Jessica L. Hite, Rachel M. Penczykowski, Marta S. Shocket, Alexander T. Strauss, Paul A. Orlando, Meghan A. Duffy, Carla E. Cáceres, and Spencer R. Hall. 2015. Parasites destabilize host populations by shifting stage-structured interactions. *Ecology* VOL:pp-pp.

APPENDIX S1. Model derivation and results.

Here we present an *SIZA* model parameterized for our focal host-parasite system. This model readily illustrates the parasite-mediated stabilization phenomenon (*H1 disease stabilizes via host mortality*, Figure S1) as shown previously for different epidemiology (Hilker & Schmitz 2008).

Our initial hypothesis (*H1 disease stabilizes via host mortality*) envisions that disease stabilizes Paradox-of-Enrichment-type host-resource cycles driven by prey escape (as proposed by Hilker & Schmitz 2008). Disease is stabilizing because parasites increase per capita mortality rate of hosts (averaged between infected and susceptible classes). This elevated mortality rate then raises the minimal resource requirement of the host and diminishes its ability to depress its resource so dramatically. Resources, in turn, reach higher densities; thus, their own negative density dependence helps to stabilize the consumer-resource system. Without parasite-inflicted mortality, consumers depress resources to low densities. At these low density levels, resources experience very high per capita mortality. When they can increase, they experience 'safety in numbers' (a strongly destabilizing form of positive density dependence). A model tailored around the natural history of our zooplankton host-algal resource-fungal parasite system can readily illustrate this parasite-driven stabilization phenomenon. The model tracks density of susceptible (S) and infected (I) hosts, free-living infectious spores of the fungus (Z), and algal

resources (A) (largely following Cáceres et al. 2014 and Hurtado et al. 2014). The model is (equ. S1, see also Table S1):

$$dS/dt = e\, f(A)\, A\, (S + \rho\, I) - d\, S - u\, f(A)\, S\, Z \quad \text{(S1.a)}$$

$$dI/dt = u\, f(A)\, S\, Z - (d + v)\, I \quad \text{(S1.b)}$$

$$dZ/dt = \sigma(A)\, (d + v)\, I - m\, Z - f(A)\, (S + I)\, Z \quad \text{(S1.c)}$$

$$dA/dt = r\, A\, (1 - A/K) - f(A)\, (S + I)\, A. \quad \text{(S1.d)}$$

Here, susceptible hosts (dS/dt, equ. S1.a) increase due to births, where e is conversion efficiency of algal carbon into host carbon, $f(A)$ is 'clearance rate' from consumer-resource theory (see below), and ρ is fecundity reduction imposed by parasites (i.e., virulence on fecundity). Susceptible hosts are then lost at background rate d and due to infection, where exposure is 'clearance rate', $f(A)$ (Hall et al. 2007), and u is per-spore susceptibility. Infected hosts then increase (dI/dt, equ. S1.b) due to these new infections, but then die at elevated rate $d + v$ due to virulence on survival (where v is the added mortality rate from parasites). Then, in the spore equation (dZ/dt, equ. S1.c), when infected hosts die, they release $\sigma(A)$ spores but are lost at background rate m (due to sinking, solar radiation [Overholt et al. 2012], consumption by other species [Hall et al. 2009c, Strauss et al. 2015]), etc.). They are also lost due to consumption by both infected and uninfected host classes. Finally, algal resources (dA/dt, equ. S1.d) increase logistically (where r is the maximal per capita growth rate, and K is the carrying capacity) but are lost due to consumption by both host classes.

