data (Table 1).

Linking the full model to data

Despite now having numerous, testable hypotheses for transmission rate (TR), we must first incorporate several other processes to the model before linking it to data. Susceptible hosts become infected hosts as they contact spores (Table 1c, eqns 8a,b; eqn 1 for the null model). Spores themselves are removed with some efficiency (here, assuming $\varepsilon = 1$) from water by both susceptible and

Table 1 Construction of hypotheses for foraging-based models of transmission of a fungal parasite (*Metschnikowia bicuspidata*) to a zooplankton host-grazer (*Daphnia dentifera*)

(A) Components of the transmission rate models

Feeding rate	Type I	Type I	Type III
	$FR_1 = \min(fA/(2k), f)$ [eqn 3a]	$FR_2 = fA/(k+A)$ [eqn 3b]	$FR_3 = fA^2/(k^2+A^2)$ [eqn 3c]
Other components	SA-dependent feeding rate	Clearance rate	Gut residence time
	$f = \hat{f}L^2$ [eqn 4]	$CR_j = FR_j/A$ [eqn 5]	$T_{G,j} = V_G/FR_j$ [eqn 6]
Gut size	Gut length	Gut surface area	Gut volume
	$L_G = g_L L$ [eqn 7a]	$SA_G = g_{SA}L^2$ [eqn 7b]	$V_G = g_V L^3$ [eqn 7c]

(B) Hypotheses for transmission rate

Hypothesis	Expression	Hypothesis	Expression
1a,b,c	$\varepsilon \times \beta \times CR_j$	5	$\varepsilon \times \beta \times CR_j \times T_{G,j} = \varepsilon \times \beta \times V_G/A$
2a,b,c	$\varepsilon \times \beta \times CR_j \times L_G$	6	$\varepsilon \times \beta \times CR_j \times T_{G,j} \times L_G = \varepsilon \times \beta \times (V_G/A) \times L_G$
3a,b,c	$\varepsilon \times \beta \times CR_j \times SA_G$	7	$\varepsilon \times \beta \times CR_j \times T_{G,j} \times SA_G = \varepsilon \times \beta \times (V_G/A) \times SA_G$
4a,b,c	$\varepsilon \times \beta \times CR_j \times V_G$	8	$\varepsilon \times \beta \times CR_j \times T_{G,j} \times V_G = \varepsilon \times \beta \times (V_G/A) \times V_G$

(C) Full ordinary differential equation model

Variable for	Equation
Susceptible hosts, S	$dS/dt = -TR \times Z \times S$ [eqn 8a]
Infected hosts, I	$dI/dt = TR \times Z \times S$ [eqn 8b]
Spores, Z	$dZ/dt = -\varepsilon \times CR \times Z \times (S+I)$ [eqn 8c]
Algal food, A	$dA/dt = -FR \times (S+I)$ [eqn 8d]

(D) Variables, parameters and synthetic quantities

Quantity	Unit	Interpretation	Value
A	mg dry L^{-1}	Algal food, density	–
I	hosts L^{-1}	Infected host, density	–
S	hosts L^{-1}	Susceptible host, density	–
t	days	Time	–
Z	spores L^{-1}	Spores, density	–
$\hat{f}$	mg dry $host^{-1}$ day^{-1} mm^{-2}	Surface-specific feeding rate	5.1×10^{-3}*
g_L	–	Ratio of lengths, gut : body	1.09‡
g_{SA}	–	Ratio of surface areas, gut : body	0.10‡
g_V	–	Ratio of volumes, gut : body	7.53×10^{-4}‡
K	mg dry L^{-1}	Half-saturation constant, host	0.6‡
L	mm	Length	1.0–2.0
β	See Table 2§	Infectivity of spores	See Table 2
ε	–	Efficiency of spore capture	1
CR_j	L $host^{-1}$ day^{-1}	Clearance rate for j = I,II,III	–
f	mg dry $host^{-1}$ day^{-1}	Maximal feeding rate ($\hat{f}L^2$)	$5.1–20.4 \times 10^{-3}$
FR_j	mg dry $host^{-1}$ day^{-1}	Feeding rate/functional response	–
L_G	mm	Length of gut ($g_L L$)	0.3–0.6
SA_G	mm^2	Surface area of gut ($g_{SA}L^2$)	0.08–0.17
$T_{G,j}$	mm^3 host day (mg dry)$^{-1}$	Residence time of gut	–
TR	L $spore^{-1}$ day^{-1}	Transmission rate	–
V_G	mm^3	Volume of gut ($g_V L^3$)	0.025–0.05

