Host nutrition and density jointly drive susceptibility and tolerance to parasite infection

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Abstract

1. Variation in host condition and population context is a major source of heterogeneity in infection risk and a key determinant of host-parasite dynamics. Host nutrition and density are drivers of this variation, yet their combined effects on host susceptibility and tolerance to parasite infection remain poorly understood. Here, we experimentally tested these interactive effects on susceptibility to parasite infections using a fish-ectoparasite system.
2. We conducted a mesocosm experiment manipulating diet quality (high vs. low-protein diets) and host density (low vs. high). Exposure to parasites was standardized by placing infected donor hosts in separated compartments, allowing transmission to non-infected target hosts through a mesh barrier. Parasite abundance per target host, mean infection intensity per tank and host body condition (scaled mass index, SMI) were modeled as a function of diet and density.
3. A high-protein diet increased parasite load across both density treatments, whereas a low-protein diet produced only weak differences between low- and high-density conditions. The combination of high-protein diet and high-density resulted in parasite loads several times greater than those observed under low-protein diet and low-density conditions, representing a sevenfold increase (from 17 to 120 parasites per host, on average). High-protein diets also doubled host body condition relative to low-protein diets.
4. Together, our results show that improved nutritional quality can increase host susceptibility while also supporting higher host condition under infection, consistent with increased tolerance. More broadly, the interaction between host diet and density is critical for understanding host-parasite dynamics and provides new insights into the mechanisms linking host condition, resistance and tolerance to infection.

Keywords: Host-parasite interactions, host density, nutritional condition, parasite transmission, infection risk

1. Introduction

Nutrition can influence the ability of hosts to overcome parasites and is considered an important driver of host-parasite interactions through both top-down and bottom-up processes (Sánchez et al., 2018; Wilson et al., 2020; Holdbrook et al., 2025). Host nutrition can improve the host immune system and control parasite infections (top-down effect), while also serving as a resource (bottom-up effect) that facilitates parasite establishment and reproduction within hosts (Vale et al., 2013; Wilson et al., 2020). As a result, variation in host nutritional status represents an important source of heterogeneity in susceptibility to parasite infection, with consequences for parasite dynamics in natural populations (Attwood, 2025; Cohen et al., 2026).

From a top-down perspective, host nutrition can affect both resistance and tolerance to parasites in different ways (Bize et al., 2008; Cressler et al., 2014; Civitello et al., 2015; Manzoli et al., 2018; Pike et al., 2019; Civitello et al., 2020; Manzoli et al., 2021). Malnutrition is expected to reduce resistance to parasitic infections through feedbacks between poor host condition and immunosuppression, often resulting in higher parasite loads (Beldomenico & Begon, 2010; Schmid-Hempel, 2021). In contrast, high host nutrition can increase tolerance to parasitic infections, as hosts generally have more resources available to repair infection-related damage without proportional fitness costs (Seppälä et al., 2008; Seppälä and Leicht, 2015). However, these effects are often context dependent and can vary with diet composition and quality, particularly protein availability (Wilson et al., 2019; Budischak et al., 2018, Blubaugh et al., 2023). For example, high-protein diets have been shown to increase host resistance to infection, whereas reductions in dietary protein can reduce host tolerance to parasites (Brunner

et al., 2014; Budischak et al., 2015; Budischak & Cressler, 2018; Sweeny et al., 2021; Wilson et al., 2020; Holdbrook et al., 2025).

From a bottom-up perspective, increases in host nutritional status can also benefit parasite fitness by enhancing growth and reproduction within hosts, thereby increasing parasite loads (Cornet et al., 2014; Holdbrook et al., 2025). Parasites depend on host-derived nutrients to establish and spread, and may directly and indirectly compete with hosts for limiting resources (Civitello et al., 2018; Holdbrook et al., 2025). In particular, parasite growth and reproductive capacity often depend on the nutritional value of host macronutrients, especially protein availability (Smyth, 1994; Wilson et al., 2020). On the other hand, starving hosts may limit parasite growth and loads (Civitello et al., 2018). Consequently, changes in host nutrition, including both nutrient quantity and quality, can strongly affect parasite establishment and proliferation, with consequences for host-parasite interactions (Becker et al., 2015; Pike et al., 2019; Wilson et al., 2020). Differences in host nutritional status may also affect parasite host choice, with parasites often exhibiting higher development and loads in well-nourished hosts (Labaude et al., 2015; Seppälä & Leicht, 2015; Johnson et al., 2019).

Despite this growing body of evidence, it remains unclear how variation in dietary protein quality (e.g. low- vs. high-protein diets) influences parasite infections in aquatic host–parasite systems (Wilson et al., 2020; Holdbrook et al., 2025). Moreover, resource availability in aquatic ecosystems can vary spatially and temporally, often increasing host density and aggregation around resource-rich areas (Budischak et al., 2015; Becker et al., 2018; Civitello et al., 2018). Such aggregation can simultaneously increase exposure to parasites through contact with infected conspecifics (Albery et al., 2020; Scherer et al., 2020; Sweeny & Albery 2022), suggesting that host nutrition and host density may interact to shape infection risk.

