Predator diversity, intraguild predation, and indirect effects drive parasite transmission

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Edited by Burton H. Singer, University of Florida, Gainesville, FL, and approved January 23, 2015 (received for review August 19, 2014)

Humans are altering biodiversity globally and infectious diseases are on the rise; thus, there is interest in understanding how changes to biodiversity affect disease. Here, we explore how predator diversity shapes parasite transmission. In a mesocosm experiment that manipulated predator (larval dragonflies and damselflies) density and diversity, non-intraguild (non-IG) predators that only consume free-living cercariae (parasitic trematodes) reduced metacercarial infections in tadpoles, whereas intraguild (IG) predators that consume both parasites and tadpole hosts did not. This likely occurred because IG predators reduced tadpole densities and anticercarial behaviors, increasing per capita exposure rates of the surviving tadpoles (i.e., via density- and trait-mediated effects) despite the consumption of parasites. A mathematical model demonstrated that non-IG predators reduce macroparasite infections, but IG predation weakens this "dilution effect" and can even amplify parasite burdens. Consistent with the experiment and model, a wetland survey revealed that the diversity of IG predators was unrelated to metacercarial burdens in amphibians, but the diversity of non-IG predators was negatively correlated with infections. These results are strikingly similar to generalities that have emerged from the predator diversity–pest biocontrol literature, suggesting that there may be general mechanisms for pest control and that biocontrol research might inform disease management and vice versa. In summary, we identified a general trait of predators—where they fall on an IG predation continuum—that predicts their ability to reduce infections and possibly pests in general. Consequently, managing assemblages of predators represents an underused tool for the management of human and wildlife diseases and pest populations.

biodiversity–ecosystem function | dilution effect | schistosomiasis | snail | trophic cascade

In the last century there has been an unprecedented global increase in infectious diseases and decline and homogenization of biodiversity (1, 2). The controversial dilution effect hypothesis suggests that these two phenomena might be linked, specifically by proposing that biodiversity often decreases disease risk (3–11). Dilution effect research, for the most part, has focused on host diversity even though there is considerable evidence that selective predation on infected or uninfected hosts can strongly affect parasite transmission (7, 8) and that predation on parasites is widespread (9). As an example, in the well-studied Carpinteria Salt Marsh food web, 44% of trophic links are believed to involve predation on parasites (12). Despite the likely importance of predators to disease dynamics, we lack evidence supporting (*i*) the importance of predation to disease relative to more well-established factors known to affect parasite transmission, (*ii*) knowledge of environmental contexts that affect the impact of predators on disease, and (*iii*) the traits of predators that make them strong or weak "diluters" of disease risk [any species that reduces infections per focal host by removing parasites (equivalent to the solute] or serving as a less competent host than the focal host [equivalent to the solvent)]. This latter point is particularly important because it might facilitate identifying types of predators that can be managed to increase or decrease disease.

Many predators can consume both parasites and hosts (9), creating intraguild (IG) "predation" [IGP; predation can be substituted with natural enemy attack to capture both predators and parasites (13)] modules in food webs, defined as the killing and eating of potential competitors (14). These modules combine competition with predation and/or infection because the predator and parasite compete for a shared resource, the host, but at least one of the natural enemies can also benefit from consuming or infecting the other (13, 14). IGP is widespread, and it can structure and potentially stabilize communities (15, 16). However, it complicates predicting the impacts of predators on parasite transmission (9, 17, 18). For instance, by reducing the density of hosts, IG predators can increase the per capita exposure of the remaining hosts to parasites (10, 19), which could make IG predators weaker diluters of disease risk than predators that consume parasites but not the focal hosts (hereafter referred to as non-IG predators). In contrast, selective predation on infected hosts should reduce disease spread (7, 8). Additionally, IG predators often induce changes in traits of prey, such as behavior, growth, or morphology, which can also modulate parasite transmission (10, 17, 18). These effects can oppose or reinforce the reduction in parasite transmission associated with IG

Significance

Humans are altering biodiversity globally and infectious diseases are on the rise; thus, there is considerable interest in understanding how changes to biodiversity affect disease risk. We show that the diversity of predators that consume parasites was the best negative predictor of infections in frogs, suggesting that predation on parasites can be an important mechanism of disease reduction. Follow-up experiments, field data, and mathematical models revealed that intraguild predators, predators that consume both hosts and parasites, decrease macroparasite infections per host less than predators that only consume parasites, representing a general trait of predators that predicts their ability to reduce disease. Consequently, managing assemblages of non-intraguild and intraguild predators is an underutilized tool to minimize human and wildlife diseases.

Author contributions: J.R.R., D.J.C., P.W.C., A.M.S., and V.R.B. designed research; J.R.R., D.J.C., P.W.C., N.T.H., A.D.M., A.M.S., C.S., L.B.J., and V.R.B. performed research; J.R.R. and D.J.C. contributed new reagents/analytic tools; J.R.R. and P.W.C. analyzed data; and J.R.R. and D.J.C. wrote the paper.

The authors declare no conflict of interest.

This article is a PNAS Direct Submission.

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This article contains supporting information online at www.pnas.org/lookup/suppl/doi:10.1073/pnas.1415971112/-/DCSupplemental.

predators consuming parasites and it is the net effect of these potentially countervailing trait- and density-mediated indirect effects [TMIEs and DMIEs, respectively (20)] that will dictate the overall effect of predation on disease risk (18, 21).

Here, we use field surveys, experiments, and mathematical models to identify the mechanisms driving predation-dependent patterns of infection in a trematode–amphibian system. In this system, free-living, parasitic trematode cercariae are transmitted from snails, the first intermediate host, to tadpoles, the second intermediate host (22) (see *SI Background* for a description of the life cycle) and several vertebrate and invertebrate taxa are known to consume cercariae (e.g., refs. 9 and 23–25; see *SI Background* for more details). To develop our hypotheses, we turned to the rich literature of predator–prey interactions because it has a longer history than the host–parasite literature (18). Consistent with both the dilution effect hypothesis (3–5) and a meta-analysis that revealed that predator diversity on average supresses prey (26), we hypothesized that, at ecologically relevant densities, increased diversity of parasite predators would decrease trematode infections in frogs. We postulated that that this effect of parasite predators would be of a strength comparable to that of more well-established factors known to affect trematode abundance (discussed below). Finally, there is considerable evidence that the efficacy of predator diversity in controlling pest species is dependent on IG predation and nonconsumptive or trait-mediated effects (26–29). Consequently, we hypothesized that the effects of predators on parasite transmission would depend on the sum of density- and trait-mediated effects and the relative abundance of IG versus non-IG predator species.

Results and Discussion

Wetland Survey. We surveyed 18 wetlands in Minnesota (see Fig. S1 for map) to evaluate whether the taxonomic richness of potential cercarial predators predicted the numbers of metacercariae (encysted cercariae) per frog per wetland and to evaluate its predictive ability relative to other plausible predictors of these infections, such as host (frog and snail) species richness; snail abundance; melanomacrophage densities in frogs (immune cells that fight trematodes); and concentrations of nitrate, phosphate, calcium, and the herbicide atrazine. The multimodel inference analyses revealed that taxonomic richness of potential cercarial predators was the best predictor of metacercarial infections per frog per wetland. The numbers of metacercariae per tadpole were lower in wetlands with more species of cercarial predators (sum of seven species of metacercariae; model averaged coefficient ± SE = −0.344 ± 0.121, $F_{1,16} = 12.83$, $R^2 = 0.45$, $P = 0.002$; Fig. 1*A* and Table S1). Richness of cercarial predators appeared in >90% of the models with ΔAICc <4 and had a relative importance score of 0.96 (Table S1). A jackknife analysis revealed that the significance and direction of this effect was robust to the removal of individual taxa or even all dragonflies and damselflies (odonates) (Table S2), highlighting that an assemblage of cercarial predators was associated with the decline in metacercarial infection in frogs. The herbicide atrazine was the only other significant predictor of infections (Table S1), but it was a positive predictor supporting previous findings (30).

Given that the diversity of cercarial predators predicted infections in frogs, we next sought to elucidate mechanisms by which predators affect these infections. We focused on larval odonates as our predator guild because they are important predators of cercariae in ponds (23–25) and some species can also be predators of tadpole hosts (e.g., ref. 31).

Foraging Experiment Without Interspecific Interactions. We first conducted laboratory experiments to quantify the cercarial foraging rates of several odonate species in the presence and absence of interspecific interactions (see below for results of interspecific interactions). All four larval odonate species significantly reduced cercariae through foraging (df = 4, $\chi^2 = 63.45$, $P < 0.0001$; all pairwise comparisons with control $P < 0.0001$; Fig. S2). The damselfly, *Ischnura verticalis*, had a higher cercarial foraging rate than that of the other three odonate species (all $P < 0.0001$; Fig. S2), which did not differ from one another ($0.212 < P < 0.759$; Fig. S2). Hence, based on cercarial foraging rates alone, *I. verticalis* might be expected to be the strongest diluter of disease risk.

