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Rebecca Cloud April 8, 2022

Parasite transmission in size-structured host populations

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Modeling parasite transmission in size-structured host populations

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Abstract

Modeling parasite transmission in size-structured host populations
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Parasite infection success can depend on host characteristics such as size, age, or genotype. Transmission theory that ignores variation and treats hosts as uniform individuals with identical infection risk matches data poorly and cannot address critical themes in disease ecology, such as superspreading and parasite aggregation. For the host snail *Biomphalaria glabrata* and its obligate parasite *Schistosoma mansoni*, larger snails experience higher rates of exposure to parasites but are less susceptible to infection. These size-dependencies are known for individual hosts in isolation, but their effects within size-structured populations remain unknown. To assess this relationship, I created mathematical models that can predict transmission dynamics at the population-level and test the strength of these models using experimental data. My results show that size-dependent models accounting for differences in both exposure and susceptibility with host size outperformed current or null models and were able to predict differences in population prevalence amongst different size-structures. Understanding how variation in host traits drives transmission is critical for increasing our ability to predict disease dynamics. Incorporating host body size in population-level parasite transmission models may enable researchers to improve decision making surrounding human schistosome risk in endemic areas.

Modeling parasite transmission in size-structured host population

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Table of Contents

1 Introduction

The infection success of parasites in host populations can depend on traits of their hosts, such as size, age, or genotype (Strauss et al. 2019). Host heterogeneity, i.e., variation in these traits, appears in many contexts in disease ecology. For example, a superspreader host is an infected individual who produces a disproportionately high number of secondary cases, creating a skewed pattern of individual-level infectiveness that differs greatly from average-based approaches to estimate population-level epidemic spread (Lloyd-Smith et al. 2005). Similarly, the infection success and aggregation of parasites among hosts can be a result of individual hosts traits, such as behavior (Johnson and Hoverman 2014). Despite known differences in infection success, many current models of disease transmission treat hosts as homogenous with identical infection risks (Levin et al. 1997, McCallum et al. 2001). In order to more accurately predict disease dynamics in natural populations, it is important to consider the sources of host variation and their consequences for transmission because hosts can differ in many ways (Hall et al. 2007, Strauss et al. 2019).

One example of a heterogenous trait known to influence transmission is body size. Body size-dependent transmission could be important for the natural dynamics and control of the human parasite *Schistosoma mansoni* in its intermediate host snail, *Biomphalaria glabrata*. Blood flukes in the genus *Schistosoma* cause human schistosomiasis, which affects approximately 200 million people worldwide (Colley et al. 2014, Hotez et al. 2014), particularly young children (Clennon et al. 2006), and can cause symptoms ranging from fever to abdominal pain to liver fibrosis and eventual liver failure (Clerinx and Van Gompel 2011, Colley et al. 2014). *S. mansoni* obligately cycles between humans and freshwater snails in the genus *Biomphalaria* (Fig. 1), using free-swimming life stages to infect both hosts. Infection success of *S. mansoni* is known to be governed

by snail body through the processes of both exposure and susceptibility (Niemann and Lewis 1990, Théron et al. 1998). We define exposure as a parasite's irreversible contact with a host, and susceptibility as the risk of infection given an exposure event (Civitello and Rohr 2014). Larger snails experience greater exposure to parasites, presumably due to stronger chemical gradients that the snails emit, which free-living parasites are able to detect (Théron et al. 1998). Conversely, larger snails are less susceptible to infection once exposed to the parasites, potentially due to better equipped immune systems (Niemann and Lewis 1990). These size-dependencies have been shown in individual hosts in isolation, but their consequences in size-structured populations of hosts remain unknown. Natural populations of freshwater snails in the genus *Biomphalaria* have been observed to show changes in size structure on a monthly basis (Loreau and Baluku 1987), so being able to predict changes in population dynamics within populations of varying size structures may aid in the understanding of schistosome risk at different points in the transmission season.

