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Plastic pollution reshapes snail intermediate host life history: implications for schistosomiasis transmission

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Abstract

Background

Plastic debris is increasingly recognized as an ecological driver of mosquito-borne disease by creating freshwater breeding habitat, yet its consequences for snail hosts of human schistosomiasis transmission remain poorly understood. For these snail hosts, including *Bulinus truncatus*, plastic pollution may alter population dynamics by modifying life-history processes through surface biofilm-derived nutritional subsidies, habitat alteration, and toxicity-induced reproductive trade-offs.

Methods

We performed two laboratory experiments that investigated the effects of macro- and microplastics, individually and jointly, on *B. truncatus'* life-history traits (survival, growth, egg mass production, hatching) by using environmentally realistic sizes, polymers, concentrations, and biofouling status. To assess the overall impact of plastic pollution on snail demography, we derived a measure of reproductive output under each plastic treatment, i.e., the expected number of cumulative viable offspring produced over the experiment.

Results

We found that macroplastic boosted somatic growth, egg mass production, and hatching, consistently playing the most critical role in the demographic increase driven by biofilm-derived nutritional subsidies. Microplastics reduced hatching success but improved survival and growth, resulting in a marginal population decline due to toxicity-induced maintenance-reproduction trade-offs. Combined exposure elevated survival, growth, and egg mass production while mitigating microplastic-induced hatching costs, generating positive but attenuated gains due to nutrition-buffered toxicological costs. Collectively, these results demonstrate that plastic exposure can shift snail population trajectories by altering life-history processes that scale to net reproductive output, with biofouling emerging as the primary driver of demographic response.

Conclusions

Our findings show that plastic pollution increases *B. truncatus* demographic output by altering its key life-history traits, which prior work has linked to increased cercarial production with critical implications for human transmission risk. These findings identify plastic pollution as a previously unrecognized pathway and demonstrate that ecological transformation of plastic debris is the dominant determinant of host demographic response with implications for integrating environmental management into disease control strategies.

Background

Plastic pollution has emerged as an Anthropocene-driven crisis, resulting from escalating plastic production and waste accumulation with slow degradation. Global plastic production increased from 5 million tons in the 1950s to 300 million tons in 2020(1), and currently, 79% of the plastic waste ends up either in landfills or the natural environment(2). In recent years, a large body of research has found that plastic pollution increases human malaria and dengue transmission, because this debris creates additional breeding habitats that support the freshwater life stages of the mosquito vectors responsible for the disease spread(3–5). By contrast, little is asked about whether other medically important freshwater hosts similarly benefit from plastic pollution. Studies on the effects of plastic on snail-borne disease, including schistosomiasis, remain extremely limited. In particular, the effects of pollution on key snail life-history traits (i.e., survival, growth, fecundity, and hatching) are largely unknown, even though these traits govern population persistence and even minor shifts could influence transmission potential. To address this knowledge gap, it is necessary to focus on specific disease-relevant snail hosts where changes in life history could directly alter human health risk.

Human schistosomiasis is among the most prevalent neglected tropical diseases worldwide, second only to malaria in global burden(6), affecting approximately 250 million individuals, placing approximately 800 million at risk, and disproportionately impacting impoverished rural communities(7, 8). Urogenital schistosomiasis (caused by *Schistosoma haematobium*) is transmitted by snails in the genus *Bulinus*, while intestinal schistosomiasis (caused by *Schistosoma mansoni*) is transmitted by *Biomphalaria* snail species. Because urogenital schistosomiasis is often more prevalent, particularly among school-aged children, *Bulinus* snails become a priority for investigating life-history traits and demography(9, 10). Because no effective vaccines are available, current control strategies rely on snail control(11–13) and mass drug administration of praziquantel(14), which does not prevent reinfection(14), making accurate risk assessment crucial. Within this context, identifying how plastic pollution may drive snail host life history reveals three major mechanisms: (i) macroplastics function as substrates that promote biofilm growth and provide nutritional and habitat subsidies, (ii) microplastics exert direct toxicological effects following ingestion, and (iii) interactions between macroplastic-associated nutrition and microplastic-induced toxicity jointly shape *Bulinus* life-history traits.

Plastic pollution has the potential to influence *Bulinus* snails' life history via multiple mechanisms. These mechanisms differ by plastic form, with macroplastics (> 5 mm) and microplastics (from < 5 mm to > 1 μm) exerting distinct effects(15). Macroplastics may act as substrates for biological enrichment. Macroplastic surfaces can support dense microbial biofilms (also known as biofouling(16, 17), periphyton(17, 18), or plastisphere(19)) that serve as nutritious food for freshwater grazers like snails(20). These biofilms are rich in long-chain polyunsaturated fatty acids and sterols, which are essential nutrients that increase both food abundance and diversity and could enhance snail growth, survival, and reproduction(21). Moreover, macroplastic may create a novel habitat and refugia that support larger snail populations. By contrast, the dominant mechanism associated with microplastics is physiological toxicity following ingestion. Ingested microplastics can bioaccumulate and induce

oxidative stress, histopathological alterations, developmental toxicity, and behavior changes(22) that may reduce snail survival, fecundity, and hatching success. Notably, the nutritional properties of macroplastic-associated biofilms have the potential to mitigate these effects, as polyunsaturated fatty acids are known antioxidants that can support antioxidant defenses and increase total antioxidant capacity(23, 24). Therefore, co-exposing snails to nutrient- and antioxidant-rich biofouled macroplastic could potentially buffer microplastic-induced oxidative stress and alter key life-history outcomes. Plastics may also exert indirect environmental effects, such as shifting the water microbial communities and water parameters such as dissolved oxygen, pH, conductivity, etc(25, 26). Snails depend on these specific water parameters and, therefore, could be affected. Existing research typically analyzes macro- and microplastics separately, emphasizes acute exposure to high doses or abundance, or focuses on adult organisms(22, 27). Here, we address this gap with two sequential randomized controlled experiments exposing *Bulinus truncatus* to macro- and microplastics, systematically varying sizes, biofouling status, and abundance. We quantified key life-history traits and modeled demographic outcomes to assess the long-term effects of plastic. Our findings provide novel insights into how plastic pollution shapes the population dynamics of *Bulinus* snails with implications for schistosomiasis risks.

Materials and Methods

Lab experimental reagent and setup

We conducted two sequential rounds of experiments (Fig. 1) to investigate the long-term effects of plastic pollution on *B. truncatus*'s life-history response, with consideration of space constraints and personnel capacity. In the first round, we assessed the effect of plastic pollution on 4 treatment groups: negative control, virgin microplastic, biofouled macroplastic, and a combined treatment from September to December 2024. In the second round, we focused only on macroplastic and used 5 treatment groups: negative control, virgin macroplastic of high and low abundance, and biofouled macroplastic of high and low abundance from April to June 2025.

We used *B. truncatus* snails from the Biomedical Research Institute, which originated from Egypt. *B. truncatus* snails were born and raised under controlled laboratory conditions (~ 25°C, the ambient temperature of the laboratory, 12-hour light/12-hour dark, artificial pond water). In the first round, we housed 3 snails per jar, starting with 75 jars per treatment. In the macroplastic-only treatment and the combination treatment, 57 jars in each treatment were eliminated due to metal contamination from staples, which was intended for experimenting with biofouled macroplastic shaping (Text S1 and Table S1-S2). Therefore, only 18 jars in the macroplastic-only treatment and the combination treatment remained for the analysis. In the second round, we housed 3 snails per jar, 50 jars per treatment for negative control and biofouled macroplastic of high or low abundance, and 29 jars per treatment for virgin macroplastic of high or low abundance due to the limited total number of snails available from the Biomedical Research Institute. Each glass jar was 470 mL in size and contained 250 mL of water. The snails were fed organic romaine lettuce *ad libitum* based on the number of snails alive in each jar. We

covered each jar with mesh that allowed air exchange but limited evaporation. We changed the water in each jar weekly to maintain water quality(28).

Macroplastic reagent and periphyton profiling

We picked 500 mL single-use plastic bottles made from Polyethylene Terephthalate (PET) from Crystal Geyser®, given that it is one of the most common macroplastic types found in waterways(29, 30). All bottles were initially processed by removing labels and cutting them vertically in half to generate plastic sheets. To biofoul macroplastic, plastic sheets were submerged in freshwater, allowing surface microbial growth to develop(19, 31). Field biofouling was conducted at Jasper Ridge Biological Reserve (Searsville Lake) during experiment round one and the latter half of round two. During the first half of round two, biofouling was conducted under laboratory conditions due to unsuitable field conditions (extreme precipitation, high water velocity, and low temperatures). More details on the field and lab macroplastic biofouling protocols are available in Text S2. All plastic sheets (field- or lab-biofouled or virgin) were trimmed into uniform 5 cm x 4 cm pieces. To explicitly test the effect of macroplastic abundance, we manipulated the number of macroplastic pieces per jar, using three pieces to represent high abundance and one piece to represent low abundance. Round one and two included both abundance levels. Biofouled macroplastic pieces were replaced when 80–100% of jars showed visible periphyton depletion during weekly monitoring, and virgin macroplastic pieces were not replaced throughout the experiment.