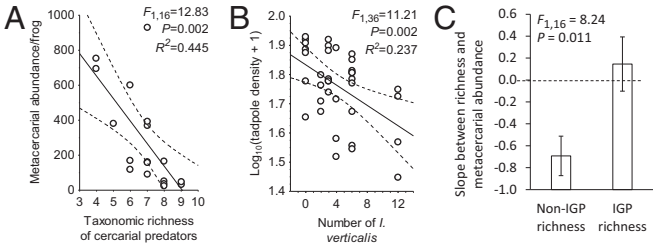

Fig. 1. The relationship between the taxonomic richness of potential cercarial predators in a wetland and the number of metacercariae per frog per wetland (*A*), effects of the density of *I. verticalis* on the survival of *R. clamitans* tadpoles in a mesocosm experiment (*B*), and the standardized slope parameters (±1 SE) between the number of metacercariae per frog per wetland and either the taxonomic richness of non-IG (those that only eat cercariae) or IG predators (those that regularly eat cercariae and tadpoles) in a wetland (*C*) (see *A* for the relationship of the two groups combined). In the scatterplots, best-fit lines and 95% confidence bands are presented.

Mesocosm Experiments. We conducted an experiment to elucidate mechanisms by which density and diversity of larval odonates affect abundance of three species of parasitic trematodes in *Rana clamitans* (green frog) tadpoles. Each of these three trematode species was found commonly in our wetland survey (see ref. 27). This experiment used a 2 × 2 × 2 × 2 fully factorial design, which crossed the presence or absence of three species of odonates [late instar *I. verticalis* (damselfly), or early instar *Pachydiplax longipennis* or *Sympetrum semicinctum* (dragonfly)], with one of two odonate densities, 6 or 12 larvae. *I. verticalis* was the only IG predator. This design resulted in three odonate densities (0, 6, and 12 larvae) and four odonate diversity treatments (0–3). In our wetland survey, all wetlands had one to three odonate species (27.8%, 55.6%, and 16.7%, respectively) and the lower of the two densities in our experiment is more ecologically relevant (32); thus, we focus on the richness and density levels most commonly found in the field (see *SI Results and Discussion* for a discussion of the other richness and density levels).

We first tested for main and interactive effects of odonate density and diversity on total metacercariae per tadpole [excluding the zero diversity treatment so it was not a missing cells design; multivariate ANOVA (MANOVA) on all three trematode species and univariate ANOVAs produced similar results, Table S3]. At the lowest odonate density, total metacercariae per tadpole decreased with increasing odonate richness from one to three species (Fig. 2*A*). At the highest odonate density, total metacercariae per tadpole decreased as odonate richness went from one to two species but increased from two to three species (density: $F_{1,45} = 5.82$, $P = 0.020$; diversity: $F_{1,45} = 4.17$, $P = 0.047$; interaction: $F_{1,45} = 4.07$, $P = 0.050$; Fig. 2*A*).

The previous statistical analysis ignored odonate species identity. To elucidate which odonate species were driving the density and diversity patterns in infections, we tested for the main effects of overall odonate density (a continuous predictor), each odonate species, and all two-way interactions between odonate species. There were no significant interactions between odonate species ($P > 0.115$), suggesting that interspecific interactions were not driving the infection patterns. Additionally, each odonate species had similar effects on each trematode species, providing little evidence that odonates specialized or had search images for

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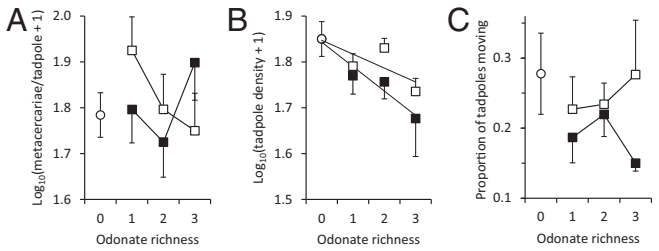

Fig. 2. Results of the mesocosm experiment showing effects of larval odonate density (0, 6, or 12 individuals; ○, □, and ■, respectively) and diversity (zero to three species) on (*A*) metacercarial infections per tadpole, (*B*) tadpole survival (i.e., density), and (*C*) tadpole activity. Shown are means ±1 SE. See text for sample sizes and statistics.

particular cercarial species. Hence, in this experiment, we had little evidence of niche complementarity (see refs. 28 and 33) as a mechanism for the predator diversity effects on parasite transmission. The MANOVA revealed significant negative effects of densities of the non-IG predators, *S. semicinctum* (Wilk's $F_{4,42} = 3.28$, $P = 0.020$; Fig. 3*A*) and *P. longipennis* (Wilk's $F_{4,42} = 2.83$, $P = 0.036$; Fig. 3*B*), on total metacercariae per tadpole. However, despite being the most voracious cercarial predator in our foraging experiment (Fig. S2), *I. verticalis* densities surprisingly did not reduce metacercarial infections in tadpoles (Wilk's $F_{4,42} = 0.44$, $P = 0.777$; Fig. 3*C*).

Next, we sought to elucidate the mechanisms for the observed patterns in metacercariae per tadpole (Fig. 2). We postulated that the observed patterns in metacercarial infections were driven by some combination of differences among treatments in (*i*) tadpole densities that affected per capita exposure to cercariae (10), (*ii*) tadpole growth rates and thus resources available for immunity (34), (*iii*) odonate interspecific interactions that affected their cercarial foraging rates, (*iv*) tadpole anticercarial behaviors (35–37), and (*v*) the relative abundance of diluting versus nondiluting odonates species. We conducted several additional analyses and experiments—taking a hypothetico-deductive approach—to gather support for or against each of these hypotheses.

Tadpole survival at the end of the experiment was negatively associated with both odonate density ($F_{1,55} = 6.03$, $P = 0.017$) and diversity ($F_{1,55} = 6.03$, $P = 0.017$; includes the zero diversity controls; Fig. 2*B*), but this entire effect seemed to be driven by *I. verticalis*, which was the only odonate species that was observed depredating tadpoles and was the only odonate for which its density was negatively associated with final tadpole density ($\beta \pm SE = -0.487 \pm 0.146$, $F_{1,36} = 11.21$, $P = 0.002$; Fig. 1*B*; other two species $P > 0.805$). Metacercariae per tadpole was a nonmonotonic function of diversity and density (Fig. 2*A*), whereas tadpole densities seemed to decline monotonically with odonate density and diversity, suggesting that tadpole density alone could not completely account for the pattern in metacercarial infections (Fig. 2*B*). Additionally, we found no evidence that available resources for immunological resistance or odonate interspecific interactions on cercarial foraging rates could account for the observed infection patterns across treatments (*SI Results and Discussion* and Fig. S3).

We also hypothesized that the pattern of infections across treatments was a function of tadpole antiparasite behaviors and the relative abundance of odonate species that did and did not reduce infections. Given that *I. verticalis* consumed tadpoles, that many amphibians possess alarm chemicals that can reduce their activity and affect their space use (38, 39), and that activity and cercarial avoidance are well-documented anticercarial behaviors (35–37), we hypothesized that *I. verticalis* density would decrease tadpole activity, increasing metacercarial infections. Indeed, *I. verticalis* was the only species that significantly reduced activity in monospecific tanks relative to controls (Fig. 4*A*). Importantly, the general pattern of metacercariae per tadpole as a function of diversity and density treatments (Fig. 2*A*) was the inverse pattern of tadpole activity (Fig. 2*C*), suggesting a cause–effect relationship. This is likely a product of the relative abundance of *I. verticalis* generally decreasing and the relative abundance of the "diluters," *S. semicinctum* and *P. longipennis*, generally increasing as diversity increased (Figs. 2*A* and 4), a mechanism that also seems to drive the classic dilution effect observed in the Lyme disease system (albeit, in that case, via increased relative abundances of competent hosts) (4, 40, 41). Indeed, as *I. verticalis* density rose, tadpole activity generally decreased and metacercariae per tadpole generally increased (Fig. 4*B*). Furthermore, infections per tadpole declined with the proportion of tadpoles active in *I. verticalis* treatments ($F_{1,5} = 5.23$, $P = 0.035$; Fig. 4*C*). These findings are consistent with other studies that have shown that predator-induced reductions in host activity can increase metacercarial infections (10, 37, 42) and suggest that there are tradeoffs between defenses against parasites and predators that warrant further research (18, 34, 43).

In summary, the reduction in trematode infections in tadpoles at higher densities of odonates (density: $F_{1,45} = 5.82$, $P = 0.020$) seems to be best explained by the abundance of *S. semicinctum* and *P. longipennis*, species that did not directly affect tadpole densities or behaviors (Fig. 4*A*) but that reduced tadpole exposure to parasites by consuming cercariae (Fig. 3 *A* and *B*). The diversity and diversity-by-density effects seem to be driven predominantly by the relative abundance of the non-IG predator species relative to *I. verticalis*, an IG predator that had opposing DMIEs and TMIEs on metacercarial infections, which likely explains the lack of a significant relationship between *I. verticalis* densities and numbers of metacerciae in tadpoles (Fig. 3*C*), despite its being the most voracious cercarial predator tested. Hence, the overall relationship between predator diversity and parasite transmission was a product of IG predation, nonconsumptive predator effects, and sampling (the increasing probability that species with traits that suppress pests increase as predator richness increases) rather than niche complementarity effects (see refs. 28 and 33). Although we did not find evidence for niche complementarity, it is possible that under natural conditions with more habitat complexity than in our mesocosms niche complementarity might be important.

Mathematical Model. Given that the IG predator in our mesocosm experiment reduced infections less than the non-IG predators, we developed a mathematical model to evaluate whether IG predators generally caused weaker reductions in disease risk than non-IG predators. The model was derived from the classic macroparasite model by Anderson and May (44) and it is similar to a macroparasite–host–predator model examined by Packer et al. (8).

