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Resources, key traits and the size of fungal epidemics in *Daphnia* populations

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Summary

1. Parasites can profoundly affect host populations and ecological communities. Thus, it remains critical to identify mechanisms that drive variation in epidemics. Resource availability can drive epidemics via traits of hosts and parasites that govern disease spread.

2. Here, we map resource–trait–epidemic connections to explain variation in fungal outbreaks (*Metschnikowia bicuspidata*) in a zooplankton host (*Daphnia dentifera*) among lakes. We predicted epidemics would grow larger in lakes with more phytoplankton via three energetic mechanisms. First, resources should stimulate *Daphnia* reproduction, potentially elevating host density. Secondly, resources should boost body size of hosts, enhancing exposure to environmentally distributed propagules through size-dependent feeding. Thirdly, resources should fuel parasite reproduction within hosts.

3. To test these predictions, we sampled 12 natural epidemics and tracked edible algae, fungal infection prevalence, body size, fecundity and density of hosts, as well as within-host parasite loads.

4. Epidemics grew larger in lakes with more algal resources. Structural equation modelling revealed that resource availability stimulated all three traits (host fecundity, host size and parasite load). However, only parasite load connected resources to epidemic size. Epidemics grew larger in more dense *Daphnia* populations, but host density was unrelated to host fecundity (thus breaking its link to resources).

5. Thus, via energetic mechanisms, resource availability can stimulate key trait(s) governing epidemics in nature. A synthetic focus on resources and resource–trait links could yield powerful insights into epidemics.

Key-words: epidemics, fungus, parasite *Daphnia*, reproduction, resource availability

Introduction

Virulent parasites can regulate host populations, shape ecological communities and exert strong selective pressure on hosts (Hudson, Dobson & Newborn 1998; Duffy *et al.* 2012). Thus, the emergence and resurgence of infectious diseases in wildlife presents a major challenge for conservation and the maintenance of ecosystem structure and function (Fisher *et al.* 2012). Yet, large epidemics occur infrequently, and disease varies profoundly in space and time. Better delineation of the drivers of this variation could enhance explanation of the distributions of disease, bolster predictions of emergent outbreaks and facilitate responses of wildlife managers to epidemics (Lafferty & Holt 2003; Johnson *et al.* 2010). Thus, it remains critical to identify these drivers and link them to variation in epidemics.

Environmental factors may drive variation in epidemics by modulating the traits of hosts and parasites that determine disease spread (Civitello *et al.* 2013a,b; Mordecai *et al.* 2013). A key environment–trait link starts with host resources. Resource availability could influence host immunity or other key traits that are mechanistically linked to the energetic status (condition) and/or body size

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of hosts (e.g. host fecundity or production of parasites once infected; Johnson *et al*. 2007). Resource availability could enhance epidemics by modulating reproduction and growth of hosts. Improved host condition could stimulate host fecundity, which in turn could raise host density. High host density can facilitate the start and then further spread of disease (Anderson & May 1986; Pedersen & Greives 2008). Similarly, improved condition could promote host growth. Larger hosts can have higher infection risk since they provide larger targets for vectors or free-living parasites (Théron, Rognon & Pagés 1998; Daost *et al*. 2010). Additionally, larger hosts may encounter more trophically transmitted parasites because they feed faster than smaller conspecifics (Hall *et al*. 2007). Thus, resource-mediated increases in host density or encounter rate could boost epidemic size.

Furthermore, resource-enhanced host condition could amplify or diminish parasite production in infected hosts. Resources can enhance parasite production through bottom-up mechanisms (energy availability). For example, poor host condition due to inadequate resources can limit parasite growth and reproduction (Hall *et al*. 2009a,b; Cressler *et al*. 2014). Hosts in poor condition yield fewer resources or physical space for parasite growth (Pulkkinen & Ebert 2004; Hall *et al*. 2009b). Moreover, hosts already in poor condition might die more quickly upon exposure to parasites, constraining parasite development (Krist *et al*. 2004). Thus, if parasite success hinges on resource acquisition, then parasites should grow or reproduce best when infecting hosts in good condition. However, rich resource environments might enhance top-down mechanisms (host defence), diminishing parasite success. Often, immunological defences are energetically expensive; thus, hosts in better condition may better resist, clear or control infection if they can deploy better defences (Sheldon & Verhulst 1996). Thus, if parasite success depends sensitively on host immunity, then elevated host condition should depress parasite loads, while poor condition hosts might suffer from heavier burdens.

