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LETTER

Success, failure and ambiguity of the dilution effect among competitors

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Abstract

It remains challenging to predict variation in the magnitude of disease outbreaks. The dilution effect seeks to explain this variation by linking multiple host species to disease transmission. It predicts that disease risk increases for a focal host when host species diversity declines. However, when an increase in species diversity does not reduce disease, we are often unable to diagnose why. Here, we increase mechanistic and predictive clarity of the dilution effect with a general trait-based model of disease transmission in multi-host communities. Then, we parameterise and empirically test our model with a multi-generational case study of planktonic disease. The model-experiment combination shows that hosts that vary in competitive ability (R^*) and potential to spread disease (R_0) can produce three qualitatively disparate outcomes of dilution on disease: the dilution effect can succeed, fail, or be ambiguous/irrelevant.

Keywords

Amplification, Daphnia, dilution effect, disease ecology, epidemic, friendly competition, modelling, transmission.

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INTRODUCTION

Disease outbreaks can regulate dynamics of host populations (Anderson & May 1979) and shift the outcome of competition between species (Freeland 1983; Price *et al*. 1988). However, we still struggle to uncover how interactions among host species regulate disease (Holt *et al*. 2003). The dilution effect offers potentially powerful connections between host communities and transmission. In the broadest sense (Keesing *et al*. 2006), it predicts that a decline in diversity (fewer diluter species) elevates disease risk for a more vulnerable focal host. Diluter species can decrease transmission when infected vectors waste bites on diluters, when diluters remove environmentally distributed parasites (e.g. by eating propagules), when diluters depress focal host density (e.g. by depleting shared resources), or when diluters modify host behaviour (Keesing *et al*. 2006, 2010). All of these proposed 'local dilution mechanisms' reduce contact between focal hosts and parasites. Hence, losses of diluter species can elevate host-parasite contact, transmission and the severity of disease outbreaks.

Evidence for dilution has now arisen in numerous systems. Some involve risks to human health, including Lyme disease (Ostfeld & Keesing 2000; LoGiudice *et al*. 2003), West Nile virus (Allan *et al*. 2009), Schistosomiasis (Johnson *et al*. 2009) and Hanta virus (Clay *et al*. 2009; Suzan *et al*. 2009). Other diseases strictly infect plant and wildlife hosts (Mitchell *et al*. 2002; Johnson *et al*. 2008, 2013; Hall *et al*. 2009a; Johnson & Thieltges 2010; Becker *et al*. 2014; Lacroix *et al*. 2014; Rottstock *et al*. 2014; Venesky *et al*. 2014). These examples indicate that further species losses may enhance disease risk in a variety of ecosystems. However, the dilution effect remains controversial, because higher species diversity does not always reduce disease. Sometimes diversity even amplifies disease (Keesing *et al*. 2006; Ogden & Tsao 2009; Wood *et al*. 2014). Additionally, switches between definitions of 'disease risk' (infection prevalence versus density of infected hosts) can qualitatively change observation of a dilution effect (Begon 2008; Roche *et al*. 2012). Thus, critiques of the dilution effect question its generality, robustness to the definition of 'disease risk' and spatial scale (Randolph & Dobson 2012; Salkeld *et al*. 2013; Wood & Lafferty 2013; Wood *et al*. 2014). More to the point, we still cannot predict when diversity *will* reduce disease. This problem arises especially when reports of the dilution phenomenon do not mechanistically pinpoint the underlying interactions that reduce disease (e.g. Allan *et al*. 2009; Clay *et al*. 2009). Thus, developing and testing a predictive, mechanistic framework for dilution could help us focus on *why*, rather than just *how frequently* dilution occurs.

Here, we take a modular approach to this problem, focusing on the traits and interactions among a few species. We develop and test a model of the interactions between two host species, their shared parasite and resource. Thus, we narrow our focus to the local scale (*sensu* Holt *et al*. 2003), rather than a regional one (e.g. Johnson *et al*. 2013; Mihaljevic *et al*. 2014). Extant dilution models often assume asymmetries in species' epidemiological traits/parameters (e.g. Schmidt & Ostfeld 2001; Rudolf & Antonovics 2005; Ogden & Tsao 2009; Roche *et al*. 2012), and the most convincing empirical studies measure these traits (LoGiudice *et al*. 2003; Johnson *et al*. 2013; Lacroix *et al*. 2014). However, unlike most extant models (e.g. Dobson 2004; Keesing *et al*. 2006; Johnson & Thieltges 2010; Roche *et al*. 2012) and experiments (e.g. Johnson

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et al. 2008; Becker *et al*. 2014; Venesky *et al*. 2014; Wojdak *et al*. 2014), we allow our host species to dynamically interact and mechanistically influence each other's densities via these traits. Then, we explore a range of realistic outcomes in our community module (three case studies) by measuring intraspecific variation in the traits of our focal host (*sensu* Bolnick *et al*. 2011). Finally, we parameterise and test our model with corresponding multi-generational experiments. This novel, synthetic approach, highlights key interactions overlooked by other theory and experiments. We show how community ecology (resource competition and R^*) and epidemiology (potential of disease spread, R_0) can govern the success, reveal a recurrent cost (competition), and unveil a potential byproduct (spillover) of local dilution. As a result, we push beyond the controversy towards a more mechanistic, experimentally tested evaluation of the dilution effect.

To build this model, we return to those 'local dilution mechanisms' (Keesing *et al*. 2006, 2010; Johnson & Thieltges 2010), and most importantly, their interactions. First, diluter species can *reduce encounters* between focal hosts and parasites. For parasites transmitted environmentally, this occurs via a 'vacuum mechanism': resistant diluter species remove parasites from the environment while rarely (or never) becoming sick. Through this removal, diluters lower the risk of infection for the focal host (Johnson & Thieltges 2010). Second, diluters can *regulate* focal host populations via competition for space or resources. All else equal, such regulation reduces density-dependent transmission for environmentally distributed parasites (Anderson & May 1981). These two mechanisms (encounter reduction and host regulation) operate simultaneously in the 'friendly competition module' (Hall *et al*. 2009a). Competition typically depresses fitness of both hosts; yet, in 'friendly competition' one competitor can indirectly benefit from reduced disease (i.e. parasite-mediated apparent facilitation). The friendly competition module must be widespread, since species often encounter the same parasites when competing for resources or space (Freeland 1983; Price *et al*. 1988). Examples likely include hantavirus transmitted among rodents (Clay *et al*. 2009; Suzan *et al*. 2009), *Schistosoma* among snails (Johnson *et al*. 2009), parasites in intertidal communities (Johnson & Thieltges 2010), emerging diseases in amphibians (Johnson *et al*. 2013; Becker *et al*. 2014), fungal pathogens and viruses in plant communities (Mitchell *et al*. 2002; Lacroix *et al*. 2014; Rottstock *et al*. 2014), potentially important agricultural examples (Boudreau 2013) and, at least theoretically, perhaps even Lyme disease (Ogden & Tsao 2009). Thus, a mechanistic understanding of dilution in many systems may require embracing 'friendly competition'.