Additionally, we profiled surface periphyton in experiment round two for each batch of biofouled macroplastic replacement, and 4 batches total. We measured the absolute abundance (dry mass; DM), proportion of organic matter (ash-free dry mass/ dry mass; AFDM/DM), nutritional quality (C:N), and autotroph to heterotroph ratio (chlorophyll a.:AFDM), periphyton physiological condition (chlorophyll a.:pheophytin)(32, 33), and took representative microscopy pictures, which all used standard protocols with more detail available in Text S3. We compared the lab versus field biofouling methodology across these aspects using a Mann-Whitney U test.

Microplastic reagent

We purchased Fluorescent Yellow Polyethylene (PE) microplastic spheres with a size range from 10 um to 27 um and a density of 1g/cc from Cospheric. We used virgin polyethylene microplastics, as polyethylene is the most common type of microplastic produced and found in natural systems(34, 35). Plastic is ubiquitous in the freshwater bodies, including where *Bulinus* snails are prevalent, with large heterogeneity in polymer type, shape, and size(36, 37). We used a 1000 ug/L microplastic concentration for the laboratory experiments, higher than the limited sampling report in Africa(38) and Western Europe(39), but lower than other microplastic and freshwater snail studies(40–42) to capture the effect of higher but ecologically relevant concentrations of microplastic where the *Bulinus* snails are present.

We prepared the virgin microplastic treatment using weighted PE microspheres and artificial pond water, formulated according to Biomedical Research Institute guidelines(43)

Lab experimental data collection

We recorded weekly snail survival, shell growth, and egg masses laid for 13 weeks in both rounds (Fig. 1B), while hatching success was assessed for 12 weeks in each round, as eggs enrolled during the final experimental week required a full 3-week evaluation period and could not be fully assessed within the observation window. We assessed snail mortality by whether the snail was motile, responded to a stimulus, or was obviously decaying (empty shell or shell residuals). We placed snails under the microscope to take shell pictures for length measurement in ImageJ, defined by the distance from the snail shell apex to the basal lip. Growth was calculated as the change in average snail length per jar from the previous week to the current week. We performed length measurement cross-validation with detailed data cleaning decisions reported in the Table S3 and Text S4. We then decanted the water from the jar through a metal sieve, counted and scraped off snail egg masses on the sieve surface, the macroplastic, and the sides of the jar. We disinfected the jar with 70% alcohol wipes between water changes. We placed approximately ten viable eggs in a well plate from each jar to test for hatching success with weekly water changes of the wells. We recorded the number of snails hatched at 1 week and 3 weeks after enrollment, and any egg that failed to hatch after 3 weeks of enrollment was taken as unviable.

Lab experimental statistical analysis

We estimated snail survival via a mixed-effect proportional hazard regression model using the *coxme* and *survival* packages in R. We modeled snail growth with a generalized additive model using Gaussian errors (identity link) and an AR(1) correlation structure; predictors included treatment and a smooth of the previous week's mean shell length. We analyzed snail egg mass production with a negative binomial generalized linear model (log link) with jar level random effect and analyzed hatching success with a zero-inflated binomial model with jar level random effect created in the glmmTMB package. We made all pairwise comparisons using the emmeans R package with the multivariate t distribution and a Bonferroni p-value correction for multiple comparisons.

Mathematical model on snail demography

We computed the snails' expected reproductive output as the number of live snail offspring that a snail would be expected to produce from the beginning of the experiment over the entire duration of the experiment, namely:

$$R_{obs}^{*} = \sum_{a=1}^{12} l(a) \times b(a) \times h(a)$$

where a is snail age [weeks] counting from the beginning of the experiment; $l(a)$ is age-specific survival probability at the treatment-level, i.e., the probability that a snail at the beginning of the experiment will survive until week *a*; $b(a)$ is age-specific birth rate at the treatment-level, that is, the mean number of eggs laid per snail. $h(a)$ is the age-specific hatching rate at the treatment-level, i.e., the fraction of eggs produced in week *a* that hatch.

To quantify the intrinsic variability of snail reproductive output within each treatment, we performed a nonparametric bootstrap at the jar level. Specifically, for each treatment, we (i) resampled jars with replacement while preserving the original sample sizes, (ii) computed the corresponding reproductive output R^*_{obs} for the resulting pseudo-dataset as described above, and (iii) repeated this procedure 10,000 times. This yielded (iv) a bootstrap distribution of jar-level reproductive output, which we then aggregated by treatment to obtain the mean reproductive output per treatment, and we used as a proxy for the underlying variability in reproductive performance.

To assess whether expected reproductive output differed significantly between treatments, we performed a permutation (randomization) test. For each pair of treatments *i* and *j*, we first computed the observed difference in the mean treatment-level reproductive output:

$$D_{obs} = R^*_{obs}(i) - R^*_{obs}(j)$$

Under the null hypothesis of no treatment effect, the jar labels "*i*" and "*j*" are exchangeable. We therefore pooled the jars from both treatments and repeatedly (n_{perm} = 10,000 times) reassigned the labels "i_{rep}" and "j_{rep}" at random (without replacement) while preserving the original sample sizes. For each permutation, we recalculated the expected reproductive outputs $R^*_{rep}(i)$ and $R^*_{rep}(j)$ using the precomputed jar-level R^*_{obs} for the randomized groups and obtained the permuted difference:

$$D_{rep} = R^*_{rep}(i) - R^*_{rep}(j)$$

The collection of D_{rep} values formed the null distribution against which D_{obs} was compared. Because we made no directional assumption, we used a two-tailed test, computing the *p*-value as:

$$p = \frac{\#\{|D_{rep}| \geq |D_{obs}|\} + 1}{n_{perm} + 1}$$

When evaluating multiple pairwise comparisons, we adjusted the resulting *p*-values using the Bonferroni correction to control for family-wise error (6 comparisons in round one, and 10 in round 2).

Results

Effect of plastic pollution on snail survival

Exposure to microplastics or microplastic-associated treatments increased survival probability in *B. truncatus* snails, while macroplastics with and without biofouling had no effects. A hazard ratio (HR)

near 1 indicates no difference in mortality risk compared to controls. In round one, the HR for snails in virgin microplastic relative to control was 0.55 (95% CI HR [0.40, 0.76], $p < 0.001$; Fig. 2A), corresponding to approximately a 45% lower risk of mortality compared to control snails. The HR for snails in the joint treatment with respect to control was 0.57 (95% CI HR [0.32, 0.99]), which is significant from the model output ($p < 0.05$) but nonsignificant from Tukey-adjusted pairwise comparison ($p > 0.05$). This estimate corresponds to an approximate 43% reduction in mortality risk relative to controls, which is not statistically robust. The HR for snails in biofouled macroplastic with respect to control is 1.09 (95% CI HR [0.69, 1.70], $p > 0.05$). Similarly, in the second round, survival probabilities did not differ significantly among any macroplastic treatments over the 13-week experiment ($p > 0.05$; Fig. 2A). For coefficients and pairwise comparisons, see Supplement Table S4-S9.

Effect of plastic pollution on snail shell growth

All plastic treatments significantly enhanced snail growth relative to controls, with growth varying based on the combination of treatments and abundance. In both rounds, snails in all treatment groups exhibited significantly greater weekly shell growth than the control. In round one, snails in the biofouled macroplastic produced the largest mean weekly growth compared to the control (93.3 μm, $p < 0.001$; Fig. 2B), followed by the joint treatment (51.2 μm, $p < 0.005$) and the virgin microplastic (40.1 μm, $p < 0.001$). In round two, both snails exposed to biofouled macroplastic or virgin macroplastic significantly increased weekly growth compared to control snails (33.2 um, $p < 0.005$; 27.9 μm, $p < 0.05$; Fig. 2B). Specifically, treatment groups with varying macroplastic abundance were ranked by growth from highest to lowest compared to the control as the following: high abundance virgin macroplastic (39.0 μm; $p < 0.05$), high abundance biofouled macroplastic (36.4 μm; $p < 0.01$), low abundance biofouled macroplastic (29.6 μm; $p < 0.05$), and low abundance virgin macroplastic (16.2 μm; $p > 0.05$). For coefficients and pairwise comparisons, see Supplement Table S10-S15.

Effect of plastic pollution on snail egg mass production

Exposure to macroplastic or macroplastic-associated treatments generally increased egg mass production in *B. truncatus* snails, although the significance varies by plastic biofouling and abundance. In experiment round one, the snails in the joint treatment had a significantly 20.8% higher egg mass production than the control ($p < 0.001$; Fig. 3A). The snails in virgin microplastic or biofouled macroplastic were not significantly different from the control ($p > 0.05$). In round two, both snails exposed to biofouled macroplastic or virgin macroplastic exhibited significantly more egg mass production compared to the control (17.4%, $p < 0.001$; 10.5%, $p < 0.005$; Fig. 3A). The variation in macroplastic abundance did not have a significant effect on egg mass production based on the coefficients and pairwise comparisons in Supplement Table S16-S21.