Additionally, *S. mansoni* cannot leave a snail once it has entered, therefore snails that are invaded by parasites indirectly reduce the infection risk of others in the population (King et al. 2011). By this mechanism, non-host species may contribute to a dilution effect in which increasing biodiversity is negatively correlated with prevalence of a parasite (Johnson et al. 2009, Johnson and Thieltges 2010, Civitello et al. 2015). The dilution effect typically refers to the effects of species diversity in a community context, though intraspecific genetic diversity has been shown to also have similar effects on parasite transmission (Altermatt and Ebert 2008, Ostfeld and Keesing 2012). If certain traits of a focal host, such as body size, cause the host to be more likely to be exposed to the parasite, though less likely to become infected once exposed, this may mimic the qualities of a dilution effect when transmission is observed at the population level.

Here, I aim to characterize how the size-dependent traits of exposure and susceptibility affect transmission in host populations that vary in their ratios of body size, i.e., are "size-structured". I conducted experiments across size-structured populations of *B. glabrata* with the goal extending our existing schistosome transmission model to predict overall parasite transmission and estimate host susceptibility and exposure rates for a known population size distribution of *B. glabrata.* I then assessed the fit of several competing transmission models using maximum likelihood estimation and Akaike's Information Criteria. I hypothesize that in a population of *B. glabrata*, larger snails may serve as a sink for *S. mansoni* by attracting the parasite but not becoming infected, thus shielding smaller snails from *S. mansoni* and reducing parasite prevalence at the population level. I also hypothesize that models accounting for differences in exposure and susceptibility with snail body size will outperform current or null models that ignore body size. This study expands upon the research done in the Civitello Lab and plays a role in our work to interpret the role of host body size on schistosome transmission risk for people in endemic regions.

2 Methods

2.1 Snail Maintenance

B. glabrata snails of the NMRI strain were maintained under favorable conditions. Snails were kept in HHCOMBO artificial lake water (Baer and Goulden 1998) at 26°C with a 12:12 light:dark cycle. Snails were fed a diet of fish flakes (Omega One) and chicken feed (Nutrena Meatbird Crumbles) suspended in agar *ad libidum.*

2.2 Experimental Design

The experiment was conducted using a fully factorial design (Fig. 2). Five population size-structures, each with a total of 18 snails, were exposed to three densities of parasites in 24-hour transmission trials in 15-liter tanks, creating 15 total treatment combinations. *B. glabrata* were first assigned into three size classes by shell diameter: “small” (2-3mm), “medium” (6-8mm), and “large” (12-15mm). Different size-structures were determined by the ratio of snails from each of the three size classes. The uniform size structure consisted of 18 snails from the same size class: “uniform small” (1:0:0), “uniform medium” (0:1:0), and “uniform large” (0:0:1). “Equal” size-structured mesocosms had 6 snails from each size class (1:1:1). “Small skewed” size structures contained 12 snails from the smallest size class and 3 snails from both the medium and large size classes (4:1:1). Parasite densities also varied between the tanks: 36 (2 miracidia/snail), 144 (8 miracidia/snail), and 252 parasites per tank (14 miracidia/snail). We obtained *S. mansoni* eggs from experimentally infected mice livers and hatched free-swimming miracidia via exposure to light. Four replicates of these trials were conducted between March – December 2021. To control for parasite batch variation in each of the four replicates, a control group consisting of all medium snails in individual well plates was simultaneously exposed to each of the parasite densities.

2.3 Infection Diagnosis

24 hours post-exposure, all snails were collected, sorted by size and treatment group, and maintained for 5 weeks (the prepatent period for *S. mansoni*) in favorable conditions (See Methods 2.1). Snails were diagnosed visually by shedding in individual well plates at 4- and 5-weeks post-exposure to obtain prevalence data. Shedding refers to the process by which the parasite emerges from the snail in response to specific environmental factors (light) which we mimic in the lab (Asch 1972). Snails were diagnosed as "infected" if we observed *S. mansoni* cercariae in their well plate, or "uninfected" if no cercariae were detected. Snails infected at week 4 were sacrificed, while uninfected snails were returned to their tanks to repeat this process at week 5. Snails that died while prepatent or between weeks 4 and 5 of the shedding process were not included in our results (6.25% of all snails).

To compare prevalence data amongst the various size structures and size classes, we used the glmmTMB package in R (Brooks et al. 2017) to create a generalized linear mixed model (GLMM) accounting for the random effects between individual tanks and exposure dates. We then used the emmeans package to conduct an estimated marginal means (least-squares means) *post hoc* test (Lenth 2022).

