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RESEARCH ARTICLE

# Infection with schistosome parasites in snails leads to increased predation by prawns: implications for human schistosomiasis control

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## ABSTRACT

Schistosomiasis – a parasitic disease that affects over 200 million people across the globe – is primarily transmitted between human definitive hosts and snail intermediate hosts. To reduce schistosomiasis transmission, some have advocated disrupting the schistosome life cycle through biological control of snails, achieved by boosting the abundance of snails' natural predators. But little is known about the effect of parasitic infection on predator–prey interactions, especially in the case of schistosomiasis. Here, we present the results of laboratory experiments performed on *Bulinus truncatus* and *Biomphalaria glabrata* snails to investigate: (i) rates of predation on schistosome-infected versus uninfected snails by a sympatric native river prawn, *Macrobrachium vollenhovenii*, and (ii) differences in snail behavior (including movement, refuge-seeking and anti-predator behavior) between infected and uninfected snails. In predation trials, prawns showed a preference for consuming snails infected with schistosome larvae. In behavioral trials, infected snails moved less quickly and less often than uninfected snails, and were less likely to avoid predation by exiting the water or hiding under substrate. Although the mechanism by which the parasite alters snail behavior remains unknown, these results provide insight into the effects of parasitic infection on predator–prey dynamics and suggest that boosting natural rates of predation on snails may be a useful strategy for reducing transmission in schistosomiasis hotspots.

**KEY WORDS: Parasitism, Behavior manipulation, Biological control, Schistosoma**

## INTRODUCTION

A human can become infected with schistosomiasis through a simple act: spending a few minutes in contact with a contaminated freshwater source. But the ecological processes that lead to that infection are anything but simple. Beneath the water's surface, the distribution of infective cercariae is determined by the distribution of schistosome eggs that infect snails, of snails that produce cercariae, and of physical and biological factors that move, clump or kill cercariae (Fig. 1). Here, we elucidate an important and little-studied aspect of the schistosomiasis transmission cycle: interactions between the snail intermediate hosts of the parasite and predators of these snails – predators that have the potential to control schistosomiasis transmission to people by reducing the number of infected snails in the environment.

Schistosome parasites infect more than 200 million people worldwide, with 90% of cases in sub-Saharan Africa. The disease is responsible for the loss of 70 million disability-adjusted life years (DALYs) annually, and therefore imposes a greater global disease burden than either malaria or tuberculosis (Hotez and Fenwick, 2009). The consequences of schistosome infection include chronic anemia, growth stunting, cognitive impairment, infertility, fatigue and increased susceptibility to co-infection (King and Dangerfield-Cha, 2008). Humans become infected through skin contact with schistosome larvae (cercariae) released into freshwaters by infected aquatic snails. Infected people then perpetuate the cycle by releasing urine or feces infected with schistosome parasites back into the snails' freshwater habitat (Gryseels et al., 2006; Fig. 1).

Predator–prey dynamics play in important role in altering the probability of schistosomiasis infection; in several instances, changes in the abundance of predators specializing on snail prey have been linked to changes in schistosomiasis incidence in people. In Lake Malawi, fishing-driven reductions in the abundance of molluscivorous fish may have released *Bulinus nyassanus* snails from predation; an increase in the abundance of this intermediate host increased transmission of *Schistosoma haematobium* to people (Evers et al., 2006, 2011; Madsen and Stauffer, 2011). In Egypt, an invasion by non-native Louisiana crayfish (*Procambarus clarkii*) may have contributed to coincident reductions in schistosomiasis prevalence (Khalil and Sleem, 2011). Indeed, experimental introduction of *P. clarkii* into Kenyan ponds reduced host snail abundance and lowered *S. haematobium* incidence in local schoolchildren (Mkoji et al., 1999).

Despite the importance of predators in patterns of schistosomiasis transmission, little is known about the effects of schistosome parasitism on snail predation or on snail anti-predator behavior. This knowledge could be leveraged to design effective biological control measures using predators to reduce abundance of infected snails and therefore lower risks of transmission from snails to people. The strength of schistosomiasis control via predation could be enhanced if infected host snails are consumed preferentially by predators, or hindered if infected snails are avoided. Here, we report a series of laboratory trials to investigate: (i) whether schistosome-infected snails are eaten more or less frequently than uninfected snails by sympatric native prawn predators, *Macrobrachium vollenhovenii*, and (ii) whether measurable behavioral differences exist between infected and uninfected snails when confronted with simulated predation conditions (to determine a set of plausible underlying explanations for differential consumption by prawns). We anticipated that schistosome infection could either increase or decrease infected snails' predation risk (Table S1).