Through these resource–trait links, resource availability could powerfully govern epidemic dynamics. However, tension can arise between the links. In a given host–parasite system, greater resource availability might stimulate one trait that enhances disease while simultaneously inhibiting another. For example, low host fecundity and exposure due to poor host condition and small size might inhibit epidemics. However, weak immunity caused by poor condition could counteract these effects and exacerbate disease spread. Thus, to explain and predict the influence of resources on epidemic size, we must know how sensitively resource–trait links catalyse or inhibit disease spread (Hall *et al*. 2009a; Civitello *et al*. 2013b).

Despite abundant laboratory evidence, concrete links among resources, traits and epidemics in the field remain rare for any disease system (but see, e.g. Pedersen & Greives 2008; Rohr *et al*. 2008). This prompts a critical question: Do resource-dependent traits actually matter for real epidemics in nature? Here, using a case study, we address this question by mapping resource–trait–epidemic connections to explain variation in a disease system in nature. In this planktonic system, a host/grazer (*Daphnia dentifera*) becomes infected by an obligate killer fungus (*Metschnikowia bicuspidata*). Based on the results of laboratory experiments and modelling, we predicted that epidemics would grow larger in lakes with more algal resources via three mechanisms. First, *Daphnia* reproduce more rapidly with more resources (Hall *et al*. 2009b). Thus, lakes with more resources might support higher *Daphnia* densities, facilitating disease spread (Anderson & May 1986). Secondly, *Daphnia* grow larger with greater resources. Larger *Daphnia* filter water faster and therefore more rapidly contact this trophically acquired fungus (Hall *et al*. 2007). Thus, we predict greater density and larger hosts (leading to greater transmission) in resource-rich lakes. Thirdly, we predict that infected hosts should yield more parasite spores in lakes with more resources. This positive host condition – parasite production pattern has repeatedly arisen in laboratory experiments of this system (manipulating resource quantity/quality, Hall *et al*. 2009a,b; Penczykowski *et al*. 2014b; water chemistry, Civitello *et al*. 2012, 2013b; and host genotype, Hall *et al*. 2010a, 2012). However, none of these predictions are a given in nature: based on planktonic natural history, we might also expect the opposite resource–fecundity–density, resource-size, and resource–parasite production patterns. Trophic cascades, driven by fish predators, could elevate algal resources, but decrease host density, body size and parasite load [since fishes preferential consume larger (Carpenter & Kitchell 1993) and infected *Daphnia* (Duffy & Hall 2008)].

To map these resource–trait–epidemic links, we sampled fungal epidemics in twelve lake populations of *Daphnia*. Specifically, we measured algal resource density, body size and reproduction of uninfected *Daphnia*, body size and production of fungal spores in infected *Daphnia*, *Daphnia* population density and the ultimate size of each epidemic. We found fungal epidemics grew largest in lakes with more resources [i.e. higher quantities of carbon and phosphorus in edible (<60 μm) seston]. However, since epidemic size was unrelated to a resource quality index (C:P ratio), we focus on resource quantity. Using a structural equation model, we found that each trait (size and reproduction of uninfected *Daphnia*, and spore load) increased in lakes with more resources. Yet, the model identified spore production as the critical resource-connected trait that drove variation in epidemic size among lakes. The model also revealed that hosts were more dense in lakes with larger epidemics (controlling for other traits), but host density was unconnected to resources via fecundity.

Materials and methods

DISEASE SYSTEM

Daphnia dentifera is a dominant zooplankton found in small, thermally stratified lakes in the Midwestern USA. This non-selective

grazer becomes infected with the fungal parasite, *M. bicuspidata*, after inadvertently consuming spores suspended in the water column (Ebert 2005). The fungus reproduces in the hemolymph, and spores fill the host's body cavity. Infection harms host survival and reproduction (Hall *et al.* 2009b). Spore release into the water requires host death (Ebert 2005). In our study lakes, epidemics begin in late summer and can continue into December (Overholt *et al.* 2012).

