When should we expect predator biocontrol of human schistosomes to backfire?

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Abstract

Conventional wisdom suggests any effort to control pests and parasites is better than none. However, growing evidence demonstrates weak or moderate effort can backfire, fail, and potentially worsen outcomes versus doing nothing. A central challenge is anticipating these potential failures before inducing environmental damage. We built a resource-explicit individual-based model of human schistosome and snail host transmission to evaluate biocontrol effort by simulating 22 predator stocking densities of native prawns. We test two host mortality scenarios and show intense biocontrol effort can succeed. However, weak to moderate effort can backfire by allowing large, prolific hosts to escape predation and drive overcompensation due to three interacting ecological mechanisms—resource competition among hosts, resource-dependent infectiousness, and predator gape limits. Ultimately, integrating physiology, ecology, and epidemiology can identify the risks of weak or moderate control effort when evaluating potential 'do or do not' control designs for future management of wildlife pests and diseases.

Introduction

Try not. Do or do not. There is no try.

— Jedi Master Yoda

When young Luke Skywalker was in Jedi training, the great Yoda imparted simple wisdom to help him surpass his own limitations: 'Try not. Do or do not. There is no try.'. For disease control, ecologists should also heed master Yoda's lesson—either doing nothing or committing fully could achieve the best outcome. Growing evidence of failed efforts to control ecological pests and parasites illustrates how weak or moderate intervention can backfire, leading to worse efforts than doing nothing or extremely intense control (Zipkin et al. 2009). One critical challenge when implementing these 'do or do not' control scenarios is anticipating their outcome from simple, general theory that often guides successful management recommendations (Shea et al. 1998; Punt 2006). Therefore, failed interventions are only diagnosed after the damage has been dealt, eroding the environment and our confidence in ecological and epidemiological science (Howarth 1991; Simberloff and Stiling 1996; Louda et al. 2005).

To prevent these ecological mismanagement disasters, ecologists should evaluate interventions with models that incorporate general ecological principles known to trigger target populations to either compensate or overcompensate in response to control. Overcompensation is central to 'do or do not' control scenarios for ecological pests and parasites. That is, increasing rates of mortality can counterintuitively increase biomass or production rates in some life stages or the total population (Choisy and Rohani 2006; Holt and Roy 2007; Schröder, Persson, and De Roos 2009). The driving force of overcompensation is resource competition, whereby eliminating competitors rewards remaining individuals energetically, enabling them to grow larger and reproduce more (De Roos et al. 2007; Ohlberger et al. 2011). For management, pest populations that compensate from resource competition are less sensitive to weak or moderate control (Pardini et al. 2009), whereas overcompensation generates a unimodal backfiring phenomenon where pest or pathogen problems

worsen with increasing control until sufficiently intense measures are reached (Moe, Stenseth, and Smith 2002; de Roos and Persson 2013; Preston and Sauer 2020; Zipkin et al. 2009). For example, recent work simulating different pesticide control scenarios in a snail-trematode system shows intense and frequent control as a 'do or do not' design can suppress infected host and parasite output, whereas moderate one-off intervention or weak application backfires and triggers parasite rebounds due to host populations overcompensating (Malishev and Civitello 2020). These failed attempts suggest that managers and ecologists should rigorously evaluate the merits of a binary strategy: do not intervene unless sufficiently intense actions can be sustained.

We use a case study of predatory biocontrol in a snail-trematode system to explore this binary strategy. The native African river prawn,*Macrobrachium vollenhovenii,* is a natural predator of snails and, recently, its local population collapse coincided with a large outbreak of human schistosomiasis in the Senegal River Basin (Sokolow, Lafferty, and Kuris 2014; Sokolow et al. 2015).We identify and evaluate three key ecological mechanisms that drive 'do or do not' control responses in parasites and vectors. First, strong laboratory and field evidence shows snail populations compete over resources (Perez-Saez et al. 2016; Preston and Sauer 2020; Civitello et al. 2020). Second, production of human-infectious cercariae per snail increases steeply with resources (Civitello et al. 2018), increasing host infectiousness with access to more food, as seen broadly for trematodes of humans and wildlife (Johnson et al. 2007). If moderate predator biocontrol reduces resource competition, then it can drive overcompensation in total cercarial production, the ecological dimension of human risk of exposure. In other words, the few remaining well-fed hosts could be more infectious than many starving hosts at high densities (Malishev and Civitello 2019). Third, like many predators, prawns are gape limited and disproportionately target individuals of smaller size ranges. Therefore, upper limits to prey intake size could promote overcompensatory parasite transmission from size-dependent host mortality (Ohlberger et al. 2011; de Roos and Persson 2013) that reduces competition experienced by prolific hosts large enough to escape predation risk. Combined, we hypothesize that these ecological mechanisms, namely (1) host competition for resources, (2) resource uptake promoting infectiousness, and (3) size-dependent mortality from gape limited predators can drive backfiring of weak or moderate biocontrol compared to 'do or do not' designs.