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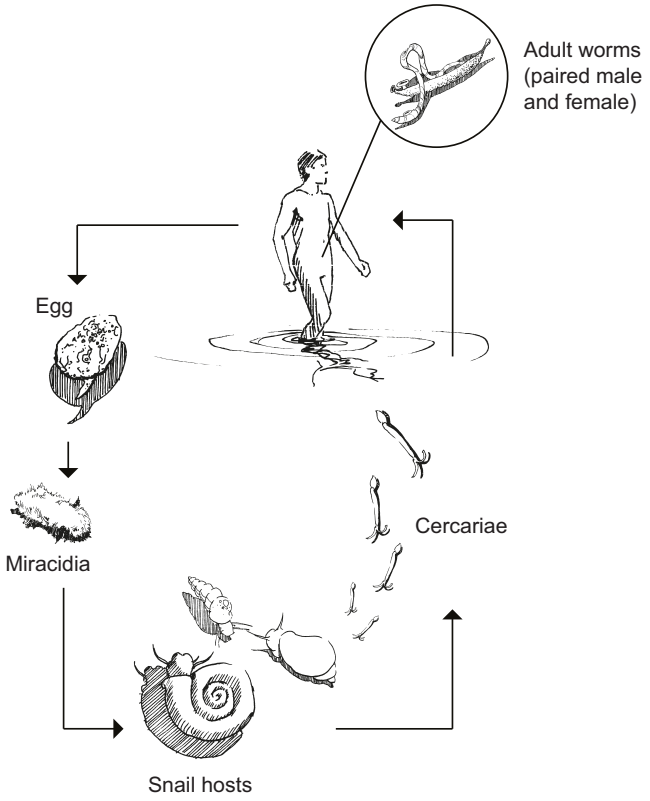


**Fig. 1. Life cycle of schistosomiasis.** Artwork by G. Galin.

Several mechanisms could increase the predation risk of infected relative to uninfected snails. Although predation kills the parasite along with its host, incidental, infection-related changes in host physiology and behavior could increase predation risk, especially if their benefit to parasite fitness outweighs the cost to the parasite in terms of increased predation. For example, infection might 'handicap' snails, generally impeding their movement and placing them at a disadvantage in reaching refuge from predation. Alternatively, parasites might physiologically alter host snails in a way that renders them more desirable or detectable by prawns. Schistosomes might also purposefully manipulate their snail hosts to position themselves in locations favorable to parasite transmission, at the expense of increased predation risk.

Conversely, because predation spells death for the parasite, worms might manipulate their hosts to evade predation. For example, parasites might force hosts to spend more time hiding and less time foraging. Physiological effects of schistosome infection could also render infected snails less detectable or desirable than uninfected snails; for example, infection might increase snail excretion of valuable nutrients, making infected snails less valuable as prey (Bernot, 2013). No natural predator of snails can contract schistosomiasis by consuming infected snails, but these predators are subject to infection by other trematode parasites; if predators have adapted to avoid eating snails infected by other trematodes, they might incidentally avoid schistosome-infected snails.

We used two schistosome-hosting snail species, *Biomphalaria glabrata* (host of *Schistosoma mansoni*) and *Bulinus truncatus* (host of *S. haematobium*) (Boelee and Madsen, 2006), to investigate the dynamics of predator–prey interactions in the presence of schistosome parasitism, largely in the pre-patent stage of infection (Table S2). We performed a predation experiment, measured snail movement rates, and observed snail anti-predator behavior after subjecting snails to simulated predation cues. This series of experiments was designed to test whether prawns preferred or avoided schistosome-infected snails and to assess the underlying behavioral differences driving snail vulnerability.

## MATERIALS AND METHODS

### Animals and aquaria

All *Bulinus truncatus* (Audouin 1827) and *Biomphalaria glabrata* (Say 1818) snails were reared and supplied by the NIH-NIAID Schistosomiasis Resource Center (Rockville, MD, USA) for distribution by BEI Resources (*Biomphalaria glabrata*, strain NMRI, unexposed, catalog no. NR-21970, and exposed to *Schistosoma mansoni*, strain NMRI, catalog no. NR-21962; and *Bulinus truncatus* subspecies *truncatus*, unexposed, catalog no. NR-21971, and exposed to *Schistosoma haematobium*, Egyptian strain, catalog no. NR-21965). Snails were either 'exposed' to *Schistosoma* spp. parasites or 'unexposed'. The success rate of exposure (defined as the percentage of snails that go on to develop patent infections) is 80–100% for *B. glabrata* exposed to *S. mansoni* (Lewis et al., 1986) and 30–70% for *B. truncatus* exposed to *S. haematobium* (Tucker et al., 2013). Not all exposed snails in the group develop full, patent infections, but we assumed that exposed snails were infected for some to all of the experimental period. We explicitly tracked patency (Table S2) but not pre-patency – snails were not dissected to confirm successful infection, as the success rates cited above were deemed sufficient to assume infection in the exposed groups.