FIELD SURVEY

We present field data from weekly surveys of small lakes in southern Indiana ($n = 12$ lakes, Greene and Sullivan counties, USA). During each sampling visit in 2010, we collected paired samples, each containing three pooled tows of a standard Wisconsin plankton net (13 cm diameter, 153-μm mesh, towed bottom to surface). We preserved one sample from each visit in 70% ethanol and later counted *D. dentifera* to estimate population density (Hall *et al.* 2011). We used log-transformed values of population density in our analyses. Using the other sample, we diagnosed infection status following Green (1974) of 400 or more live hosts from each sample using a dissecting microscope (20–50×). With the prevalence data, we then characterized epidemic size as the integrated area under the prevalence–time curve. This index of epidemic size is highly correlated with another index, maximum infection prevalence ($R = 0{\cdot}89$, $P < 0{\cdot}001$), which ranged from 8 to 47% in these lakes in 2010. From these live samples, we collected infected hosts. Typically, we measured body length (eye to base of the tail) of at least 25 infected hosts and then placed three samples of 10 infected hosts each into 2-mL plastic centrifuge tubes in 0·5 mL of filtered lake water. We then gently mashed these hosts to release spores, and we counted them using a hemocytometer at 200× on a compound microscope. We also measured body length and counted eggs of 25 or more uninfected adult hosts. We averaged weekly values of these quantities from 29 September (as many epidemics began) through 1 December (as epidemics ended) in our analysis. Mean body size of uninfected and infected hosts was extremely correlated ($R^2 = 0{\cdot}97$), indicating that these indices provide identical information. Therefore, we used only the size of uninfected hosts in our analysis.

We calculated four indices of resource availability using water collected with an integrated tube sampler. During stratified periods, we lowered the tube to the bottom of the epilimnion (as determined by temperature profiling). Once a given lake destratified in autumn, we lowered the tube to the bottom of the oxygenated layer or seven metres, whichever was shallower. We poured these water samples through a mesh sieve to obtain 'edible' (<60 μm) algae and filtered the samples onto acid-washed, preashed GF/F filters (0·7 μm pore size; Whatman, Piscataway, NJ). We then determined edible carbon and nitrogen (using a Perkin Elmer Series 2400 CHN analyzer, Waltham, MA) and particulate phosphorus (using standard colorimetric methods: Prepas & Rigler 1982). We calculated C:P ratios and log-transformed quantities of C, N and P for each sampling date. We then averaged these weekly values as above in our analysis.

DATA ANALYSIS

We hypothesized that high resource availability would simultaneously increase host size (length of uninfected hosts), reproduction (egg ratio of uninfected adults) and parasite production (spore yield from infected hosts). Thus, we hypothesized that the common underlying factor, resources, linked all of these traits. In turn, greater reproduction could stimulate host population density. Finally, increased host size, population density and parasite production should all drive larger epidemics in *Daphnia* populations. We tested this multivariate hypothesis using a structural equation model (SEM; Grace 2006). In the Appendix S1 (Supporting information), we also examine three slight variants of this SEM model, which all yield consistent results and conclusions. SEMs have several advantages for testing multivariate hypotheses over other methods, for example generalized linear models. SEMs aim to test directional, multivariate causal networks that can be represented by a path diagram (Grace 2006). SEMs use variances and covariances of measured variables to simultaneously test multiple causal relationships in a single analysis (Grace 2006). Crucially, SEM analyses can directly assess the overall fit of the hypothesized model in addition to the significance of individual relationships (Grace 2006). While the good fit of an SEM does not demonstrate causation, it can strongly bolster the support for causal hypotheses when combined with consistent theoretical or empirical evidence (Grace 2006). Thus, relative to more typical GLMs, SEMs offer superior methods to evaluate causal relationships between variables: given the *a priori* mechanistic hypotheses we sought to test, SEMs offer a vastly superior model than GLMs. SEMs can also incorporate latent variables (conceptually important factors measured indirectly: Grace 2006). In our SEM, edible carbon, nitrogen and phosphorus serve as indicator variables for a single latent factor that represents 'resource availability'. However, other factors might drive covariation in length, egg ratio and spore load. Therefore, we also estimated their residual covariances, where non-significant covariances indicate that additional (unmodelled) factors do not jointly drive relationships among them. We fit this model using the SEM function in the lavaan package in R (Rosseel 2012) and assessed its goodness-of-fit using a chi-square test ($P < 0{\cdot}05$ indicates poor fit) and the comparative fit index (CFI). The CFI (range: 0–1) robustly measures fit with small sample sizes (CFI $\geq 0{\cdot}95$ indicates good fit). While epidemic size correlated more strongly with edible C, edible P best correlated with the latent factor 'resource availability' ($R^2 = 0{\cdot}98$) in the SEM analysis. Thus, plots with P help to best visualize the relationships between the key traits and 'resource availability'. Furthermore, we used the (multivariate) SEM as the main test of the resource–trait hypotheses because this analysis controls for the effects of the other hypothesized driving factors. However, univariate plots (with univariate correlation statistics) accompany the SEM to aid interpretation. Nonetheless, readers should focus most attention on the results of the SEM.