In this model, algal density is connected to transmission in two fundamental ways. First, exposure to parasites equals 'clearance rate' from consumer-resource theory, $f(A)$:

$$f(A) = f / (h + A) \quad \text{(S2)}$$

where f is maximal feeding rate and h is the half-saturation constant of this type II functional response. Clearance rate here is the per-prey — and per-spore — risk of being eaten, with maximal rate f/h (when $A = 0$) that declines non-linearly as algal resources become more dense (i.e., the safety in numbers that generates positive density dependence for the algal resource and therefore drives consumer-resource cycles). Therefore, as algal resources become more dense (higher A), per capita exposure to parasites, $f(A)$, drops. Second, parasite production (spore yield) per infected host, $\sigma(A)$, increases proportionally to growth rate / birth rate of susceptible hosts (Hall et al. 2009a,b, Hall et al. 2010):

$$\sigma(A) = \sigma\, e\, f(A) \qquad \text{(S3)}$$

where σ converts growth rate of hosts into parasite mass. Maximal spore yield is $\sigma\, e\, f$.

This *SIZA* model (equ. S1-S3), equipped with the type II functional response, readily illustrates the parasite-mediated stabilization phenomenon (Figure S1) shown previously for different epidemiology (Hilker & Schmitz 2008). As parameterized, the host without disease (black lines, Fig. S1*a*) begins to oscillate (arrow) with its resource at lower carrying capacity (K) than during an epidemic (grey lines, showing total host density, $N = S + I$). Once it oscillates, the cycle amplitude (the difference between the maximal and minimal densities in the cycle) is smaller with disease than without disease. Algal resources also begin to oscillate at higher K in systems with parasites (Fig. S1*b*), of course, but mean algal biomass is also higher during epidemics than without parasites. Higher mean algal density reduces the destabilizing positive-dependence enjoyed by resources when they are more rare (i.e., when severely over-exploited). Prevalence of infection, p (where $p = I / (S + I)$) reaches a high level, then begins to oscillate; mean prevalence then declines with K (as the host oscillations increase: Fig. S1*c*). Mean per

capita death rate, $\bar{d}$, increases during epidemics relative to disease-free conditions (Fig S1*d*). Mean per capita death rate is simply the weighted average of death rates for both host classes:

$$\bar{d} = [\, d\, S + (d + v)\, I \,] / N \qquad \text{(S4)}$$

and largely mirrors the prevalence pattern with increasing *K*. Thus, the increased parasite-mediated per capita death rates of hosts delays the onset of cycling with *K* and produces smaller host oscillations through time once they begin. Therefore, epidemics can stabilize host dynamics.

The example shown (Fig. S1) illustrates two other points, and then we must offer a caveat. First, the examples show how a gradient of carrying capacity (*K*) in systems with disease creates some problems for the spurious prevalence-instability mechanism (*H2: nutrient enrichment destabilizes*). Imagine that higher nutrient supply (indexed by total phosphorus) correlates positively with *K* (a completely reasonable assumption in lakes). We see how mean prevalence first increases, then decreases with *K*. (Therefore, disease prevalence does not have to increase with *K per se* once systems begin to cycle; Fig. S1*a*). Then, Panels A and C combined show how higher mean prevalence does not have to necessarily correlate with higher cycle amplitude (the difference between minima and maxima of cycles) since amplitude increases with *K*. Thus, the enrichment-destabilization correlation (*H2*) makes common sense, but in this particular model, it is not an inevitable outcome. Second, as a side note, we see a hydra effect (Abrams 2009): host density during epidemics can be higher than host density without disease when systems cycle (Fig. S1*a*). However, a caveat: we must note that the *SIZA* model (equ. S1-S3) can produce other behaviors (described in detail by Hurtado et al. 2014). In particular, very large oscillations of hosts without disease can inhibit successful invasions with disease. The parameterized example does not show that behavior over the reasonable range of carrying

capacity, and we have never seen such behaviors in the field. Thus, we do not dwell on it here (since it seems more like an exotic non-linear behavior rather than a practical result).