At first glance, friendly competition seems destined to promote successful dilution. After all, friendly competition rests on two mechanisms – encounter reduction and host regulation – that both decrease transmission. Yet, interactions between these mechanisms pose four crucial uncertainties. First, focal hosts that compete strongly could constrain the density of competitor/diluters. Sparse competitor/diluters may not sufficiently reduce encounters of hosts with parasite propagules, particularly when focal hosts create large epidemics. Second, competitor/diluters (if not completely resistant) could then be overwhelmed with parasite propagules and suffer spillover (amplified disease) from uncontrolled focal host epidemics. Third, competition from diluters could strongly depress focal host density. Even in cases where competitor/diluters reduce infection prevalence, they could still decrease density of healthy (uninfected) focal hosts. Fourth, the relative cost of competition and benefit of dilution could vary by perspective, depending on the metric used to define 'disease risk' (infection prevalence versus density of infected hosts). Each uncertainty hinges on traits of species involved: how strongly focal hosts compete with diluters (R^*) and how effectively they spread disease (R_0).

Here, we compare three empirically motivated case studies to explore the above uncertainties inherent in friendly competition. By allowing feedbacks among interacting species, we reveal that the outcome of dilution (measured both in terms of infection prevalence and density of infected hosts) does not simply mirror the additive effects of host regulation (competition) and encounter reduction (parasite removal). More specifically, we show that the outcome of dilution (success, failure, or ambiguity/irrelevance) depends on the interactions between a focal host's ability to compete and its ability to spread disease. This pairing of theory and experiments offers novel insights into the friendly competition module, and brings predictive clarity to the dilution effect among competitors.

MATERIALS AND METHODS

Study system & model specification

Our focal host zooplankter (*Daphnia dentifera*) non-selectively grazes on phytoplankton, and is the dominant grazer in many North American freshwater lakes (Tessier & Woodruff 2002). Across many of these lakes, this host experiences yearly epidemics of a virulent fungus *Metschnikowia bicuspidata* in late summer and fall (Hall *et al*. 2010b). *M. bicuspidata* can infect several zooplankton species, but we have only observed severe epidemics in our focal host species (Hall *et al*. 2009a). Community assembly of zooplankton in these lakes is predominantly determined by physical constraints (lake depth) and the degree of fish predation (Tessier & Woodruff 2002). Another zooplankter grazer, *Ceriodaphnia sp*., co-occurs with our focal host in shallow lakes with some deep water refuge from fish predators (Tessier & Woodruff 2002). In lakes where our focal host and this competitor/diluter co-occur, epidemics tend to be smaller for the focal host. This observation offers tentative support for a dilution effect among these species (Hall *et al*. 2010b).

Friendly competition emerges inherently from this natural history, which we depict graphically (Fig. 1, centre) and describe mathematically (Box 1). For a robust mathematical analysis of a similar model, see Cáceres *et al*. (2014). Susceptible focal hosts (S_{FH}) filter water at a foraging rate (f) and convert their algal resource (R) into births with conversion efficiency (e). While foraging non-selectively on algae, hosts inadvertently consume spores (Z) and thus become exposed to the virulent fungus *M. bicuspidata*, also at rate (f) (Hall *et al*.