Host density is frequently associated with increased parasite infections across host-parasite systems and is considered a major driver of parasite transmission (Arneberg et al.,

1998; Poulin et al., 2013; Buck et al., 2017; McCallum et al., 2017; Hasik & Siepielski, 2022). Higher density can increase contact rates between susceptible and infected hosts, promoting parasite transmission and higher parasite burdens, particularly in aquatic systems (McCallum et al., 2001; Lafferty et al., 2015; Buck et al., 2017; Daversa et al., 2017; Albery et al., 2020). However, higher host density can also reduce infection risk through encounter-dilution effects or the frequency-dependent transmission, leading to context-dependent outcomes (Lagrue & Poulin, 2015; Buck & Lutterschmidt, 2016; Wilber et al., 2022; Henriksen et al., 2023; Johnson et al., 2024). Such context dependence may arise from heterogeneity in host traits, including nutritional condition, which can modify transmission processes and infection outcomes (Hopkins et al., 2020; Albery et al., 2025).

Although host nutrition and host density are both known to influence resistance and tolerance to parasite infections, their interactive effects remain poorly understood, particularly in aquatic systems. Integrating within-host nutrition processes with between-host density effects is therefore essential for understanding and predicting parasite dynamics under fluctuating resource and population conditions (Johnson et al., 2015; Budischak & Cressler, 2018; Hayward et al., 2019; Walsman et al., 2023). Experimental approaches that explicitly manipulate both nutrition and density are required to decouple these effects their joint influence on host-parasite interactions (Pedersen & Fenton, 2015; Agostini et al., 2017; Johnson & Wilber, 2017; Sweeny et al., 2021; Walsman et al., 2023). Mesocosm experiments offer a powerful framework for addressing this challenge by allowing controlled manipulation of ecological conditions that cannot be achieved in observational studies (Wilber et al., 2017; Wunderlich et al., 2022; Arancibia et al., 2025).

Here, we conducted a mesocosm experiment using a fish-ectoparasite system to test the interactive effects of host nutrition and host density on susceptibility to parasite infection. We manipulated diet quality (high- vs. low-protein diets) and host density (low = 2 vs. high = 6)

over a 20-day period (Figure 1A, B). We imposed density levels to capture population context rather than to quantify density dependence sensu stricto, as our aim was to test how contrasts in local aggregation interacts with host condition to shape infection outcomes. We specifically tested whether host density modifies the effects of nutritional condition on infection, consistent with theory predicting context-dependent susceptibility arising from trade-offs between energetic state, immune function, and parasite transmission. We thus predicted that a high-protein diet would increase host tolerance and parasite burden, particularly at high-density where exposure is elevated (Hopkins et al., 2020; Sweeny & Albery 2022). In contrast, a low-protein diet at low host density was expected to result in lower parasite loads. Intermediate parasite burdens were predicted under mixed diet–density scenarios (Figure 1C).

2. Methods

2.1 Host-parasite model system

We used the Nile tilapia *Oreochromis niloticus* and its gill dactylogyridean monogenean species because these parasites have a direct life cycle (i.e. monoxenous) and release eggs into the aquatic environment (Buchmann & Bresciani 2006). After egg hatching, free-swimming larvae must find a suitable host within approximately 24 h, where they develop into adults and remain for the rest of their lifespan (Buchmann & Bresciani 2006).

Cichlid fish and their monogenean parasites are widely used to experimentally test how host traits influence parasite infestation and transmission dynamics (e.g. Wunderlich et al., 2022; Kmentová et al., 2022), because these host-parasite systems are highly tractable to elucidate processes in parasite ecology studies (Vanhove et al., 2016). These systems offer important insights into host-parasite dynamics because they allow high replication and experimental control at the level of individual hosts and most hosts are usually parasitized by

several closely or distantly related monogenean species (Timi & Poulin, 2020; Krasnov et al., 2021).

2.2 Fish collection and maintenance

Naturally infected Nile tilapias were collected from a commercial fish farm (net-cage system) and transported by truck under continuous liquid oxygen aeration to experimental aquaculture facilities at São Paulo State University (UNESP, Rio Claro, Brazil). All fish were of similar age.

Upon arrival, fish were randomly assigned into two groups and acclimated for two weeks in outdoor concrete tanks (10000 L, Figure 1A) under controlled conditions. During this period, fish were fed either a high-protein, animal-based diet or a low-protein, plant-based diet. Fish in the high-protein diet received commercial stick feed (23% protein; Tropical®), twice daily, while fish in the low-protein treatment were fed with lettuce daily (1.4% protein), both at approximately 5% of body mass. Diet composition and feeding regimes are detailed in the Table 1. Water temperature, dissolved oxygen, conductivity, pH, and ammonia concentration (Ammonia Test Kit for Fresh Water - colorimetric method) in all tanks were also monitored daily (Horiba, Multi Checker U-50 series) and maintained within satisfactory for organisms during the duration of the experiment (Figure 1A).