Effect of plastic pollution on snail hatching success

Microplastic exposure reduced snail hatching success, while macroplastic exposure markedly improved hatching success, although combined treatments had no significant effect. In round one, virgin

microplastic significantly reduced offspring hatching success by increasing the probability of structural zeros by approximately 57% ($p < 0.001$) while conditional hatching success among non-zero observations did not differ from controls ($p > 0.05$; Fig. 3B). Biofouled macroplastic or joint treatment did not significantly affect either the probability of structural zeros or conditional hatching success ($p > 0.05$). In round two, biofouled macroplastic significantly reduced the probability of structural zeros by approximately 36% relative to control ($p < 0.001$) and increased conditional hatching success by 3.2-fold ($p < 0.001$; Fig. 3B), indicating concurrent effects on both hatching occurrences and performance. Virgin macroplastic significantly increased conditional offspring hatching success by 9% ($p < 0.05$) but did not affect structural zeros ($p > 0.05$). Increasing macroplastic abundance enhanced hatching responses in biofouled treatments ($p < 0.001$) but not in virgin macroplastic treatments ($p > 0.05$). For coefficients and pairwise comparisons, see Supplement Table S22-S30.

The overall effect of plastic pollution on snail expected reproductive output

Plastic pollution of various sizes and biofouling conditions significantly impacted the snail population. Biofouled macroplastic plays the most critical role in driving expected reproductive output increases, with notable variations observed between experiments (Fig. 4). In round one, the expected reproductive output of snails exposed to both biofouled macroplastic and virgin microplastic was 25.3% larger than controls ($p < 0.001$) (Fig. 4A). Similarly, snails in biofouled macroplastic alone had a 26.2% increase in viable offspring per snail ($p < 0.001$), while those in virgin microplastic had an 8.3% decrease in viable offspring per snail ($p < 0.001$). In round two, all treatments differed significantly from each other in expected reproductive output ($p < 0.001$; Fig. 4B). Snails in high or low abundance biofouled macroplastic exhibited an increase of 89.2% and 66.4%, respectively, compared to controls ($p < 0.001$). Snails in high or low abundance virgin macroplastic treatments had increases of 24.0% and 19.5%, respectively, compared to controls ($p < 0.001$). For coefficients and pairwise comparisons, see Supplement Table S31-S34. A summary table of the estimated direction and significance between plastic treatments and snail life-history results for both rounds is provided in Fig. 5.

Profiling periphyton growth on biofouled macroplastic

We profiled the periphyton characteristics on macroplastics, including abundance, representative microorganisms, organic content ratio, community composition, physiological condition, and nutritional quality. The mean quantity of periphyton on macroplastics was 38.4 mg/100cm^2 (95% CI [3.4, 56.7]) in dry mass. Microscopic pictures were shown in Figure S13 at 40 times magnification of the representative microbial community grown on the surface of biofouled macroplastic. The mean AFDM: DM ratio was 0.25 (95% CI [0.20, 0.36]), and the mean autotrophic index was 1309 (95% CI [998, 1626]). The mean Chlorophyll: pheophytin ratio of the algae was 3.2 (95% CI [2.4, 3.7]), and the mean C: N ratio of lettuce was 10.1 (95% CI [8.8, 11.4]), and of algae is 12.8 (95% CI [10.1, 11.4]). For coefficients in box plots, see Text S5 and Figure S14.

Discussion

In two controlled laboratory experiments, our results demonstrate that plastic pollution alters snail life-history traits and, in turn, has the potential to substantially reshape population dynamics, with effects contingent on plastic size, biofouling, and abundance (Fig. 4). Across experiments, biofouled macroplastics consistently produced the largest demographic increases, including nearly doubling expected viable offspring per snail at high abundance in round two. In contrast, virgin materials exerted weaker and more variable effects: virgin macroplastic yielded modest population gains, whereas virgin microplastic led to slight population declines. The joint treatment of virgin microplastic with biofouled macroplastic produced intermediate increases in per-capita viable offspring. Together, these patterns indicate that ecological transformation of plastic in the aquatic environment (surface biofilm growth and accumulation), rather than the mere presence of debris, largely determines snail population responses, with plastic size and abundance modulating the magnitude of effects. The following sections detail the specific life-history responses and mechanisms underlying these population changes for each pollution scenario (Table 1).

Biofouled macroplastic underscores the critical role of periphyton as a nutritional and habitat subsidy that elevates snail growth and reproductive performance, with survival and hatching success showing context dependence between rounds. Before deployment, our analysis showed that periphyton on biofouled macroplastic accumulated at low to medium organic loads and supported diverse, heterotroph-dominant communities with healthy autotrophs (Text S5 and Figure S13-S14). The periphyton nutritional content per dry weight was comparable to lettuce, indicating that periphyton, alongside lettuce *ad libitum*, increased both dietary abundance and diversity. Periphyton provides essential nutrients such as long-chain polyunsaturated fatty acids and sterols that enhance snail growth, survival, and reproduction(21, 44). Previous studies found that greater food diversity can optimize snail growth and fecundity(21, 45). In round one, higher snail mortality or lower hatching success coincided with degraded water quality in some biofouled macroplastic jars, potentially due to either periphyton-driven or mortality-caused water quality deterioration. Prior review documented the concern of pathogenic microorganisms' adhesion to plastics that can destabilize water quality(31). The two experimental rounds showed non-significant and opposing trends in survival and hatching success, which could reflect natural variability in water quality and shifts in microbial community composition rather than a consistent effect of treatment. These insights suggest that newly formed biofilms on nominally virgin macroplastic may provide similar but attenuated benefits, consistent with its weaker yet positive demographic effects.

Virgin macroplastic may enhance snail growth, reproduction, and hatching success by developing nutritious surface biofilms after entering aquatic systems. Plastic was virgin at deployment and not replaced during the experiment. Although we did not directly quantify surface colonization, prior work shows that plastic surfaces rapidly accumulate biofilm over days to weeks(46) that serve as a food source to grazers on timescales that vary with environmental conditions, including light availability, temperature, and dissolved oxygen(18, 47). Our results are consistent with this mechanism, suggesting

that even initially virgin macroplastic can influence snail demography over the course of the 12-week experiment. Similar processes may also occur on microplastic, although this remains untested. While biofouling on larger plastic may provide nutritional subsidies, responses to microplastic particles likely reflect different physiological mechanisms beyond nutrition.

Snails exposed to virgin microplastic exhibited increased survival and somatic growth but reduced reproductive output, indicating a trade-off consistent with energy reallocation from reproduction to maintenance. This pattern aligns with reported endocrine-disrupting effects of polyethylene microplastic(48), to which snail reproduction is particularly sensitive due to neuroendocrine regulation(49). Microplastic-associated chemicals may exacerbate oxidative stress and reproductive disturbances(50), via an imbalance between reactive oxygen species and antioxidants that damage cells in the ovum. Similar maintenance-fecundity trade-offs have been documented in polyethylene microplastic exposures(51), supporting this interpretation. Growth gains may also reflect biofilm ingestion along with the nominally virgin microplastic or more efficient food assimilation via microplastic-facilitated shredding in the gut(50). We confirmed snail microplastic ingestion and egestion through microscopy (Figure S15). Notably, this trade-off is not observed under joint exposure to biofouled macroplastic and virgin microplastic, consistent with a nutrition-dependent response to endocrine-disruptors.

Joint exposure to biofouled macroplastic and virgin microplastic attenuates the reproduction-maintenance trade-off seen with microplastic alone, yielding a concurrent increase in survival, somatic growth, and egg production. We speculate a nutrition-dependent buffering of microplastic-induced endocrine and oxidative stress, whereby periphyton-derived micronutrients like antioxidants may mitigate reproductive impairment while sustaining somatic benefits. Such nutrient-mediated modulation of contaminant toxicity is well documented across biological systems humans(52, 53), animals(54, 55), and in vitro(56, 57). Periphyton communities containing polyunsaturated fatty acids(58, 59) with antioxidant properties may enhance antioxidant defenses and increase total antioxidant capacity under oxidative stress following microplastic ingestion(23, 24). These interacting nutritional and physiological effects likely explain the joint-treatment outcomes, which scale beyond individual life-history traits to shape the population-level consequences.

Plastic pollution tips the balance toward *B. truncatus*: improved survival and fecundity, faster maturation, and could potentially provide colonizable habitat, altered predator-prey dynamics, and create positive feedback that collectively inflates snail populations. Under realistic co-occurring microplastic and macroplastic pollution, our analysis indicates that snail populations would expand, driven by increases in expected reproductive output. We also found that the next generations of snails could reach reproductive maturity earlier with increased somatic growth when exposed to pollutants. Moreover, snails have been documented to colonize macroplastic debris as a substrate for settlement(60). The additional novel habitat and surface nutrition provided by biofouled macroplastic may further increase the carrying capacity of the system if all other ecological factors remain the same, reinforcing the positive feedback loop of a surging snail population. Beyond these direct demographic effects, plastic

pollution may also influence snail population dynamics through behavioral and community-level pathways. Although such processes were beyond the scope of this study, research has shown that marine snails exposed to microplastics exhibit increased vulnerability to predation due to reduced vigilance and antipredator responses(61). Macroplastic may additionally function as a physical refuge, as artificial plastic structures are widely used to enhance aquatic habitats(62, 63). However, these mechanisms remain speculative in freshwater snail systems and would benefit from targeted investigation. Field-based studies are therefore needed to validate whether plastic debris consistently serves as habitat and/or resource subsidies for *B. truncatus* and to determine how these changes translate to population growth and, as a result, schistosomiasis transmission risk.