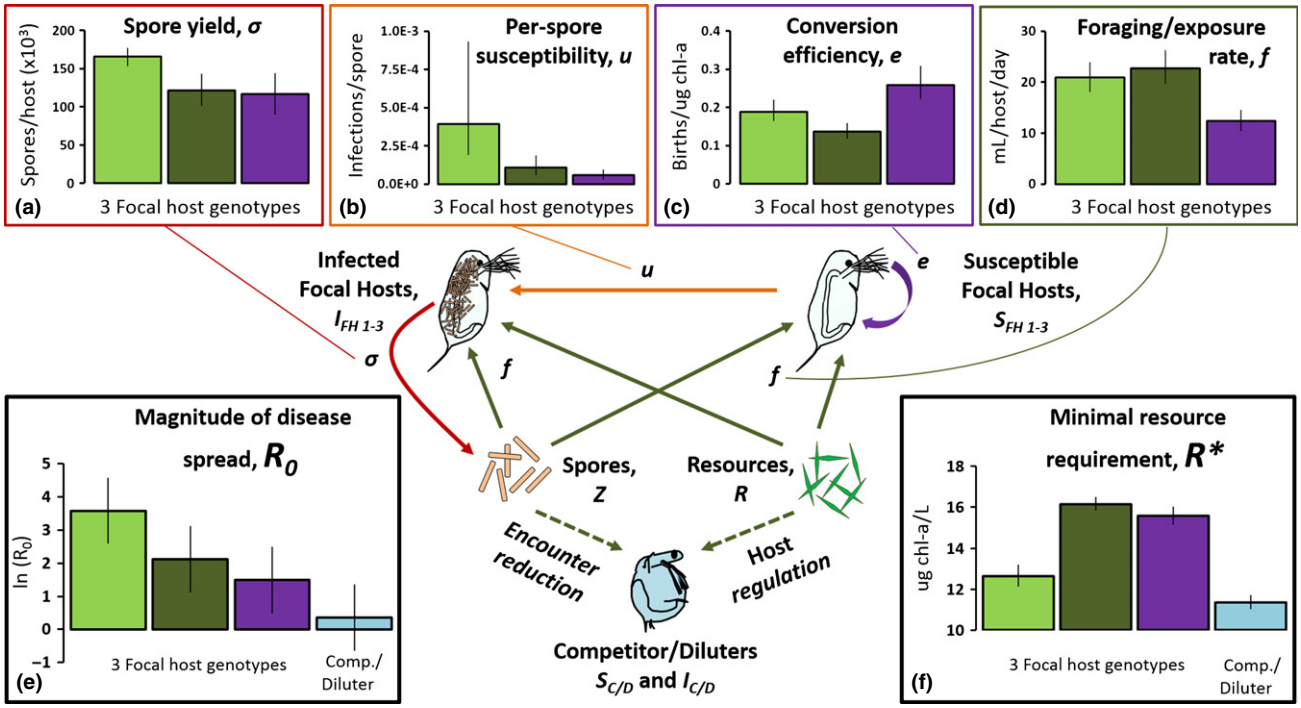

Figure 1 Focal host genotypes (indexed by FH 1–3) vary in four key traits which determine an index of disease spread (R_0) and an index of competitive ability (R^*). Infected focal hosts (a) produce spore yield σ and (b) become infected with per-spore susceptibilities u. Susceptible focal hosts (c) convert resources into births with conversion efficiencies e and (d) encounter algal resources and spores at foraging/exposure rates f. Competitor/diluters (indexed by C/D) reduce disease by consuming resources (host regulation) or spores (encounter reduction). Competitor/diluter traits are not shown. Variation in traits drives differences in focal host and competitor/diluter phenotypes, summarised as (e) the potential for disease spread, R_0, and (f) minimal resource requirements, R^*. (Strong competitors have low R^*'s). Error bars are bootstrapped 95% confidence intervals. Differences among these focal host genotypes lead to the qualitative differences seen in the model simulations (left columns in Figs. 2-4).

2007). Post-exposure, susceptible focal hosts enter the infected class (I), with per-spore susceptibility (u). Once infected, these hosts cannot recover, and host death rate increases from parasite virulence (v). After death, hosts release a number (σ) of fungal spores back into the environment, fulfilling obligate killer epidemiology, common to a variety of disease systems (Ebert & Weisser 1997). Spore yield increases with resources (see Appendix S1 in Supporting Information for modelling details; Hall *et al.* 2009b). Critically, focal host genotypes vary in these traits, translating into variation in both competitive ability (R^*) and the potential for disease spread (R_0). Susceptible competitor/diluters *Ceriodaphnia sp.* ($S_{C/D}$) compete with focal hosts for algae, but strongly resist infection from consumed spores (Hall *et al.* 2010b). Thus, this competitor/diluter could reduce disease via spore vacuuming (encounter reduction) and/or competition (regulation of susceptible hosts). Competition also constrains the density of competitor/diluters, which limits their net vacuuming rate.

Trait measurements

In our mechanistic framework for friendly competition, traits ultimately determine the fate of the dilution effect. We measured critical traits (foraging/exposure rate f, conversion efficiency e, susceptibility u, virulence v and spore yield σ) for three focal host genotypes and one diluter genotype of a separate species. All genotypes were chosen from existing laboratory cultures that had been isolated from lakes in southwestern Michigan. Using limited prior knowledge of these genotypes (Hall *et al.* 2010a), we selected our three focal host genotypes for our case studies that spanned a gradient of overall resistance to infection (exposure times susceptibility; $f \times u$). This provided us with the trait space necessary to explore a range of dilution outcomes.

Prior to trait measurement assays, all genotypes were grown in isoclonal cultures and fed high quality laboratory-cultured algae (*Ankistrodesmus falcatus.*). Cultures were maintained in filtered (Pall A/E: 1.0 μm) lake water under ideal conditions for three generations in order to standardise any maternal effects. We estimated foraging rate (f) with a foraging assay; per-spore susceptibility (u) with an infection assay (Hall *et al.* 2010a); and conversion efficiency (e), virulence (v) and spore yield (σ) with a life table experiment (see Appendix S1 for details and parameter estimation; Fig. 1a–d). We replicated trait measurement assays by genotype and bootstrapped 95% confidence intervals in R (R Development Core Team 2008).

Next, we summarised the traits of our focal host genotypes using model-derived indices of the potential for disease spread (R_0: Anderson & May 1981) and competitive ability (R^*: Tilman 1977). Strong competitors have low R^*'s (minimal resource requirements); strong disease spreaders have high

Box 1 A dynamical model describing changes in host, parasite and resource densities

Susceptible hosts (S) are non-selective feeders and encounter parasites (Z) while foraging for resources (R). Parasites are obligate killers and hosts do not recover. Infected hosts (I) also forage and reproduce. Spore yield (σ) is a function of resources (Fig. S2). i = Focal Host 1–3 or the Competitor/Diluter. Traits (parameters for e_i, f_i, u_i, σ_i and v_i) were measured with laboratory assays (Fig. 1). These differences cause qualitative differences in simulations (Figs 2–4). For all simulations, background death rate $d = 0.05$; spore death rate $m = 0.2$; resource growth rate $r = 0.9$; resource carrying capacity $K = 250$.

Host dynamics:

$$\frac{dS_i}{dt} = \overbrace{e_i f_i R(S_i + I_i)}^{\text{Births}} - \overbrace{dS_i}^{\text{Deaths}} - \overbrace{u_i f_i Z S_i}^{\text{Transmission}}$$

$$\frac{dI_i}{dt} = \overbrace{u_i f_i Z S_i}^{\text{Transmission}} - \overbrace{(d + v_i) I_i}^{\text{Increased mortality}}$$

Parasite dynamics:

$$\frac{dZ}{dt} = \sum_i \overbrace{\sigma_i(R)(d+v_i)I_i}^{\text{Parasite release}} - \sum_i \overbrace{f_i Z(S_i+I_i)}^{\text{Parasite removal}} - \overbrace{mZ}^{\text{Parasite mortality}}$$

Resource dynamics:

$$\frac{dR}{dt} = \overbrace{rR\left(1 - \frac{R}{K}\right)}^{\text{Resource growth}} - \sum_i \overbrace{f_i R(S_i + I_i)}^{\text{Resource removal}}$$

Definitions and units for parameters and variables:

S (susceptible host density; L^{-1})
I (infected host density; L^{-1})
Z (spore density; L^{-1})
R (resource density; μg chl-α L^{-1})
e (conversion efficiency; births μg chl-α^{-1})
f (foraging rate; L day^{-1})
d (death rate; day^{-1})
u (susceptibility; infections $spore^{-1}$)
v (virulence; day^{-1})
σ (spore yield; spores $host^{-1}$)
m (spore loss; day^{-1})
r (resource growth; day^{-1})
K (resource carrying capacity; μg chl-α L^{-1})

R_0's (basic reproductive ratios of the parasite). When combined, these two indices delineated three distinct phenotypes of the focal host (R_0: Fig. 1e; R^*: Fig. 1f). We featured these three phenotypes in the three case studies discussed below. Case 1 uses a focal host with low R^* and high R_0 (Fig. 1, first [light green] bars); case 2 uses a focal host with high R^* and moderate R_0 (Fig. 1, second [dark green] bars); case 3 uses a focal host with high R^* and low R_0 (Fig. 1, third [purple] bars). The diluter had the lowest R^* and lowest R_0, indicating that it competed strongly but spread disease very poorly (without complete resistance; Fig. 1e, f, fourth [blue] bars).

Model predictions

Using our dynamical model (Box 1), we assessed whether the addition of the competitor/diluter reduced disease for each focal host, both in terms of infection prevalence and density of infected focal hosts. We simulated our model using the deSolve package in R. Parameters are defined in Box 1. Estimates for conversion efficiency e, foraging/exposure rate f, susceptibility u, virulence v and spore yield σ varied among genotypes and were estimated with the assays described above (Fig. 1 and Fig. S1). Other parameter estimates (maximum algal growth rate r, algal carrying capacity K, spore loss rate m and background death rate d) are described in Appendix S1. All simulations began with low density of focal hosts and/or diluters (S_{FH} = 1 L^{-1}, $S_{C/D}$ = 0 or 1 L^{-1}, R = 35 μg chl-α L^{-1} and Z = 0 L^{-1}) and allowed hosts to increase in density for 15 days (as in the experiment below). Differences in densities on day 16 arose from differences in traits between genotypes. On day 16, we simulated epidemics by adding spores (Z = 5000 L^{-1}). We plotted infection prevalence and log-transformed infected host density and uninfected host density over the first 31–35 days of the epidemics, according to the length of each corresponding mesocosm experiment.

Mesocosm experiments

Parallel experiments grew isoclonal populations of each focal host genotype, both alone and with the competitor/diluter. Mesocosm experiments were housed in 75-litre acid-washed polyethylene tanks in a climate-controlled room and grown under a 16 L: 8 D light cycle. Tanks were filled to 60 litres with a mixture of 80% tap water (detoxified with Kordon Amquel Plus and Novaqua Plus) and 20% filtered lake water. Evaporated water was replaced throughout the experiments. Initial doses of nitrogen and phosphorus were added to the tanks in the form of sodium nitrate and potassium phosphate (300 μg L^{-1} N as $NaNO_3$ and 20 μg L^{-1} P as K_2HPO_4.). We subsequently replenished 5% of this initial nutrient dose per day throughout the experiment. We inoculated all tanks with 50 mg dry weight of *Scenedesmus acutus* and let this algae grow for 1 week prior to introducing any hosts.

The experiment was conducted in two blocks: the case 1 genotype in 2009 and cases 2 and 3 in 2012. Both experiments crossed focal host genotype with presence/absence of the diluter and included diluter-only tanks. The 2012 experiment also included algae-only tanks. All treatments were replicated 4–6 times. In 2012, tanks were inoculated with low densities of focal hosts (S_{FH} = 15 L^{-1}) and allowed to increase in density for 2 weeks. Then, appropriate tanks were inoculated with equivalent densities of competitor/diluters ($S_{C/D}$ = 100 L^{-1}) and allowed to increase in density for an additional 2 weeks. In

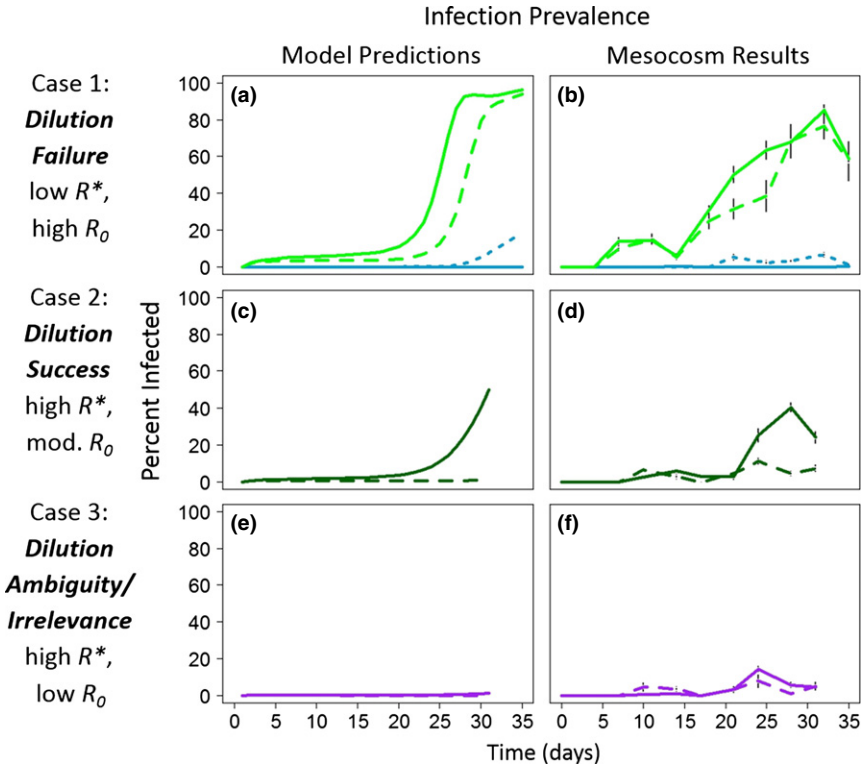

Figure 2 Variation in infection prevalence and the outcome of dilution depend on competitive ability (R^*) and the potential for disease spread (R_0) among three focal host genotypes. Parameterised model simulations (left column) qualitatively predict experimental results (right column). (a, b) Competitor/diluters ***fail*** to significantly reduce infection prevalence for focal hosts that compete strongly and spread disease extensively. Moreover, disease spills over into the diluter population, presenting an amplification effect. (c, d) Competitor/diluters ***succeed*** in significantly reducing infection prevalence for focal hosts that compete weakly and spread disease moderately. (e, f) Dilution is ***irrelevant*** (in terms of infection prevalence) for focal hosts that compete weakly and spread disease poorly. Solid lines: focal hosts alone; dashed lines: focal hosts with competitor/diluters; blue solid lines: competitor/diluters alone; blue dotted lines: competitor/diluters with focal hosts. Competitor/diluters shown only in (a, b). Error bars are standard errors.