2.3 Diet treatments and parasite manipulation

Before the manipulation phase of the experiment, a subset of fish from each treatment was disinfected using 15-20 min salt baths to remove gill parasites following Wunderlich et al. (2022). These fish served as uninfected target hosts. The remaining fish were kept naturally infected with gill parasites and served as donor hosts.

This procedure resulted in three experimental host categories: uninfected target hosts fed with a low-protein diet (THL), uninfected target hosts fed with a high-protein diet (THH), and infected donor hosts fed with a high-protein diet (DHH; Figure 1A). All groups were kept in separated tanks until the start of the experiment.

2.4 Experimental design

Our experimental unit consisted of a 500 L circular polyethylene tank divided into three equal compartments separated by mesh partitions (three-patch arena; Figure 1B). One infected donor host (DHH) was placed in one compartment, while uninfected target hosts (THL or THH) were placed in the remaining compartments. Throughout the manuscript, we use the term "host density" to refer to experimentally imposed differences in local aggregation (crowding), which we interpret as a component of population context rather than a demographic process.

We manipulated host density at two levels (low density: two fish; high density: six fish) and crossed this factor orthogonally with diet treatment. We replicated the four treatments (T1-T4) eight times, resulting in 32 experimental arenas:

T1: DHH | THL and THH at low density;

T2: DHH | THL and THH at high density;

T3: DHH | THL at low density and THL at high density;

T4: DHH | THH at low density and THH at high density (Figure 1B).

The nutritional manipulation was designed to alter host energetic condition rather than induce large differences in body size over the experimental timeframe, allowing us to isolate condition-mediated effects on infection. Also, because our focus was on target host susceptibility, not donor infectivity, donor hosts were standardized (single diet type) to reduce variation in parasite supply. All tanks were filled with dechlorinated tap water (pH ~ 7.6),

aerated continuously, and maintained under controlled water temperature (~ 26 °C) and photoperiod (12 h light : 12 h dark). The experiment was carried out for 20 days, during which fish were monitored daily for signs of stress, disease, or abnormal behavioral.

2.5 Parasite quantification, host condition, and tolerance assessments

At the end of the experiment, fish were euthanized and gills were removed and examined under a compound microscope (Zeiss Primo Star, Germany), following Wunderlich et al. (2022). Parasites were counted and identified based on the morphology of sclerotized haptoral and copulatory structures (Pariselle & Euzet 2009).

Host body condition was assessed using the scaled mass index (SMI; Peig & Green 2009, 2010). SMI estimates individual body condition relative to structural size and is widely used in studies linking parasitism and host condition (Macedo-Veiga et al. 2016; Wunderlich et al. 2025). Here, SMI is interpreted as an integrated outcome of diet and infection, not a baseline covariate. The index was calculated as:

Eq. (1) $\mathbf{[}SMI = Mi \times (L0/Li)^{bSMA}\mathbf{]}$,

where Mi and Li are the body mass (g) and length (cm) of individual fish, L0 is the mean body length for each diet treatment, and bSMA is the slope of a standardized major axis regression of mass on length. The bSMA was estimated separately for each diet treatment using disinfected fish prior to the experiment, following recommendations by Lagrue & Poulin (2015) and Macedo-Veiga et al. (2016).

To measure host tolerance, we used SMI as a proxy of host fitness against parasite loads between host diet quality (i.e. low-protein vs. high-protein diets) and host density (i.e. low-density vs. high-density), where a positive slope indicated a higher tolerance (Råberg et al., 2009; Kutzer & Armitage, 2016; Kutzer et al., 2019).

2.6 Data analyses

We assessed the effects of host nutrition and density on parasite infection using generalized linear mixed models with a negative binomial error distribution, implemented in the *glmmTMB* package (Magnusson et al., 2020). Parasite abundance per target host and mean infection intensity per tank were used as response variables. Following standard parasitological terminology (Reiczigel et al., 2019), parasite abundance was defined as the number of parasites per individual host, whereas mean infection intensity was calculated as the mean number of parasites per infected host within each experimental tank. Host diet, density, and their interaction were included as fixed effects. Host body condition (SMI) was also analyzed as a response variable to assess treatment effects on host condition. To account for excess zeros, hurdle and zero-inflated negative binomial models were also evaluated in model selection (Chipeta et al., 2014; Harrison, 2014).

To control for potential non-independence and overdispersion, we included individual fish identity, nested within tank identity, as random effects (Harrison, 2014; Zuur & Ieno, 2016). Multicollinearity among predictors was examined using variance inflation factor (VIF), with values >5 indicating problematic collinearity (Zuur et al., 2013), in the *performance* package (Lüdecke et al., 2021). To perform post-hoc comparisons, we used estimated marginal means (EMMs), implemented in the *emmeans* package (Lenth & Piaskowski, 2025).