To test this hypothesis, we used an individual-based model of schistosome transmission based on bioenergetics theory to simulate size-dependent predation risk and examine potential biocontrol success and failure. We simulated two size-dependent predation scenarios (host mortality) over a range of predator densities: discrete host mortality, where hosts were categorized as either within or outside of the predator gape limits; and continuous host mortality, where predation risk was a decreasing function of host size based on experimental data. We propose both mortality scenarios present two constraints to biocontrol success: 1) a host vulnerability window to predation introduces the potential for a non-linear response to predator stocking density, where increasing densities first fail before reaching a successful threshold for eliminating infected hosts; and 2) a size class refuge from predation suggests predator gape limits can lead to failed biocontrol by shifting the infected host size distribution to larger, more prolific hosts. Our simulations propose management scenarios that attribute the success of 'do or do not' biocontrol designs to how gape limited biocontrol interacts with ecological mechanisms, while identifying common practices central to robust biocontrol success, i.e. no predator stocking vs. high stocking. Using these testable ecological mechanisms to explore the drawbacks, thresholds, and success of biocontrol design can explicitly evaluate management plans and features of potential biocontrol agents for enhancing pest and parasite control more broadly.

Methods

Study system Schistosomiasis, a global, tropical disease, is caused by schistosome blood flukes in the genus *Schistosoma* that cycle between freshwater snails and mammals and infect more than 200 million people worldwide (Hotez et al. 2014). Transmission is seasonal and relies on intermediate snail hosts primarily from three genera,*Biomphalaria, Bulinus* , and *Oncomelania* . Snails are infected by miracidia, the free-living juvenile stage of the schistosome that hatch from eggs excreted by humans into water bodies. Schistosomes reproduce asexually within the snail host and later emerge as aquatic cercariae. Humans become infected following dermal exposure to cercariae, which then mature into adults and lay eggs, completing the life cycle

(Gryseels et al. 2006). Therefore, the density of schistosome cercariae in aquatic environments represents an important ecological dimension of the human risk of exposure.

The Schistosome Individual-Based Dynamic Energy Budget (SIDEB) model

We built an individual-based epidemiology model for the human schistosome, *S. mansoni* , infecting a size-structured *B. glabrata* host population (Malishev and Civitello 2019). The overall model is a complete within-host dynamic energy budget (DEB) model (Kooijman 2010) for *B. glabrata* host biomass and *S. mansoni* parasite biomass integrated with a resource production model and a between-host parasite transmission model of infection contact between hosts and schistosome miracidia, the free-living life stage excreted by humans into freshwater environments.

Both susceptible and infected hosts follow a DEB model for growth, reproduction, survival, and production of parasite cercariae (Civitello and others 2018). The model uses coupled differential equations to track changes in resource inputs (either logistically growing algae or a detritus subsidy), host growth, somatic maintenance, maturity/reproduction, reserve density, parasite biomass and reproduction, and complementary traits of the infection pathway, such as damage density to hosts. A detailed breakdown of state variables and parameters for the sub-models and resource growth equations are found in (Malishev and Civitello 2019).

The infection rate of susceptible snail hosts is based on a constant daily introduction rate of free-living miracidia and host and parasite DEB parameters are from experiments on manipulated resource supply rates and periodic starvation periods (Civitello et al. 2020). We simulated a 150-day season of host and parasite infection rates and densities resembling the typical schistosomiasis transmission season. Simulations were seeded with an initial host population of 50 individuals and either logistically growing algae or a detritus resource subsidy. Resource supply levels determine infected host density and parasite output rates as host competition for resources weakens feeding pressure and increases overall mortality and per-capita intake rate, allowing remaining hosts to become more infectious by capitalizing on available food. This means for logistically growing algae, host density reduces resource growth rates, whereas a resource subsidy such as detritus continues to increase irrespective of host density. The complete model, including equations for the within-host energetics, between-host transmission, and population level dynamics are provided in Appendix 1.