Subscript j corresponds to type of functional response.
*Mourelatos & Lacroix (1990).
‡A.J. Tessier, C. Becker, and W. R. DeMott, unpublished data.
§Scheffer *et al.* 2000.

infected hosts proportional to clearance rate (Table 1c, eqn 8c). This assumption differs from Anderson & May's (1981) Model G, where only susceptible hosts clear parasites. Algal food is consumed by both classes of hosts following the hosts' feeding rate, but algae do not reproduce (Table 1c, eqn 8d). Thus, food levels decrease with time in the model. Such a decrease alters contact between hosts and spores.

To link these models with data, we found values of the infectivity rate parameter (β) that best predicted prevalence of infection observed in all of the data from each of our two laboratory experiments. To predict infection prevalence, $p_I(t)$ for a given β, we integrated the model for the duration of exposure to spores (as explained above in *Model building and fitting: the null model and general approach*; eqn 2). We added stochasticity to these predictions by assuming that infection most naturally followed the binomial distribution (where model-generated $p_I(t)$ and experiment-generated data for I and S provided the required inputs; thus, the model could account for different numbers of individuals between the experiments). This stochastic version of the model offered a testable statistical hypothesis and suggested a likelihood function (the binomial distribution) that can be optimized (Hilborn & Mangel 1997). More specifically, we searched for the value of the infectivity parameter (β), using Matlab's downhill simplex algorithm (MathWorks 1999), which minimized the negative log-likelihood of the data, given the model and literature- and laboratory-based parameters. We profiled 95% confidence intervals surrounding infectivity parameters of the best models (following Hilborn & Mangel 1997).

Finally, once each model was fit, we needed to compare and rank their relative performance. We used techniques based on Akaike information criterion (AIC; Burnham & Anderson 2002), where the best performing model had the lowest AIC value. The first AIC-based statistic involved relative AIC differences ($\Delta_j = \mathrm{AIC}_j - \mathrm{AIC}_{min}$), where higher Δ_j indicates worse performance of the model, the best performing model has an AIC difference of zero, and differences greater than 20 conservatively indicate poor performance of the model (Burnham & Anderson 2002). The second statistic is Akaike weight, w_j (see Burnham & Anderson 2002, p. 75). This statistic gives the relative weight of evidence in favour of a model j, given other models. Similar to a probability, higher w_j (i.e. closer to one) indicates stronger support for the model being considered. (Readers can consult Burnham & Anderson 2002 and Johnson & Omland 2004 to learn more about advantages of model selection based on information-theoretic approaches).

RESULTS

The two experiments highlighted the importance of body size but also food density (Food Density experiment) and spore density (Spore Density experiment) for infection of *Daphnia* with its fungal parasite. In general, the food experiment signalled that prevalence of infection decreased as algal food became more dense, particularly at high levels (2.0 mg dry/L; Fig. 2a). Further, infection increased as size of host animals increased. In particular, the jump from small to medium-small size classes yielded much higher prevalence. Differences among larger sizes became less notable as already high infection prevalence could not increase much further. At the largest size class, prevalence reached levels that approached 100% infection, regardless of food density. In the experiment that varied spore concentration, infection did increase with spores (as expected; Fig. 1f) but also body size (Fig. 2a). Notably, the null model (where infection occurs at a constant per capita rate) could not capture dependence of infection prevalence on body size and food density in the experiments (Fig. 2a). It performed terribly – the worst of the competing models (i.e. it had very high AIC, highest Δ_j, and tiny w_j).