Model selection was based on Akaike's Information Criterion (AIC; Burnham & Anderson 2002), as implemented in *performance* package (Lüdecke et al., 2021). Conditional (R^2_c) and marginal (R^2_m) coefficients of determination were calculated following Nakagawa & Schielzeth (2013), using the glmmTMB package. We used the *DHARMa* package (Hartig, 2025) to assess model fit based on a simulation-based residual diagnostics approach. For the best-supported model, we checked variation of the individual predictors using semi-partial coefficients of determination (partR^2) (Stoffel et al., 2021). PartR^2 decompose the variance of

R^2 into components uniquely explained by individual predictors (Stoffel et al., 2021). All analyses were performed in R v. 4.4.1 (R Development Core Team, 2024), and figures were produced using *ggplot2* (Wickham, 2016).

3. Results

We found a positive effect of the high-protein diet on parasite abundance (Figure 2a) and mean infection intensity (Figure 2b) at both density levels, with the strongest effects observed under the combined high-protein and high-density treatment (Table 2). Target hosts receiving the high-protein diet treatment showed higher nutritional condition, as demonstrated by a scaled mass index (Figure 2c), and greater tolerance to infection compared with hosts fed the low-protein diet (Figure 3a) and those maintained at low density (Figure 3b).

Model selection based on Akaike's Information Criterion (AIC) supported the full model including host diet, host density, and their interaction as the best-supported model for parasite abundance and mean infection intensity (ΔAIC > 2 relative to reduced models; Table 2). This model indicated that both parasite abundance (R^2_c = 0.86; R^2_m = 0.65) and mean infection intensity (R^2_c = 0.84; R^2_m = 0.55) were positively related to high-protein diet, high host density and their interaction (Table 2). The best supported model for the scaled mass index indicated that it was influenced solely by host diet, with no detectable effect of density or the interaction term (R^2_c = 0.96; R^2_m = 0.95, Table 2). Post-hoc comparisons based on estimated marginal mean (EMMs) showed significant differences among treatments (Figure S1A,B), with the highest mean infection intensity observed under the high-protein, high-density treatment (mean = 5.14±0.143, FDR-adjusted, Figure S1B). Multicollinearity analyses indicated low correlation among predictors, with VIFs below five for models (Figure S2).

We further decomposed the relative importance of predictors in the best-supported GLMMs to evaluate the contribution of individual predictors to explained variation (Table 3;

Figure S3). Host diet and host density each explained a moderate proportion of the variation in parasite abundance in target hosts (aproximately 44% and 33%, respectively). Their combined effects, however, explained aproximately 64% of the fixed-effect variation (R^2_m = 0.64, CI_lower = 0.61, CI_upper = 0.75, nboot = 1000, Table 3; Figure S3a), and showed a variance for each predictor separately (Table 3; Figure S3b), the strongest structure coefficients (Table 3; Figure S3c), and the largest standardized regression coefficients (Table 3; Figure S3d). Beta weights show that the two predictors seem to have an effect (positive) on parasite abundance, because none of the confidence intervals overlaps zero (Table 3; Figure S3d).

4. Discussion

Our study demonstrates that the interaction between host nutrition and host density strongly influences infection outcomes by increasing parasite load while host nutritional condition promotes tolerance to infection. Although host density is often assumed to affect parasite transmission primarily through increased exposure (Anderson & May, 1978; McCallum et al., 2001; McCallum et al. 2017; Albery et al., 2020; Wilber et al. 2022; Albery et al., 2025), our experimental design isolated density-associated changes in infection outcomes that arise from host condition and interaction context, rather than exposure alone. Specifically, we found that a high-protein diet amplified the effects of high host density, resulting in higher parasite loads while simultaneously increasing host tolerance to infection in a freshwater fish host. While our study focuses on a single fish–ectoparasite system, the mechanisms we examine (condition-dependent susceptibility and density-mediated interaction context) are general features of host–parasite systems across taxa. Our findinds our consistent with a condition-dependent framework in which increased resource availability enhances host quality without necessarily reducing parasite load. Such responses differ from the prevailing

295 expectation for vertebrate hosts that improved nutrition strengthens immune resistance and
296 limits parasite proliferation (e.g. Pike et al., 2019).