2.4 Model Creation and Maximum Likelihood Estimation of Parameters

We then built deterministic size-dependent transmission models for parameterization and competition using the experimental data (Fig. 3). The fully size-dependent model, which varies both exposure (ε) and susceptibility (σ) with host body size, predicts that different size classes within a single size-structured population will experience different levels of infection prevalence

and that populations of differing size-structures will differ in their overall population infection prevalence. In the null model, all functions of length are constants, therefore no variation in prevalence by size-structure can be predicted. We also created two hybrid models: (1) the size-dependent exposure-only, in which only exposure is a function of host body size, and (2) the size-dependent susceptibility only, in which only susceptibility is a function of body size. We used the bbmle package in R to conduct maximum likelihood estimation to estimate parameters for ε and σ for each model using the binomial error distribution (Strauss et al. 2019, Bolker and R Development Core Team 2021).

2.5 Model Competition

Using corrected Akaike's information criteria (AICc) in R (R Core Team 2021), we competed the fully size-dependent model against the size-dependent exposure only, the size-dependent susceptibility only, and the size-independent null model. A batch-controlled fully size-dependent model was also competed, which controlled for differences in host susceptibility between the 4 experimental trials.

3. Results

3.1 Experimental Results

Average prevalence for snails varied by size structure and parasite density (Fig. 4). Of the size structures, the uniform small size structure had the greatest prevalence at each parasite density (36 parasites: M = 0.37, SD = 0.26; 144 parasites: M = 0.69, SD = 0.078; 252 parasites: M = 0.83, SD = 0.034), and was significantly greater than all size structures except for the small skewed (generalized linear mixed model (GLMM), uniform large: $p<.0001$; uniform medium: $p=0.0018$; equal: $p= 0.0005$). The uniform large size structure had the smallest prevalence of the tanks at the lowest and highest parasite densities (36 parasites: M = 0.13, SD = 0.11; 252 parasites: M = 0.32, SD = 0.13), and prevalence for the uniform large size structure was significantly less than the small skewed (GLMM, $p= 0.0065$) and the uniform small (GLMM, $p <.0001$).

Average prevalence for snails in each size class also varied by population size structure and parasite density. Infection success of small snails in the uniform small size structure was significantly higher than in the equal treatment (GLMM, $p<.0001$). Infection success of medium snails and large snails did not significantly differ between size structures.

In our experiment, small snails were significantly more likely to be infected than large snails (GLMM, $p=0.0031$). There were no significant differences in infection success between small and medium or between medium and large snails.

3.2 Model Results

The results from the maximum likelihood estimation of parameters predicted exposure (ε) to be an increasing function of body size and for susceptibility (σ) to be a decreasing function of body size, accurately reflecting our predictions of these relationships.

Based on the results from the AIC model competition (Table 1), the batch-controlled fully size-dependent was the strongest model fit to the experimental data (Akaike weight, W_{AIC}=0.88), followed by the original fully size-dependent model (Akaike weight, W_{AIC}=0.12). The size-dependent exposure-only, size-dependent susceptibility-only, and size-independent null models were very poor fits to the experimental data (Akaike weights, W_{AIC}<0.001). Figure 5 illustrates the predictive abilities of both the fully size-dependent and size-independent models against the experimental results.

4. Discussion

Individual host heterogeneities play a role in determining population-level disease transmission dynamics. For the parasite *S. mansoni* and its intermediate host snail *B. glabrata*, body size is a key host trait that influences infection success of the parasite and, in turn, influences population prevalence within a size-structured population. Our experimental results provide evidence that *B. glabrata* populations of varying size-structure vary in population infection prevalence. Furthermore, we observed differences in infection prevalence of *B. glabrata* of varying body sizes within the size-structured populations.

We originally hypothesized that larger snails may act as a sink to remove parasites from the water while not becoming infected themselves, thereby decreasing the infection prevalence for the smaller individuals in the population and also decreasing the overall population prevalence. While the larger snails did not have significant differences in prevalence when in their uniform size structure compared to their size structures that included smaller snails, the small snails benefitted significantly from the presence of the larger snails in the equal size structure when compared to their uniform structure. Additionally, overall population prevalence was significantly lower in the equal size distribution than the uniform small (Fig 4). However, because our measurements of infection prevalence only show successful parasite infections, our experimental design does not allow for us to definitively determine if larger snails acted as a sink to remove parasites from the water while not becoming infected themselves. Regardless, our results provide evidence that the presence of larger snails produced an indirect protective effect by lowering the infection prevalence for their smaller counterparts.