Predator biocontrol

We simulated varying rates of snail host mortality representing predators as biocontrol agents feeding on hosts based on their body size under different predator densities. We used 22 predator densities of 0–50 N m^{-2}, extending beyond realistic densities used in aquaculture of congeneric species and field experiments of prawn predation on schistosome host snails (Sokolow, Lafferty, and Kuris 2014).

We simulated two scenarios of host mortality by predators,*discrete host mortality* and *continuous host mortality* . The discrete risk scenario represents predation exclusively on individuals falling within a given size range in which a lower bound of this size range at zero represents gape-limited predation. In contrast, the continuous risk scenario represents predation risk as a decreasing function of host size, consistent with laboratory studies on size-selective feeding of snail hosts by prawn predators (Sokolow, Lafferty, and Kuris 2014). Hosts not consumed by predators died naturally at baseline mortality rates (hazard rate,$h_b = 0.001$ d^{-1}).

Discrete host mortality

We simulated host mortality as a discrete vulnerability window to potentially gape-limited predators selectively feeding on host size cohorts: 0–5 mm, 0–10 mm, 0–15 mm, 0–25 mm, and 0–50 mm. Each snail experienced predation risk only when its size (diameter, mm) fell within the size range. The first two size ranges represent juveniles and juveniles plus newly reproductive adults, respectively, while the remaining three size ranges extend to larger adults that disproportionately contribute to total reproduction. The final size range encompasses all snails, since the largest host observed in any simulation was approximately 28 mm.

We modeled predation as an increase in the per capita hazard (instantaneous mortality) rate, h_t , for snails within the predator's size range according to a Type II functional response (Sokolow, Lafferty, and Kuris 2014)

$$h_t = h_b + \frac{p_A \ p_N}{1 + \ p_A \ p_H \quad H_N} H_C$$

where h_b is the baseline host mortality rate,p_A , p_N , andp_H are predator attack rate, predator population density, and predator handling rate, respectively,H_N is snail host population density, andH_C is an indicator variable for whether a given snail is within the host size class cohort vulnerable to predation, defined as H_{Cmin} $< L_i$ [?]H_{Cmax} where H_{Cmin} andH_{Cmax} are minimum and maximum host sizes in the cohort (mm) and L_i is the size of the currently predated individual host (mm). Biologically, this assumption means predators are equally likely to contact and handle any snail, but snails outside the size range always escape these encounters without being eaten.

Each host size range was run as separate simulations (n = 5) for each predator density to determine how predation impacts total transmission potential, represented as cumulative cercariae output over the 150-day transmission season.

Continuous host mortality

In the second scenario, we parameterized a negative exponential function of snail size on predator attack rate, p_A , based on laboratory feeding trials across a range of prawn and snail sizes (Sokolow, Lafferty, and Kuris 2014). We held the handling time,p_H , independent of snail size. Biologically, this assumption corresponds to predators being equally likely to contact and handle any snail, but larger snails more frequently escape these encounters without being eaten. These feeding data reported snail wet weights, which we converted to length (mm) using a size relationship of length to mass (L^3) (Thompson 1988)

$$h_t = h_b + \frac{p_A \ p_N}{1 + \ p_A \ p_H \quad H_N} e^{-0.237 \ \ L_i}$$

where L_i is individual host length (mm). Our previous analyses of the underlying SIDEB model show resource productivity interacts with sources of snail mortality to drive parasite output rates. Therefore, we simulated a range of resource supply rates of 0, 0.01, 0.05, 0.1, 0.2, and 0.25 for algae (d^{-1}) and detritus (mg C L^{-1} d^{-1}). We varied resource growth rates for each resource type as separate simulations (n = 5). Complete and reproducible model code is found in Appendix 2.

Results

Discrete host mortality

Increasing predator density triggered overcompensation in total cercariae output from infected hosts for both algae (Fig. 1) and detritus (Fig. 2) resource scenarios. For algae, compared to no predators (Fig. 1A), intermediate predator density generated peak parasite outputs (Fig. 1B) before a successful predator threshold eliminated infected hosts and parasites (Fig. 1C). Increasing predator density promoted higher parasite yields until an intermediate density of 10 N m^{-2}, after which predator densities > 13 N m^{-2} successfully eliminated infected hosts and parasites (Fig 1D). Similarly, for detritus, mean cumulative parasite output peaked at 7 N m^{-2} before successful predator densities of > 14 N m^{-2} (Fig. 2).