297 While increased host nutrition has more commonly been associated with higher parasite
298 loads in invertebrate hosts (Cressler et al., 2014; Pike et al., 2019), our results show that similar
299 dynamics can arise in vertebrate hosts when additional resources promote tolerance rather than
300 resistance. In this context, increased energetic reserves may allow hosts to withstand the
301 physiological costs of infection without reducing parasite load, thereby maintaining
302 performance while simultaneously benefiting parasite fitness ("parasite tolerance mechanism";
303 Singh & Best 2021). This mechanism is particularly plausible for monogenean parasites, which
304 can evade immune responses even in hosts with good body condition (Buchmann &
305 Lindenstrøm, 2002; Bize et al., 2008; Kaur et al., 2018; Ilgová et al., 2021; Attwood, 2025).
306 Importantly, our results indicate that the highest parasite loads occurred under the combination
307 of high density and high nutritional availability, consistent with a tolerance-dominated
308 response when hosts have access to high resources. High density likely increases opportunities
309 for parasite establishment, while high nutritional status buffers hosts against infection costs,
310 shifting the balance away from resistance and toward tolerance. Comparable patterns have been
311 observed in aquatic mesocosms, where high nutrient conditions favored the evolution of
312 tolerance over resistance in zooplankton hosts (Walsman et al., 2023). Together, these findings
313 suggest that tolerance may be an energetically advantageous strategy under high-resource
314 conditions compared with costly immune-mediated resistance (Budischak et al., 2018;
315 Budischak & Cressler, 2018; Perrine et al., 2025).

316 Parasite establishment in our experiment is also likely shaped by host-recognition
317 mechanisms. Many aquatic parasites use chemosensory cues, such as glycoproteins, fatty acids,
318 carbohydrates, ammonia, or volatile organic compounds, to identify and select suitable hosts
319 (Buchmann & Lindenstrøm, 2002; Buchmann & Bresciani, 2006; Chaisson & Hallem, 2012).

Previous studies showed that parasites preferentially select better-nourished hosts (Sukhdeo & Sukhdeo, 2004; Christe et al., 2003; Chaisson & Hallem, 2012; Seppälä & Leicht, 2015; Johnson et al., 2019). Monogenean oncomiracidia can also use glycoprotein-based cues to detect host quality and susceptibility (Wunderlich et al., 2022; Attwood, 2025). Fish on high-protein diets excrete more ammonium as a by-product of protein metabolism (Hardison & Eliason, 2024; Zimmer, 2024), and this nitrogenous waste can serve as a chemical cue guiding parasites toward high-quality hosts (Haberl et al., 2000; Chaisson & Hallem, 2012). Thus, in our system, hosts fed a high-protein diet may have emitted stronger or more detectable cues, increasing their attractiveness to parasites (Seppälä & Leicht, 2015; Johnson et al., 2019). Collectively, these processes suggest that both tolerance and host-choice mechanisms could have contributed to the elevated parasite loads observed under high-protein, high-density conditions.

The emergence of tolerance under high-resource conditions is also consistent with expectations for specialist parasites. Theory predicts that specialist parasites can achieve high abundances and efficient transmission while imposing relatively low damage on their hosts, thereby favoring tolerance-mediated host responses (Armour et al., 2025; Park et al., 2025). Comparative analyses showed that specialist lineages often reach parasite burdens several times higher than generalist lineages (Armour et al., 2025), and global analyses of helminths reveal that specialists commonly achieve higher abundances on their primary hosts (Park et al., 2025). Monogeneans fit this profile as they often attain high loads in fish hosts, particularly when high-quality hosts are available to track and infect (Paredes-Trujillo et al., 2021; Paredes-Trujillo & Mendoza-Carranza, 2022; Wunderlich et al., 2022). These parallels suggest that the patterns we observed (i.e., high parasite loads coupled with high host tolerance) are consistent with predictions for specialist parasites in aquatic host–parasite systems.

These host-level processes have important implications for parasite transmission. Heterogeneity among hosts in quality and nutritional state can strongly influence transmission dynamics (McCallum et al., 2017), and variation in macronutrient availability can amplify differences in host susceptibility and parasite growth rates (Cotter et al., 2011; Vale et al., 2013; Wilson et al., 2020; Blubaugh et al., 2023). When parasites preferentially infect high-quality hosts and those hosts exhibit elevated tolerance, parasite transmission could increase even when host resistance does not. Such combined effects of parasite choice and host tolerance could enhance parasite persistence and alter outbreak dynamics in natural populations (Christe et al., 2003; Vale et al., 2013; Penczykowski et al., 2014; Sanchez-Thirion et al., 2019).

Our experiment captured short-term infection outcomes and did not incorporate longer-term demographic or eco-evolutionary feedbacks, which may amplify or dampen density- and condition-dependent dynamics in natural systems. Evidence for diet-mediated tolerance in vertebrates remains limited, and additional studies are needed to disentangle the relative effects of food quality, food quantity, and host condition in aquatic host–parasite systems (Becker et al., 2018; Sanchez-Thirion et al., 2019). Nonetheless, both empirical and theoretical work suggest that tolerance can be favored under high-resource conditions (Dean et al., 2024), and that bottom-up processes can strongly influence transmission pathways in host–parasite interactions (Holdbrook et al., 2025).

By revealing how nutritional condition and population density act together to influence infection trajectories, our study underscores that host–parasite dynamics are fundamentally context dependent. Extending this framework across host taxa and ecological settings will be important for predicting parasite responses in a changing world.