Results

Epidemics grew larger in lakes with more edible carbon ($n = 12$ lakes, $R^2 = 0{\cdot}49$, $P = 0{\cdot}011$, Fig. 1a) and phosphorus ($n = 12$, $R^2 = 0{\cdot}36$, $P = 0{\cdot}039$, Fig. 1b). However, epidemic size was not related to the C:P ratio of edible seston ($n = 12$, $R^2 = 0{\cdot}08$, $P = 0{\cdot}37$, Fig. 1c). The SEM linking resource availability to epidemic size through condition-dependent traits fit the observed data well (χ^2 goodness-of-fit test, d.f. = 15, $P = 0{\cdot}20$; CFI = 0·951). Moreover, the model explained 76% of the variation in epidemic size (SEM $R^2 = 0{\cdot}76$, Fig. 2). The three

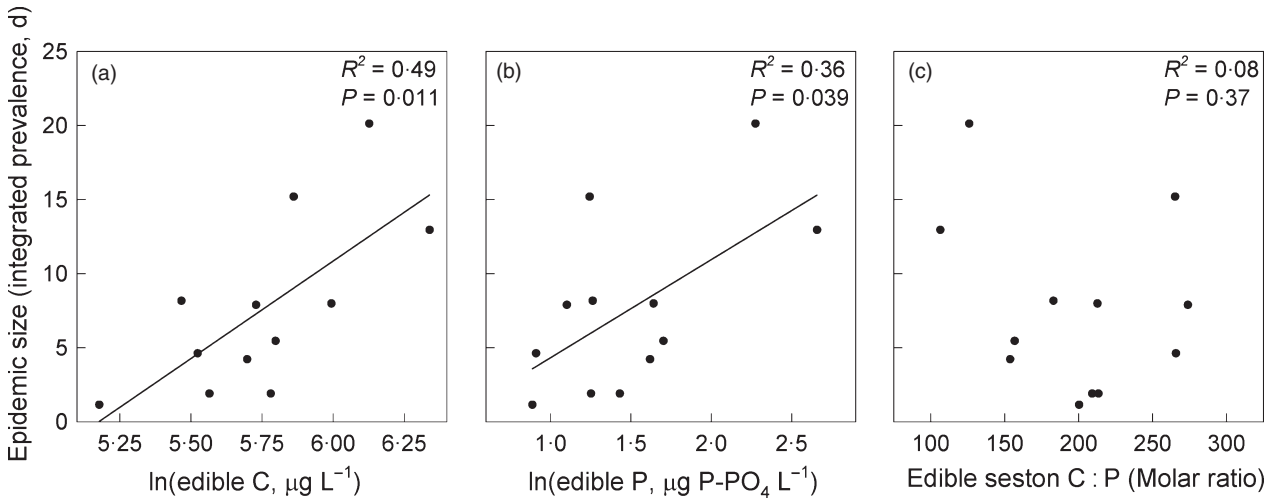

Fig. 1. Epidemics of a virulent fungus, *Metschnikowia bicuspidata,* infecting a zooplankton host, *Daphnia dentifera*, grew larger in lakes with more edible (a) carbon and (b) phosphorus. However, (c) epidemic size was not related to an index of resource quality, carbon: phosphorus ratio of edible seston. This result justifies subsequent focus on resource quantity, not this quality index, in the multivariate (SEM) and univariate analyses. Epidemic size is indexed as area under the time series of infection prevalence, while resource metrics average over weekly samples during epidemic season (late September–late November 2010). Thus, each point is a lake mean, with significant relationships noted with solid lines.