Several models based on feeding biology vastly outperformed the mass-action model. Actually, the Food Density experiment permitted clear statistical differentiation among these competing hypotheses (Table 2), but the Spore Density data yielded virtual statistical ties among several of the candidate models (Table S1 in Appendix S2). This latter result emerged because food density did not vary among treatments, so clearance of spores could not vary with food levels. Stated alternatively, without food-dependent variation in clearance rate, the Spore Density experiment yielded insufficient information to differentiate among the top models. Therefore, variation in body size mattered most for this experiment, and many models were sufficiently flexible to capture the body size signal that emerged.

However, given variation in food density, and hence food-dependent clearance rate, transmission models build on Type I-based clearance rate and gut size (surface area or length, Hypotheses 3A and 2A) performed best (i.e. lowest AIC values, etc.; Fig. 2a, Table 2). These models predicted flat response of prevalence until food density reached levels of food saturation for the host (Fig. 1c). After this saturation level was surpassed, clearance rate declined with the inverse of food density (Table 1). This decline with clearance rate yielded little drop in infection prevalence for the largest size class; given spores levels in the experiment, these large animals already consumed too many spores for drops in clearance rate to matter much. The third and fourth ranked models (Hypotheses 3C and 2C respectively) performed worse (Table 2) when fit to the Food Gradient experiment, given literature-based estimates of the feeding rate ($\hat{f}$) and half-saturation (K) parameters. These models, constructed with a Type III-based clearance rate, contained surface area or length of the host's gut. Both Type III-based models captured several features of the Food Gradient data

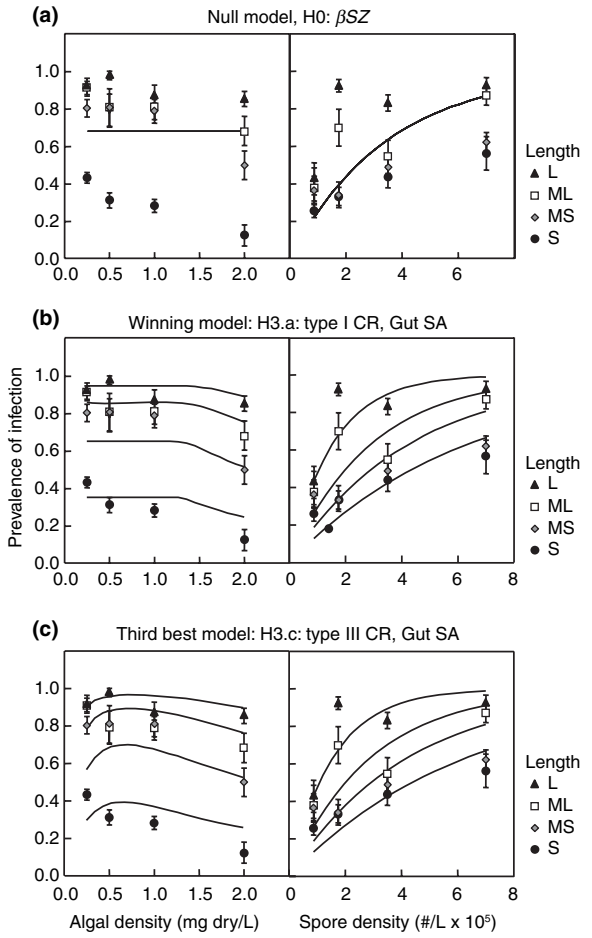

Figure 2 The two top performing models for size- and food-dependent transmission of the fungus to the *Daphnia* host (see Table 2 and Table S1 in Appendix S2 for results of model competition) and a null hypothesis for transmission rate. (a) In the null model case, transmission occurs at the constant rate β. While this model correctly anticipates increasing infection prevalence with initial spore density, it cannot predict the size- and food-dependent signals apparent in the laboratory experiments. (b) and (c) In the superior models, transmission rate (TR) is proportional to clearance rate (CR_J) of spores times surface area of the host's gut (SA_G), following Hypothesis 3 (see Table 1). (b) The top performing model has clearance rate based on a Type I functional response; the second best model (also with Type I clearance) behaves very similarly, so it is not shown; and (c) the third best model (shown) uses CR based on a Type III functional response. (Note that relative rankings among these top three models can change; see Appendix S2). These two models performed best relative to competing models using data from the Food Gradient experiment, but they produced virtually identical results to each other and several others using data from the Spore Gradient experiment. Body size labels correspond to large, medium large, medium small and small categories from each experiment (see Laboratory experiments for actual sizes). Lines are predictions of the model calculated with best-fit parameters; points are mean ± 1 SE.