Plastic pollution may elevate urogenital schistosomiasis risk by increasing *B. truncatus* abundance and per-capita parasite output, but its direct effects on cercarial production, survival, and infectivity remain a key knowledge gap. *B. truncatus* snails are the obligate intermediate host of human urogenital schistosomiasis. Independent of plastic contamination, growth in the snail population increases the total number of free-swimming cercariae released into water, raising human infection risk with routine water contact(64). Prior work also shows that faster snail growth and higher resource availability, which are observed in our experiment, boost cercarial production per capita once infected(65). These factors imply a higher human infection risk through both more contributing hosts and greater parasite loads per host. This is assuming the schistosome cercarial production, morphology, longevity, and infectivity remain unchanged despite plastic pollutant exposure, which remains untested. However, comparative studies report divergent cercarial outcomes across systems, which may plausibly reflect differences in the snail organs where parasites develop and reproduce. One study found that high nanoplastic diets yielded the greatest cercarial production in freshwater snail *Stagnicola elodes* infected with trematode *Plagiorchis* sp., which develops primarily in gonadal tissues(66). These findings are consistent with compensatory feeding that offsets the energy costs of rediae and sporocysts(67) and sustains elevated cercarial emergence(68). By contrast, another study reported that nanoplastic reduced parasite production in a marine snail *Zeacumantus subcarinatus*on infected with the trematode *Maritrema novaezealandensis*, which develops primarily in the guts and other soft tissue(69). It is likely because nanoparticle exposure imposes energetic and tissue-repair costs on the snail that divert resources from intramolluscan parasite development, and/or nanoparticles inflict direct physical or cellular damage on sporocysts, impairing their capacity to asexually produce cercariae. They also found that cercarial morphology and infectivity were unchanged, but survival declined under high concentrations due to oxidative stress, tegumental damage, or impaired osmoregulation. Conversely, high concentrations of microplastics lowered infection prevalence and intensity for tadpole hosts in an echinostome-*Helisoma trivolvis*-*Rana sylvatica* model(70). It likely arises because of draining cercarial energy upon microplastic contact and delaying host encounters beyond the brief infectious window. Given these divergent outcomes, the net impact of plastic pollution on *S. haematobium* transmission remains uncertain. Future studies are needed to quantify how plastic exposure alters snail susceptibility to infection and subsequent cercarial production, morphology, longevity, and infection success.

This experiment has several limitations and strengths. The macroplastics were biofouled in a location not inhabited by *B. truncatus*, but our profiling of periphyton traits provides a basis for comparison and for evaluating potential effects on snail life-history in different ecological contexts. Snails may have ingested trace microplastic scraped from the macroplastic surfaces, but we consider this exposure negligible relative to the orders-of-magnitude higher doses that produced the effects observed in our microplastic-related treatments. Due to laboratory constraints, we did not quantify the number of eggs per egg mass for each treatment on a weekly basis. Instead, we used the mean number of eggs enrolled in the hatching assay for each treatment and week as a proxy (around 10 eggs, which typically corresponds to one egg mass(71)). Because egg masses cannot be subdivided without structural damage, this approach provides a reasonable and consistent approximation across treatments and time points. Despite these limitations, our study is the first to quantitatively examine how plastic pollution shapes the key life-history traits of *B. truncatus*, an intermediate host of human schistosomiasis. Using various plastic size classes, polymers, biofouling status, and abundance, we capture key dimensions of environmental heterogeneity while isolating their biological effects. We measured life-history traits using standardized protocols to ensure comparability. We found that plastic pollution meaningfully altered the *B. truncatus* population dynamics: biofouled macroplastic strongly enhances reproductive output; virgin microplastic produces a more subtle demographic decrease; the joint exposure yields concurrent improvement in life-history. This highlights macroplastic biofouling as a key ecological driver of snail population growth, with implications for schistosomiasis transmission. Future field studies are needed to confirm whether *B. truncatus* utilizes plastic debris, alongside laboratory experiments quantifying snail susceptibility and cercarial production under plastic exposure, to determine how plastic pollution influences the disease transmission risk to inform public health interventions.

Declarations

Ethics approval and consent to participate

Not applicable, as this study did not involve human participants or vertebrate animals.

Consent for publication

Not applicable.

Competing Interest

The authors have declared that no competing interests exist.

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Author Contribution

AY, KRM, JTV, and GDL designed the research; AY performed the research; AY contributed new reagents and analytical tools; AY, KRM analyzed the data; AY, KRM, JTV, and GDL interpreted the data; AY wrote the paper; AY, KRM, JTV, and GDL all edited and revised the manuscript.

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Data Availability

All data and analysis code supporting the findings of this study are available in the Zenodo repository at the following URL: https://doi.org/10.5281/zenodo.19685289

References

1. Padha S, Kumar R, Dhar A, Sharma P. Microplastic pollution in mountain terrains and foothills: A review on source, extraction, and distribution of microplastics in remote areas. Environmental Research. 2022;207:112232. doi:10.1016/j.envres.2021.112232
2. Jung YS, Sampath V, Prunicki M, Aguilera J, Allen H, LaBeaud D, et al. Characterization and regulation of microplastic pollution for protecting planetary and human health. Environmental Pollution. 2022;315:120442. doi:10.1016/j.envpol.2022.120442
3. Maquart PO, Froehlich Y, Boyer S. Plastic pollution and infectious diseases. The Lancet Planetary Health. 2022;6(10):e842–5. doi:10.1016/S2542-5196(22)00198-X PubMed PMID: 36208647.
4. Abdullah MS, Islam MJ, Hasan MM, Sarkar D, Rana MS, Das SS, et al. Impact of Waste Management on Infectious Disease Control: Evaluating Strategies to Mitigate Dengue Transmission and Mosquito

Breeding: A Systematic Review. Journal of Angiotherapy. 2024;8(8):1–12. doi:10.25163/angiotherapy.889850

5. Rosser JI, Tarpenning MS, Bramante JT, Tamhane A, Chamberlin AJ, Mutuku PS, et al. Development of a trash classification system to map potential Aedes aegypti breeding grounds using unmanned aerial vehicle imaging. Environ Sci Pollut Res. 2024;31(28):41107–17. doi:10.1007/s11356-024-33801-0
6. Hotez PJ, Kamath A. Neglected Tropical Diseases in Sub-Saharan Africa: Review of Their Prevalence, Distribution, and Disease Burden. PLOS Neglected Tropical Diseases. 2009;3(8):e412. doi:10.1371/journal.pntd.0000412
7. Gray DJ, McManus DP, Li Y, Williams GM, Bergquist R, Ross AG. Schistosomiasis elimination: lessons from the past guide the future. The Lancet Infectious Diseases. 2010;10(10):733–6. doi:10.1016/S1473-3099(10)70099-2 PubMed PMID: 20705513.
8. WHO. Schistosomiasis [Internet]. 2025 [cited 2025 Mar 13]. Available from: https://www.who.int/news-room/fact-sheets/detail/schistosomiasis
9. Léger E, Borlase A, Fall CB, Diouf ND, Diop SD, Yasenev L, et al. Prevalence and distribution of schistosomiasis in human, livestock, and snail populations in northern Senegal: a One Health epidemiological study of a multi-host system. The Lancet Planetary Health. 2020;4(8):e330–42. doi:10.1016/S2542-5196(20)30129-7
10. Ojo JA, Adedokun SA, Akindele AA, Olorunfemi AB, Otutu OA, Ojurongbe TA, et al. Prevalence of urogenital and intestinal schistosomiasis among school children in South-west Nigeria. PLOS Neglected Tropical Diseases. 2021;15(7):e0009628. doi:10.1371/journal.pntd.0009628
11. Rohr JR, Sack A, Bakhoum S, Barrett CB, Lopez-Carr D, Chamberlin AJ, et al. A planetary health innovation for disease, food and water challenges in Africa. Nature. 2023;619(7971):782–7. doi:10.1038/s41586-023-06313-z
12. Sokolow SH, Huttinger E, Jouanard N, Hsieh MH, Lafferty KD, Kuris AM, et al. Reduced transmission of human schistosomiasis after restoration of a native river prawn that preys on the snail intermediate host. Proceedings of the National Academy of Sciences. 2015;112(31):9650–5. doi:10.1073/pnas.1502651112
13. King CH, Sutherland LJ, Bertsch D. Systematic Review and Meta-analysis of the Impact of Chemical-Based Mollusciciding for Control of Schistosoma mansoni and S. haematobium Transmission. PLOS Neglected Tropical Diseases [Internet]. 2015. Available from: https://journals.plos.org/plosntds/article?id=10.1371/journal.pntd.0004290
14. Wang W, Wang L, Liang YS. Susceptibility or resistance of praziquantel in human schistosomiasis: a review. Parasitol Res. 2012;111(5):1871–7. doi:10.1007/s00436-012-3151-z
15. Wang Y, Liu G, Wang Y, Mu H, Shi X, Wang C, et al. The Global Trend of Microplastic Research in Freshwater Ecosystems. Toxics. 2023;11(6):539. doi:10.3390/toxics11060539 PubMed PMID: 37368639.