indicators of resource availability (edible C, N and P) were highly correlated (all $R > 0{\cdot}85$), supporting their use as indicators of one latent factor. As predicted, resource availability simultaneously increased the size (SEM: $P < 0{\cdot}001$, $R^2 = 0{\cdot}63$) and reproduction (SEM: $P = 0{\cdot}012$, $R^2 = 0{\cdot}35$) of uninfected *Daphnia* as well as the production of fungal spores within infected *Daphnia* (SEM: $P = 0{\cdot}001$, $R^2 = 0{\cdot}50$; Figs 2, 3a,b and 4a). Since no significant residual covariances arose among these three traits (all $P > 0{\cdot}1$), unmodelled factors likely did not jointly drive variation in them. However, *Daphnia* population density was unrelated to host fecundity (SEM: P = 0·80, Figs 2 and 4b). Regardless, epidemics grew largest in lakes with greater host density (SEM: $P = 0{\cdot}001$, Figs 2 and 4c) and spore production per host (SEM: $P < 0{\cdot}001$, Figs 2 and 3d). However, body size of hosts (SEM: $P = 0{\cdot}56$) was uncorrelated with epidemic size (Figs 2 and 3c) in the SEM. The three SEM variants that we examined all yielded quantitatively similar results to the SEM presented here (see Appendix S1).

Discussion

In this study, we argue that variation in a key environmental factor (resource availability) should predictably drive variation in epidemic size among natural populations. Previous theory and laboratory experiments indicate that resources can modulate key epidemiological traits (Hall *et al*. 2009a,b; Arsnoe, Ip & Owen 2011; Cressler *et al*. 2014). However, which, if any, of these resource–trait links matter for epidemics in nature has remained poorly understood. By connecting resource availability with key epidemiological traits, this study establishes that resources are relevant drivers of natural epidemics through these mechanistic connections with traits. Uninfected *Daphnia* grew larger and reproduced more in lakes with more algal resources (indexed by edible carbon, nitrogen and phosphorus). Simultaneously, infected *Daphnia* had greater spore loads in lakes with more resources. These resource–trait relationships echo those seen in laboratory experiments that manipulated laboratory-reared or field-collected algae (Hall *et al*. 2009a,b). We observed clear signals of resource-dependent traits across lakes despite variation in other factors that might have influenced these phenotypes (e.g. genetic variation, water chemistry or temperature). Thus, resource availability robustly drives spatial variation in epidemiological traits in nature.

In principle, each of the traits could drive larger epidemics (e.g. Hall *et al*. 2007, 2009a). However, we did not know which, if any, of these resource–trait links were actually relevant in natural populations. Thus, our SEM analysis revealed novel insight for the relevance of these resource-dependent traits for the size of natural epidemics. Resource-dependent parasite production emerged as the most important trait driving epidemic size. Compared with the other traits, spore load was most sensitive to resources, and it varied 4·2-fold among lakes. This sensitivity to resource quantity could explain why spore load best predicted epidemic size in nature. Crucially, the resource-parasite production pattern in nature stems directly from results frequently seen with this host–parasite system in laboratory experiments (e.g. Hall *et al*. 2009b). In the laboratory, similarly positive, resource–parasite production links also arise across many host–parasite systems (e.g. snail–trematode: Krist *et al*. 2004; insect–mite: Ryder, Hathway & Knell 2007; bird–virus: Arsnoe, Ip & Owen 2011; fish–monogean: Tadiri, Dargent & Scott 2013). Thus, based on the *Daphnia*-fungus example, resource-dependent parasite production could generally