set, but also predicted increasing, then decreasing prevalence of infection. Note, however, that the relative ranking of these four top models changed if we simultaneously estimated β and K but held $\hat{f}$ constant (Table S2 in Appendix S2). In this latter case, models containing surface area of the gut and Type I or III-based clearance rate behaved equivalently; models based on gut length performed less well. Despite all of these details, a key point emerged: transmission models that included clearance rate (Type I or III) and gut size (especially surface area) performed consistently better than other foraging-based models – and especially the null (mass action) model.

What do the best-fitting models tell us about transmission rate, body size and food density? First, transmission rate depends sensitively upon body size. At a given food level, low spore densities (e.g. the 0.5×10^4 level in Fig. S3 of Appendix S2) might infect very few small animals but a notable proportion of large animals. Second, transmission rate varies with food density mainly at intermediate levels of infection prevalence (Fig. S3). At lower and higher prevalence of infection, changes in food-dependent clearance rate should not sufficiently increase or decrease contact with/ingestion of spores to alter infection.

Examination of other losing models illuminates the winners' strengths. First, the clearance-rate-only models (Hypothesis 1) failed because they underestimated the importance of body size for disease transmission, despite that clearance rate scaled with surface area of the host (Fig. 3). Second, a model containing Type II-based clearance rate and gut surface area seemed to capture food dependence of transmission rate for smaller animals. However, it under-predicted infection prevalence in large size classes, and it paid a heavy price statistically for that problem. The model based purely on clearance rate and gut residence time (Hypothesis 5) overestimated the importance of food density for clearance rate (Fig. 3). Similarly, Hypotheses 7 and 8, both involving clearance rate, gut residence time, and gut size, overemphasized the importance of food density but qualitatively captured that of body size.

DISCUSSION

In many disease systems, hosts become infected by consuming spores of their parasite. Thus, epidemiology of such systems should intimately link with feeding processes. In fact, a merger of foraging theory with host–parasite models may someday yield a powerful understanding of disease dynamics. Here, we started this merger by developing models of parasite transmission that embraced simple yet fundamental components of the feeding biology of our focal host, *Daphnia*. Similar efforts also developed mechanistic understanding of other transmission models, e.g.

Table 2 Results of competition among models for transmission rate (TR) for the Food Gradient experiment