Parameterization of the fully size-dependent transmission model reflected the known relationships between *B. glabrata* body size and exposure and susceptibility, supporting prior research (Niemann and Lewis 1990, Théron et al. 1998). Furthermore, the results from the AIC test shows that the fully size-dependent model strongly outperforms the size-dependent susceptibility-only, the size-dependent exposure only, and the size-independent null model (Table 1). When the size-dependent model incorporates batch effects between the 4 experimental trials, the model performs even better. This evidence supports our second hypothesis that models accounting for differences in exposure and susceptibility with snail body size will outperform current or null models. We hope that this finding emphasizes the importance of incorporating host heterogeneity in models predicting population level dynamics.

Natural populations of freshwater snails in the genus *Biomphalaria* fluctuate in size structure with seasonality, birth pulses, and washout/flooding events (Loreau and Baluku 1987). Changes in population size structure could be the result of temperature (Mccreesh et al. 2014), flooding (Loreau and Baluku 1987), resource fluctuations (Civitello et al. 2020), or any combination of these factors. Our study provides evidence that size structure may play a role in parasite transmission. Because size structure can change so frequently, understanding the mechanisms behind changes in population size structure can help to predict parasite transmission in endemic areas and inform best practices for parasite and mollusk control.

A potential area for future expansion upon this project is to adopt a community ecology perspective through the addition of a non-host species. As explored briefly in the introduction, a non-host species may impact the host population transmission dynamics by creating a dilution effect via removing parasites from the water while not becoming infected, thus lowering infection prevalence

for the focal host population (Johnson et al. 2009, Johnson and Thieltges 2010, Civitello et al. 2015). However, non-hosts also vary in their traits and types of interactions with focal hosts and parasites, and unveiling the complex mechanisms by which non-host species can create this dilution effect is critical to our understanding of parasite transmission in these systems (Shaw and Civitello 2021). Our results naturally lead to the hypothesis that larger individuals of non-host species are likely to be stronger diluters than smaller individuals because they will encounter, and therefore remove, more free-living parasites. Integrating a non-host species may give us greater insight into the natural dynamics of this host-parasite system.

5. Figures

5.1 Figure 1

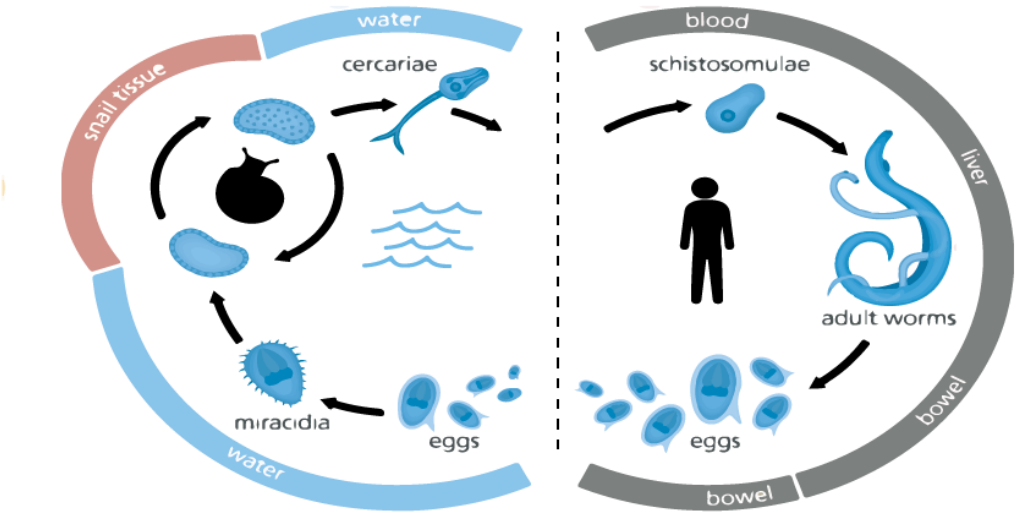

Figure 1. Schistosomes undergo a complex life cycle. Human hosts excrete schistosome eggs into water, where eggs hatch as free-swimming miracida. Miricida must enter a snail vector, in which they reproduce asexually. Schistosomes exit their snail vectors as free-swimming cercariae, which go on to infect human hosts. Within humans, schistosomes develop futher, reproduce sexually, producing more eggs and continuing the cycle.