Snails were housed in 25 l freshwater holding tanks before and after trials, and were fed with deionized water-rinsed romaine lettuce. For replacement and depletion experiments (see below), infected and uninfected snails were distinguished by applying spots of either blue or pink nail varnish to shell surfaces. Half of each trial was performed with blue denoting infected and pink denoting uninfected snails; the other half was performed vice versa. We compared consumption rates between colors to verify that prawns did not have a systematic color preference.

*Macrobrachium vollenhovenii* prawns were bred from wild Cameroonian stock in 2011, and shipped to UC Santa Barbara from the Kentucky State University Aquaculture department (Frankfort, KY, USA). Prawns were kept in a 400 l holding tank between trials and were fed a 40% protein commercial shrimp crumble diet (Rangen Corporation, Buhl, ID, USA) at ~3% of the cumulative body mass of all prawns in the holding tank per day. To ensure that prawns in predation trials would be hungry, they were not fed on days when experimental trials were being conducted.

All experimental trials were performed at the UC Santa Barbara Marine Biotechnology Laboratory (Goleta, CA, USA), in a facility with 27, 10 l polyethylene tanks, plumbed in three, nine-tank recirculating freshwater systems, each with a common sump, pump and filter. Each of these 10 l tanks was filled to 6 l with conditioned tapwater. Each contained one vinyl strip (~25 $cm^2$) as simulated substrate and one piece of deionized water-rinsed lettuce (~50 $cm^2$), and came equipped with an overflow drain at the water line.

All statistical tests were performed using R.2.11.0 (CRAN), and data processing/visualization was performed with JMP Version 10 (SAS, Cary, NC, USA).

### Experiment 1: replacement experiment

Experiment 1 involved predation of snails by co-housed prawns, where consumed snails were replaced every 2 h, to test whether there was any preference for or avoidance of infected or uninfected snails. Each independent predation trial consisted of one prawn housed in a 10 l tank with 10 snails (with a ratio of 8 uninfected:2 infected snails, to mimic natural conditions where the majority of snails in the environment are typically uninfected). Each trial day lasted 12 h for *B. glabrata*, and 10 h for *B. truncatus*. Every 2 h, the number and location (out of the water, under the substrate, on the lettuce, or on the bulkhead drain) of each snail were recorded. All snails consumed by prawns were replaced at the earliest possible observation point. Trials were repeated over the course of 2 2/3 days (*B. glabrata*) or 4 2/5 days (*B. truncatus*). For *B. truncatus*, $N$=8 independent tank trials were performed (8 experimental tanks, 5 time points per day, 4 2/5 days=176 total tank observations). For *B. glabrata*, $N$=4 independent tank trials were performed (4 experimental tanks, 6 time points

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per day, 2 2/3 days=64 total tank observations). The difference in consumption of infected and uninfected snails was computed using a linear mixed effects regression (LMER) in R.2.11.0 (package='lme4'), with binomial error term, and tank number, time and experimental day included as random effects.

### Experiment 2: depletion experiment

Experiment 2 involved consumption of snails by prawns over 24 h, without replacement of consumed snails at each observation point. Depletion trials included 1 prawn, 8 uninfected snails and 2 infected snails in each 10 l tank. Observations of the same metrics as in experiment 1 were taken at 0.5, 1, 2, 3, 4, 6, 8, 12 and 24 h. The trial was carried out twice in 8 tanks over a 24 h period for *B. truncatus*, and once in 7 tanks over a 24 h period for *B. glabrata*. Nail varnish colors to indicate infection status were switched for the second day of *B. truncatus* trials.

We fitted a Cox mixed effects model to the survival curves of infected versus uninfected snails in R.2.11.0 (package='coxme'), with tank number and species included as random effects. Species were combined for model fitting after determining the non-significance of the infection status×species interaction term.