H	Formula*: TR=	$\beta \times 10^{-3}$	Units of β	NLL	AIC	Δ_i	w_i
3a	$\varepsilon \times \beta \times CR_1 \times SA_G$	60.0†	host sp^{-1} mm^{-2}	167.4	336.9	0.0	0.88
2a	$\varepsilon \times \beta \times CR_1 \times L_G$	0.89†	host sp^{-1} mm^{-1}	169.4	340.9	4.0	0.12
3c	$\varepsilon \times \beta \times CR_3 \times SA_G$	68.9†	host sp^{-1} mm^{-2}	176.0	354.0	17.1	1.68×10^{-4}
2c	$\varepsilon \times \beta \times CR_3 \times L_G$	1.02†	host sp^{-1} mm^{-1}	178.3	358.6	21.7	1.67×10^{-5}
4a	$\varepsilon \times \beta \times CR_1 \times V_G$	679.5	host sp^{-1} mm^{-3}	179.7	361.4	24.6	4.09×10^{-6}
1a	$\varepsilon \times \beta \times CR_1$	1.28	host sp^{-1}	185.3	372.6	35.8	1.51×10^{-8}
1c	$\varepsilon \times \beta \times CR_3$	1.47	host sp^{-1}	194.6	391.1	54.2	1.48×10^{-12}
3b	$\varepsilon \times \beta \times CR_2 \times SA_G$	15.0	host sp^{-1} mm^{-2}	211.5	425.0	88.1	6.47×10^{-20}
6	$\varepsilon \times \beta \times CR_j \times T_{G,j} \times L_G$	1.87	mg day^{-1} sp^{-1} mm^{-4}	211.5	425.0	88.1	6.47×10^{-20}
2b	$\varepsilon \times \beta \times CR_2 \times L_G$	0.22	host sp^{-1} mm^{-1}	216.3	434.6	97.8	5.21×10^{-22}
5	$\varepsilon \times \beta \times CR_j \times T_{G,j}$	2.71	mg mm^{-3} sp^{-1} day^{-1}	216.3	434.6	97.8	5.20×10^{-23}
4b	$\varepsilon \times \beta \times CR_2 \times V_G$	176.7	host sp^{-1} mm^{-3}	218.1	438.2	101.3	8.85×10^{-23}
7	$\varepsilon \times \beta \times CR_j \times T_{G,j} \times SA_G$	14.8	mg day^{-1} sp^{-1} mm^{-5}	218.1	438.2	101.3	8.85×10^{-23}
1b	$\varepsilon \times \beta \times CR_2$	0.31	host sp^{-1}	234.2	470.4	133.5	9.03×10^{-23}
8	$\varepsilon \times \beta \times CR_j \times T_{G,j} \times V_G$	1435	mg day^{-1} sp^{-1} mm^{-6}	234.5	471.0	134.1	6.58×10^{-30}
4c	$\varepsilon \times \beta \times CR_3 \times V_G$	977.9	host sp^{-1} mm^{-3}	235.2	472.4	135.5	3.24×10^{-30}
0	β	6.73×10^{-3}	L sp^{-1} day^{-1}	263.6	529.2	192.3	1.73×10^{-42}

Fits of each model yielded estimates of the infectivity parameter (β), negative log-likelihood (NLL), the Akaike information criterion (AIC), AIC differences for each model *i* (Δ_i) and Akaike weights (w_i). Models are sorted by AIC differences, from best to worst. Units of the infectivity parameter vary with the hypothesis considered.

*See Table 1 for details.

†Best-fitting suite of models, as described in the text. Profiled 95% confidence intervals respectively: [54.3, 66.3]; [0.81, 0.98]; [62.3, 76.0]; [0.93, 1.13].

frequency-dependence for sexually transmitted diseases (Lloyd-Smith *et al.* 2004). Prior work (summarized by Ebert 2005) provided us with the qualitative foundation for this foraging-transmission link: grazing *Daphnia* hosts become parasitized after consuming spores. Because the classic mass-action model of disease transmission ignores feeding biology, it could not capture food- and size-dependent transmissions seen in our experimental and field data.

We found that transmission rate was best described as a function of two general, broadly applicable aspects of foraging ecology: clearance rate and gut size. Clearance rate is the amount of habitat from which a host removes food (and spores) per unit time (Grover 1997), and it decreases with food density. Here, we found support for clearance rate based on both Type I and Type III functional responses, and the magnitude of support depended on specifics of the procedure to estimate parameter(s). (The selection of models with Type I and III over Type II-based functional responses might interest some biologists, particularly those who study foraging by *Daphnia* (Sarnelle 2003; Jeschke *et al.* 2004); however, detailed discussion of particular functional responses would probably involve over-interpretation of the present data set). Even though clearance rate depends upon body size of the host (Kooijman 1993), we needed to add another size-dependent factor, gut size, to best fit the data. This addition makes intuitive sense, as larger guts should hold more spores and provide more area for penetration of host tissue. These mechanisms helped to explain a strong body-size signal in epidemics of lake *Daphnia*. Of course, other factors might also contribute to these patterns in nature, particularly for the juvenile–adult contrast (i.e. observational biases for incubation periods, longer exposure time of adults to spores). However, the foraging-transmission link offered here provided a potent (if partial) explanation for this signal in nature.