5.2 Figure 2

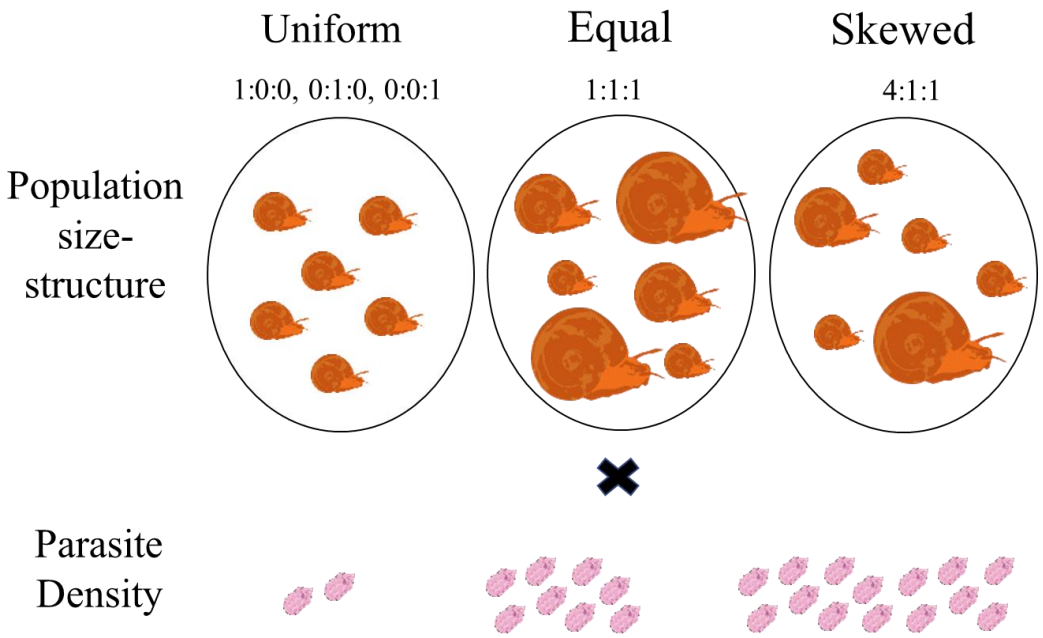

Figure 2. Fully factorial design of size-structured *B. glabrata* populations and *S. mansoni* treatments. Five population size structures snail: (1) uniform small, (2) uniform medium, (3) uniform large, (4) equal, and (5) skewed were exposed to each of three parasite densities: 2 parasites/snail (36), 8 parasites/snail (144), and 14 parasites/snail (252).

5.3 Figure 3

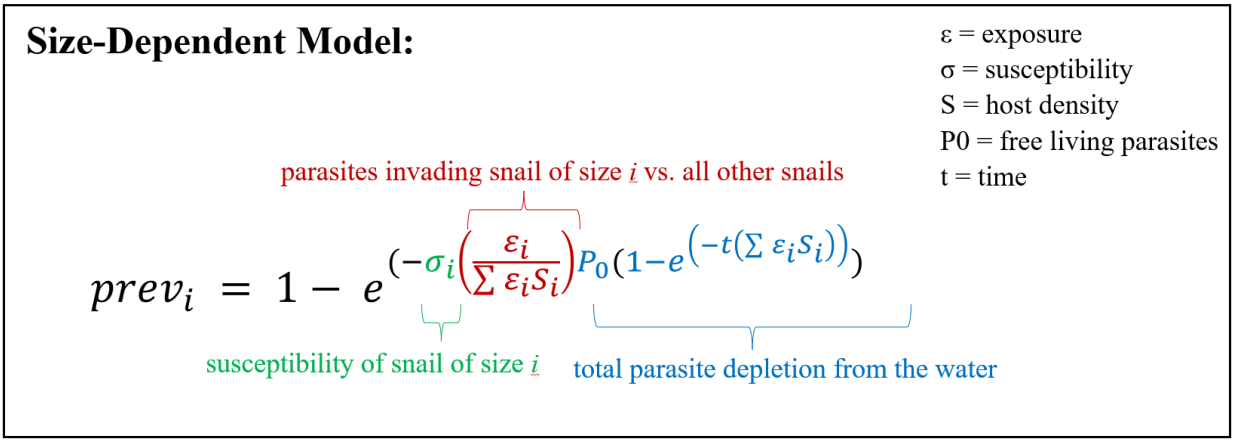

Figure 3. The size-dependent transmission model for a single time-step transmission experiment predicts infection prevalence of a snail of size *i*. σ_i is a decreasing function of body size, and ε_i is an increasing function of body size.