### Experiment 3: movement trials

In experiment 3, we tested for differences in movement between infected and uninfected snails. Each trial group consisted of 2 infected and 2 uninfected snails, placed in shallow polyethylene trays filled to a depth of 2 cm with deionized water. To begin each trial, snails were placed in a line at one end of the tray, with four grains of 40% protein commercial shrimp crumble diet (Rangen Corporation, Buhl, ID, USA) at the other end as an incentive to move. The number of snails moving and their speeds, if moving, were recorded every 30 min for 6–10 consecutive observations. Speed was measured as the net distance traveled (in mm) by each snail over the course of 1 min. There were a total of $N$=70 observations for *B. glabrata* (10 observation points per tank, 7 tanks) and $N$=24 observations for *B. truncatus* (6 observation points per tank, 4 tanks). The difference between the number of infected and number of uninfected snails moving was tested using a binomial generalized linear mixed effects regression (GLMER) in R.2.11.0 (package='lme4'); differences in speed between infected and uninfected snails were tested using a Gaussian linear mixed effects test (LME) in R.2.11.0 (package='nlme'). Both models included tank group as a random effect. For both metrics, *B. truncatus* and *B. glabrata* data were combined to yield a single test for whether infected or uninfected snails differed in movement speed or frequency; this combination was validated by first performing the GLMER and LME with species×infection status as an interaction term, then excluding the interaction after verifying that it was not significant.

### Experiment 4: snail anti-predator behavior

In experiment 4, we compared the response to simulated predation conditions between infected and uninfected snails. This experiment was performed on groups of 6 snails, co-housed in 10 l tanks. We assigned either 6 infected or 6 uninfected snails to each tank. After a 24 h acclimation period, the location of each snail in each tank was recorded at 0.5, 1, 2, 4, 6, 8, 10 and 12 h (out of the water, under the substrate, on the lettuce, on the bulkhead drain, or in any other open area of the tank) on a control day (un-manipulated) and on a day of simulated predation treatment. The simulated predation treatment consisted of 2–5 manually crushed conspecific snails added at time 0 and 7 h, plus caged prawns added at time 0 and left in the treatment tanks for the entire trial. Each treatment was randomly assigned among 18 tanks. Snails were not painted with nail varnish, as tanks housed infected and uninfected snails separately.

We tested the response to predation conditions using a Poisson GLMER in R.2.11.0 (package='lme4'), which assessed the difference in behavioral metrics between the simulated predation treatment and control. Here, the interaction term infection status×treatment was of interest; that is, the interaction term addresses whether the change in behavior in response to predation was different depending on the snails' infection status. The time (hour of observation) was used as a random effect to account for the repeated measures over time and correct for any systematic difference through the course of the trial.

## RESULTS

Infected snails were preferentially consumed by prawn predators for both *B. glabrata* snails exposed to *S. mansoni* (attack rate for infected versus uninfected snails: 20.3±4.12% versus 10.4±1.67%, respectively, means±s.e.; linear mixed effects regression $P$=0.0015, d.f.=123) and *B. truncatus* snails exposed to *S. haematobium* (attack rate for infected versus uninfected snails: 31.5±3.13% versus 25.1±2.15%; linear mixed effects regression $P$=0.01, d.f.=316; Fig. 2). These were the results of the predation experiment (experiment 1), whereby all consumed snails were recorded and replaced every 2 h for 12 h. We obtained similar results from the depletion experiment (experiment 2), in which we tracked the number of consumed snails without replacement. A survival analysis showed that infected snails were consumed significantly faster than uninfected snails ($P$=0.03, d.f.=3, 14, mixed effect Cox model; Fig. 3).

In order to assess whether infected snails were 'handicapped' and therefore easier targets for predators, we compared movement rates between infected and uninfected snails. We found that significantly fewer infected snails were moving during observation periods (30.2±3.6% of the infected snails, $N$=86, compared with 40.7±4.1% of the uninfected snails, $N$=86, means±s.e.; $P$=0.03, linear mixed effects regression, d.f.=166). Among those snails that were moving, infected snails moved slower than uninfected snails (25.2±3.1 mm min$^{-1}$ for uninfected snails, $N$=41, versus 18.9±2.7 mm min$^{-1}$ for infected snails, $N$=31, means±s.e.; $P$=0.1, linear mixed effects model, d.f.=17). Trends were consistent for both of the snail–parasite pairs tested: *B. glabrata*/*S. mansoni* and *B. truncatus*/*S. haematobium* (Fig. 4).

Lastly, we performed trials to monitor and record anti-predator responses of infected versus uninfected snails under simulated predation conditions. Here, we defined anti-predator behavior as aversion to open areas of the tanks, with a preference for hidden areas underneath the substrate (black strips of vinyl) or out of the water (water quitting) (DeWitt et al., 1999). Predation threat was simulated by including a caged prawn and crushed conspecific snails in each tank.