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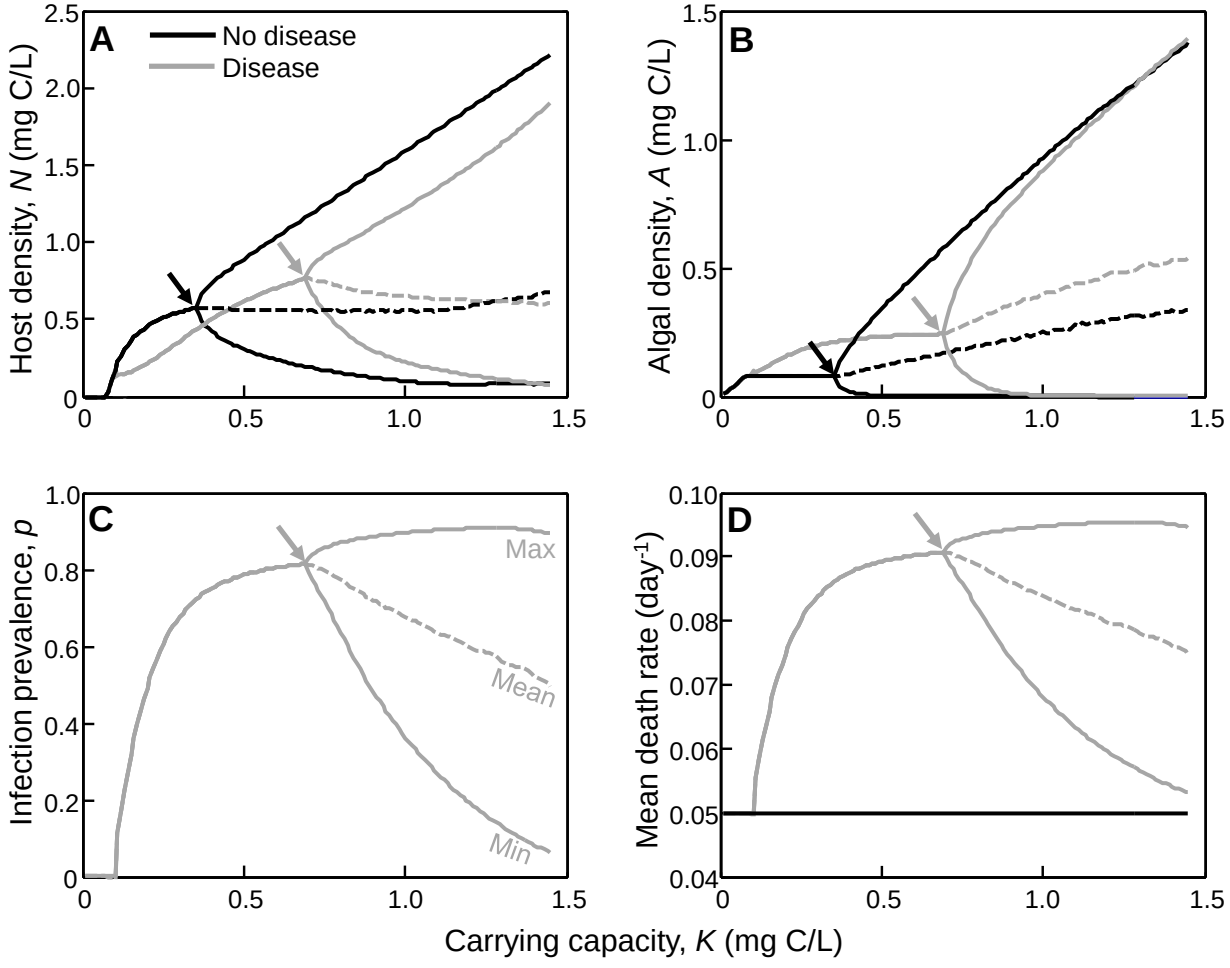
Table S1. Description of state variables, parameters, and functions in the *SIZA* model (equ. S1-S3) used to illustrate *H1 disease stabilizes via host mortality* (following Cáceres et al. 2014).