16. Amaral-Zettler LA, Zettler ER, Mincer TJ, Klaassen MA, Gallager SM. Biofouling impacts on polyethylene density and sinking in coastal waters: A macro/micro tipping point? Water Research. 2021;201:117289. doi:10.1016/j.watres.2021.117289
17. Bixler GD, Bhushan B. Biofouling: lessons from nature. Philos Trans A Math Phys Eng Sci. 2012;370(1967):2381–417. doi:10.1098/rsta.2011.0502
18. Azim ME, Verdegem MCJ, Dam AA van, Beveridge MCM. Periphyton: ecology, exploitation and management [Internet]. CABI; 2005 [cited 2025 Nov 3]. Available from: https://research.wur.nl/en/publications/periphyton-ecology-exploitation-and-management/
19. Amaral-Zettler LA, Zettler ER, Mincer TJ. Ecology of the plastisphere. Nat Rev Microbiol. 2020;18(3):139–51. doi:10.1038/s41579-019-0308-0
20. Bakenhus I, Jongsma R, Michler-Kozma D, Hölscher L, Gabel F, Holert J, et al. A domesticated photoautotrophic microbial community as a biofilm model system for analyzing the influence of plastic surfaces on invertebrate grazers in limnic environments. Front Microbiol. 2023;14. doi:10.3389/fmicb.2023.1238913
21. Bilka RH, Edward P. L. The Invasive New Zealand Mud Snail (Potamopyrgus antipodarum) Grows Faster When Consuming Periphyton Compared to Detritus. Journal of the Pennsylvania Academy of Science [Internet]. 2013 [cited 2025 Nov 3];87(3). Available from: https://dx.doi.org/10.5325/jpennacadscie.87.3.0125
22. Rodrigues CC, Salla RF, Rocha TL. Bioaccumulation and ecotoxicological impact of micro(nano)plastics in aquatic and land snails: Historical review, current research and emerging trends. Journal of Hazardous Materials. 2023;444:130382. doi:10.1016/j.jhazmat.2022.130382
23. Aiello A, Medoro A, Accardi G, Calabrò A, Carru C, Cannavo A, et al. Polyunsaturated fatty acid status and markers of oxidative stress and inflammation across the lifespan: A cross-sectional study in a cohort with long-lived individuals. Experimental Gerontology. 2024;195:112531. doi:10.1016/j.exger.2024.112531
24. Richard D, Kefı K, Barbe U, Bausero P, Visioli F. Polyunsaturated fatty acids as antioxidants. Pharmacol Res. 2008;57(6):451–5. doi:10.1016/j.phrs.2008.05.002 PubMed PMID: 18583147.
25. Yu H, Liu M, Gang D, Peng J, Hu C, Qu J. Polyethylene microplastics interfere with the nutrient cycle in water-plant-sediment systems. Water Research. 2022;214:118191. doi:10.1016/j.watres.2022.118191
26. Liu S, Qiu Y, He Z, Shi C, Xing B, Wu F. Microplastic-derived dissolved organic matter and its biogeochemical behaviors in aquatic environments: A review. Critical Reviews in Environmental Science and Technology. 2024;54(11):865–82. doi:10.1080/10643389.2024.2303294
27. Ding R, Tong L, Zhang W. Microplastics in Freshwater Environments: Sources, Fates and Toxicity. Water Air Soil Pollut. 2021;232(5):181. doi:10.1007/s11270-021-05081-8
28. Yu A, Vannatta JT, Gutierrez SO, Minchella DJ. Opportunity or catastrophe? effect of sea salt on host-parasite survival and reproduction. PLOS Neglected Tropical Diseases. 2022;16(2):e0009524. doi:10.1371/journal.pntd.0009524

29. Khairul Anuar NFS, Huyop F, Ur-Rehman G, Abdullah F, Normi YM, Sabullah MK, et al. An Overview into Polyethylene Terephthalate (PET) Hydrolases and Efforts in Tailoring Enzymes for Improved Plastic Degradation. IJMS. 2022;23(20):12644. doi:10.3390/ijms232012644
30. Dhaka V, Singh S, Anil AG, Sunil Kumar Naik TS, Garg S, Samuel J, et al. Occurrence, toxicity and remediation of polyethylene terephthalate plastics. A review. Environ Chem Lett. 2022;20(3):1777–800. doi:10.1007/s10311-021-01384-8
31. Bocci V, Galafassi S, Levantesi C, Crognale S, Amalfitano S, Congestri R, et al. Freshwater plastisphere: a review on biodiversity, risks, and biodegradation potential with implications for the aquatic ecosystem health. Front Microbiol. 2024;15. doi:10.3389/fmicb.2024.1395401
32. Vannatta JT, Minchella DJ. The influence of parasitism on producers and nutrients in mesocosm ecosystems. Food Webs. 2021;28:e00204. doi:10.1016/j.fooweb.2021.e00204
33. Biggs BJF, Kilroy C. Stream Periphyton Monitoring Manual [Internet]. New Zealand Ministry for the Environment; 2000. Available from: https://niwa.co.nz/sites/default/files/import/attachments/peri_complete.pdf
34. Li C, Busquets R, Campos LC. Assessment of microplastics in freshwater systems: A review. Science of The Total Environment. 2020;707:135578. doi:10.1016/j.scitotenv.2019.135578
35. Akanyange SN, Zhang Y, Zhao X, Adom-Asamoah G, Ature ARA, Anning C, et al. A holistic assessment of microplastic ubiquitousness: Pathway for source identification in the environment. Sustainable Production and Consumption. 2022;33:113–45. doi:10.1016/j.spc.2022.06.020
36. Talbot R, Chang H. Microplastics in freshwater: A global review of factors affecting spatial and temporal variations. Environmental Pollution. 2022;292:118393. doi:10.1016/j.envpol.2021.118393
37. Li C, Busquets R, Campos LC. Assessment of microplastics in freshwater systems: A review. Science of The Total Environment. 2020;707:135578. doi:10.1016/j.scitotenv.2019.135578
38. Aragaw TA. Microplastic pollution in African countries' water systems: a review on findings, applied methods, characteristics, impacts, and managements. SN Appl Sci. 2021;3(6):629. doi:10.1007/s42452-021-04619-z PubMed PMID: 34002166; PubMed Central PMCID: PMC8116826.
39. Lots FAE, Behrens P, Vijver MG, Horton AA, Bosker T. A large-scale investigation of microplastic contamination: Abundance and characteristics of microplastics in European beach sediment. Marine Pollution Bulletin. 2017;123(1):219–26. doi:10.1016/j.marpolbul.2017.08.057
40. Qu H, Ma R, Barrett H, Wang B, Han J, Wang F, et al. How microplastics affect chiral illicit drug methamphetamine in aquatic food chain? From green alga (*Chlorella pyrenoidosa*) to freshwater snail (*Cipangopaludian cathayensis*). Environment International. 2020;136:105480. doi:10.1016/j.envint.2020.105480
41. Rodrigues CC, Harayashiki CAY, S. Pereira E, Rodrigues GLS, Neves BJ, Rocha TL. How do microplastics alter molluscicidal activity? Effects of weathered microplastics and niclosamide in developing freshwater snails. Science of The Total Environment. 2024;922:171165. doi:10.1016/j.scitotenv.2024.171165