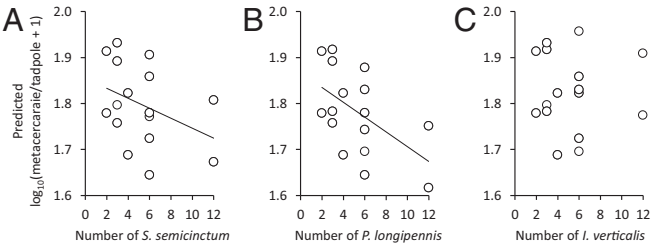

Fig. 3. Relationships between metacercariae per tadpole and the densities of two non-IG predators (those that only eat cercariae), (*A*) *S. semicinctum* and (*B*) *P. longipennis* and (*C*) an IG-predator (eats cercariae and tadpoles), *I. verticalis*. Shown are the predicted values from a model with temporal block and the main effects of each species, the presence-only treatments for each species, and best-fit lines for significant relationships.

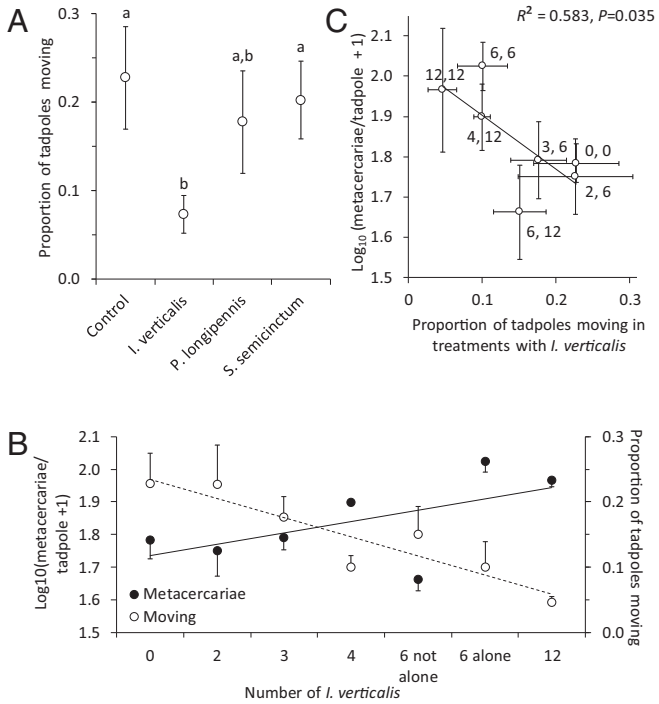

Fig. 4. Relationships between tadpole activity and metacercarial infections in *R. clamitans* tadpoles. (*A*) Effects of two non-IG predators (those that only eat cercariae), *S. semicinctum* and *P. longipennis*, and an IG-predator (eats cercariae and tadpoles), *I. verticalis*, in monospecific treatments on tadpole activity. Points with different letters are significantly different from one another ($P < 0.05$). (*B*) Tadpole activity and metacercarial loads as a function of the density of the IG predator *I. verticalis*, the only odonate species that significantly reduced tadpole activity. (*C*) Relationship between tadpole activity in treatments with *I. verticalis* and metacercarial infections per tadpole. The first number next to each point represents the number of *I. verticalis* in that treatment and the second number represents the total number of odonate larvae in that treatment. In each panel, means and 1 SE are displayed.

The model includes differential equations for populations of intermediate hosts, focal hosts, infections in focal hosts, and free-living parasites (analogous to the snails, tadpoles, metacercariae in tadpoles, and cercariae in our experimental system, respectively), and incorporates top-down effects of a predator guild that can consume hosts and/or free-living parasites. Although in our experimental system antipredator behaviors (reduced activity) are opposite of antiparasite behaviors (increased activity), this might not be the case in other host–parasite systems and thus we conservatively did not include trait-mediated effects in our model.

Predation on the intermediate host had effects qualitatively similar to predation on free-living parasites that are released from these hosts (*SI Results and Discussion*), and thus we focus on contrasting the effects of predation on free-living parasites (non-IG predator) versus predation on both free-living parasites and focal hosts (IG predator). Intuitively, the model demonstrated that non-IG predators that only consumed free-living parasites increased focal host densities, reduced the population of parasites infecting focal hosts, and reduced mean burdens in focal hosts (Fig. 5 *A* and *B*; see also Fig. S4 and *SI Results and Discussion*). In contrast, the effect of IG predators was nonlinear. If IG predators preferred free-living parasites more than focal hosts or preferred infected focal hosts to uninfected focal hosts [i.e., healthy herd effect 7, 8)], they only weakly reduced infections on focal hosts, but if they strongly preferred focal hosts over free-living parasites, they could increase mean parasite abundance per focal host by increasing the per capita exposure rate of the surviving hosts (Fig. 5*C*; see also Fig. S4 and *SI Results and Discussion*). Mechanistically, this occurs because predation on either focal hosts or free-living parasites decreases the total population size of adult parasites (P^*, Fig. 5*B*), but predation on hosts causes host density to decrease rapidly (H^*, Fig. 5*A*). If host density decreases more rapidly than parasite density, then mean burdens can increase (P^*/H^*, Fig. 5*C*). This amplification effect is further supported by a partial solution for mean parasite burden at equilibrium (*SI Results and Discussion*). Hence, consistent with the experimental results, the model indicates that non-IG predators should generally reduce macroparasite infections per host more so than IG predators.

Test of IG Predator Predictions in the Field. Given that our experiment and mathematical model suggest that non-IG predators should reduce infections per host more so than IG predators, we returned to our wetland survey to test this hypothesis in the wild. As predicted, trematode infections in frogs decreased much more steeply with the richness of non-IG predators than with the richness of IG predators, which did not differ from zero (Fig. 1*C*).

Conclusions

Our wetland survey uncovered a negative relationship between the diversity of potential cercarial predators and the total abundance of metacercariae in tadpoles. To our knowledge, this is the first field study comparing the predictive strength of predators of parasites to other factors known to affect parasite transmission. Our findings indicate that cercarial predators might have a larger influence on metacercarial infections in frogs than the richness and abundance of first and second intermediate hosts, frog immunity, and nutrients associated with elevated snail populations and trematode infections (30, 45) (Table S1). Moreover, the influence of cercarial predators on metacercarial infections in frogs was of comparable strength (but in the opposite direction) to the previously reported impacts of the herbicide atrazine on such infections (Table S1) (30).

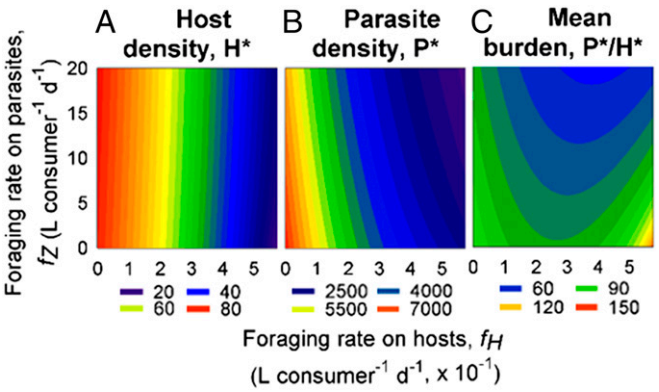

Fig. 5. Epidemiological consequences of simultaneous predation on focal hosts and free-living parasites (correspond with tadpoles and cercariae in our experimental system) at equilibrial values. (*A*) Increasing predation on free-living parasites increases the equilibrium density of focal hosts. However, increasing predation on focal hosts strongly reduces their equilibrium density. (*B*) Both types of predation reduce the equilibrial density of parasitic infections within focal hosts. (*C*) Predation on free-living parasites causes a monotonic decrease in equilibrial mean burden of infection for individual focal hosts. In contrast, predation on focal hosts causes a unimodal response. Initially, predation on focal hosts slightly reduces mean burdens. However, as predation on focal hosts increases, equilibrial mean burden rises, eventually surpassing burdens in the absence of predation. Thus, predators of free-living parasites monotonically reduce mean burdens for definitive hosts. However, predators of focal hosts can only weakly reduce mean burden or even amplify infection risk for hosts. Simulation parameters are identical to those in Fig. S4.

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Our experiment demonstrated that larval odonate density, diversity, and species composition, IGP, TMIEs, DMIEs, and a sampling effect (*sensu* ref. 33) all affected the abundance of metacercarial infections in anuran larvae. More specifically, under conditions that were most common in the field, increasing odonate richness reduced metacercarial infections in tadpoles, a pattern consistent with what was observed in our field survey. However, in the experiment and in the field, most of the decrease in infections per host was driven by the non-IG predators and our mathematical model suggests that non-IG predators should generally reduce macroparasite infections per host more so than IG predators.

Our findings have many similarities to the generalities that have emerged from the predator diversity–biocontrol literature. For example, similar to evidence that guilds of predators on average control pests better than single predator species (26–28), our findings suggest that entire guilds of predators can also regulate infections in hosts. Thus, managing predator assemblages might be more effective than managing single predator species to control disease (see *SI Results and Discussion* for examples). Again similar to our results, the predator diversity–biocontrol literature provides evidence that non-IG predators exhibit stronger biocontrol than IG predators and that biocontrol can be influenced by nonconsumptive effects of predators (26–29; see *SI Results and Discussion* for additional details on similarities). Overall, these similarities suggest that there might be general mechanisms for pest control regardless of whether the pest is a pathogen or consumer; thus, the general conclusions for disease control might match those for pest control. Specifically, releasing multiple non-IG predators will likely provide better pathogen suppression than releasing a single control agent (26), but the release of IG predators could decrease or increase pathogens, and thus the release of the single best control agent might provide better suppression of pathogen populations on average than the release of IG predators (29). Regardless of whether these conclusions hold, it seems clear that biocontrol research might inform disease management, and vice versa (18).