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Authors' contributions

A.C.W. and T.S. conceived the hypotheses and designed the experiment. A.C.W. ran the experiment and identified the material. A.C.W. and T.S. analysed the data and wrote the first draft of the manuscript. All authors agreed on the final version of the manuscript.

Data accessibility

Data and R code to reproduce manuscript analyses will be made available via the Dryad Digital Repository upon manuscript acceptance.

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620

Table 1. Variation in diet composition and feeding regime used in experimental parasite exposure.

Host diet	Composition (g)	Feeding regime per cage/treatment			
		Treatment codes	Density/cage	Cage (%, weight)	Frequency/day
Low-protein diet					
Protein (g)[a]	1.4	THL[c]	Low=2	5%/weight	1 time
Fat (g)[a]	0.2	THL[c]	High=6	5%/weight	1 time
Carbohydrates (g)[a]	1.7				
Kcal (g)[a]	0.2				
High-protein diet					
Protein (g)b	23.0	THH[c]	Low=2	5%/weight	2 times
Fat (g)b	3.0	THH[c]	High=6	5%/weight	2 times
Carbohydrates (g)[b]	57.5				
Kcal (g)[b]	3.5				

[a]Values determined for lettuce from the Brazilian Table of Composition Foods (TBCA), USP. Food Research Centre (FORC). Version 7.2. São Paulo, 2023 [www.fcf.usp.br/tbca].
[b]Values determined by Tropical-Pond stick Light (Floating type-pellet) [Tropical Tadeusz Ogrodnik – www.tropical.pl].
[c]THL=Uninfected target host with low-protein diet; THH=Uninfected target host with high-protein diet.

Table 2. Outputs from generalized linear mixed models assessing how host diet (i.e. low-protein and high-protein) and host density (i.e. low-density and high-density) affect the parasite abundance per individual target host, the mean intensity of infection (MII) per tank, and the scaled mass index (SMI) in individual target host after a parasite exposure experiment in mesocosm conducted for 20 days. Parameter estimates, standard errors (SE), Wald z-values, *p*-values, 95% confidence intervals (CI), and total marginal (R^2_m), conditional (R^2_c) are provided for each predictor and model. Bold values indicate significant variables. The confidence intervals (CI) excluding zero are also indicated in bold.

Parameters	Parameter estimate	SE	z-value	*p*	95% CI	
					Lower	Upper
Parasite abundance model						
(Intercept)	2.17	0.18	11.81	**<0.001**	**1.81**	**2.53**
High-protein diet	1.22	0.17	7.13	**<0.001**	**0.89**	**1.56**
High-density	1.32	0.16	8.40	**<0.001**	**1.01**	**1.63**
High-protein diet:High-density	0.42	0.17	2.38	**0.017**	**0.07**	**0.76**
R^2_c	0.86					
R^2_m	0.65					
Mean infection intensity model						
(Intercept)	2.18	0.14	15.72	**<0.001**	**1.91**	**2.46**
High-protein diet	1.08	0.06	17.94	**<0.001**	**0.96**	**1.20**
High-density	1.44	0.07	20.98	**<0.001**	**1.31**	**1.58**
High-protein diet:High-density	0.49	0.06	7.62	**<0.001**	**0.37**	**0.62**
R^2_c	0.84					
R^2_m	0.55					
Scaled mass index model						
(Intercept)	4.49	0.02	248.15	**<0.001**	**4.45**	**4.52**
High-protein diet	0.76	0.03	33.54	**<0.001**	**0.71**	**0.80**
High-density	-0.03	0.02	-1.49	0.136	-0.07	0.009
High-protein diet:High-density	-0.02	0.02	-095	0.344	-0.08	0.03
R^2_c	0.96					
R^2_m	0.95					

Table 3. Results from part (semi-partial) R^2 (i.e. estimate the explained variance uniquely by each predictors and combinations of predictors), inclusive R^2 (i.e. estimate the variance explained by a predictor independent of all other predictors), structure coefficients r (i.e. correlation between a predictor and the fitted response, ranging from -1 to 1) and beta weights (i.e. standardized slopes) for the best GLMM model (i.e. parasite abundance). Statistical significance was assumed when 95% of confidence intervals (CI) did not overlap with zero. CI for all estimates were used parametric bootstrapping (nboot = 1000).