In 5 of 6 trials, infected snails showed depressed anti-predator behavior responses compared with uninfected snails. In all but one case, for each species, and each of three behavior metrics (number in open areas, number under the substrate, number out of the water), results were consistent with the hypothesis that infection led to a muted anti-predator response. In this experiment, the same snails

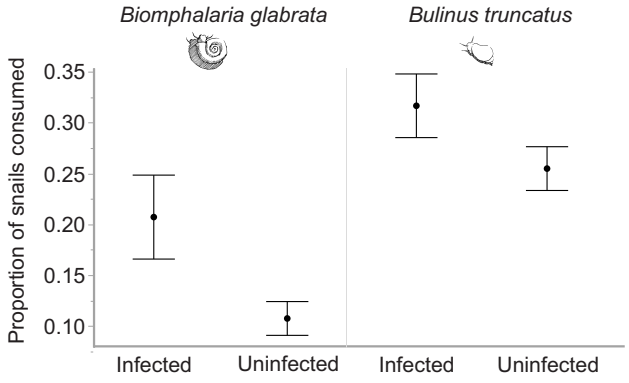


**Fig. 2 . Experiment 1: mean proportion of snails consumed by prawns.** In each tank, 8 uninfected and 2 infected snails were housed with 1 prawn; all snails consumed were replaced every 2 h for 12 h. Points represent the mean percentage of infected or uninfected snails consumed by prawns. Error bars represent 1 s.e.m.



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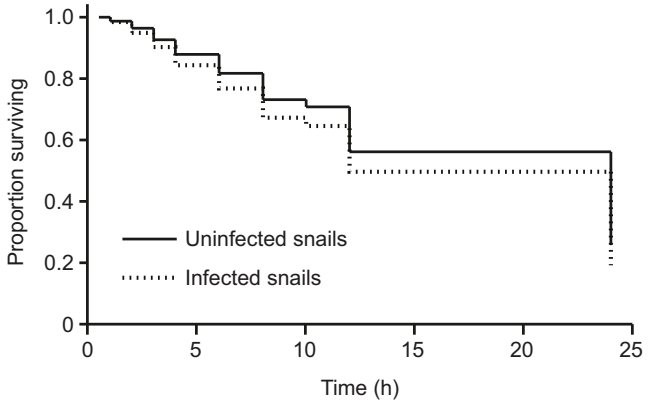


**Fig. 3. Experiment 2: average proportion of snails remaining over time.** In each tank, 8 uninfected and 2 infected snails were housed with a prawn for a 24 h observation period; the average percentage of snails of each infection status remaining alive at each observation point are represented in the survival plots.

were monitored before and after simulated predation, so that each snail acted as its own control. For *B. glabrata*, uninfected snails demonstrated significantly more water quitting [$P<0.0001$, generalized linear mixed model (Poisson family), d.f.=129], a non-significant aversion to open areas ($P=0.185$, d.f.=129) and a non-significant preference for hiding under substrate ($P=0.0758$, d.f.=129) when confronted with simulated predation conditions. For *B. truncatus*, the water quitting response was significantly stronger under simulated predation in uninfected snails [$P=0.0073$, generalized linear mixed model (Poisson family), d.f.=219], while exiting open areas tended (non-significantly) to be stronger in uninfected snails ($P=0.209$, d.f.=219). Anomalously, the response of hiding under substrate was stronger in infected *B. truncatus* snails than in uninfected *B. truncatus* snails in our trials, but the trend was not significant ($P=0.282$, d.f.=219; Fig. 5). This may have been caused by a high proportion of infected *B. truncatus* snails under the substrate at time=0 of the treatment day (Fig. S1).

## DISCUSSION

Our results demonstrate both reduced movement and reduced anti-predator behavior in schistosome-infected snails compared with uninfected snails, along with preferential consumption of infected snails by prawns. These findings support the hypothesis that parasitism renders schistosome-infected snails more susceptible to predation. Behavioral modification associated with infection could be a simple 'handicapping' of infected snails that reduces their ability to exhibit anti-predator behavior, or a 'side effect' of infection that leads infected snails to be more desirable or detectable. Alternatively, the behavior change could be 'strategic' for the parasite, in that it may increase parasite fitness at the cost of predation risk; this might occur if, for example, remaining in the water and in open areas increases the dispersal and transmission potential for schistosome larvae emitted from the snail host. The observed behavioral changes are not necessarily the result of a single, exclusive mechanism; a combination of the listed mechanisms could drive increased susceptibility to predation following infection.