Finally, whereas we identified a general trait of predators—where they fall on an IG predation continuum—that predicts their ability to reduce disease and possibly pests in general, recent studies suggest that there might also be general traits of host species that predict their ability to dilute or amplify disease risk (46, 47) and herbivory (48). Consequently, to enhance infectious disease management and biocontrol we encourage further work that searches for traits of host and nonhost species that might be useful indicators of species that can increase or decrease parasite and pest populations.

Materials and Methods

Wetland Survey. Methods of our wetland survey were reported in Rohr et al. (30) and Schotthoefer et al. (49) and thus we relegated a summary of these methods to *SI Materials and Methods*. The following taxa were considered potential predators of cercariae because they either filter-feed or actively feed on planktivorous prey of similar size to cercariae, or consume snails that can harbor cercariae: the insects Dytiscidae, Hydrophilidae, Chaoboridae, Belostomatidae, Corixidae, Nepidae, Notonectidae, Pleidae, Aeshnidae, Coenagrionidae, Libellulidae, other dragonfly and damselfly families, amphipods, and crayfish. Dytiscidae, Aeshnidae, damselflies, and crayfish were considered IG predators because they also regularly consume tadpoles.

Odonate Foraging Experiments. To quantify the foraging rates of odonate larvae on cercariae, we exposed *Echinostoma trivolvis* cercariae to one of five odonate predator treatments: a nonpredator control to estimate background mortality of cercariae, one *Anax junius* larva, one *E. simplicicolis* larva, one *S. semicinctum* larva, or one *I. verticalis* larva (head widths of all odonates ranged from 2.11 to 3.21 mm). Fifty cercariae were transferred to a plastic predation arena (8.5 × 6.5 × 2.3 cm) containing 100 mL of water and an odonate larva. After 1 h we counted the number of cercariae that remained (see *SI Materials and Methods* for details).

We conducted a follow-up experiment to test whether interspecific interactions among the odonate species affected their cercarial foraging rates. The experiment had the same methods as the previous experiment except that there were eight treatments: a nonpredator control, one or three *A. junius* larvae, one or three *P. longipennis* larvae, or one or three *I. verticalis* larvae, and an interspecific interaction treatment where there was one of each of the three odonate species.

Mesocosm Experiment. To examine whether larval odonate density and diversity influence trematode infections in amphibians, we conducted a 2 × 2 × 2 × 2 experiment in which the first three factors were the presence or absence of three species of odonates and the last factor was one of two odonate densities, 6 or 12 larvae per replicate. Each treatment was replicated two times in each of two temporal blocks (9-d duration each), with the exception of four replicates of the nonpredator control in each block. The experiment was conducted in clear rectangular plastic tubs (38 × 25 × 15 cm filled with 10 L of filtered pond water) each with 10 *R. clamitans* tadpoles (Gosner stage 25). To expose the tadpoles and odonates to cercariae, replicates received a single *Planorbella trivolvis* snail that was infected with one of three trematode species: *E. trivolvis*, *Ribeiroia ondatrae*, or a species from family Plagiorichiidae. The snails were rotated through the replicates so that tadpoles were exposed to each snail and each trematode species for the same amount of time, thus homogenizing tadpole exposure to the cercariae. There was no snail mortality during the experiment but some odonate larvae died and were replaced to maintain consistency in the predator treatments.

Daily scan samples (9:00 AM and 4:00 PM) were used to quantify tadpole activity levels (number of tadpoles moving in 10 s). Tadpoles that died during the experiment were preserved in 70% ethanol and were not replaced. At the end of the experiment, the remaining tadpoles were counted, weighed, killed in 0.05% benzocaine, preserved in 70% ethanol, and cleared and stained to quantify the number of metacercarial infections of each trematode species, as described by Rohr et al. (21) (see *SI Materials and Methods* for additional details).

Mathematical Model. To capture long-term dynamics and feedbacks that are important to IGP (50), our model used ordinary differential equations to track changes in the densities of focal (could be second intermediate or definitive) hosts, H, parasites that successfully infected hosts, P, intermediate hosts, I, and free-living parasites, Z. (Eqs. **1a**–**1d**):

$$\frac{dH}{dt} = b_H\left(1-\frac{H}{K_H}\right)H - d_H H - f_H C H - vP \quad \textbf{[1a]}$$

$$\frac{dP}{dt} = \varepsilon\sigma H Z - (d_H + f_H C + \mu + v)P - v\frac{P^2(\theta+1)}{H\theta} \quad \textbf{[1b]}$$

$$\frac{dI}{dt} = b_I\left(1-\frac{I}{K_I}\right)I - d_I I - f_I C I \quad \textbf{[1c]}$$

$$\frac{dZ}{dt} = \gamma\left(\frac{P}{P+q}\right)I - \varepsilon H Z - f_Z C Z - d_Z Z. \quad \textbf{[1d]}$$

Focal hosts increase through density-dependent births, as determined by a maximum rate, b_H, and a host carrying capacity, K_H (Eq. **1a**). Focal hosts are lost because of background deaths, at mortality rate, d_H, predation by consumers, C, at per capita feeding rate, f_H, and from parasitic infection, with virulence on survival, v. Parasitic infections of focal hosts, P, increase when hosts become infected by free-living parasites, Z. Following exposure (at per capita exposure rate, ε), hosts can become infected according to their per-parasite susceptibility, σ ($0 \le \sigma \le 1$; Eq. **1b**). Parasitic infections decrease when hosts die (from background mortality, predation by consumers, or parasite virulence). The final term in Eq. **1b** accounts for additional losses from parasite-induced mortality that occur because parasites are aggregated in focal hosts, indexed by θ, the aggregation parameter of the negative binomial distribution. Intermediate hosts also increase through density-dependent births, as determined by a maximum rate, b_I, and their carrying capacity, K_I (Eq. **1c**). They are also lost from background deaths, at mortality rate, d_I, and predation by consumers, C, at per capita feeding rate, f_I. Finally, free-living parasites increase because of release (at per capita rate γ) by infected intermediate hosts (with infections assumed to be a saturating function of P, governed by the half-saturation constant, q; Eq. **1d**). Free-living parasites are then lost following contact with focal hosts, ε, predation by consumers, f_Z, or from background mortality, d_Z.

The model assumes that the intermediate host population produces free-living parasites at a constant per capita rate. Additionally, we chose not to dynamically couple the predator guild to host or free-living parasite densities because most, if not all, of the predators in our focal system are broad

generalists. A full analytical solution for this model is intractable, but we gained insight by examining a partial solution for mean parasite burden at equilibrium, P^*/H^*, by setting Eq. **1a** equal to zero. Finally, we numerically simulated the model across a range of reasonable values for the predation rate on hosts, f_H, and free-living parasites, f_Z, with the lsoda function from the deSolve package in R statistical software and determined the equilibrium values of (*i*) focal host density, (*ii*) parasite density, and (*iii*) mean parasite burden in focal hosts for each simulation. See Fig. 5 and Fig. S4 for the state variables and parameters used in the epidemiological model.

Statistical Analyses. For our field study, we used a multimodel inference approach in R statistical software (dredge and model.avg functions in the MuMIn package) to evaluate the importance of the taxonomic richness of potential cercarial predators to the number of metacercariae per frog per wetland (treating the wetland as the replicate) relative to other plausible predictors of this response variable (see *Materials and Methods*, *Wetland Survey* above). We limited the maximum number of variables in any model to three. To assess whether IG predators were generally weaker diluters than non-IG predators, we tested for a significant difference in the slope parameters between metacercariae per frog per wetland and the richness of IG and non-IG predators (nesting IG and non-IG predator richness within wetland).

For the two odonate foraging experiments, the response variable was the number of cercariae that were missing out of 50, the error distribution was binomial, each cercaria was nested within its test arena/replicate, and replicate was treated as a random variable to ensure proper error structure (lmer function in the lme4 package). For the mesocosm experiment, analyses were conducted with a general linear model with log trematode load as a response variable, temporal block as a random effect, and log diversity, log density, and their interaction as predictors for analyses focusing on richness and log density of each of the three odonate species as predictors for analyses focusing on species composition. We did not use a generalized linear model with a negative binomial error distribution because we were interested in conducting a multivariate analysis that incorporated all three, nonindependent trematode species in each tank as response variables and we are unaware of any multivariate analog for negative binomial error distributions. To test whether the richness, density, and odonate species had different effects on the abundance of the three metacercarial species, we conducted the same analyses as above, but nested trematode species within tank and tested for interactions between the predictors and this within-tank, trematode species factor.

ACKNOWLEDGMENTS. We thank M. Piwoni, J. Murphy, C. Johnson, A. Tremle, C. Lieske, N. Mateus-Pinilla, and the many highly skilled and dedicated technicians and undergraduate students who worked in our expansive field and/or laboratory studies, and B. Sears, T. McMahon, and anonymous reviewers for comments. This work was supported by grants to J.R.R. from the US Department of Agriculture (NRI 2008-00622 20 and 2008-01785), US Environmental Protection Agency (STAR R83-3835 and CAREER 83518801), National Institutes of Health (R01GM109499), and National Science Foundation (NSF) (DEB 0516227 and EF-1241889), and to V.R.B. from the US Environmental Protection Agency (Grant STAR R825867). P.W.C. was supported by a Research Opportunity Award Supplement to NSF Grant DEB 0516227.