2009, tanks started with similar conditions to 2012. We used greater starting host densities than in the simulations because hosts in the simulations approached their equilibria much more rapidly than in the experiment. In both experiments, epidemics were initiated with the addition of fungal spores after 4 weeks ($Z = 5000$ L^{-1}). Host densities at this point corresponded qualitatively to host densities in simulations when spores were added (Fig. 2). We sampled one litre from each tank twice per week with 80 μm mesh sieves. We tracked infected and uninfected host densities as well as infection prevalence through time (using microscopes to quantify samples and visually diagnose infections [50×]). Epidemics lasted 3–5 host generations, and approximately three parasite generations.

We quantified epidemics for each tank in our experiments by integrating the area under time series of infection prevalence and log-transformed infected host density. Then, we compared epidemics with and without the competitor/diluters (and among focal host genotypes) with *t*-tests. Similarly, we quantified uninfected host density for each tank by integrating the area under the log-transformed density curves, and compared these quantities with integrated density *t*-tests. Visually, these tests compare the areas under the curves presented in Figs 2–4. Total host densities are also shown in Appendix S2 (Fig. S3). We also used *t*-tests to compare the density of diluters competing with our different focal hosts at the time of spore addition.

RESULTS

Overall, model predictions qualitatively matched experimental results (Figs 2–4). We cannot test for block differences between the 2009 and 2012 mesocosm experiments. However, the agreement between parameterised model predictions and experimental results allows us to focus our argument on variation among the traits of our focal host genotypes. Our trait measurements revealed that the competitor/diluter was the superior competitor (lowest R^*: Fig. 1), and was thus predicted to outcompete all focal host genotypes over long periods of time. However, R^*'s were similar enough that competitive replacement was slow (Grover 1997), and did not occur in any experiments. Indeed, our simulations predicted that competitive replacement ($S_{FH} < 1$ L^{-1}) would only occur after 216 days of competition (~ 22–30 generations), even for our weakest competing focal host. With these points in mind, during the 31–35 days of our experimental epidemics, we show three trait-dependent outcomes of dilution among competing hosts: dilution failure (case 1), dilution success (case 2) and dilution ambiguity/irrelevance (case 3).

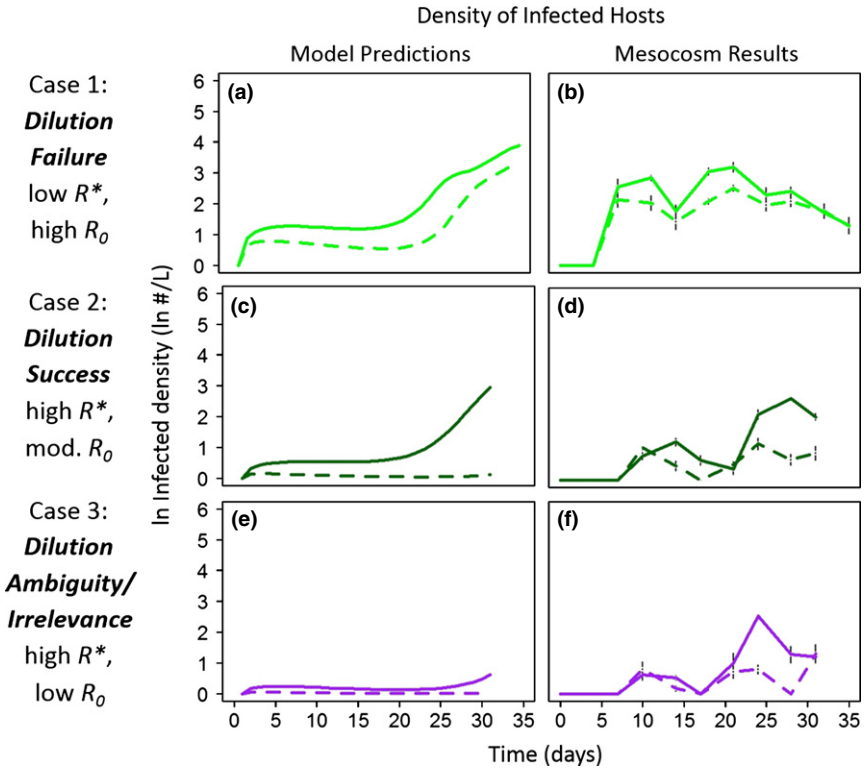

Figure 3 Variation in density of infected focal hosts depends on competitive ability (R^*) and the potential for disease spread (R_0) among three focal host genotypes. Parameterised model simulations (left column) qualitatively predict experimental results (right column). (a, b) Competitor/diluters **fail** to significantly reduce the density of infected focal hosts that compete strongly and spread disease extensively (although they do marginally reduce the density of these infected hosts). (c, d) Competitor/diluters **succeed** in reducing the density of infected focal hosts that compete weakly and spread disease moderately. (e, f) Competitor/diluters also succeed in reducing the density of focal hosts that compete weakly and spread disease poorly. However, competitor/diluters were irrelevant in terms of infection prevalence for this host (Fig. 2); thus, dilution is **ambiguous**. Solid lines: focal hosts alone; dashed lines: focal hosts with competitor/diluters. Error bars are standard errors.