Symbol	Units	Meaning	Value
S	mg C/L	Susceptible host density	
I	mg C/L	Infected host density	
Z	mg C/L	Spore density	
A	mg C/L	Algal resource density	
t	days	Time	
d	day^{-1}	Background death rate of hosts	0.05[a]
e	-	Conversion efficiency	0.6[b]
f	$(mg\ C/L \cdot day)^{-1}$	Maximal feeding rate	0.3[c]
h	mg C/L	Half-saturation constant	0.2[d]
K	mg C/L	Carrying capacity of resource	0.01-1.4[e]
m	day^{-1}	Background loss rate of spores	0.9[f]
r	day^{-1}	Maximal growth rate of algal resource	0.8[b]
u	-	Per spore infectivity (susceptibility)	10[c]
v	day^{-1}	Added death rate due to infection	0.05[c]
ρ	-	Fecundity reduction due to infection	0.9[c]
σ	days	Spore release conversion parameter	15[c]
$\bar{d}$	day^{-1}	Mean death rate during epidemics (equ. A4)	

$f(A)$	day^{-1}	Type II clearance rate: $f(A) = f/(h + A)$
N	mg C/L	Total host density: $N = S + I$
p	-	Prevalence of infection: $p = I/N$
$\sigma(A)$	mg C/L· day^{-1}	Spore release: $\sigma(A) = \sigma\, e\, f(A)$

[a] Assuming a natural death rate plus some background mortality due to predation. [b] Reasonable for higher quality algae (Andersen 1997). [c] Measured for adult sized hosts, converted to a per carbon basis (Hall et al. 2009a, Hall et al. 2010). [d] Reasonable for focal host (Hall et al. 2007). [e] A range relevant to the study lakes. [f] A high loss rate, envisioning high losses due to sinking, solar radiation, and consumption by other species (Hall et al. 2009c, Overholt et al. 2012).

Figure S1. Examples of the stabilizing effects of disease (*H1 disease stabilizes via host mortality*), illustrated with a one dimensional bifurcation diagram along a gradient of resource carrying capacity (*K*). The disease free example is in black; systems with epidemics are drawn in grey. Oscillations start at the arrows. Once they begin, maxima, minima, and means of cycles are shown (as labeled in panel C). (*A*) Total host density, *N* (where $N = S + I$); (*B*) density of algal resources, *A*; (*C*) prevalence of infection, *p* (where $p = I/N$, or proportion infected); (*D*) weighted mean per capita death rate, $\bar{d}$ (equ. A4).



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APPENDIX S2. Additional details regarding methods, results for the field survey and experiments, and the effect of nutrient addition in the lake enclosure experiment on total phosphorous (TP) levels, host density, and infection prevalence.

Here, we provide additional methods for birth rate calculations (field survey and experiments) and for other aspects of the field experiment. We also show additional results from the lake enclosure experiment for total phosphorous (TP), host density (integrated over the epidemic season), and infection prevalence (integrated over the epidemic season).

Methods: Birth rate calculations

In the field survey, we calculated temperature-dependent birth rate in a way that incorporates diel migration of the host. This species of host typically migrates below the thermocline (into the 'metalimnion') of lakes during the day into deeper, colder, but still oxygenated (> 1.0 mg/L dissolved O_2 [DO]) waters. Then, at night, it moves above the thermocline into upper, warmer habitat (the 'epilimnion') (e.g., Duffy *et al.* 2005, Hall *et al.* 2005). Therefore, using temperature data, we calculated depth of the thermocline (during periods of stratification) by: (1) converting temperature data into densities (following Chen and Millero [1977]); (2) then calculating buoyancy frequency, $N = (g/\rho(d\rho/dz))^{1/2}$ [where g is acceleration due to gravity, ρ is the mean density and $d\rho/dz$ is the vertical density gradient], at 0.1 m depths

by differentiating piece-wise cubic splines fit through the density-depth data (with pchip.m in Matlab); and (3) finding the thermocline as the depth of maximum buoyancy frequency. We found the oxygenation threshold (1.0 mg DO) using cubic splines fit through DO-depth data. With temperature, thermocline depth, and oxygen threshold information, we calculated mean development time in the oxygenated metalimnion (day, D_M) and epilimnion (night, D_E).