Foraging-dependent transmission rate may have important implications for the community ecology of disease in this system. For instance, it might indirectly mediate interactions between fish predators, *Daphnia* hosts and various parasites. We have hypothesized that predators can control disease in these systems by preferentially preying on infected hosts (Duffy *et al.* 2005; Hall *et al.* 2005a, 2006a; see also Johnson *et al.* 2006a). Such selective predation probably reduces fitness and spread of parasites. The results presented here suggest two other, indirect pathways through which fish could reduce parasitism. First, fish might indirectly elevate food resources by preying upon grazers (i.e. the trophic cascade; Carpenter & Kitchell 1993). Higher food densities should lower transmission rate. Second, fish typically consume larger *Daphnia* (Carpenter & Kitchell 1993). All else being equal, smaller hosts should encounter (clear) fewer spores, and therefore experience less infection.

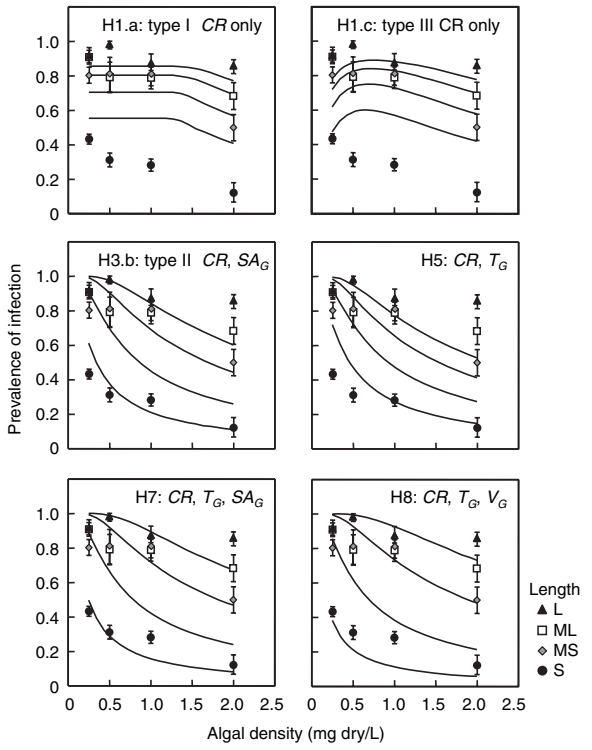

Figure 3 Performance of several of the losing models, shown compared with data from the Food Gradient experiment. Each hypothesis number (H_n) corresponds to one summarized in Table 1. Body size labels follow those from Fig. 2. Lines are predictions of the model calculated with best-fit parameters; points are mean ± 1 SE.

Thus, fish may indirectly influence host–parasite interactions via effects on food levels and body size of hosts.

Of course, such predictions for this system built around transmission rate alone will remain speculative until we consider other aspects of the host–parasite interaction. For instance, many parasites of *Daphnia* virulently reduce fecundity and survivorship of their hosts (Mangin *et al.* 1995; Stirnadel & Ebert 1997; Bittner *et al.* 2002; Ebert 2005; Hall *et al.* 2006a; Johnson *et al.* 2006b). Thus, theory predicts that these parasites should harm their host populations (Anderson & May 1981, 1991). However, the magnitude of these virulent effects depends upon food concentration (Pulkkinen & Ebert 2004) and spore dose (Regoes *et al.* 2002; Ebert 2005). In our own system, we see stronger reduction in fecundity, higher death rates, and increased production of spores released after death in high food treatments (J. Simonis, S.R. Hall, and C.E. Cáceres, unpublished data). Therefore, the net effect of variation in food levels on this host–parasite interaction becomes hard to predict. However, previous modelling efforts that incorporated roughly similar details (Hochberg 1991; Fenton *et al.* 2002; Regoes *et al.* 2002) concluded that they can have huge effects on disease dynamics. Also, like in other disease systems (Burdon 1980; Lively 1989), *Daphnia* genotypes often show variation in resistance to parasites (Ebert *et al.* 1998, 2000; Carius *et al.* 2001; Ebert 2005; Mitchell *et al.* 2005), including in the *D. dentifera–Metschnikowia* system (Duffy & Sivars-Becker 2006; this genetic issue does not apply to our experiments, as we used one clone of *Daphnia*). Some systems even show trade-offs of feeding rate vs. resistance among genotypes (e.g. Fellowes *et al.* 1999). Such a trade-off could yield interesting results from a model of competition among genotypes.