Case 1: Dilution failure (strong competitor, large epidemic)

The dilution effect failed for the focal host predicted to compete strongly (low R^*) and spread disease extensively (high R_0). When alone, these hosts drove large epidemics. Infection prevalence and infected density were both higher than the other focal host genotypes (Figs 2 and 3a, b; *t*-tests, all $P < 0.001$). Meanwhile, at the start of epidemics, diluters reached lower densities with this focal host than with the other two (Fig. 4 a, b; *t*-tests, both $P < 0.01$). Due in part to this competitive constraint, competitor/diluters failed to significantly reduce infection prevalence during epidemics (Fig. 2a, b; *t*-test, $P > 0.3$). Most likely, competitor/diluters were not dense enough to inhibit disease by 'vacuuming' the large number of spores released by this focal host (Fig. 1a). Although they marginally reduced the density of infected focal hosts, this effect was not statistically significant (Fig. 3a, b; *t*-test, $P < 0.1$). Presence of competitor/diluters did lower mean densities of uninfected focal hosts (*t*-test, $P < 0.01$), although focal host populations crashed during epidemics regardless (Fig. 2c, d). Finally, spillover from the large focal host epidemics even caused a small outbreak (i.e. amplified disease) in the diluter population (Fig. 2a, b; *t*-test, $P < 0.05$). Thus, when focal hosts compete strongly and spread disease extensively, friendly competition can produce a double failure: uncontrolled disease for focal hosts and spillover of disease into the competitor/diluters (i.e. an amplification effect).

Case 2: Dilution success (weak competitor, moderate epidemic)

The dilution effect succeeded for the focal host with weak competitive ability (high R^*) and moderate potential to spread disease (moderate R_0). When alone, these focal hosts drove intermediate epidemics (Figs 2 and 3c, d). Infection prevalence was lower than case 1 ('failure'; *t*-test, $P < 0.001$) and higher than case 3 ('ambiguity/irrelevance'; *t*-test, $P < 0.05$). Furthermore, density of infected hosts was lower than case 1 (*t*-test, $P < 0.0001$) and equivalent to case 3 (*t*-test, $P > 0.7$). At the start of epidemics, diluters reached higher density than in case 1 (Fig. 4c, d; *t*-test, $P < 0.01$), but were equivalent to case 3 ($P > 0.7$). Because of the moderate epidemic size and their high density, diluters reduced both infection prevalence (Fig. 2c, d, *t*-test, $P < 0.05$) and density of infected hosts (Fig. 3 C,D, *t*-test, $P < 0.01$) in the focal host population. No spillover was detected (*t*-test, $P > 0.5$). The model also predicted a small reduction in uninfected host density with competitor/diluters (especially relative to case 3; Fig. 3c). However, this reduction was too small in the experiment for us to detect statistically (*t*-test, $P > 0.4$). Thus, for focal hosts with weak competitive

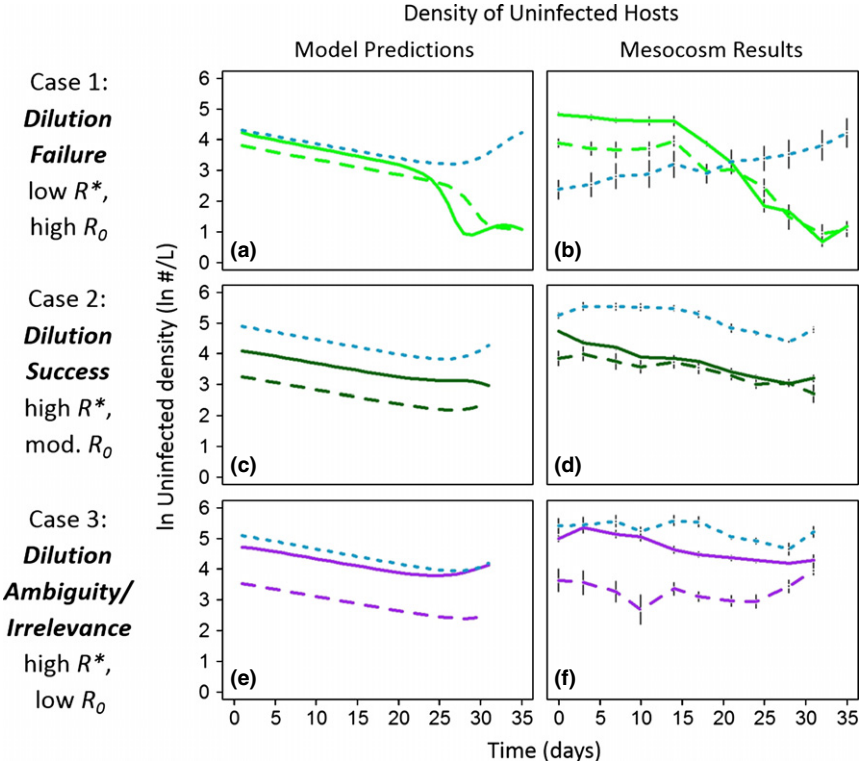

Figure 4 Variation in density of uninfected (susceptible) focal hosts and competitor/diluters depends on competitive ability (R^*) and the potential for disease spread (R_0) among three focal host genotypes. Parameterised model simulations (left column) qualitatively predict experimental results (right column). Competitor/diluters significantly reduce the density of (a, b) uninfected focal hosts that compete strongly and spread disease extensively and (e, f) focal hosts that compete weakly and spread disease poorly. (c, d) Density of uninfected focal hosts that compete weakly but spread disease moderately is unaffected by competitor/diluters. Competitor/diluter density prior to the epidemic is lower when competing with (a, b) the strong-competitor focal host than when competing with (c–f) the two weak-competitor focal hosts. Solid lines: focal hosts alone; dashed lines: focal hosts with competitor/diluters; blue dotted lines: competitor/diluters with focal hosts. Error bars are standard errors.

ability and moderate R_0, the dilution effect succeeded with minimal density cost and no spillover (no amplification).