42. Phuong NN, Zalouk-Vergnoux A, Poirier L, Kamari A, Châtel A, Mouneyrac C, et al. Is there any consistency between the microplastics found in the field and those used in laboratory experiments? Environmental Pollution. 2016;211:111–23. doi:10.1016/j.envpol.2015.12.035
43. Alina. Artificial Pond Water - Biomedical Research Institute [Internet]. 2022 Jun 1 [cited 2025 Mar 11]. Available from: https://www.afbr-bri.org/schistosomiasis/standard-operating-procedures/artificial-pond-water/
44. Labed-Veydert T, Danger M, Felten V, Bec A, Laviale M, Cellamare M, et al. Microalgal food sources greatly improve macroinvertebrate growth in detritus-based headwater streams: Evidence from an instream experiment. Freshwater Biology. 2022;67(8):1380–94. doi:10.1111/fwb.13924
45. El-Emam MA, Madsen H. The effect of temperature, darkness, starvation and various food types on growth, survival and reproduction of Helisoma duryi, Biomphalaria alexandrina and Bulinus truncatus (Gastropoda: Planorbidae). Hydrobiologia. 1982;88(3):265–75. doi:10.1007/BF00008506
46. Ramsperger AFRM, Stellwag AC, Caspari A, Fery A, Lueders T, Kress H, et al. Structural Diversity in Early-Stage Biofilm Formation on Microplastics Depends on Environmental Medium and Polymer Properties. Water. 2020;12(11):3216. doi:10.3390/w12113216
47. Mulholland PJ, Steinman AD, Marzolf ER, Hart DR, DeAngelis DL. Effect of periphyton biomass on hydraulic characteristics and nutrient cycling in streams. Oecologia. 1994;98(1):40–7. doi:10.1007/BF00326088
48. Jeong E, Lee JY, Redwan M. Animal exposure to microplastics and health effects: A review. Emerging Contaminants. 2024;10(4):100369. doi:10.1016/j.emcon.2024.100369
49. Koene JM. Neuro-Endocrine Control of Reproduction in Hermaphroditic Freshwater Snails: Mechanisms and Evolution. Front Behav Neurosci. 2010;4. doi:10.3389/fnbeh.2010.00167
50. Colpaert R, Petit dit Grézériat L, Louzon M, de Vaufleury A, Gimbert F. Polyethylene microplastic toxicity to the terrestrial snail Cantareus aspersus: size matters. Environ Sci Pollut Res. 2022;29(20):29258–67. doi:10.1007/s11356-021-15824-z
51. Colpaert R, de Vaufleury A, Fritsch C, Rieffel D, Gimbert F. Ecotoxicological impacts of polyethylene, polystyrene and polyamide on the land snail Cantareus aspersus in a life cycle experiment. Journal of hazardous materials. 2025;491. doi:10.1016/j.jhazmat.2025.137968 PubMed PMID: 40120262.
52. Noguera-Navarro C, Candela-González J, Orenes-Piñero E. Nutritional Changes to Improve Female Fertility: Role of Obesity, Hormones, Dietary Patterns and Endocrine Disrupting Chemicals. Obstetrical & Gynecological Survey. 2025;80(1):44. doi:10.1097/OGX.0000000000001330
53. Skoracka K, Ratajczak AE, Rychter AM, Dobrowolska A, Krela-Kaźmierczak I. Female Fertility and the Nutritional Approach: The Most Essential Aspects. Advances in Nutrition. 2021;12(6):2372–86. doi:10.1093/advances/nmab068
54. Hornos Carneiro MF, Shin N, Karthikraj R, Barbosa F Jr, Kannan K, Colaiácovo MP. Antioxidant CoQ10 Restores Fertility by Rescuing Bisphenol A-Induced Oxidative DNA Damage in the Caenorhabditis elegans Germline. Genetics. 2020;214(2):381–95. doi:10.1534/genetics.119.302939

55. Zhang J, Liu W, Cui F, Kolehmainen M, Chen J, Zhang L, et al. Exploring the potential protective role of anthocyanins in mitigating micro/nanoplastic-induced reproductive toxicity: A steroid receptor perspective. Journal of Pharmaceutical Analysis. 2025;15(2):101148. doi:10.1016/j.jpha.2024.101148
56. Tripathi SK, Nandi S, Gupta PSP, Mondal S. Antioxidants supplementation improves the quality of in vitro produced ovine embryos with amendments in key development gene expressions. Theriogenology. 2023;201:41–52. doi:10.1016/j.theriogenology.2022.11.048
57. Huang G, Cui Z, Gao Q, An Z, Liu P, Wang H, et al. Tryptophan Metabolism as a Key Target in PFOS-Mediated Decline of Porcine Oocyte Quality. Environ Sci Technol. 2025;59(21):10182–93. doi:10.1021/acs.est.5c01550
58. Guo F, Ebm N, Bunn SE, Brett MT, Hager H, Kainz MJ. Longitudinal variation in the nutritional quality of basal food sources and its effect on invertebrates and fish in subalpine rivers. Journal of Animal Ecology. 2021;90(11):2678–91. doi:10.1111/1365-2656.13574
59. Gmeiner I, Vitecek S, Wassenaar LI, Kainz MJ, Pilecky M. Selective Retention of Long-Chain Polyunsaturated Fatty Acids in Heptageniidae (Ephemeroptera) of Subalpine Streams. Freshwater Biology. 2025;70(6):e70052. doi:10.1111/fwb.70052
60. De-la-Torre GE, Dioses-Salinas DC, Pérez-Baca BL, Millones Cumpa LA, Pizarro-Ortega CI, Torres FG, et al. Marine macroinvertebrates inhabiting plastic litter in Peru. Marine Pollution Bulletin. 2021;167:112296. doi:10.1016/j.marpolbul.2021.112296
61. Seuront L. Microplastic leachates impair behavioural vigilance and predator avoidance in a temperate intertidal gastropod. Biology Letters. 2018;14(11):20180453. doi:10.1098/rsbl.2018.0453
62. Grutters BMC, Pollux BJA, Verberk WCEP, Bakker ES. Native and Non-Native Plants Provide Similar Refuge to Invertebrate Prey, but Less than Artificial Plants. Armitage AR, editor. PLoS ONE. 2015;10(4):e0124455. doi:10.1371/journal.pone.0124455
63. Sass GG, Shaw SL, Fenstermacher CC, Porreca AP, Parkos JJ III. Structural Habitat in Lakes and Reservoirs: Physical and Biological Considerations for Implementation. North American Journal of Fisheries Management. 2023;43(2):290–303. doi:10.1002/nafm.10812
64. Wood CL, Sokolow SH, Jones IJ, Chamberlin AJ, Lafferty KD, Kuris AM, et al. Precision mapping of snail habitat provides a powerful indicator of human schistosomiasis transmission. Proceedings of the National Academy of Sciences. 2019;116(46):23182–91. doi:10.1073/pnas.1903698116
65. Malishev M, Civitello DJ. Linking Bioenergetics and Parasite Transmission Models Suggests Mismatch Between Snail Host Density and Production of Human Schistosomes. Integrative and Comparative Biology. 2019;59(5):1243–52. doi:10.1093/icb/icz058
66. Babaran D, Koprivnikar J, Parzanini C, Arts MT. Parasites and their freshwater snail hosts maintain their nutritional value for essential fatty acids despite altered algal diets. Oecologia. 2021;196(2):553–64. doi:10.1007/s00442-021-04944-5
67. Sorensen RE, Minchella DJ. Snail–trematode life history interactions: past trends and future directions. Parasitology. 2001;123(7):S3–18. doi:10.1017/S0031182001007843

68. Balsdon MKC, Koprivnikar J. Effects of microplastics and nanoplastics on host–parasite interactions in aquatic environments. Oecologia. 2024;204(2):413–25. doi:10.1007/s00442-023-05502-x
69. Mayeur L, Poulin R, Li CH. Plastic pollution and parasitism: Impact of nanoplastics on the transmission of a marine trematode parasite. Science of The Total Environment. 2025;1003:180735. doi:10.1016/j.scitotenv.2025.180735
70. Buss N, Sander B, Hua J. Effects of Polyester Microplastic Fiber Contamination on Amphibian–Trematode Interactions. Environmental Toxicology and Chemistry. 2022;41(4):869–79. doi:10.1002/etc.5035
71. Davis AA, Vannatta JT, Gutierrez SO, Minchella DJ. Lack of trade-offs in host offspring produced during fecundity compensation. Journal of Helminthology. 2021;95:e66. doi:10.1017/S0022149X21000651

Tables

Table 1 is not available with this version.