In the meantime, we should highlight that the best-performing and the rejected models developed here might prove useful for other host–parasite interactions that involve foraging. Of course, one must verify that the foraging components that we summarized reflect biology of the particular host (e.g. caveats about gut residence time in the Methods section). Assuming that these foraging components apply and/or have been modified appropriately, the clearance rate–gut size or clearance rate-only models should also extend to other grazers that eat their parasites (snails infected by trematodes; Lively 1989; many pathogens of insect defoliators, such as gypsy moths: Evans & Entwistle 1987; worm and anthrax infection of grazing mammals: Willams & Barker 2001; Wobeser 2006) or become infected by vectors that wait for browsing hosts in vegetation (ticks vectors on vertebrate hosts: Wobeser 2006). Additionally, we quickly rejected a model in which contact with spores varied proportionately to feeding rate, rather than clearance rate. However, the feeding rate assumption might apply if parasites physically attach to or live within food of hosts (i.e. tropically transmitted parasites: Lafferty 1999; parasites transmitted by cannibalism: Knell *et al.* 1996, 1998). Therefore, one can imagine many variations on these foraging-based models for transmission rate, designed according to the underlying foraging biology of the specific system.

Finally, it is important to remember that transmission biology does not always involve consumption of free-living parasites or may include other factors not considered here. For instance, transmission of sexually transmitted diseases and those relying on social contact with hosts typically follows a frequency-dependent model, and transmission of macroparasites often involves terms that capture aggregation of parasites (Begon *et al.* 1999; McCallum *et al.* 2001; Lloyd-Smith *et al.* 2004). The choice of function for transmission rate can strongly shape inferences yielded from epidemiological models (Hochberg 1991; McCallum *et al.* 2001; Fenton *et al.* 2002; Regoes *et al.* 2002; Keesing *et al.* 2006). Therefore, it becomes critical to choose transmission functions infused with relevant, underlying

biology. Furthermore, other factors may still influence transmission of free-living parasites to their hosts. These include (but are not limited to): heterogeneities in behaviour and susceptibility of hosts (Dwyer & Elkinton 1993; Dwyer *et al.* 1997), saturation of infection of hosts by extremely abundant parasites (Knell *et al.* 1996), and other spatial clumping of hosts and parasites (Keeling & Grenfell 2000). Additionally, our models ignore immunology (i.e. 'clearance' of initial spore invasion) because we never see infected *Daphnia* that recover. In systems where immunity/clearance of infection by hosts is important, larger/older hosts may be less rather than more vulnerable to infection (Wobeser 2006).

With these caveats in mind, our main message is that basic tenets of foraging ecology can produce more powerful epidemiology when hosts encounter free-living parasites while eating. Functions for transmission are challenging to craft, and much progress continues to arise from the use of the mass-action model and more phenomenological, nonlinear relatives (Hochberg 1991; McCallum *et al.* 2001; Fenton *et al.* 2002). However, we developed models for transmission rate that mechanistically connected feeding biology of a *Daphnia* host with infection by its fungal parasite. A somewhat similar model links feeding biology of vectors with transmission of disease to host plants (Ferrari *et al.* 2006). Our models were built around general components of foraging biology of the host, and these components should apply broadly across taxa of animals (when modified appropriately). Furthermore, these results provided a step towards synthesis of *Daphnia* as pivotal grazer of algae and as host to parasites. Such a synthesis should help catalyse understanding of the community–disease interface in this system and others in which consumers inadvertently eat parasites.

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SUPPLEMENTARY MATERIAL

The following supplementary material is available for this article:

Appendix S1 Signature of body size in field data.

Appendix S2 More statistical and empirical results.

This material is available as part of the online article from: http://www.blackwell-synergy.com/doi/full/10.1111/j.1461-0248.2006.01011.x

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