Low (1 N m^{-2}) to moderate (10 N m^{-2}) predator densities consistently failed to suppress parasite output from all host size classes for both algae and detritus (Fig. 3). Here, targeting small- to medium-sized hosts between 0 and 15 mm was unsuccessful in mitigating parasite emergence irrespective of predator stocking density. That is, targeting infected juvenile and adult hosts <15 mm failed to control cercariae emergence for both algae and detritus. In contrast, targeting large adult hosts >15 mm successfully eliminated parasites for stocking predators >10 N m^{-2}, demonstrating a minimum threshold needed to control parasite output.

Combined, this unimodal backfire response of increasing control pressure prior to reaching a sufficiently intense measure illustrates the risk of moderate intervention.

Continuous host mortality

Under size-dependent host mortality from a negative exponential feeding rate by predators, increasing predator density failed to suppress cumulative cercariae density at appreciable resource supply levels for both algae (d^{-1}) and detritus (mg C L^{-1} d^{-1}) (>= 0.05) (Fig. 4). As seen before, high algal growth rates or detritus supply rates promoted greater parasite output. Predators only effectively suppressed cercariae output at unrealistic predator densities of 50 N m^{-2} and > 20 N $m^{-2}$for algae and detritus, respectively, shown on a log + 1 scale in Figure 4.

Discussion

We show that the success and failure of predatory biocontrol of schistosomes depends on key ecological mechanisms, namely resource competition, increased infectiousness with resource uptake, and size-dependent host mortality. These combined mechanisms result in overcompensation of parasite output to predator biocontrol on intermediate hosts and thus a unimodal response of ecological risk of human schistosome exposure. Compared to no predators (Fig. 1A & 2A), weak to moderate predator biocontrol performs increasingly worse (Fig. 1B & 2B) before eventually succeeding at extremely high levels of investment (Fig. 1D & Fig. 2D). Here, low predation pressure reduces resource competition and enables some juvenile snails to more rapidly grow, reach an invulnerable size, and produce more parasites, i.e. escape an otherwise strong juvenile bottleneck (de Roos and Persson 2013). Therefore, low predator stocking densities backfire because they only partially reduce host density, which relaxes competition for food and triggers a trophic cascade to facilitate higher parasite yields from fewer remaining hosts. In contrast, high predator densities kill more hosts to control parasite output, first by reducing infected snail lifespan below the prepatent, i.e. developmental period of schistosomes (˜4 weeks), and then by extirpating snail populations (Fig. 1C & 2C). This result is consistent when predation is size-independent or categorically depends on host size (*discrete host mortality*). The consistent pattern of backfiring until ultimate success exemplifies the challenges of 'do or do not' biocontrol by highlighting overcompensation based on resource competition as a primary driver.

Size-dependent predation drives parasite output by introducing a host size refuge from predation. We show when predators are gape limited (*discrete host mortality*), biocontrol success depends on the upper limit of the binary host vulnerability window: there is a strong relationship between the upper limits to this window and a successful predator stocking density because predator gape limits shift the infected host size distribution to larger, more prolific hosts (Fig. 3, lighter curves). When predators kill almost all hosts (0–25 mm vulnerable; H_{Cmax} = 25 mm), they trigger host overcompensation and thus boost parasite output for both algae and detritus until eventually succeeding at high predator stocking densities (Fig. 3). In contrast, predator densities below this critical threshold not only underperform, but become increasingly worse and riskier the closer they approach it (Fig. 3, darker curves). However, when predators are unable to consume intermediate or large snail hosts, (H_{Cmax} = 5, 10, or 15 mm), total parasite production increases linearly. In other words, even at unrealistically high densities, predators that are small-snail specialists reduce resource competition, but do not eliminate large, prolific hosts. Therefore, if predator biocontrol programs fail to target large, disproportionately infectious hosts, they may be prone to backfire, regardless of management intensity.