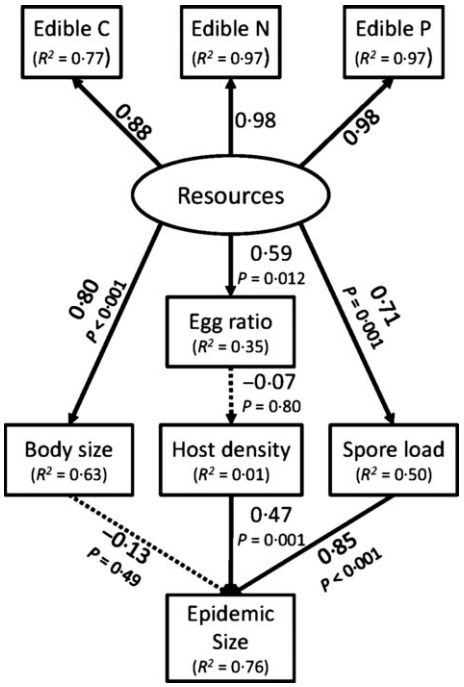

Fig. 2. Diagram of the hypothesized structural equation model relating resources, traits of uninfected and infected *Daphnia* hosts, host density and size of fungal epidemics. All measured quantities are represented by rectangles. Resource availability, a latent variable, is represented by an oval and was estimated using edible (<60 μm) carbon (μg C L^{-1}), nitrogen (μg N L^{-1}) and phosphorus (μg P L^{-1}). Traits include mean body size of uninfected adult hosts (mm), adult egg ratio (an index of fecundity, eggs/adult female) and fungal spore load per infected host (spores/host). Arrows indicate hypothesized causal relationships, represented by standardized regression coefficients and P values. Solid arrows indicate significant relationships ($\alpha = 0{\cdot}05$), and dotted arrows indicate non-significant relationships. The model fits the observed data well (χ^2 goodness-of-fit test, d.f. = 15, $P = 0{\cdot}20$; CFI = 0·951). Coefficients of determination (R^2) are presented for each measured quantity.

drive epidemics in natural populations in many host–parasite systems. However, in other systems, the opposite pattern might arise: greater resource availability could stimulate host defences, diminishing parasite production. For example, increased consumption of protein can alleviate the costs of immunological resistance in a caterpillar–virus system (Lee *et al*. 2006). Similarly, carotenoids can stimulate immune defences in vertebrate and invertebrate hosts (Blount *et al*. 2003; Babin, Biard & Moret 2010). Given the crucial role of parasite production (a key determinant of infectiousness) in driving epidemic size, the direction of the resource–parasite production relationship could broadly predict patterns of epidemics across populations or time.

In contrast, epidemic size did not correlate significantly with host size or fecundity. Body size influences exposure to parasites: larger hosts contact more spores, all else equal (Hall *et al*. 2007). Adult *Daphnia* grew larger in lakes with more resources. This result suggests that resource availability (energetics), rather than size-selective predation from fishes, may drive variation in host body size in these lakes (Carpenter & Kitchell 1993). Nonetheless, body size (a driver of exposure) did not correlate with epidemic size. However, relative to the spore load trait, adult size varied only slightly in our focal lakes (1·1–1·4 mm) along the algal resource gradient. Thus, even if larger host size could boost exposure to parasites, it likely varied too little in nature to influence epidemic size, at least at the across-lake scale. We also predicted that host fecundity could stimulate epidemics by increasing host density. Indeed, epidemics grew larger in lakes with dense populations of *Daphnia*. Positive host density–disease relationships are anticipated by classic epidemiological models (Anderson & May 1986) and arise in an array of case studies (e.g. mammal–nematodes: Arneberg *et al*. 1998; lion–viruses: Packer *et al*. 2001; human–measles: Keeling & Grenfell 2002). However, previous surveys in this *Daphnia*–fungus system found either no relationship between host density and disease (Hall *et al*. 2010b; Penczykowski *et al*. 2014a) or a unimodal one (Civitello *et al*. 2013a). Thus, host density–disease relationships in this system may vary temporarily and may be mediated by other factors/traits. Moreover, host density was not correlated with host fecundity. Thus, in these lakes, host density may be driven by environmental factors other than fecundity (e.g. predation: Carpenter & Kitchell 1993).