$$D_M = \exp[\ \ln(a) + b\ln(T_M) + c\,(\ln(T_M))^2\ ] \quad \text{(S1.a)}$$

$$D_E = \exp[\ \ln(a) + b\ln(T_E) + c\,(\ln(T_E))^2\ ] \quad \text{(S1.b)}$$

where T_M and T_E are mean temperatures in the metalimnion and epilimnion, respectively, and coefficients $\ln(a) = 3.4$, $b = 0.22$, and $c = -0.3$ come from Botrell *et al.* (1977). Mean development time at each lake-date, D_{ave}, is then just the weighted average of D_E and D_M:

$$D_{ave} = \varphi_M D_M + \varphi_E D_E \quad \text{(S2)}$$

where φ_M and φ_E are the proportion of time per day spent in the metalimnion and epilimnion, respectively (taking into account waning of daylight as autumn progresses).

In the field exposure experiment, we used a simpler procedure. During the experiment, University Lake was not stratified (i.e., the thermocline was weak), so we did not have to account for temperature differences between habitat layers used during day and night. In the indoor mesocosm experiment room temperature was maintained at approximately 22°C day and night throughout the experiment. Thus, for these two experiments we calculated development time, D, with a simpler form of equ. S1:

$$D = \exp[\ \ln(a) + b\ln(T) + c\,(\ln(T))^2\ ] \quad \text{(S3)}$$

where we used mean water column temperature of the lake for T (field enclosure: temperature decreased through time during the experiment) or $T = 22$°C (indoor mesocosm). We calculated the average weighted egg ratio, E_{ave}, using data on infected and uninfected adult host classes.

Then, we calculated the population-level egg ratio, E_p, by multiplying E_{ave} times the percentage of asexual females in the population. Finally, we calculated the per capita birth rate, *b*:

$$b = \ln(E_p + 1)/ D_{ave} \quad \text{(S4)}$$

Additional Methods: Field enclosures

The lake enclosure experiment also included a mixing treatment, where half of the enclosures for each productivity x parasite treatment were mixed with a Secchi disk while the other half were not. The effects of mixing on disease dynamics will be presented elsewhere (Penczykowski et al., in prep *b*) We also had to exclude a total of 5 replicates from the analyses for the following reasons: One replicate was accidentally contaminated with extra nutrients during the first week of the experiment, in one replicate the host population crashed before the experiment began, two replicates were infested with high densities of host predators resulting in dramatic population declines, and one bag was destroyed by an anchor line malfunction.

Additional Results: Field enclosures

Nutrient additions significantly increased total phosphorous (ANOVA; N-effect: $F_{1,24} = 27.46$, $p < 0.001$, Fig S1*a*) and were consistent across disease treatments (E-effect: $F_{1,24} = 0.09$, $p = 0.76$; N x E: $F_{1,23} = 0.13$, $p = 0.73$). However, neither nutrient enrichment (N-effect: $F_{1,24} = 2.43$, $p = 0.13$, Fig. B1*b*), disease (E-effect: $F_{1,24} = 3.40$, $p = 0.08$), nor their interaction (N x E: $F_{1,23} = 1.23$, $p = 0.28$) significantly altered host density integrated over the course of the epidemic. Epidemics tended to be higher in the enriched treatments. However, this effect was not statistically significant (one-sided t-test; t = 1.45, df = 8.27, p-value = 0.09, Fig. S1*c*).