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Supporting Information

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SI Background

Trematode Life Cycle. In our focal trematode–amphibian system, adult trematodes are found in vertebrate definitive hosts, where they reproduce sexually. Trematode eggs are released into waterbodies in the excrement of the definitive host. Miracidia hatch from the eggs and infect snails, the first intermediate host. These miracidia develop into sporocysts. Through asexual reproduction, the sporocysts produce, and the snail sheds free-living trematode cercariae. The cercariae, in turn, swim through the water searching for tadpoles, the second intermediate host. Two of the three focal cercariae in our experiment use proteolytic enzymes to encyst as metacercariae s.c. The last trematode, *Echinostoma trivolvis*, crawls up the amphibian cloaca and encysts as metacercariae in the kidneys. Finally, if an infected tadpole is consumed by a suitable definitive host, the life cycle is completed (1).

Taxa That Consume Cercariae. Several taxa are known to consume several species of free-living cercariae. Many of the seminal studies were conducted on *Schistosoma mansoni* cercariae, a parasite that infects humans. Rowan (2) and Knight et al. (3) reported that the guppy *Lebistes reticulatus* consume *S. mansoni* cercariae. Christensen (4) found that *Daphnia pulex* and *Daphnia longispina* (Cladocera), *Notodromas monacha* and *Cypria ophthalmica* (Ostracoda), and *L. reticulatus* were predators of *S. mansoni* cercariae. Additionally, experiments conducted by Christensen (4) and Christensen et al. (5) showed that predation of cercariae reduced transmission of *S. mansoni* to laboratory mice.

Kaplan et al. (6) presented six species of native estuarine fishes with 10 native trematode species. Many of the fishes engorged on cercariae. Moreover, they found evidence that fish also consumed cercariae under field conditions.

Schotthoefer et al. (7) showed that *Hydra* spp., damselfly (Odonata, Coenagrionidae) larvae, dragonfly (Odonata, Libellulidae) larvae, and copepods (Cyclopoida) consumed *Ribeiroia ondatrae* cercariae. Damselfly and dragonfly larvae were particularly voracious, in some cases consuming 80–90% of the cercariae offered within 10 min. In most cases, the foraging rates of predators on cercariae were not significantly affected by alternative prey.

Orlofske et al. (8) revealed that California newt larvae (*Taricha torosa*), western mosquitofish (*Gambusia affinis*), damselfly larvae (*Enallagma* spp. or *Lestes* spp.), and California clam shrimp (*Cyzicus californicus*) all depredated *R. ondatrae* cercariae. Moreover, in laboratory experiments, newt larvae and damselfly larvae reduced transmission of cercariae to tadpole hosts. Bioassays indicated that these predators consumed cercariae even in the presence of alternative prey.

SI Materials and Methods

Wetland Survey.

Overview. To quantify the diversity of amphibians and macroinvertebrates we conducted daytime visual time- and area-constrained dip-net sampling, adjusting effort according to the size of the wetland. Vegetation was quantified by randomly placing three 10-m transects within each community type around each wetland, and the line-intercept method was used to record relative cover of each plant species under or over the line. Macroinvertebrate and vegetation sampling occurred during three visits (March–April, May–June, and July–August). Snails were identified to species and arthropods were identified to family or below. We wanted to avoid destructively sampling macroinvertebrates and thus obtained presence–absence information for each snail and arthropod captured and abundance information for the dominant snail species, *P. trivolvis*.

On two visits (one in April–May and a second in June–July) we obtained water samples and attempted to collect a minimum of 15 recently metamorphosed *Rana pipiens* for parasite assessments and 25 for pathology studies including immune cell quantification. We quantified nitrate, phosphate, calcium (necessary for shell production), and atrazine (herbicide) levels from the water samples because each can promote snail population growth (9, 10). Amphibians were necropsied and their macroparasites were identified and quantified. Additionally, we quantified melanomacrophages from the hematoxylin and eosin-stained and sectioned livers of the amphibians because they are important immune cells for fighting trematodes (10).

Wetland selection and biotic sampling. Candidate wetlands were identified on National Wetland Inventory maps and selected for inclusion in the study following field reconnaissance within the Broadleaf Forest Ecoregion in Minnesota. Final criteria for inclusion in the analyses were (*i*) classification as a palustrine aquatic bed or emergent wetland, (*ii*) 0.5–5.0 ha in size, (*iii*) degree of landscape disturbance perceived by field assessments, (*iv*) landowner permission, (*v*) the presence of *R. pipiens*, and (*vi*) intermediate host (snail) abundance. Attempts were also made to include only wetlands that were at least 2 km apart to reduce spatial autocorrelation.

Analyte sampling and quantification. Water samples were taken just below the surface in the deepest area of the wetlands using a pole sampler (Nasco Swing Sampler 3228) and an amber glass collecting bottle, with care to avoid surface plants and other floating matter. Water was then decanted into a series of dedicated bottles specifically prepared for groups of analytes, including base neutral organics, acid herbicides, paraquat/diquat, metals, glyphosate, carbamate insecticides, and inorganic ions. Water for metals analysis was stabilized with ultra-grade nitric acid. Water for diquat and paraquat assays was stabilized with reagent-grade sulfuric acid. Finally, water for carbamate analysis was stabilized with monochloroacetic acid buffer. The samples were immediately chilled on ice and transferred on cold packs to the laboratory every 2 to 3 d.

Seven sediment cores were collected from each wetland in 0.6 m of water at approximately equidistant points around the wetland edge using a soil sampler auger (AMS basic soil sampler 3106) fitted with a 25- × 5-cm acrylic plastic sleeve. Samples were immediately placed and remained on ice to maintain 4 °C. Samples were sent to the analytical laboratory within 3 d of collection. The coring technique allowed us to collect sediment from the sediment–water interface, which is most likely to be actively engaged in exchange with the overlying water and in contact with amphibians. For three of the seven cores, the top 15 cm of each core was sent to the South Dakota State University Soil Analysis Laboratory for determination of organic matter content, phosphorus, and texture. Loss-on-ignition was used to measure organic matter content. Extractable P was determined using the sodium method. The remaining four cores were sent to the Illinois Waste Management and Research Center (recently renamed the Illinois Sustainable Technology Center) at the University of Illinois for quantification of elements and organic contaminants.

The following standard US EPA methods were used to quantify analytes with occasional minor modifications: 200.8 for metals and elements in tissue; 1631 for mercury in digested samples; 525.2 for base neutral organic compounds in water; 3545, 3630C, and 3640A for base neutral organic compounds in sediment and tissue; 8151A, 515.1, and 515.2 for acid herbicide compounds in water and sediment; 547 for glyphosate in water; 549.1, Revision

1.0 for paraquat and diquat in water; and 531.1, Revision 3.0 for N-methylcarbamoyloxamines and N-methylcarbamates in water.

Quality assurance in analyte quantifications. Duplicate or triplicate water samples were collected in several ponds during each sampling survey at the same location as the pond samples. An additional "mix" sample was taken, well away from the usual collection site, in selected ponds to test the assumption that the water of the ponds was well mixed. Trip blank water samples were carried to the field, stored with the samples, and delivered with the samples to the laboratory.

During the analytical process, several types of quality assurance samples/analyses were used, including analytical and matrix spikes, analytical replicates, and laboratory blanks. Instruments were calibrated according to manufacturer and/or method guidelines. Calibration curves were prepared from the reporting limit through most of the linear range of the instruments. Samples yielding results greater than the highest standard were diluted and rerun. Calibration check standards were run for most of the analyses. Internal standards were used in all GC/MS and ICP/MS analyses. Surrogate organic compounds were added to GC/MS samples before extraction and were monitored for recovery as an overall measure of the performance and consistency of the entire analytical procedure. Recoveries typically were above 90%.

Frog collection, pathology, and parasitology. Frogs were delivered within three days of collection to either the National Wildlife Health Center for parasitology evaluations or the University of Illinois, College of Veterinary Medicine for pathology assessments. Collections occurred in July and August of 1999. Pathology and parasitology examinations occurred after frogs were killed and followed standard protocols. Melanomacrophage aggregates were identified on hematoxylin and eosin-stained sections with a light microscope. Encysted metacercariae in the musculature were identified and enumerated by examining the cleared and stained specimens under a dissecting microscope (Fig. S2). Fixed parasite specimens were prepared for identification following standard protocols. Voucher specimens of parasites were deposited in the USDA National Parasite Collections, Beltsville, Maryland and cleared and stained frogs were deposited in the Bell Museum of Natural History, University of Minnesota, Minneapolis–St. Paul, Minnesota (collection numbers 14624–15168).

Odonate Foraging Experiment. For each trial, we used a pipette and a dissecting microscope to collect 50 cercariae from a mixture of seven infected *Planorbella trivolvis* snails. Cercariae were transferred to a plastic predation arena (8.5 × 6.5 × 2.3 cm) containing 100 mL of double-filtered (through 75-μm Nitex to remove any cercariae, which measure >200 μm) pond water and an odonate larva. After 1 h, we counted the number of cercariae that remained. The number of replicates per odonate species ranged from 11 to 28 depending on their availability. Odonates and snails were collected from ponds in Boyce and Hume, Virginia and were maintained on a 14:10 light-dark cycle. Odonates were not fed the day before the trials. In the first foraging experiment, we used *E. trivolvis* cercariae. In the second foraging experiment, we used a Plagiorchid cercariae.