In addition to a binary host vulnerability window, we also simulated*continuous host mortality* , where predation risk follows a continuous negative-exponential function derived from experiments (Sokolow, Lafferty, and Kuris 2014). Under this scenario, amassing predator stocks fails to suppress parasite density until reaching unrealistic predator densities (Fig. 4), even when compared with densities simulated in a recent study that tested these prawns experimentally (dotted line; (Sokolow, Lafferty, and Kuris 2014)) and the maximum recommended density for aquaculture practice (dashed line; (Upstrom 1986)). Here, large snails are never completely invulnerable to predation, but experience a substantially lower risk than smaller individuals. Cumulative parasite output in these scenarios also stems from overcompensation. As seen previously,

parasite output increases with resource supply rate (Civitello et al. 2018; Malishev and Civitello 2019) and overcompensation emerges under two conditions: whenever resource supply is high enough to support any parasite production and when the steepness of this response increases with supply rate. Combined, these results confirm how the aforementioned mechanisms shape overcompensation and further suggest resource type and supply exacerbate its role in size-dependent mortality under predator biocontrol.

Our model results contrast other simulations of predatory biocontrol of schistosomes (Sokolow et al. 2015; Hoover et al. 2019) that predict consistent success with increasing predator stocking density. Despite several differences between our individual-based model and these ordinary differential equation (ODE) models, one central difference drives these divergent forecasts: the positive effect of resource uptake on schistosome production by individual infected snails. Indeed, an extremely simplified ODE model of snail-schistosome dynamics illustrates how resource-dependent cercariae output can drive overcompensation due to increased background mortality (Civitello et al. 2018), supported by the SIDEB model. The positive effects of food quantity and quality are pervasive among schistosomes and other trematodes, infecting snails (Keas and Esch 1997; Civitello et al. 2018), suggesting a critical need to incorporate this general phenomenon when analysing disease mitigation strategies involving snails.

Given these divergent conclusions and the stakes involved in control intervention exacerbating or eliminating infection risk in vulnerable human populations, it is critical to evaluate existing evidence. Currently, only one published field study evaluates prawn-based biocontrol of human schistosomes (Sokolow et al. 2015). However, the study introduced an unspecified density of prawns into a water access point at one village in the Senegal River Basin and suggested this lowered schistosome infection in the following year compared to a control village not receiving prawns. While encouraging, these data cannot rigorously attribute this effect to the prawn manipulation, given the sample size of one per treatment and no follow up estimation of realized prawn density or survival. Despite a dearth of laboratory studies evaluating the population dynamics of snails and schistosomes in response to a range of prawn densities, one laboratory study shows gape-limited predation by prawns can relax competition in populations of uninfected snails, yielding fewer, larger individuals that produce many eggs, consistent with predictions from the SIDEB model (Sokolow, Lafferty, and Kuris 2014). Although this experiment did not incorporate infection, recent experiments on resource competition and food supply suggest these conditions could promote high parasite yields from the remaining larger hosts (Civitello et al. 2018, 2020). Data on predator densities, feeding selectivity, dynamics of snail population densities and size-structure, cercariae shedding, and human infection are needed to critically evaluate these alternative hypotheses and thus effective predator biocontrol of human schistosomes.

Limitations and future work

The underlying benefit to biocontrol is mitigating the higher economic and environmental costs of conventional control programs, such as chemical control of snail hosts and human mass drug administration (MDA). For example, MDA, such as administering praziquantel, relies on extensive monitoring to evaluate success (Gurarie et al. 2015), while chemical programs, such as molluscicide, incur high environmental costs (Coelho and Caldeira 2016) and rely on precise timing and careful interventions (Malishev and Civitello 2020). Further, collecting field data to improve models, such as host demography, can be cheaper than MDA intervention and supplement model forecasts with detailed ecological consequences needed to sustain long-term biocontrol (Louda 2003), bolster existing programs (King et al. 2020), or revisit alternatives, i.e. the lottery model (Myers 1985). Another major barrier is program logistics and scaling biocontrol programs constrained or complicated by the ecology and physiology of the biocontrol agent. For example, prawns farmed for biocontrol may alter diet preferences to meet growth demands, as seen in cichlid fish shifting to species other than snails due to changes in jaw morphology (Slootweg, Malek, and McCullough 1994). In nature, predators may also target narrower snail size cohorts, i.e. 5–10 mm versus 15–20 mm. In our simulations, we chose a wide range of size cohorts that encompasses the entire size distribution of the host lifecycle (Fig. 3) and represents a realistic host mortality rate at the population level. The snail and schistosome relationship also spans many host genera and three major schistosome species, so future models targeting specific snail-schistosome pairs could further tease apart the subtleties of the ecological mechanisms we identify in

this study. We encourage future lab and field work to explicitly consider testable ecological mechanisms that will help separate successful and resourceful biocontrol designs from risky and wasteful ones and provide the data to identify economic and ecological red flags.