Case 3: Dilution ambiguity/irrelevance (weak competitor, small epidemic)

The presence of diluters had ambiguous effects (due to multiple definitions of 'disease risk') for focal hosts with weak competitive ability (high R^*) and low potential to spread disease (low R_0). Simulated epidemics spread very slowly, remaining below 1% infection prevalence (Fig. 2e) and one infected host per litre (Fig. 3e). In the experiment, infection prevalence was lower for this host alone than in case 1 ('failure'; t-test, $P < 0.001$) and case 2 ('success'; t-test, $P < 0.05$) (Fig. 2f). Density of infected hosts was also lower for this host alone than in case 1 (t-test, $P < 0.0001$), but not case 2 (t-test, $P > 0.1$) (Fig. 3f). The model did not predict this detail (Fig. 3e). Competitor/diluters did not significantly reduce infection prevalence in this focal host (Fig. 2f; t-test, $P > 0.7$), likely because competitor/diluters were nearly as good at spreading disease as these low-R_0 focal hosts (similar R_0's: Fig. 1e). Thus, diluters were irrelevant in terms of infection prevalence, despite reaching densities similar to case 2 (Fig. 4f; t-test, $P > 0.7$). With so little disease, spillover (i.e. amplification) was neither predicted nor detected (t-test, $P > 0.5$). Nevertheless, competitor/diluters did significantly reduce density of infected focal hosts during the epidemic (Fig. 3f; t-test, $P < 0.05$). This effect was likely driven by the competitive interaction between host species rather than vacuuming, since infection prevalence was not significantly different between treatments ($P > 0.7$). Indeed, competitor/diluters vastly outnumbered this focal host overall, and uninfected focal host density was also strongly reduced by competition (Fig. 4e, f; t-test, $P < 0.05$). For focal hosts with these traits, the outcome of dilution is ambiguous and depends on the definition of disease risk (infection prevalence versus density of infected hosts). From a density perspective, dilution was successful. However, from a prevalence perspective, dilution was irrelevant.

DISCUSSION

Our three case studies mathematically predicted and experimentally confirmed three qualitatively different outcomes of the friendly competition module. To predict these differences, we mechanistically linked competition, disease spread and outbreak size (both in terms of prevalence and number of infected hosts). More specifically, the outcome of dilution among competitors—success, failure, or ambiguity/irrelevance—depended predictably on encounter reduction (i.e. vacuuming), host regulation (i.e. the strength of competition, R^*), and the magnitude of disease spread (indexed by R_0). In case

1, the focal host genotype was a strong competitor (low R^*) and a strong spreader of disease (high R_0). The dilution effect failed for this focal host, because competition constrained the diluter population (limiting vacuuming and constraints on the focal host), while large epidemics overwhelmed diluters with infective spores. Disease even spread to competitor/diluters via spillover from the focal host epidemic (i.e. an amplification effect). In case 2, the focal host genotype was a weak competitor (high R^*) and a moderate spreader of disease (moderate R_0). The dilution effect succeeded here, because more diluters (i.e. stronger host regulation) sufficiently vacuumed the moderate density of infective spores. In case 3, the focal host genotype was a weak competitor (high R^*) and a weak spreader of disease (R_0). Here, the dilution outcome became ambiguous, because competitor/diluters significantly lowered the density of infected focal hosts but were irrelevant regarding infection prevalence (because prevalence was so low). These three case studies emphasise the range of dilution outcomes (success, failure and ambiguity/irrelevance) that can occur even within a simple community module (Bolnick *et al*. 2011). Yet, using measured traits of our hosts as a mechanistic guide, we have explained – and even predicted – these seemingly idiosyncratic outcomes (e.g. Salkeld *et al*. 2013).

Our dynamical model and multi-generational experiments enabled novel synthesis of encounter reduction and host regulation (but see Keesing *et al*. 2006; Johnson *et al*. 2008, 2012a; Wojdak *et al*. 2014). These dilution mechanisms do not act independently, for two reasons. First, competition between focal hosts and diluters determines regulation of focal hosts (potentially reducing net disease spread), and also the magnitude of the net vacuuming (encounter reduction) provided by the competitor/diluters. Net release of infective spores is the product of infected focal host density and their per-capita spore yield (Fig. 1). Likewise, 'net vacuuming' is the product of competitor/diluter density and their per-capita vacuuming rate. Focal hosts which compete strongly (case 1) do not receive the disease-mediating benefits of either strong regulation or strong net vacuuming. Weaker competitors (cases 2 and 3) experience some combination of stronger regulation *and* higher net vacuuming. Thus, the outcomes of dilution over multiple host generations could hinge sensitively on relatively small differences in competitive ability.

A second dilution mechanism interaction, density-mediated feedbacks, also likely contributed to the outcomes in our model and experiment. Consider, for example, the following hypothetical four-step feedback cycle: (1) Disease outbreaks kill focal hosts, (2) As hosts die, diluters are released from competition and increase in density, (3) A higher density of diluters enhances their net vacuuming rate, (4) Higher net vacuuming reduces disease spread and prevents focal hosts from dying. We cannot directly track this four-step process in our model and experiments, because all four steps occur simultaneously. Therefore, we cannot fully disentangle the effects of host regulation and encounter reduction. However, our model and experiments suggest that the net outcome of this feedback cycle likely depends on traits of the interacting species: their relative competitive abilities, diluters' per-capita vacuuming rate and the ability of focal hosts to spread disease. These feedbacks cannot occur in experiments that only last a single host generation, even though they likely operate in host communities in nature. Thus, these dynamics need to become part of the conceptual repertoire for the dilution effect.

The competition component of our 'friendly competition' model may unify some existing theory for dilution. Competition in extant dilution theory has been modelled as an interaction coefficient among hosts (Schmidt & Ostfeld 2001), the effect that a diluter species has on overall species density (Rudolf & Antonovics 2005; Ogden & Tsao 2009), and how host density scales with richness (Roche *et al*. 2012; Mihaljevic *et al*. 2014). These various modelling forms and assumptions have obscured the recurrent role that competition has played in the dilution effect literature. Simultaneously and independently however, they have emphasised the importance of competition in modulating the dilution effect. Model assumptions (e.g. specifically *how* host richness scales with density) can fundamentally change whether or not a dilution effect is predicted (Rudolf & Antonovics 2005; Ogden & Tsao 2009; Mihaljevic *et al*. 2014). This result is synonymous with ours: the outcome of dilution can hinge on the strength of competition among host species. We argue that parameterised resource competition (either explicit or phenomenological) is a preferable, clear alternative to cryptic and weighty model assumptions about the densities of interacting species. Parameterised competition can mechanistically determine – as an outcome, not an assumption – the strength of competition and its importance for dilution.