Mesocosm Experiment. This experiment was conducted in clear rectangular plastic tubs (38 × 25 × 15 cm filled with 10 L of filtered pond water) with 6 10-cm pieces of black nylon rope attached to the bottom of each tub in a uniform distribution to provide perches for the larval odonates. Each tub had 10 *R. clamitans* tadpoles (Gosner stage 25; hatched from two egg masses ordered from Charles D. Sullivan Co. Inc.), a single *P. trivolvis* snail rotated among tubs, and food for the tadpoles and snails (1 g of coarsely ground rabbit food and a 1- × 1-cm portion of frozen spinach). The experiment was conducted at the University of Virginia's Blandy Experimental Farm, on a 14:10 light-dark cycle, and included a total of 64 experimental units.

SI Results and Discussion

Mesocosm Experiment. We hypothesized that odonate exposure might reduce foraging activity and thus the resources available for immunological resistance to cercariae that could account for the observed infection patterns across treatments. We assumed that any significant reduction in tadpole body mass per individual would reflect a reduction in overall resources that could be dedicated toward immunity. We found no evidence of any differences in tadpole body mass as a function of odonate density, diversity, or their interaction ($F_{1,53} < 0.321$, $P > 0.573$) or the densities of any specific odonate species or interactions between species ($F_{1,49} < 2.467$, $P > 0.122$). Although we cannot discount a reduction in relative investment in immunity, or, in other words, a redistribution of resources from immunity to antipredator defenses, our data provide little evidence that any of the treatments reduced the absolute level of resources for immunological defenses against cercariae.

We then examined the results of our second cercarial foraging experiment to assess whether interactions among odonate species affected cercarial foraging and thus infections in tadpoles. Tripling odonate density did not triple the overall foraging rate within a species (Fig. S3), suggesting that the relationship between odonate density and cercarial foraging is nonlinear and odonate interactions, regardless of species, can reduce cercarial foraging rates (11). Nevertheless, cercarial foraging rates were generally independent of interspecific interactions among odonates (Fig. S3), consistent with the lack of interactions among odonate species on metacercarial infections per tadpole and suggesting that odonate interspecific interactions were not capable of explaining the observed pattern in metacercarial infections per tadpole as a function of diversity.

Increasing odonate diversity from two to three species at the high odonate density caused an increase in metacercarial infections and a concomitant drop in tadpole activity that was surprisingly inconsistent with the number of *I. verticalis* in this treatment (Fig. 2 *A* and *C*). For whatever reason, tadpoles seemed to perceive this treatment as the most dangerous (apart from 12 *I. verticalis*) as reflected by their activity (Figs. 2*C* and 4 *B* and *C*). Nevertheless, such a high density of odonates seems unlikely to be common in the field (12).

Although we did not encounter any wetlands in our field survey without odonates, indicating that this treatment is also of rare ecological relevance, we would be remiss if we did not briefly discuss the lower-than-expected metacercarial infections in this treatment. Metacercarial loads likely increased from zero to one odonate species at low odonate densities because the reduction in cercarial exposure associated with the cercarial foraging was not as great as the increase in cercarial rates of contact with tadpoles caused by the decrease in tadpole activity (Fig. 2*C*). At the high density, we suspect that monospecific odonate treatments provided a sufficient amount of cercarial foraging to offset the effects of reduced anticercarial behaviors of tadpoles associated with this treatment relative to controls (Fig. 2 *A* and *C*).

Mathematical Model. Here we reiterate and further explain the mathematical model that we used to capture long-term dynamics and feedbacks that are important to IGP. The model tracks changes in the densities of focal (could be second intermediate or definitive) hosts, H, parasites that successfully infected hosts, P, intermediate hosts, I, and free-living parasites, Z using differential equations (Eqs. **1a–1d**):

$$\frac{dH}{dt} = b_H\left(1 - \frac{H}{K_H}\right)H - d_H H - f_H C H - vP \qquad \textbf{[1a]}$$

$$\frac{dP}{dt} = \varepsilon\sigma HZ - (d_H + f_H C + \mu + v)P - v\frac{P^2(\theta+1)}{H\theta} \qquad \textbf{[1b]}$$

$$\frac{dI}{dt} = b_I\left(1 - \frac{I}{K_I}\right)I - d_I I - f_I CI \qquad [1c]$$

$$\frac{dZ}{dt} = \gamma\left(\frac{P}{P+q}\right)I - \varepsilon HZ - f_Z CZ - d_Z Z. \qquad [1d]$$

Focal hosts increase through density-dependent births, as determined by a maximum rate, b_H, and a host carrying capacity, K_H (Eq. **1a**). Focal hosts are lost because of background deaths, at mortality rate, d_H, predation by consumers, C, at per capita feeding rate, f_H, and from parasitic infection, with virulence on survival, v. Parasitic infections of focal hosts, P, increase when hosts become infected by free-living parasites, Z. Following exposure (at per capita exposure rate, ε), hosts can become infected according to their per-parasite susceptibility, σ ($0 \le \sigma \le 1$; Eq. **1b**). Parasitic infections decrease when hosts die (from background mortality, predation by consumers, or parasite virulence). The final term in Eq. **1b** accounts for additional losses from parasite-induced mortality that occur because parasites are aggregated in focal hosts, indexed by θ, the aggregation parameter of the negative binomial distribution. Intermediate hosts also increase through density-dependent births, as determined by a maximum rate, b_I, and their carrying capacity, K_I (Eq. **1c**). They are also lost from background deaths, at mortality rate, d_I, and predation by consumers, C, at per capita feeding rate, f_I. Finally, free-living parasites increase because of release (at per capita rate γ) by infected intermediate hosts (with infections assumed to be a saturating function of P, governed by the half-saturation constant, q; Eq. **1d**). Free-living parasites are then lost following contact with focal hosts, ε, predation by consumers, f_Z, or from background mortality, d_Z.

The model assumes that the intermediate host population produces free-living parasites at a constant per capita rate. Additionally, we chose not to dynamically couple the predator guild to host or free-living parasite densities because most, if not all, of the predators in our focal system are broad generalists. Although a full analytical solution for this model is intractable, we can simplify the model somewhat. The dynamics of the intermediate host population, I, do not depend on any of the other state variables in the system. Therefore, we can solve for the equilibrium density of intermediate hosts, I^*, place this term in the equation for free-living parasites (dZ/dt, Eq. **1d**), and reduce the system from four dimensions to three:

$$\frac{dH}{dt} = b_H\left(1 - \frac{H}{K_H}\right)H - d_H H - f_H CH - vP \qquad [2a]$$

$$\frac{dP}{dt} = \varepsilon\sigma HZ - (d_H + f_H C + \mu + v)P - v\frac{P^2(\theta+1)}{H\theta} \qquad [2b]$$

$$\frac{dZ}{dt} = \gamma\left(\frac{P}{P+q}\right)I^* - \varepsilon HZ - f_Z CZ - d_Z Z. \qquad [2c]$$

Solving for I^* from Eq. **1c** yields

$$I^* = \left(\frac{b_I - (d_I + f_I C)}{b_I}\right)K_I, \qquad [3]$$

which is the carrying capacity of intermediate hosts tempered by the relative population growth rate of intermediate hosts (numerator: birth rate minus total loss rate) divided by the birth rate of intermediate hosts.

Although the model remains intractable, we gained insight by examining two partial solutions for mean parasite burden at equilibrium, P^*/H^* and Z^*, by setting Eqs. **1a** and **1b** equal to zero, respectively. We also numerically simulated the model across a range of reasonable values for the predation rate on hosts, f_H, and free-living parasites, f_Z, with the lsoda function from the deSolve package in R statistical software and determined the equilibrium values of (*i*) focal host density, (*ii*) parasite density, (*iii*) free-living parasite density, and (*iv*) mean parasite burden in focal hosts for each simulation. See Fig. 5 and Fig. S4 for the state variables and parameters used in the epidemiological model.

Consumption of free-living parasites increases equilibrial densities of focal hosts and reduces the total number and mean burden of parasites among focal hosts, as well as the density of free-living parasites (Fig. S4 *A*–*D*). The consequences of predation on parasites can be illustrated by calculating the proportion of free-living parasites that contact focal hosts before dying, $P(\textit{Contact any focal host})$. This quantity is determined, by definition, as the ratio of the overall contact rate with focal hosts and the total loss rate of free-living parasites. All else equal, the proportion of parasites that successfully contact focal hosts decreases monotonically with predation on parasites, fz:

$$P(\textit{Contact any focal host}) = \frac{\varepsilon H}{\varepsilon H + d_Z + f_Z C}. \qquad [S1]$$

Predation on focal hosts directly reduces equilibrial focal host density (Fig. S4*E*). It also reduces the total density of parasites (Fig. S4*F*). This occurs for two reasons. First, consumption of focal hosts destroys parasites that had successfully infected those hosts. Second, the reduction in focal host density reduces the probability that a free-living parasite will successfully contact a focal host before death (because this probability is a monotonically increasing function of focal host density, Eq. **S1**). Initially, predation on focal hosts reduces the equilibrial mean burden. However, as predation on focal hosts increases, the equilibrial density of free-living parasites and the mean burden increases as well (Fig. S4 *G* and *H*). This unimodal response can even lead to equilibrial mean burdens that exceed the predation-free case.