The fact that infected snails were (i) less likely to be moving and (ii) slower when they did move supports the handicap explanation – that infected snails are debilitated by their parasites, rendering them less likely or less able to seek refuge and, thus, easier targets for predators (Wood et al., 2007). The metabolic cost of schistosome parasitism in snails is evidenced by the fact that *Biomphalaria* spp. show stunted growth immediately following infection (Gérard and Théron, 1997), though they can exhibit gigantism as the infection progresses (Sturrock, 1966). Following infection, energy allocation shifts from the host to the parasite (Gérard and Théron, 1997), resulting in the cessation of snail reproduction and possible effects on behavior (Crews and Yoshino, 1989). Additionally, in our behavior-response experiments, infected snails hid under the substrate and quit the water less frequently than did uninfected snails; this could be a result of debilitation, handicapping snails by hindering efficient movement to safety.

Another possible explanation for this pattern is that the parasite deliberately invests in altering host behavior to enhance its own fitness – potentially in a way that increases the probability of transmission at the cost of increased risk of predation. Little is

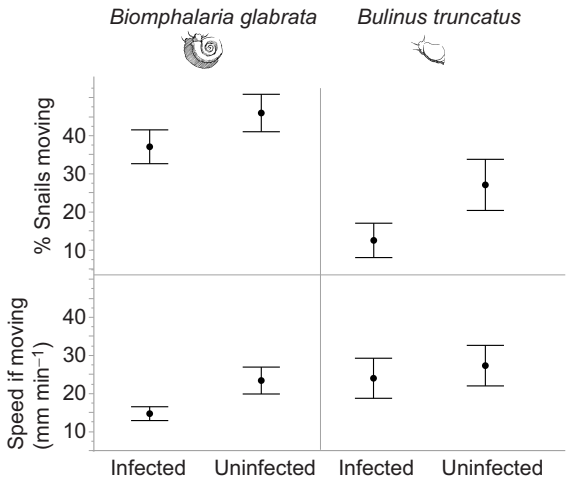


**Fig. 4. Experiment 3: snail movement.** For snail movement trials, the mean percentage of snails moving at each observation point and the mean speed at observation are presented. Error bars represent 1 s.e.m.

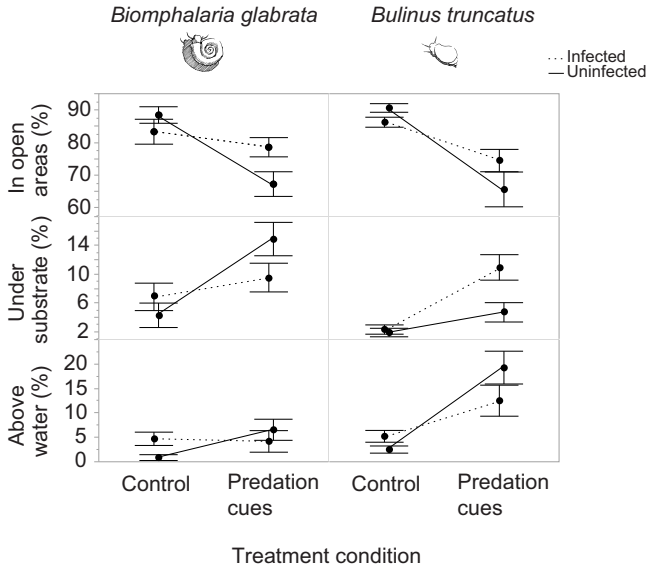


**Fig. 5. Experiment 4: mean proportion of snails observed out of the water, under the substrate and in the open.** Predation condition included exposure to crushed conspecific snails and a caged prawn. The plots represent the proportion of snails observed in each location before and after introduction of predation-threat cues, for infected compared with uninfected snails. Error bars represent 1 s.e.m.

known about strategic behavioral modification by schistosomes in their snail hosts. Some studies have shown that schistosomes induce increased feeding responses in parasitized snails (Williams and Gilbertson, 1983). Schistosome-parasitized snails are observed to seek out other infected snails and therefore aggregate within the environment; this is hypothesized to benefit parasite fitness because when snails with mono-sex larval parasite infections aggregate, the probability of successful parasite mate finding in the definitive host increases (Bernot, 2003).