Figures

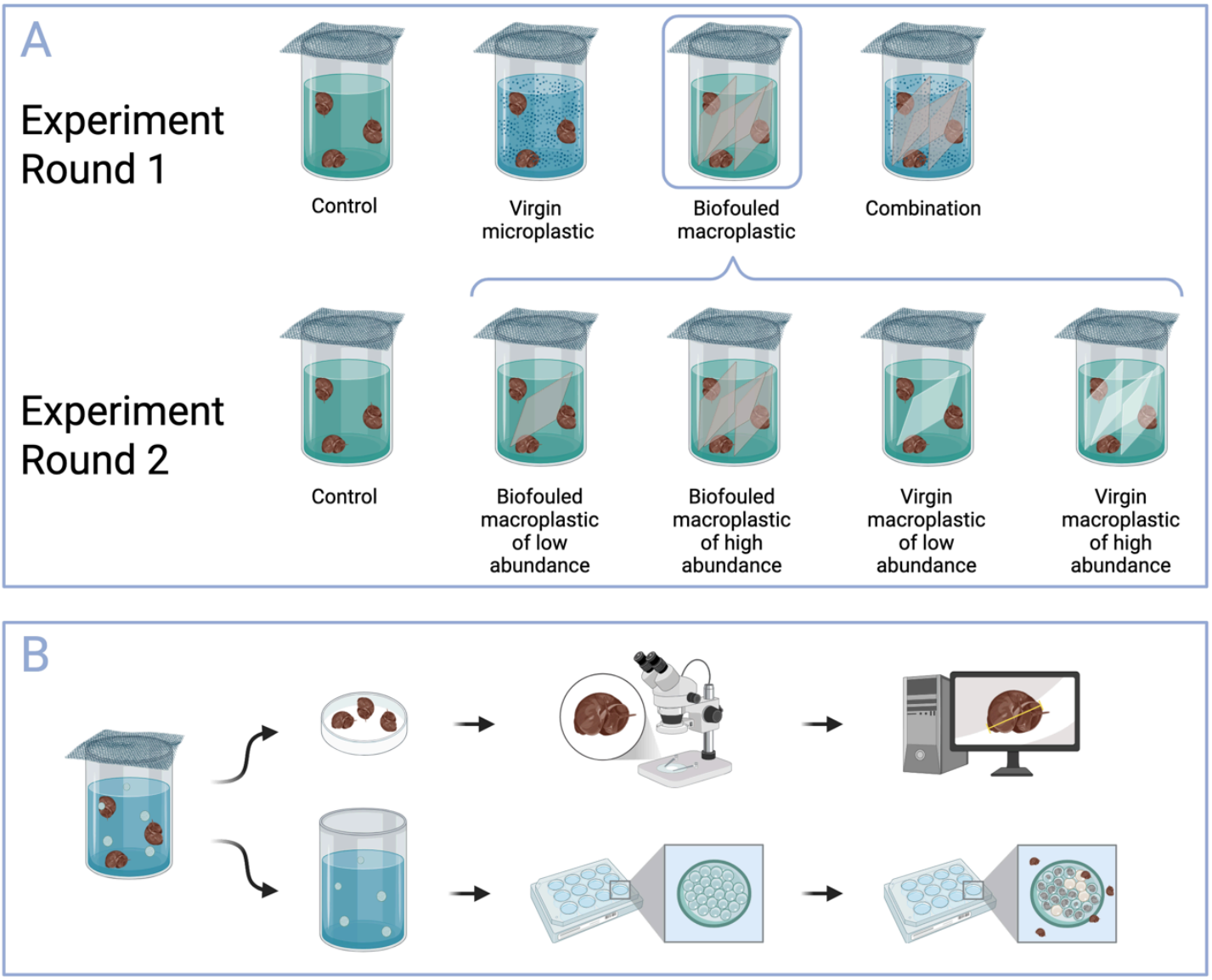


Figure 1

Setup for two rounds of experiments (A) and weekly data collection workflow diagram (B). In panel A, Experiment round 1 includes negative control (75 jars), virgin microplastic (75 jars), biofouled macroplastic (18 jars), and a combined treatment (18 jars). In panel B, each week, we recorded snail survival, shell growth, and egg masses, and assessed hatching success. We checked snail mortality and took live snail pictures under the microscope for snail shell measurements using ImageJ. Then, we collected and counted all egg masses from the container. We placed approximately ten viable eggs in a well plate from each jar to test for hatching success, where we recorded the number of snails hatched.

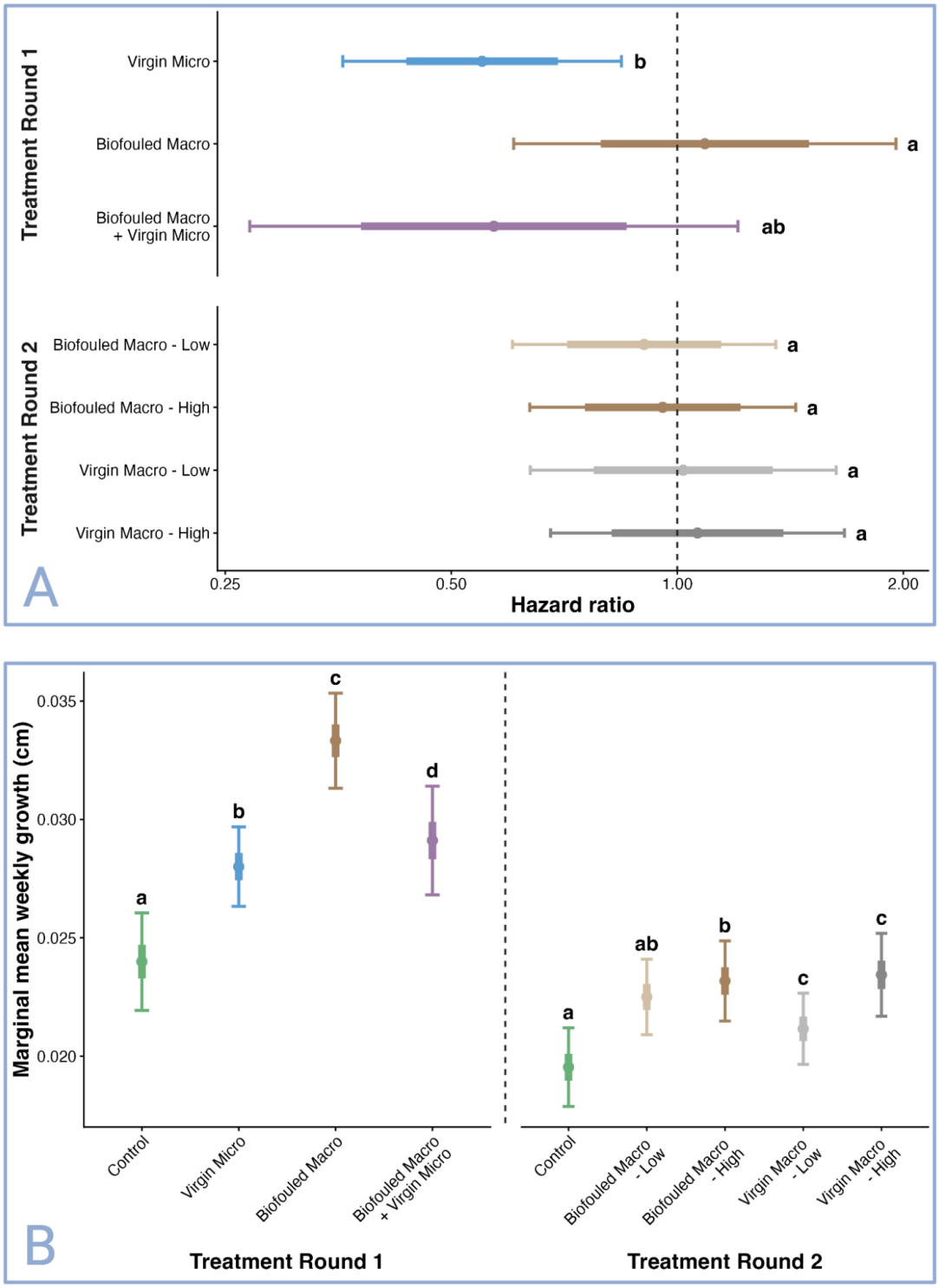


Figure 2

Effects of plastic treatments on snail survival (A) and growth (B) across experimental rounds. Letters above or adjacent to bars denote statistically significant differences among treatments (Tukey-adjusted pairwise comparisons). Hazard ratios (HRs) show the effect of each treatment relative to the control with HR = 1. Model coefficients and full pairwise comparison results are provided in Tables S4-S15. Weekly snail survival and shell growth for experimental rounds 1 and 2 are shown in Figure S1-S6.