In principle, resource quality could also have influenced the size of fungal epidemics. For example, P-limitation reduces reproduction of the bacterium *Pasteuria ramosa* in infected *Daphnia* (Frost, Ebert & Smith 2008). Similarly, highly defended algae can limit the growth and reproduction of uninfected *Daphnia* and the production of *M. bicuspidata* (Hall *et al*. 2012; Penczykowski *et al*. 2014b). *Daphnia* can also reduce their foraging rates when confronted with lower quality food (e.g. *Microcystis*), decreasing exposure to parasites (Penczykowski *et al*. 2014b). However, epidemic size was not related to one index of quality, C:P ratio, perhaps because algae remained fairly P-rich (i.e. C:P was low) during these autumnal epidemics (Sterner & Elser 2002). In addition, C:P ratio was not correlated with spore load, the strongest trait-based correlate of epidemic size (linear regression: $n = 12$ lakes, $R^2 = 0{\cdot}17$, $P = 0{\cdot}18$, not shown). Still, resource quality could drive disease in other systems [e.g. toxins in milkweeds influence protozoan production in monarch butterflies (de Roode *et al*. 2008); see Smith & Holt (1996)]. Thus, resource quantity and/or quality might drive disease dynamics in many systems.

Here, resources stimulated epidemics by enhancing three traits that could promote disease spread. In other systems, opposing resource–trait links may create tension for disease dynamics. This tension most likely involves host immunity. Condition-dependent immunity should lower parasite production and enhance clearance of infection (Cressler *et al*. 2014). All else equal, these effects should depress epidemics. However, host fecundity and