5.4 Figure 4

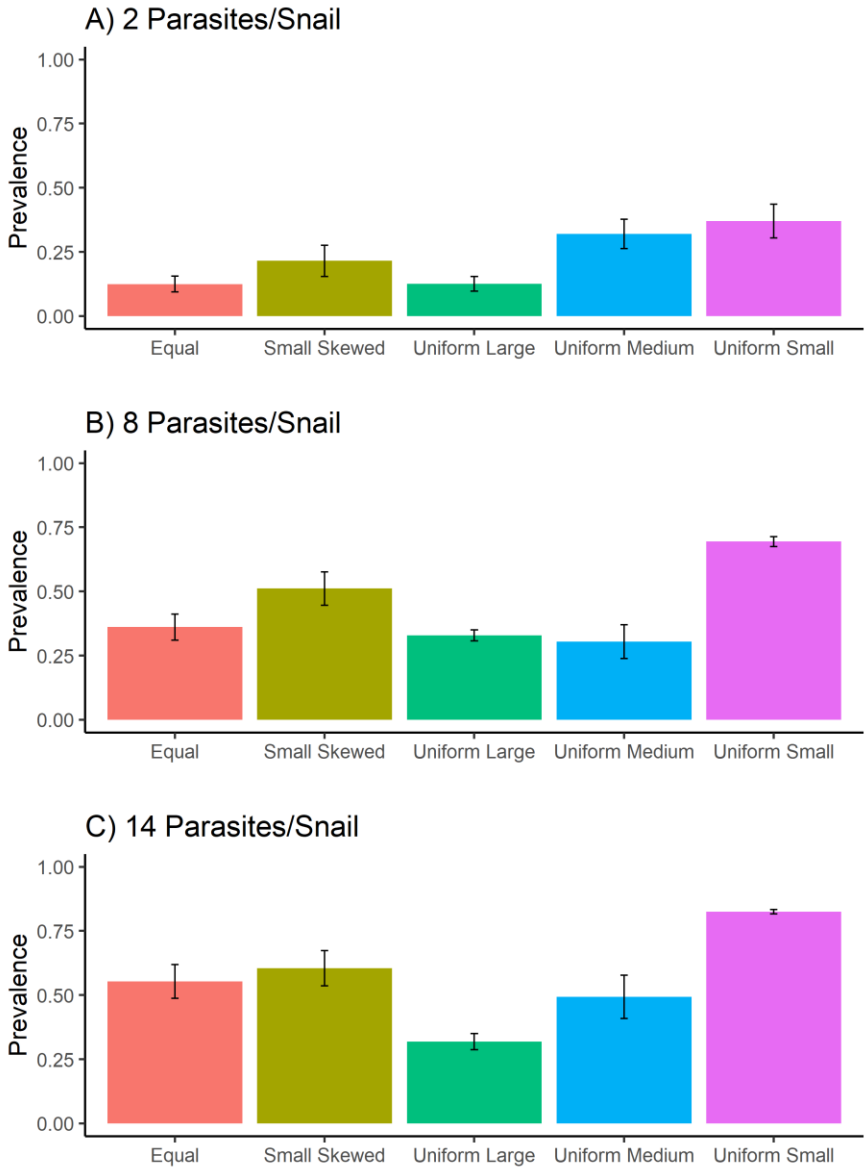

Figure 4. Experimental results for population prevalence of size-structured populations. Population prevalence varies between size-structured populations. For each parasite density (A-C), the uniform small size structure shows the greatest prevalence. The uniform large size structure had the least prevalence at the lowest and highest parasite densities (A, C).