Parameters	Estimate	95% CI	
		Lower	Upper
Part (semi-partial) R^2			
R^2_m (full model)	0.64	0.61	0.75
High-protein diet	0.44	0.41	0.53
High-density	0.33	0.29	0.40
High-protein diet:High-density	0.64	0.61	0.75
Inclusive R^2			
High-protein diet	0.36	0.32	0.44
High-density	0.25	0.19	0.30
High-protein diet:High-density	0.53	0.48	0.63
Structure coefficients r			
High-protein diet	0.75	0.70	0.81
High-density	0.62	0.55	0.67
High-protein diet:High-density	0.91	0.88	0.95
Beta weights			
High-protein diet	1.26	0.89	1.51
High-density	1.32	0.96	1.52
High-protein diet:High-density	0.41	0.17	0.88

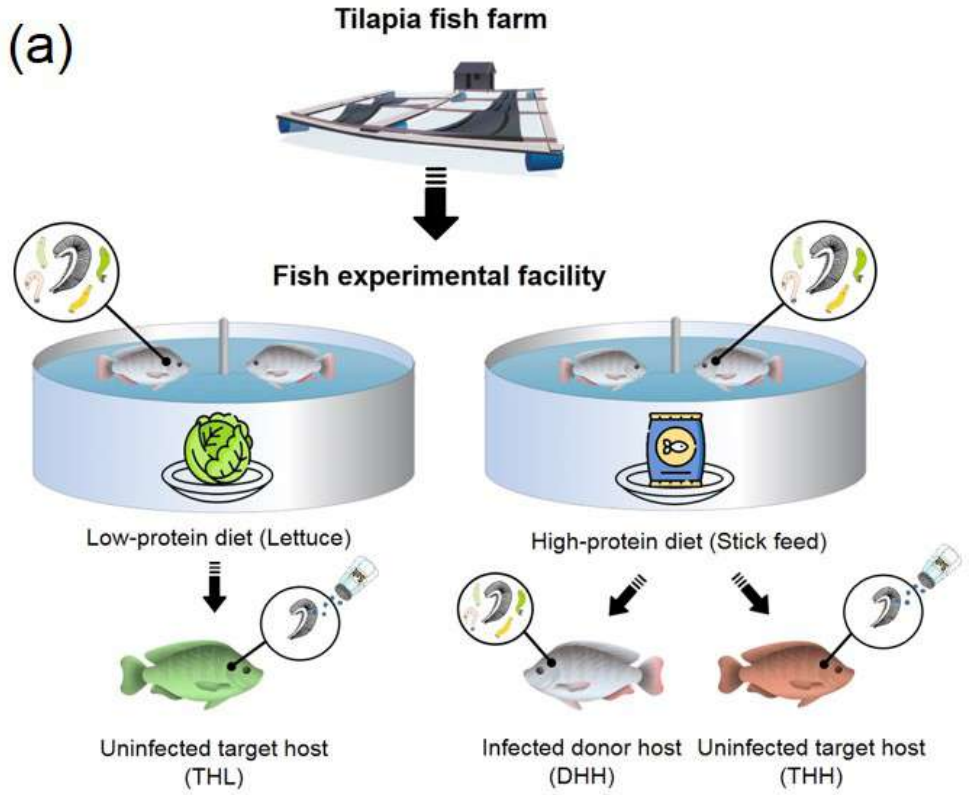
Figure legends

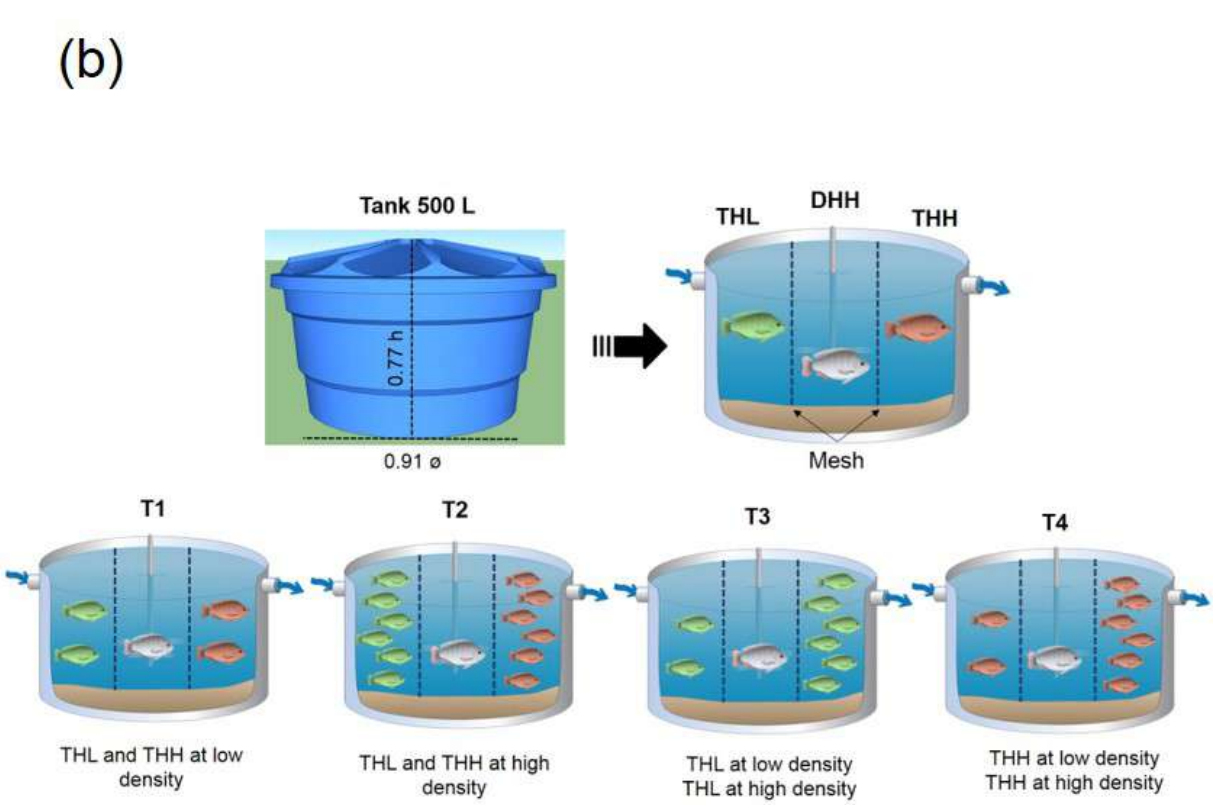
Figure 1. a) Schematic overview of the steps from the fish farm to the tanks of the experimental fish facility where the fish were separated into two groups (lettuce = low-protein diet and stick feed = high-protein diet) to the acclimation to the experiment. Before the experiment, the fish of each diet treatment/tank (THL = target non-infect hosts; THH = target non-infect hosts) received a salt bath to remove the ectoparasites, while a part of the fish feeding with stick feeding had to be kept with the parasites (DHH = donor infected hosts is represented by gray); b) Illustration of the 500L experimental unit (i.e. tank with three patch arenas) separated by mesh in three isolated compartments of the same volume. The four treatments (T1-T4) are demonstrated by a combination of host nutrition and host density (THL = target host with low-protein diet is represented by green and THH = target host with high-protein diet is represented by orange); c) Predictions show that high-protein-diet increases the host tolerance to parasites, while low-protein diet reduces the host tolerance to parasites. An intermediate tolerance is expected between the treatments 3 and 4.