In our experiments, infected snails exited the water less frequently than uninfected snails in response to predation cues. Parasite-induced reductions in water-quitting might benefit parasite larvae (cercariae), which require an aquatic medium to leave the snail host and seek the next host in the life cycle (humans). It is possible that the trade-off between water quitting and predation risk to the parasite favors having the snails remain in the water. This is consistent with analogous systems, such as *Potamopyrgus antipodarum* freshwater snails infected with *Microphallus* sp. trematodes; infected snails are found atop rocks, with greater access to food but higher risk of predation, more frequently than their uninfected counterparts (Levri, 1998). Although we did not record where in the tanks each snail was residing at each observation point, we noticed that many infected snails lingered just underneath the water's surface, perhaps as a behavioral modification to increase the probability of cercarial survival or transmission. Schistosome cercariae are negatively geotropic and positively phototropic, suggesting they prefer the water's surface (Standen, 1950). Our results were consistent with – and could not rule out – deliberate behavioral modification by the parasite as a potential contributing mechanism to the differential predation rates by prawns, but we acknowledge that this mechanism remains speculative and warrants further investigation. Similar behavioral modification theories have been posited in various analogous systems (Poulin, 2013; Bernot, 2003; Levri, 1999; Belgrad and Smith, 2014).

Another potential explanation for preferential consumption of infected snails is that infection might initiate a physiological change that renders infected snails either more detectable or more desirable to predators. Specifically, in trematode-infected snails, the nitrogen to phosphorus (N:P) excretion ratios are higher in infected snails than in uninfected snails, an effect that may be detectable by predators (Bernot, 2013). Analogous phenomena have been observed in other systems. For example, red grouse (*Lagopus lagopus scoticus*) with high parasitic worm burdens are more frequently found by hunting dogs than those with reduced infections, and this is probably due to greater scent emissions in the infected birds (Hudson et al., 1992; Isomursu et al., 2008).

Our findings strengthen the case for biological control of schistosomiasis through predation. Initial field trials in Senegal have shown fewer infected snails in waters stocked with predatory prawns, and reduced schistosomiasis prevalence in an adjacent village (Sokolow et al., 2015). In several other case studies, changes in the abundance of snail predators have been linked to changes in schistosomiasis incidence in people. Our results suggest that one reason for the efficacy of prawn predators in controlling schistosome-hosting snail populations may be their preference for infected snails.

More research is needed to elucidate the specific physiological pathways and underlying mechanisms driving behavioral alterations associated with schistosome infection in snails – notably, further studies should investigate any difference between consumption of snails with patent versus pre-patent infections. Nonetheless, our experiments demonstrate that prawn predators preferentially consume schistosome-infected snails, including snails in the pre-patent stage of infection, and that this is probably due to altered anti-predator behavior. The findings are relevant to future efforts to model this and similar host–parasite–predator systems and to leverage predators in the global fight to reduce human schistosomiasis transmission.

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#### Competing interests
The authors declare no competing or financial interests.

#### Author contributions
S.H.S. conceived and designed the experiments. S.J.S. and S.H.S. performed the experiments and analyzed the results. S.J.S., G.A.D.L., C.L.W., and S.H.S. wrote the paper.

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#### Supplementary information
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#### References
**Belgrad, B. A. and Smith, N. F.** (2014). Effects of predation and parasitism on climbing behavior of the marine snail, *Cerithidea scalariformis*. *J. Exp. Mar. Biol. Ecol.* **458**, 20-26.
**Bernot, R. J.** (2003). Trematode infection alters the antipredator behavior of a pulmonate snail. *J. North Am. Benthol. Soc.* **22**, 241-248.
**Bernot, R. J.** (2013). Parasite–host elemental content and the effects of a parasite on host-consumer-driven nutrient recycling. *Freshw. Sci.* **32**, 299-308.
**Boelee, E. and Madsen, H.** (2006). Irrigation and schistosomiasis in Africa: ecological aspects. *Int. Water Manag. Inst.* **99**, 39.
**Crews, A. E. and Yoshino, T. P.** (1989). *Schistosoma mansoni*: effect of infection on reproduction and gonadal growth in *Biomphalaria glabrata*. *Exp. Parasitol.* **68**, 326-334.
**Dewitt, T. J., Sih, A., and Hucko, J. A.** (1999). Trait compensation and cospecialization in a freshwater snail: size, shape and antipredator behaviour. *Anim. Behav.* **58**, 397-407.
**Evers, B. N., Madsen, H., McKaye, K. M. and Stauffer, J. R.** (2006). The schistosome intermediate host, Bulinus nyassanus, is a 'preferred' food for the cichlid fish, *Trematocranus placodon*, at Cape Maclear, Lake Malawi. *Ann. Trop. Med. Parasitol.* **100**, 75-85.
**Evers, B. N., Madsen, H. and Stauffer, J. R.** (2011). Crush-resistance of soft-sediment gastropods of Lake Malawi: implications for prey selection by molluscivorous fishes. *J. Freshw. Ecol.* **26**, 85-90.
**Gérard, C. and Théron, A.** (1997). Age/size- and time-specific effects of *Schistosoma mansoni* on energy allocation patterns of its snail host *Biomphalaria glabrata*. *Oecologia* **112**, 447-452.
**Gryseels, B., Polman, K., Clerinx, J. and Kestens, L.** (2006). Human schistosomiasis. *Lancet* **368**, 1106-1118.
**Hotez, P. J. and Fenwick, A.** (2009). Schistosomiasis in Africa: An emerging tragedy in our new global health decade. *PLoS Negl. Trop. Dis.* **3**, e485.
**Hudson, P. J., Dobson, A. P. and Newborn, D.** (1992). Do parasites make prey vulnerable to predation? Red grouse and parasites. *J. Anim. Ecol.* **61**, 681-692.
**Isomursu, M., Rätti, O., Helle, P. and Hollmén, T.** (2008). Parasitized grouse are more vulnerable to predation as revealed by a dog-assisted hunting study. *Ann. Zool. Fennici* **45**(6), 496-502.
**Khalil, M. T. and Sleem, S. H.** (2011). Can the freshwater crayfish eradicate schistosomiasis in Egypt and Africa? *J. Am. Sci.* **7**, 457-462.
**King, C. H. and Dangerfield-Cha, M.** (2008). The unacknowledged impact of chronic schistosomiasis. *Chronic Illn.* **4**, 65-79.
**Levri, E. P.** (1998). Perceived predation risk, parasitism, and the foraging behavior of a freshwater snail (*Potamopyrgus antipodarum*). *Can. J. Zool.* **76**, 1878-1884.
**Levri, E. P.** (1999). Parasite-induced change in host behavior of a freshwater snail: parasitic manipulation or byproduct of infection? *Behav. Ecol.* **10**, 234-241.