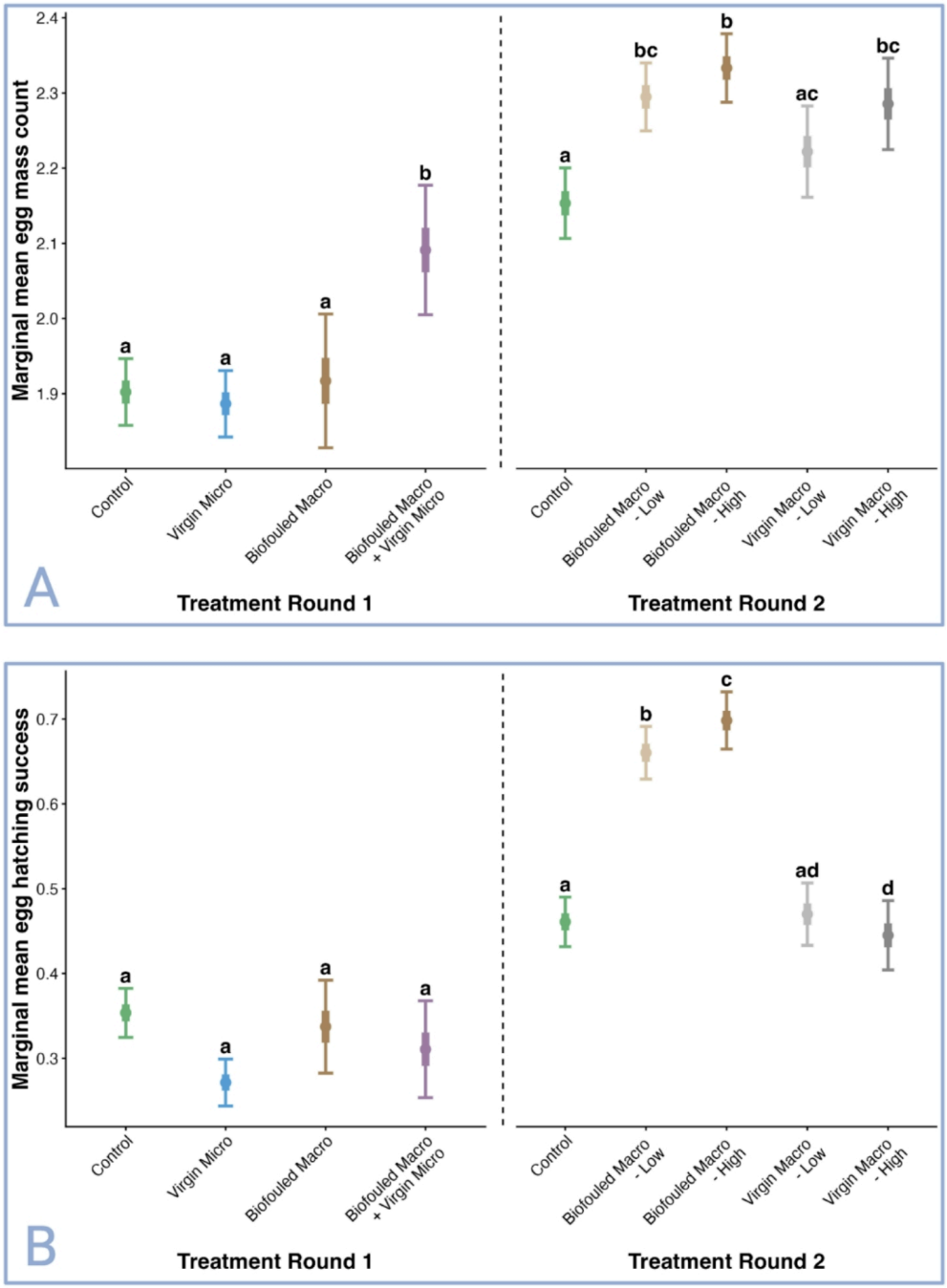


Figure 3

Effects of plastic treatments on snail egg mass production per jar per week (A) and hatching success (B) across experimental rounds. Values represent model-based marginal means, averaged over variation in snail size and density. Letters above or adjacent to bars denote statistically significant differences among treatments (Tukey-adjusted pairwise comparisons). Model coefficients and pairwise comparison

results are provided in Tables S16-S30. Weekly snail egg mass production and hatching success for experimental rounds 1 and 2 are shown in Figure S7-S12.

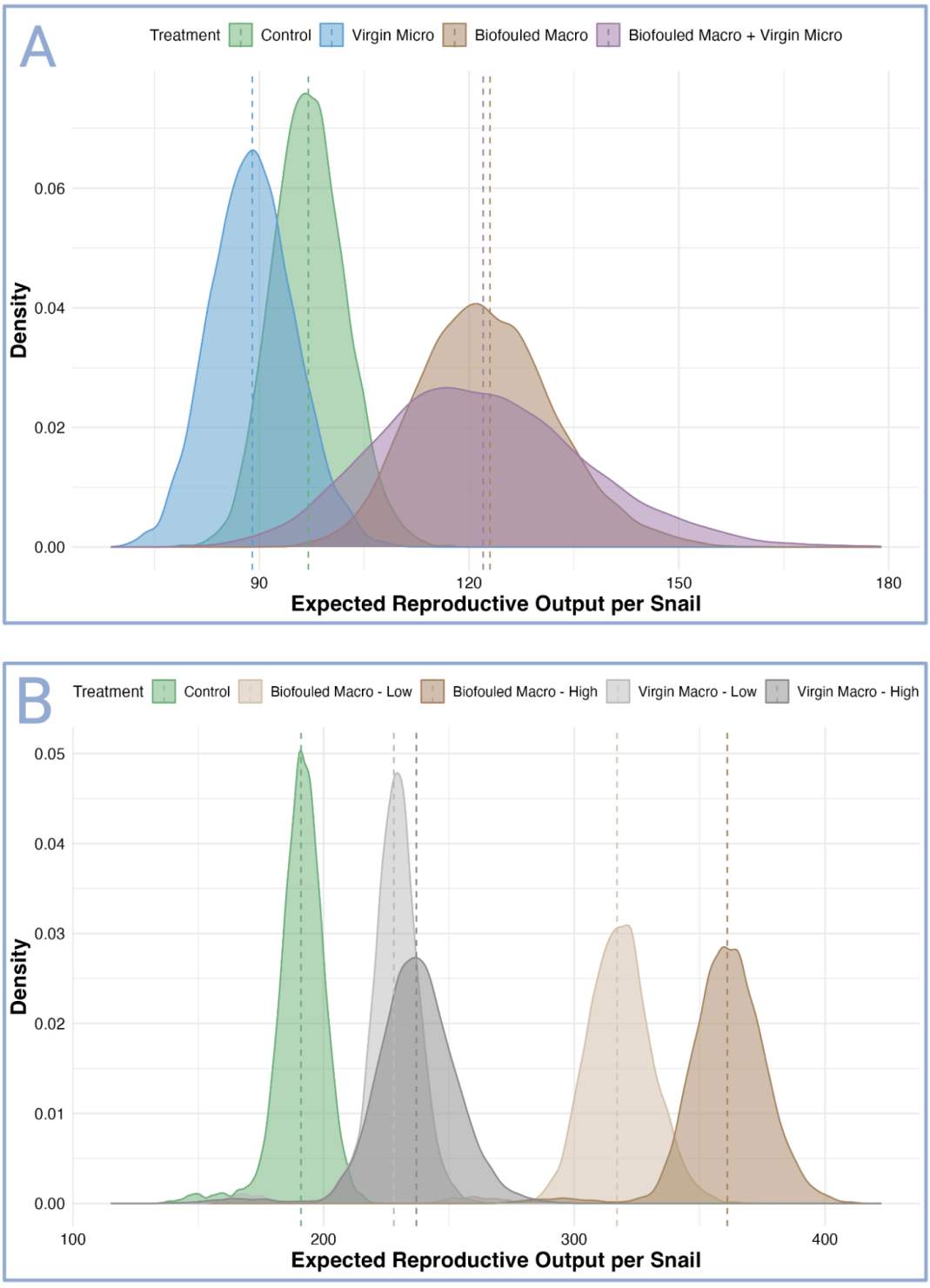


Figure 4

Distribution of the per-snail cumulative viable offspring by the end of the experiment. A) Experiment round 1 with control, biofouled macroplastic only, virgin microplastic only, and joint of both biofouled macroplastic and virgin microplastic. B) Experiment round 2 with control, biofouled macroplastic of high

or low abundance, and virgin macroplastic with high or low abundance. Model coefficients and pairwise comparison results are provided in Tables S31-S34.

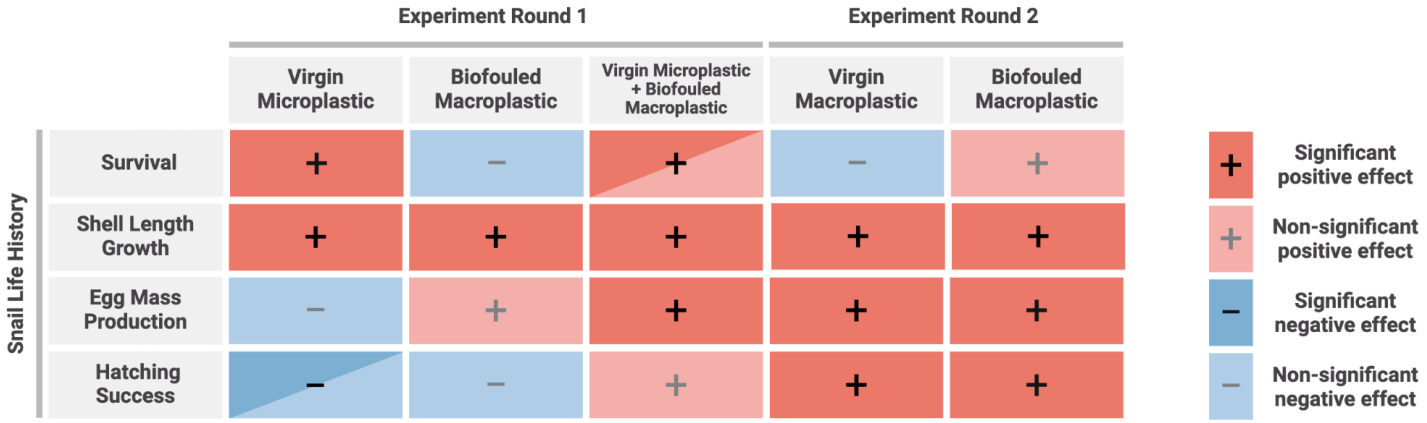


Figure 5

A summary table of the snail life history response under each pollution treatment in both experimental rounds. The dual-color box indicates the significant output of the model estimate is no longer significant under the Tukey-adjusted multiple pairwise comparison ($p > 0.05$).

Supplementary Files

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