5.5 Figure 5

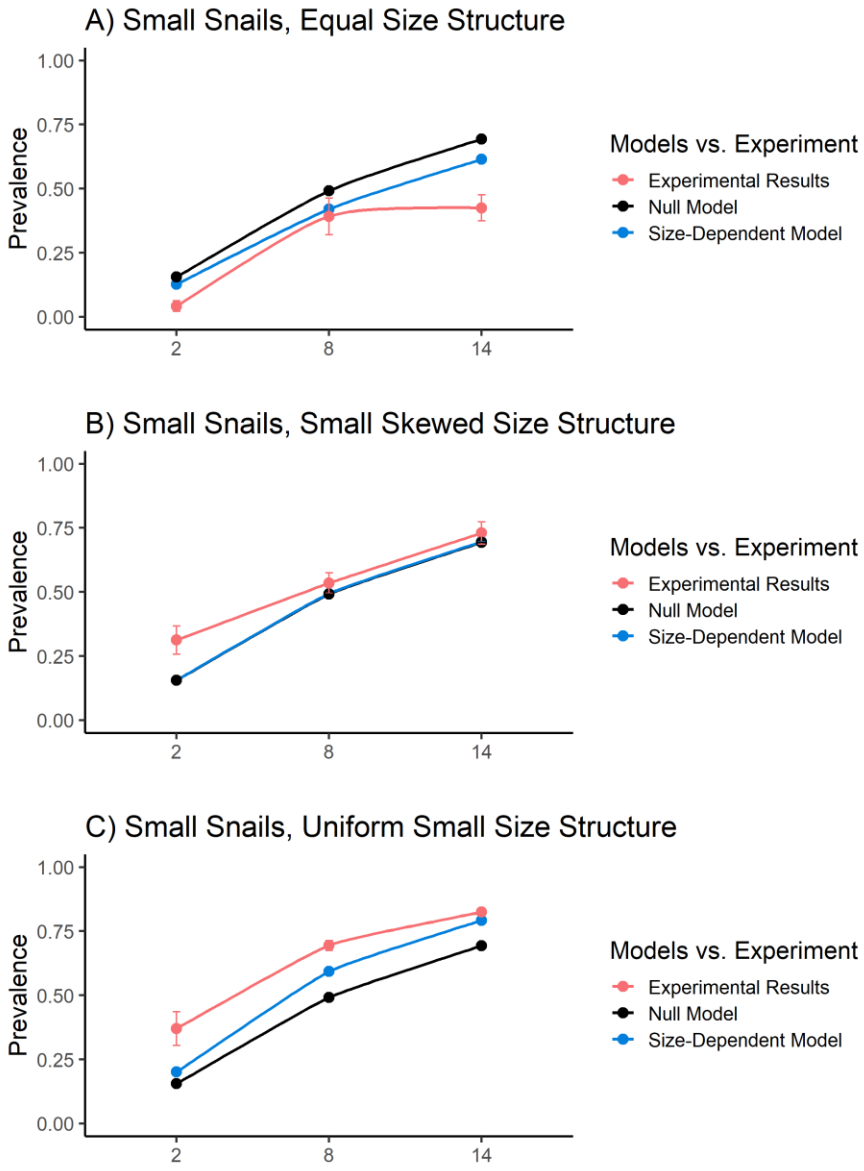

Figure 5. Small snail prevalence across size structures as predicted by the fully size-dependent model, size-independent (null) model, and experimental results. The fully size-dependent model strongly outperformed the null model in predicting prevalence for small snails. The null model predictions did not vary with body size, thus are the same for panels A-C.

5.6 Table 1

	AIC	ΔAIC	df	weight
Batch-Controlled, Fully Size-Dependent Model	997.1	0.0	8	0.88
Fully Size-Dependent Model	1001.0	3.9	4	0.12
Size-Dependent Susceptibility Only	1042.8	45.7	3	<0.001
Size-Dependent Exposure Only	1098.5	101.4	3	<0.001
Size-Independent (Null Model)	1134.6	137.5	2	<0.001

Table 1. Batch-controlled, fully size-dependent model proves stongest model using the Akaike's Information Criterion test. By definition, the ΔAIC score for the winning model = 0. Other models in the table are sorted by increasing ΔAIC. Typically, a ΔAIC < 10 indicates a relatively strong model performance. ΔAIC scores also correspond to the model's Akaike weight, which denotes the probability that the model is the best model fit when compared to the rest.

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