Conclusions

Our results urge caution when implementing gape-limited biocontrol in parasite transmission systems that meet similar conditions to these prawns, snails, and schistosomes. Resource uptake by intermediate hosts that increases transmission, intense resource competition among hosts, and size-selective predators are all ingredients for potential biocontrol failure that may also worsen with greater effort. Here, we illustrate how resource competition as an underlying mechanism pervades trophic levels and population changes to drive potentially counterintuitive outcomes in the control of a major human parasite. Overcompensation as a key element of these systems is emerging as a recurrent theme in the ecology of pest control (Choisy and Rohani 2006; Bolzoni, Real, and De Leo 2007; De Roos et al. 2007), resulting in a growing list of scenarios that suggest 'do or do not' management responses. Importantly, we emphasize that not adopting this strategy does not suggest predatory biocontrol is hopeless, but rather weak or inconsistent control efforts seem unlikely to succeed. One critical management suggestion is that control programs should start small so that effort can be sustained and gradually expanded, rather than spread too thin. Ultimately, we stress the clear need for more experimental testing of predator biocontrol of human schistosomes and encourage more physiology and ecology detail in pest management more broadly. These details legitimise the biological data needed for successful control design that can extend to other host-parasite systems.

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Figure legends

Figure 1. Simulation model outputs with *discrete host mortality*(Eq. 1) showing failure and success of varying predator density (N m^{-2}) for projected snail host density (N L^{-1}, purple solid line), human-infectious cercariae density (N 0.1 L^{-1}, red line), resource density (mg C L^{-1}, green line), and infected snail host density (N L^{-1}, purple dashed line) for periphyton algae ($r = 0.25$ d^{-1}) over the 150-day transmission season: A) no predators (0 N m^{-2}), B) intermediate predator stocking density (10 N m^{-2}), and C) high predator stocking density (15 N m^{-2}). D) Mean (±SE) cumulative human-infectious cercariae density (N L) for predator stocking densities of 0–15 and 20 N m^{-2}. Letters represent cumulative cercariae density for top panel figures. Baseline host mortality rate $h_b = 0.001$ d^{-1}.

Figure 2. Simulation model outputs with *discrete host mortality*(Eq. 1) showing failure and success of varying predator density (N m^{-2}) for projected snail host density (N L^{-1}, purple solid line), human-infectious cercariae density (N 0.1 L^{-1}, red line), resource density (mg C L^{-1}, green line), and infected snail host density (N L^{-1}, purple dashed line) for a detritus resource subsidy ($det = 0.25$ mg C $L^{-1}d^{-1}$) over the 150-day transmission season: A) no predators (0 N m^{-2}), B) intermediate predator stocking density (7 N m^{-2}), and C) high predator stocking density (15 N m^{-2}). D) Mean (±SE) cumulative human-infectious cercariae density (N L) for predator stocking densities of 0–15 and 20 N m^{-2}. Letters represent cumulative cercariae density for top panel figures. Baseline host mortality rate$h_b = 0.001$ d^{-1}.

Figure 3. Mean (±SE) cumulative cercariae density (N L) with*discrete host mortality* (Eq. 1) under increasing predator stocking densities (0–15, 20, 25, and 30 N m^{-2}) targeting infected host size cohorts of 0–5, 0–10, 0–15, 0–25, and 0–50 mm for A) algae (0.25 d^{-1}) and B) detritus (0.25 mg C L^{-1} d^{-1}). The 0–50 mm host size cohort represents non-discriminate host mortality (all possible host sizes) and is the same output in Fig. 1D and Fig. 2D for algae and detritus, respectively. Size cohorts capture the entire size distribution of the host lifecycle.

Figure 4. Mean (±SE) cumulative cercariae density (N L) with*continuous host mortality* (Eq. 2) under increasing log + 1 predator stocking densities (N m^{-2}) for non-discriminate host predation (0–50 mm hosts)

for varying resource productivity levels of 0, 0.01, 0.05, 0.1, 0.2, and 0.25 for A) algae (d^{-1}) and B) detritus (mg C $L^{-1}d^{-1}$). Dotted line represents the current recommended predator density from (Sokolow, Lafferty, and Kuris 2014). Dashed line represents current maximum stocking densities of aquaculture river prawns *Macrobrachium rosenbergii* from http://www.aquacultureoftexas.com/tanks.htm.

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