Why do mean burdens increase with predation on focal hosts even when the total density of parasites within focal hosts decreases? Again, the answer is related to rates of contact among focal hosts and free-living parasites. Eq. **S1** represents the probability that a free-living parasite contacts any focal host before dying. However, mean parasite burden is more directly related to the per-host contact rate (i.e., the probability that a parasite contacts a particular focal host before dying). This per-host contact rate, $P(\textit{Contact a given focal host})$, is the total contact rate (Eq. **S2**) divided by focal host density:

$$P(\textit{Contact a given focal host}) = \frac{\varepsilon}{\varepsilon H + d_Z + f_Z C}. \qquad [S2]$$

This is analogous to the fact that per capita mortality rates of prey equal the predator's functional response divided by prey density (13). This per capita contact rate (Eq. **S2**) is now a monotonically decreasing function of focal host density. This occurs specifically because exposure of hosts depletes free-living parasites from the environment (i.e., no one parasite can invade or infect two hosts). Therefore, as predators reduce the total density of focal hosts from the environment, the per capita risk of exposure (and therefore infection) can actually increase for focal hosts that remain. This result is supported by a partial analytical solution for equilibrium mean parasite burden, P^*/H^*, derived from Eq. **1a**:

$$\frac{P^*}{H^*} = \frac{b_H\left(1 - \frac{H^*}{K_H}\right) - d_H - f_H C}{v}. \qquad [S3]$$

Predation on hosts, $f_H C$ (the third term in the numerator of Eq. **S3**), directly reduces mean burden. However, predation can also depress equilibrium host density, H^*, increasing the density dependent birth rate of hosts (the first term in the numerator of Eq. **S3**). If the increase in density-dependent birth rate outweighs the direct effect of predation, then predation on hosts increases mean burdens. Thus, predators can increase mean burdens by causing greater relative decreases in host density than parasite density (Fig. 5 and Fig. S4 *E–H*).

In our model, density dependence in the focal host population facilitates this pattern. Here we chose to represent density dependence among hosts with logistic population growth. However, intraspecific density dependence can be nonlinear, and over some ranges of population density for some species it can act weakly. If density dependence only weakly affects the focal host population, then IGP-driven increases in parasitism might not occur as strongly. Thus, the specific details regarding density dependence may influence the potential and magnitude of IGP-driven increases in parasitism across systems.

Another effect (involving the density of free-living parasites) arises in this model that contributes to increasing parasite burdens with predation. Predation on hosts causes a reduction in the total number of parasites in focal hosts (P^*) but simultaneously causes the density of free-living parasites in the environment (Z^*) to increase (Fig. S4 *F* and *G*). Again, we turn to a partial solution for Z^* using Eq. **1b**. First, assuming H is positive, we divide both sides by H:

$$\frac{1}{H}\frac{dP}{dt} = \varepsilon\sigma Z - (d_H + f_H C + \mu + \nu)\frac{P}{H} - \nu\left(\frac{P}{H}\right)^2\frac{(\theta+1)}{\theta}. \quad \textbf{[S4]}$$

At equilibrium, the right-hand side of this equation is equal to zero:

$$0 = \varepsilon\sigma Z^* - (d_H + f_H C + \mu + \nu)\frac{P^*}{H^*} - \nu\left(\frac{P^*}{H^*}\right)^2\frac{(\theta+1)}{\theta}. \quad \textbf{[S5]}$$

Rearranging this equation for Z^* yields this partial solution:

$$Z^* = \left((d_H + f_H C + \mu + \nu)\frac{P^*}{H^*} + \nu\left(\frac{P^*}{H^*}\right)^2\frac{(\theta+1)}{\theta}\right) \Big/ (\varepsilon\sigma). \quad \textbf{[S6]}$$

Here Z^* is an increasing function of both predation on hosts ($f_H C$) and mean equilibrial burden (P^*/H^*). If we assume that Z^* is constant, then as $f_H C$ increases, P^*/H^* would have to decrease. However, this does not occur; P^*/H^* increases, and therefore Z^* also increases with predation on hosts (Fig. S4*G*). This occurs because hosts deplete free-living parasites during the transmission process. If there are fewer hosts, then there is less depletion via transmission. This decreases the total loss rate of free-living parasites from the environment (denominator of Eq. **S1**), and therefore increases the density of free-living parasites. Thus, despite a lower total density of parasites in focal hosts, predators can cause an increase in the density of free-living parasites in the environment (Fig. 5 and Fig. S4 *E–H*). This provides a testable prediction of this model: Predators that disproportionately attack hosts could simultaneously boost mean parasite burden and the density of free-living parasites.

Some predator species can consume free-living parasites and focal hosts (i.e., IG predators). Therefore, we simulated our model across a 2D gradient of predation rates on parasites and focal hosts. As in the simpler cases above, predators that only consumed free-living parasites reduced disease efficiently. In fact, for all rates of predation on focal hosts, increasing the predation rate on parasites resulted in higher focal host density, reduced the population of parasites infecting focal hosts, and reduced mean burdens (Fig. 5). Increasing predation on focal hosts always reduced the total number of parasites successfully infecting focal hosts (Fig. 5*B*). In contrast, increasing predation rates on hosts had a nonlinear effect on equilibrial mean parasite burden (Fig. 5*C*), even though increasing predation on hosts always reduced host density (Fig. 5*A*). This recaptured the pattern from the previous simulation (Fig. S4 *E–H*). Initially, increases in predation on focal hosts reduce mean burden, but further increases in predation minimally reduce mean burdens. High rates of predation on focal hosts can elevate equilibrial mean burdens substantially, especially if predation on free-living parasites is low (Fig. 5*C*, lower right).

Similarities Between Predator Diversity–Biocontrol Literature and Results of This Study. Our findings have many similarities with the generalities that have emerged from the predator diversity–biocontrol literature, suggesting that our work might inform both disease management and pest control. First, similar to evidence that guilds of predators on average control pests better than single predator species (14–16), our findings suggest that entire guilds of predators can also regulate infections in hosts. Thus, managing predator assemblages might be more effective than managing single predator species to control disease. As an example, schistosomiasis is a debilitating trematode disease of humans in the tropics and management efforts have focused on introducing single fish or crustacean predator species to control this disease (17, 18). Our results suggest that research should compare the value of management that maximizes the abundance and/or diversity of snail and cercarial predators to that of current approaches focused on single predator species.

Second, and again similar to our results, the predator diversity–biocontrol literature provides evidence that IG predation and nonconsumptive effects influence the strength of biocontrol offered by predator diversity (14–16, 19). In our model, the strength of predator control of parasites was not considerably different if the predator consumed the parasite directly or coincidentally by consuming infected snails. Likewise, in a meta-analysis, biocontrol of herbivore pests was not reduced when IG predators consumed parasitized herbivores, thereby consuming both the herbivore and developing parasitoid (20). Our model also revealed that IG predators that strongly prefer free-living parasites over hosts suppress parasite densities, whereas IG predators that strongly prefer hosts over free-living parasites could increase parasite densities per host by increasing the per capita exposure rate of the surviving hosts. Similarly, and consistent with IG predation theory (21), a meta-analysis revealed that the addition of non-IG predators generally suppressed pest prey populations, but IG predators, especially those that consumed one another, could even increase pests (19).

These similarities between the effects of IG predators and predator diversity on the control of both pathogens and pests suggest that there may be general mechanisms for pest control regardless of whether the pest is a pathogen or consumer. Indeed, our results suggest that the general conclusion for disease control might match that for pest control. That is, releasing multiple non-IG predators will likely provide better pathogen suppression than releasing a single control agent (14), but the release of IG predators could decrease or increase pathogens and thus the release of the single best control agent might provide better suppression of pathogen populations on average than the release of IG predators (19). Regardless of whether this conclusion holds, it seems clear that biocontrol research might inform disease management, and vice versa (22).

Disclaimer. Although the research described herein has been funded by several granting agencies, this report has not been subjected to their review and therefore does not necessarily reflect their points of view. Accordingly, no official endorsement should be inferred.

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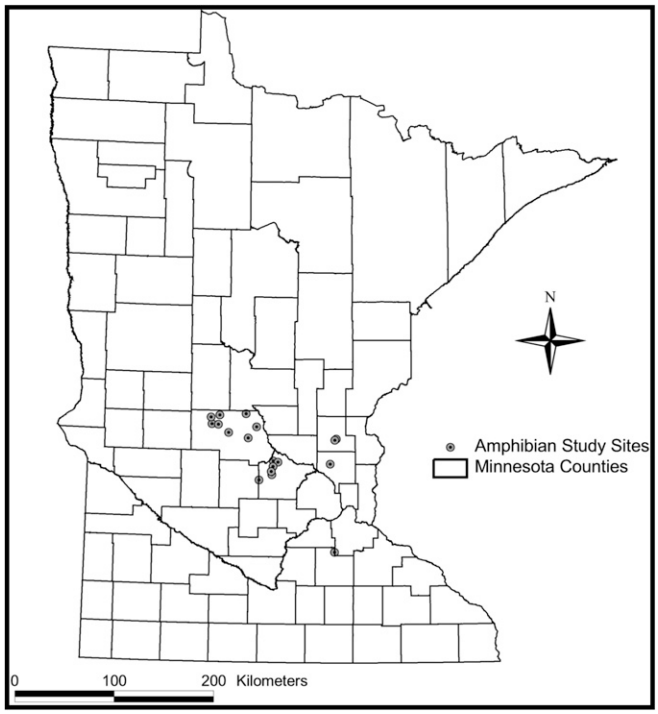

Fig. S1. Map of wetlands used in this study.