**Lewis, F. A., Stirewalt, M. A., Souza, C. P. and Gazzinelli, G.** (1986). Large-scale laboratory maintenance of *Schistosoma mansoni*, with observations on three schistosome/snail host combinations. *J. Parasitol.* **72**, 813-829.
**Madsen, H. and Stauffer, J. R.** (2011). Density of *Trematocranus placodon* (Pisces: Cichlidae): a predictor of density of the schistosome intermediate host, *Bulinus nyassanus* (Gastropoda: Planorbidae), in Lake Malaŵi. *Ecohealth* **8**, 177-189.
**Mkoji, G. M., Hofkin, B. V., Kuris, A. M., Stewart-Oaten, A., Mungai, B. N., Kihara, J. H., Mungai, F., Yundu, J., Mbui, J., Rashid, J. R. et al.** (1999). Impact of the crayfish Procambarus clarkii on *Schistosoma haematobium* transmission in Kenya. *Am. J. Trop. Med. Hyg.* **61**, 751-759.
**Poulin, R.** (2013). Parasite manipulation of host personality and behavioural syndromes. *J. Exp. Biol.* **216**, 18-26.
**Sokolow, S. H., Huttinger, E., Jouanard, N., Hsieh, M. H., Lafferty, K. D., Kuris, A. M., Riveau, G., Senghor, S., Thiam, C. and N'Daye, A. et al.** (2015). Reduced transmission of human schistosomiasis after restoration of a native river prawn that preys on the snail intermediate host. *Proc. Natl. Acad. Sci. USA* **112**, 31.
**Standen, O. D.** (1950). The concentration of cercariae of *Schistosoma mansoni* for the preparation of cercarial antigen. *Trans. R. Soc. Trop. Med. Hyg.* **43**, 527-530.
**Sturrock, B. M.** (1966). The influence of infection with Schistosoma mansoni on the growth rate and reproduction of *Biomphalaria pfeifferi*. *Ann. Trop. Med. Parasitol.* **60**, 187-197.
**Tucker, M. S., Karunaratne, L. B., Lewis, F. A., Freitas, T. C. and san Liang, Y.** (2013). Schistosomiasis. *Curr. Protoc. Immunol.* **103**, 19.1:19.1.1-19.1.58.
**Williams, C. L. and Gilbertson, D. E.** (1983). Altered feeding response as a cause for the altered heartbeat rate and locomotor activity of *Schistosoma mansoni*-infected *Biomphalaria glabrata*. *J. Parasitol.* **69**, 671-676.
**Wood, C. L., Byers, J. E., Cottingham, K. L., Altman, I., Donahue, M. J. and Blakeslee, A. M. H.** (2007). Parasites alter community structure. *Proc. Natl. Acad. Sci. USA* **104**, 9335-9339.