Likewise, we argue that competition (manifested as host densities) is an important design component in experiments that test for dilution effects. Competition among hosts is a prominent feature in empirical plant and animal dilution systems (Mitchell *et al*. 2002; Johnson *et al*. 2008, 2009; Clay *et al*. 2009; Hall *et al*. 2009a; Johnson & Thieltges 2010; Becker *et al*. 2014; Lacroix *et al*. 2014; Rottstock *et al*. 2014). Substitutive experimental designs are most appropriate when hosts compete strongly, thus reducing disease (e.g. Mitchell *et al*. 2002; Rottstock *et al*. 2014). Especially in single generation experiments, the strength of host regulation is artificially imposed (via densities of hosts in the experimental design). Substitutive designs can confound host regulation with other mechanisms (e.g. encounter reduction), and artificially strong host regulation could overshadow the relevant mechanisms that reduce disease in nature. Great care must therefore be taken to ensure that experimental densities reasonably resemble natural communities. Designs that manipulate both host density and community composition can decouple the effects of host regulation and encounter reduction (Johnson *et al*. 2008; Wojdak *et al*. 2014). However, these designs still obscure the dynamical feedbacks and interactions described above. Thus, we urge more experimental tests of dilution theory that incorporate multi-generational competition (e.g. Mitchell *et al*. 2002; Johnson *et al*. 2012a; Rottstock *et al*. 2014).

Focusing on density of infected hosts versus infection prevalence might change the interpretation of friendly competition here. For instance, competitor/diluters reduced infection *prevalence* in only one of our case studies. However, they reduced *density* of infected focal hosts in two of our three case studies (and marginally reduced it in the third). Such a density-focused outcome might herald unequivocal success in systems involving wildlife reservoirs of human disease, such as schistosomiasis

(Johnson *et al.* 2009) and hantavirus (Clay *et al.* 2009; Suzan *et al.* 2009). In these systems, reduced density of wildlife hosts infected with human parasites would signal a favourable outcome of dilution, as long as there is no compensatory *increase* in infection prevalence (e.g. Ogden & Tsao 2009). Case 3 ('ambiguity/irrelevance') would be a success under these criteria. However, this same outcome (reduced density of infected *and* susceptible hosts) might prove too costly for wildlife diseases like amphibian chytrid (Bd; Venesky *et al.* 2014) and trematode infections (Ribeiroria; Johnson *et al.* 2013), or in agriculture (Boudreau 2013). For such hosts of economic or conservation concern, the regulatory component of friendly competition may unacceptably depress density of uninfected hosts, even if competitor/diluters do reduce infection prevalence (as in case 2, 'success'). Thus, the costs and benefits of friendly competition depend sharply on perspective (i.e. from human disease control vs. conservation/agriculture). Unless we clearly define our definition of 'dilution success' on a case-by-case basis, this ambiguity could clearly propagate more confusion in the dilution effect literature.

Our results also prompt a set of questions best framed over broader parameter space, temporal scale and spatial scales. First, a thorough mathematical analysis of our model would allow us to freely manipulate traits, eliminating the constraints of our three guiding empirical case studies (e.g. Cáceres *et al.* 2014). We could analyse the sensitivity of friendly competition's outcomes to variation in each host trait independently, and use our inferences to better disentangle the effects of host regulation and encounter reduction. Second, as parameterised in the model, our competitor/diluter can outcompete all focal hosts over long enough time periods. Theory for long-term dynamics of friendly competition therefore requires better representation of species niches that could promote coexistence between focal hosts and competitor/diluters. After all, these two hosts do coexist in nature (Tessier & Woodruff 2002; Hall *et al.* 2010b). Third, such realistic long-term theory may require embracing evolutionary changes in hosts. Both competition (Pimentel 1968) and disease (Duffy *et al.* 2012) can drive rapid evolutionary changes in genetically diverse host populations; however it is unclear how selection could regulate friendly competition and dilution through feedbacks (e.g. if all three of our focal host genotypes occurred together in a genetically diverse population). Fourth, armed with explicit dilution models, community ecologists could expand friendly competition to larger spatial scales. Do competition-colonisation tradeoffs (Tilman 1990) or life history-pathogen defence tradeoffs (Johnson *et al.* 2012b) link traits that both regulate local dilution *and* determine regional assembly of host communities? All four of these extensions (parameter space exploration, coexistence, evolution, and community assembly) require estimating the variation and covariation of host and diluter phenotypes in nature. With these data, we could search for traits that promote host coexistence, aid in dispersal and persistence among sites, and determine competitive ability (R^*) and the potential for disease spread (R_0). Insight into the variances and covariances among these traits in focal hosts and diluters in nature could ultimately catalyse a mechanistic eco-evolutionary framework for the dilution effect across landscapes and through ecological time.

Even without these extensions, friendly competition speaks to some immediate conservation and disease management concerns. For instance, when hosts compete for resources (e.g. Becker *et al.* 2014; Lacroix *et al.* 2014), reintroduction of diluters to control disease could exact an undesirable cost on density of focal hosts. Alternatively, diluters constrained by competition might fail to control disease in hosts that drive severe epidemics. In extreme cases of failure, diluters could even suffer disease, via spillover/amplification themselves. These hazards prompt precise delineation of potential future goals for disease management using the dilution effect. Sometimes, the goal might centre on boosting density of healthy focal hosts (e.g. in threatening wildlife diseases like amphibian chytridiomycosis: Becker *et al.* 2014; Venesky *et al.* 2014). In these cases, management decisions must balance the inherent cost of competition with the potential benefit of reduced disease. Alternatively, human disease control efforts (e.g. for hantavirus: Clay *et al.* 2009; Suzan *et al.* 2009) may warrant great reductions of the density of focal hosts through competition with diluters. In these instances, the inherent cost of competition from diluters might reap management benefits. All of these possibilities arise because local species interactions can potentially interfere with disease transmission but exact other ecological consequences. Thus, a more tested, dynamical, and mechanistic theory will push the dilution effect beyond its phenomenological foundation and help us better anticipate its success, failures, ambiguity, or irrelevance.

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AUTHORSHIP

ATS, CEC and SRH designed the study. ATS and DJC performed the trait measurement assays. ATS, DJC and SRH implemented the model. ATS led data collection and analysis. ATS wrote the first draft of the manuscript, and all authors contributed substantially to revisions.

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