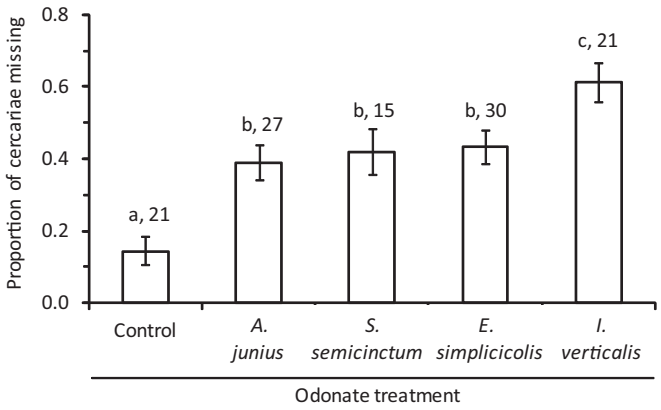

Fig. S2. Mean (± SE) predation rates of four species of odonate larvae (*Anax junius, Erythemis simplicicolis*, *Sympetrum semicinctum*, *Ischnura verticalis*) on cercariae of *Echinostoma trivolvis* during 1-h trials. Bars with different letters are significantly different from one another ($P < 0.05$) and numbers above the bars indicate sample sizes.

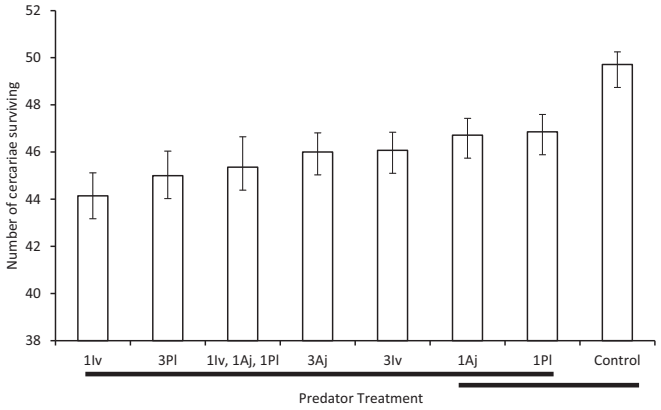

Fig. S3. Survival of cercariae in each treatment (Iv = *Ischnura verticalis*, Pl = *Pachydiplax longipennis*, Aj = *Anax junius*). Treatments connected by bars are not significantly different as determined by post hoc test. Shown are means ±1 SE.

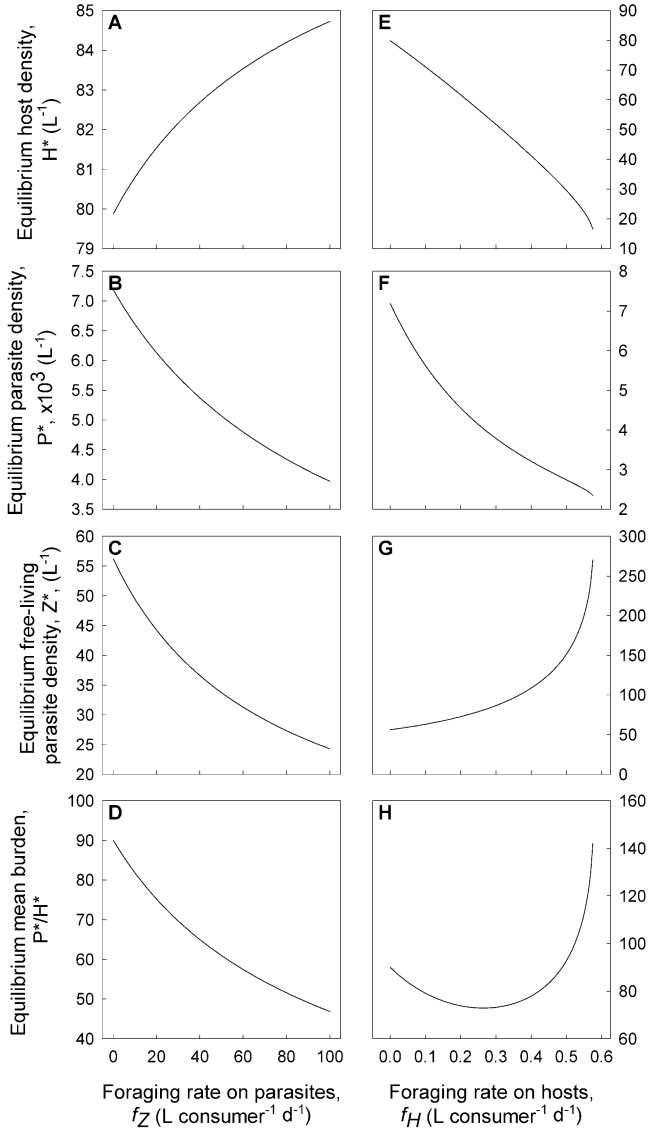

Fig. S4. Epidemiological consequences of predation on (*A–D*) free-living parasites and (*E–H*) focal hosts. Increasing predation on free-living parasites (*A*) increases the equilibrium density of focal hosts, (*B*) decreases the equilibrium density of parasitic infections in focal hosts, (*C*) decreases the density of free-living parasites, and (*D*) reduces the equilibrium mean burden of parasites within focal hosts. In contrast, predation on focal hosts decreases (*E*) equilibrial focal host density and (*F*) the equilibrium density of parasitic infections in focal hosts. However, predation on focal hosts can cause a substantial increase in the (*G*) density of free-living parasites and (*H*) mean parasite burden for focal hosts at equilibrium (especially at high predation rates). No predation on focal hosts in *A–D* or on parasites in *E–H*. All other simulation parameters: $b_H = 1$, $K_H = 100$, $d_H = 1$, $C = 1$, $v = 0.001125$, $\varepsilon = 1$, $\sigma = 1$, $\mu = 0.1$, $\theta = 10$, $b_I = 1$, $K_I = 100$, $d_I = 0.1$, $\gamma = 50$, $q = 10$, and $d_Z = 0.05$.

Table S1. Results of multimodel inference conducted in the MuMIn package of R statistical software and treating metacercariae per *Rana pipiens* metamorph as the response variable

Effect	Estimate	SE	Adjusted SE	z value	Pr(>|z|)	Relative importance
Intercept	0.821	1.186	1.250	0.657	0.511	
Taxonomic richness of potential cercarial and snail predators	−0.344	0.121	0.131	2.619	0.009	0.96
Atrazine concentration	3.146	1.218	1.319	2.385	0.017	0.94
Phosphate concentration	0.380	0.236	0.253	1.502	0.133	0.19
Pigmented melanomacrophage score	−1.278	0.936	1.025	1.248	0.212	0.11
Wetland area	0.257	0.213	0.233	1.102	0.270	0.09
Species richness of snails	0.116	0.110	0.120	0.963	0.336	0.07
No. of *Planorbella trivolvis*	−0.001	0.013	0.014	0.054	0.957	0.07
Species richness of amphibians	0.062	0.103	0.112	0.550	0.582	0.05
Nitrate concentration	−0.088	0.190	0.208	0.422	0.673	0.04
Species richness of vegetation	0.011	0.028	0.030	0.358	0.720	0.04
Calcium concentration	<0.001	<0.001	<0.001	0.239	0.811	0.04

Table S2. Jackknife results for the relationship between larval trematode abundance per frog per wetland (n = 18 wetlands) and the richness of potential cercarial predators when all taxa of potential cercarial predators were included and when taxa were serially removed from the dataset to evaluate the sensitivity of the results to the presence of particular taxa

Dataset	r	P
All taxa included	−0.667	0.002
All taxa but		
Amphipods	−0.631	0.005
Dytiscidae	−0.614	0.007
Hydrophilidae	−0.605	0.008
Chaoboridae	−0.619	0.006
Belostomatidae	−0.664	0.003
Corixidae	−0.566	0.014
Nepidae	−0.646	0.004
Notonectidae	−0.693	0.001
Pleidae	−0.654	0.003
Aeshnidae	−0.736	0.001
Coenagrionidae	−0.545	0.019
Unidentified dragonfly	−0.607	0.008
Libellulidae	−0.691	0.001
Damselflies	−0.707	0.001
Dragonflies and damselflies	−0.678	0.002

Table S3. Results of general linear models examining the effects of odonate density (6 or 12 odonates) and odonate diversity (one to three species) on the abundance of three species of metacercariae and total metacercariae per *Rana clamitans* tadpole

			Log Plagiorchidae				Log *Echinostoma trivolvis*				Log *Ribieroia ondatrae*				Log total metacercariae			
Effect	(F/R)	df	β	SE	F	P	β	SE	F	P	β	SE	F	P	β	SE	F	P
Block	Random	1,45	0.414	0.126	10.79	0.002	0.171	0.137	1.55	0.219	−0.708	0.099	51.14	0.000	0.321	0.130	6.47	0.014
Log density	Fixed	1,45	−1.195	0.499	5.73	0.021	−1.230	0.545	5.10	0.029	−0.270	0.393	0.47	0.495	−1.309	0.517	5.82	0.020
Log diversity	Fixed	1,45	−1.883	0.926	4.14	0.048	−2.187	1.010	4.68	0.036	−0.307	0.728	0.18	0.676	−2.129	0.959	4.16	0.047
Log density * log diversity	Fixed	1,45	2.096	1.044	4.03	0.051	2.646	1.140	5.39	0.025	0.188	0.821	0.05	0.820	2.354	1.082	4.08	0.050