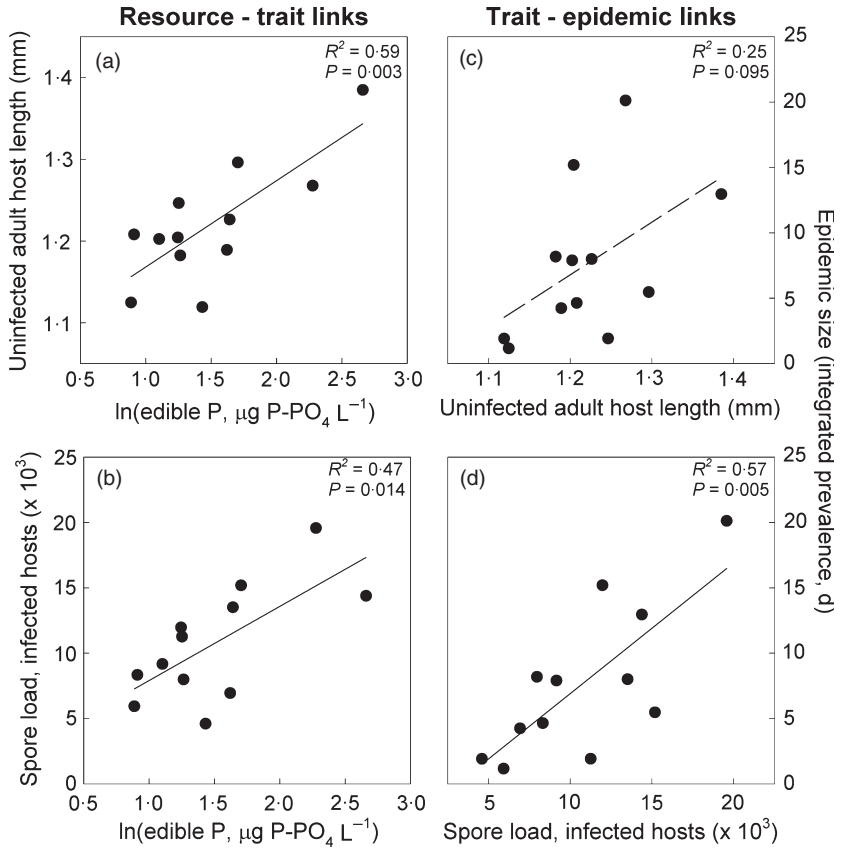

Fig. 3. Univariate links between resources, traits and size of fungal epidemics across lakes help to interpret the multivariate, causal hypothesis (Fig. 2). Each point represents the mean value for one lake (n = 12) during 2010 epidemics. (a, b) *Resource–trait links*: uninfected *Daphnia* were (a) larger in lakes with more edible phosphorus [a close correlate of the latent 'resource' variable in the SEM model (Fig. 2)]. Simultaneously, (b) infected *Daphnia* contained more fungal spores in lakes with more edible phosphorus. (c, d) *Trait–epidemic links*: epidemic size was not related to (c) size of uninfected, adult hosts. However, (d) epidemics grew larger in lakes with higher mean spore loads. Coefficients of determination (R^2) correspond to univariate regressions between pairs of variables. Significant ($P < 0{\cdot}05$) and marginally significant ($P < 0{\cdot}10$) univariate relationships are indicated by solid and dashed lines, respectively. These univariate goodness-of-fit statistics (R^2, P-value) mirror fit results from the SEM (Fig. 2).

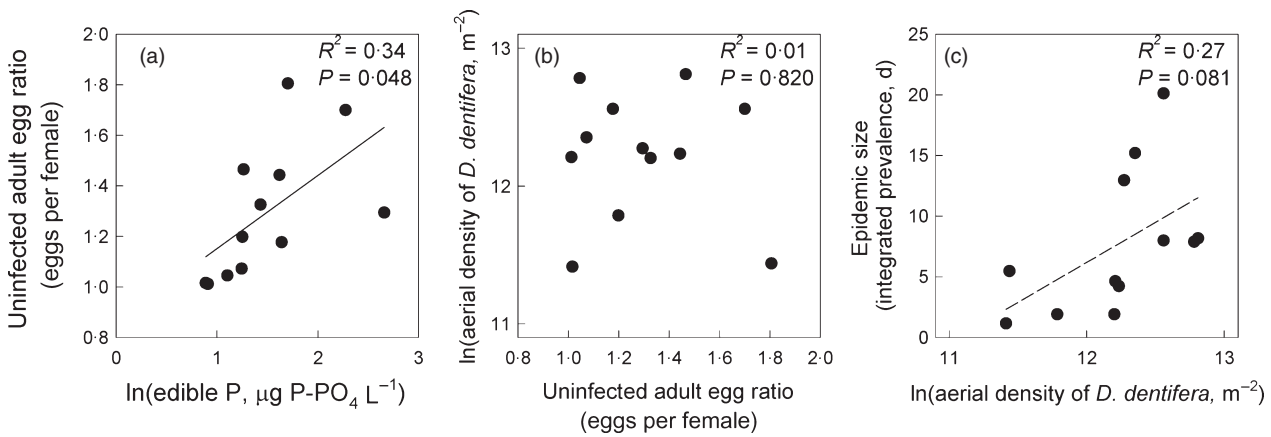

Fig. 4. Univariate relationships among host reproduction (indexed by egg ratio), population density and epidemic size. Each point represents the mean value for one lake (n = 12) during 2010 epidemics. (a). Uninfected *Daphnia* reproduced more in lakes with more edible phosphorus [a close correlate of the latent 'resource' variable in the SEM model (Fig. 2)]. (b) However, *Daphnia* population density was not related to the index of per capita reproduction. (c) Regardless, epidemics grew larger in lakes containing denser populations of hosts. Coefficients of determination (R^2) correspond to univariate regressions between pairs of variables. Significant ($P < 0{\cdot}05$) and marginally significant ($P < 0{\cdot}10$) univariate relationships are indicated by solid and dashed lines, respectively. These univariate goodness-of-fit statistics (R^2, P-value) mirror fit results from the SEM (Fig. 2), although the density-epidemic size relationship was stronger (more significant) in the focal, multivariate SEM analysis.

growth could still increase with resource availability/host condition (Kooijman 2009). Therefore, favourable conditions could still increase disease spread by increasing host density and exposure to parasites – despite any immune response (Anderson & May 1986; Hall *et al.* 2007). Resolution of this tension between immunity and other traits lies at the heart of future development of a trait-centred, resource-dependent theory for disease. Ultimately, if ecologists can understand the drivers of variation in resource availability (e.g. productivity and predation) and map links between resources, traits and epidemics, they may glean powerful insights into disease outbreaks.

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Data accessibility

Data available from the Dryad Digital Repository: http://dx.doi.org/10.5061/dryad.8n344 (Civitello *et al*. 2015).

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Supporting Information

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

Appendix S1. Considering additional structural equation models.