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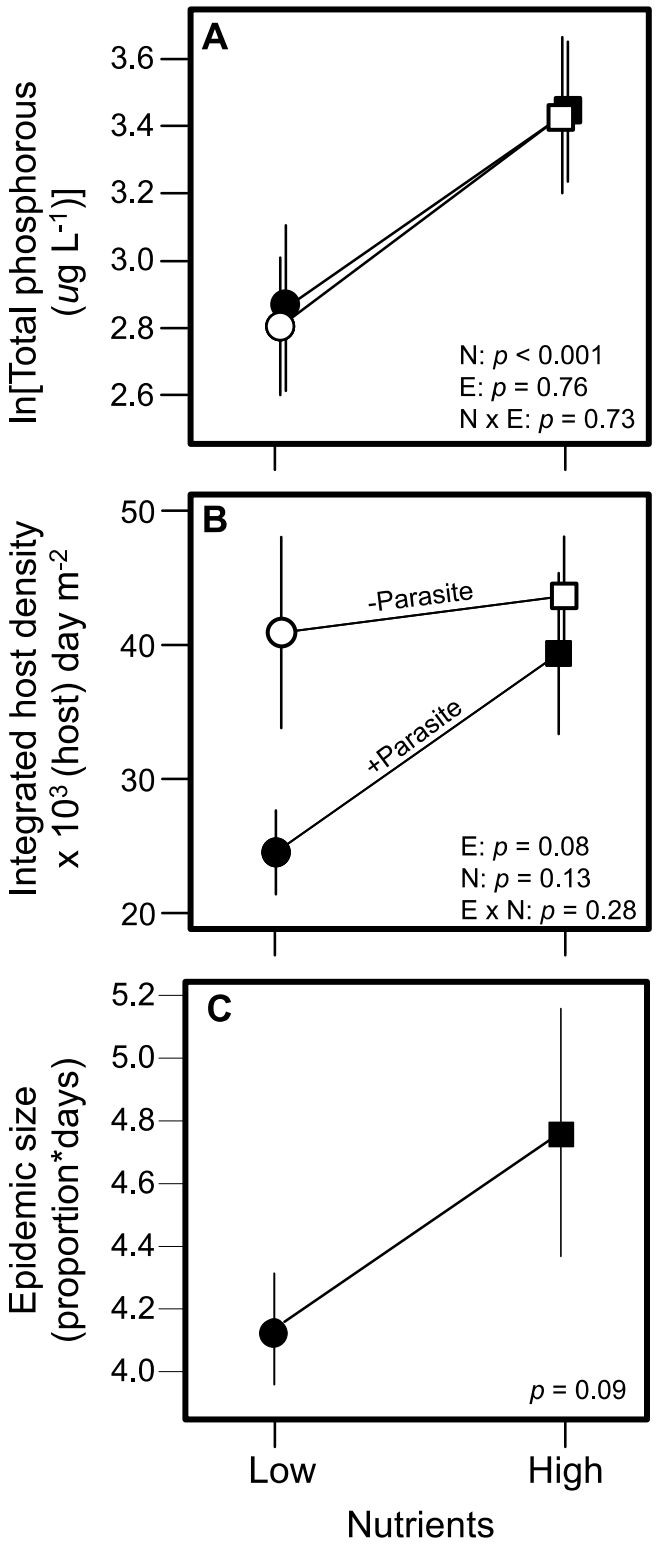
Figure S1. Results from the lake enclosure experiment. (A) Nutrient additions consistently and significantly increased total phosphorous (TP). (B) Neither nutrient enrichment nor disease significantly altered host density over the course of the epidemic. *P*-values of ANOVA are presented with "E*"* indicating epidemic effects, "N*"* indicating nutrient effects and E x N indicating their interaction. (C) Epidemics tended to be higher in the enriched treatments. However, this effect was not statistically significant. *P*-value is from a one-tailed t-test. Filled symbols are + parasite treatments and unfilled symbols are – parasite treatments.


A
ln[Total phosphorous (ug L-1)]
3.6
3.4
3.2
3.0
2.8
2.6
N: p < 0.001
E: p = 0.76
N x E: p = 0.73
B
Integrated host density x 10^3 (host) day m-2
50
40
30
20
-Parasite
+Parasite
E: p = 0.08
N: p = 0.13
E x N: p = 0.28
C
Epidemic size (proportion*days)
5.2
5.0
4.8
4.6
4.4
4.2
4.0
p = 0.09
Low
High
Nutrients

223

224
225