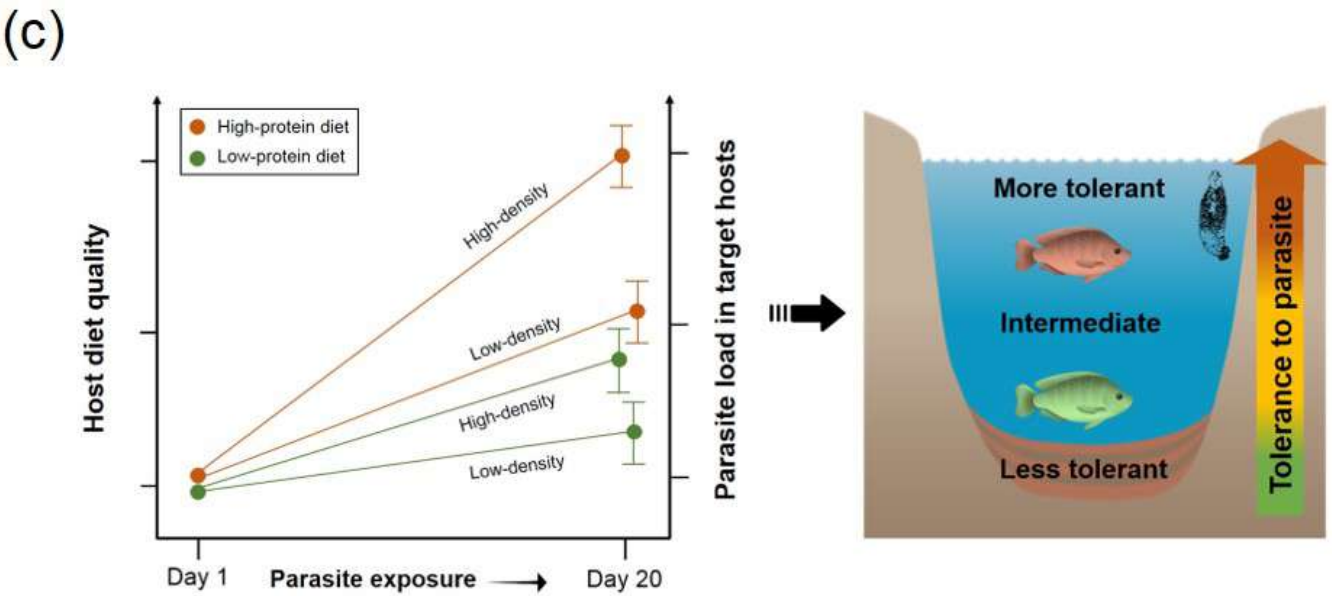
Figure 2. a) Variation of parasite abundance in individual target hosts among the treatments (nutrition and density); b) Variation of mean infection intensity per tank in target hosts among the treatments (nutrition and density); c) Variation of scaled mass index (SMI) among the treatments (nutrition and density). Each box corresponds to the 25^{th} and 75^{th} percentiles; the dark line within each box represents the median; error bars show the minimum and maximum values.

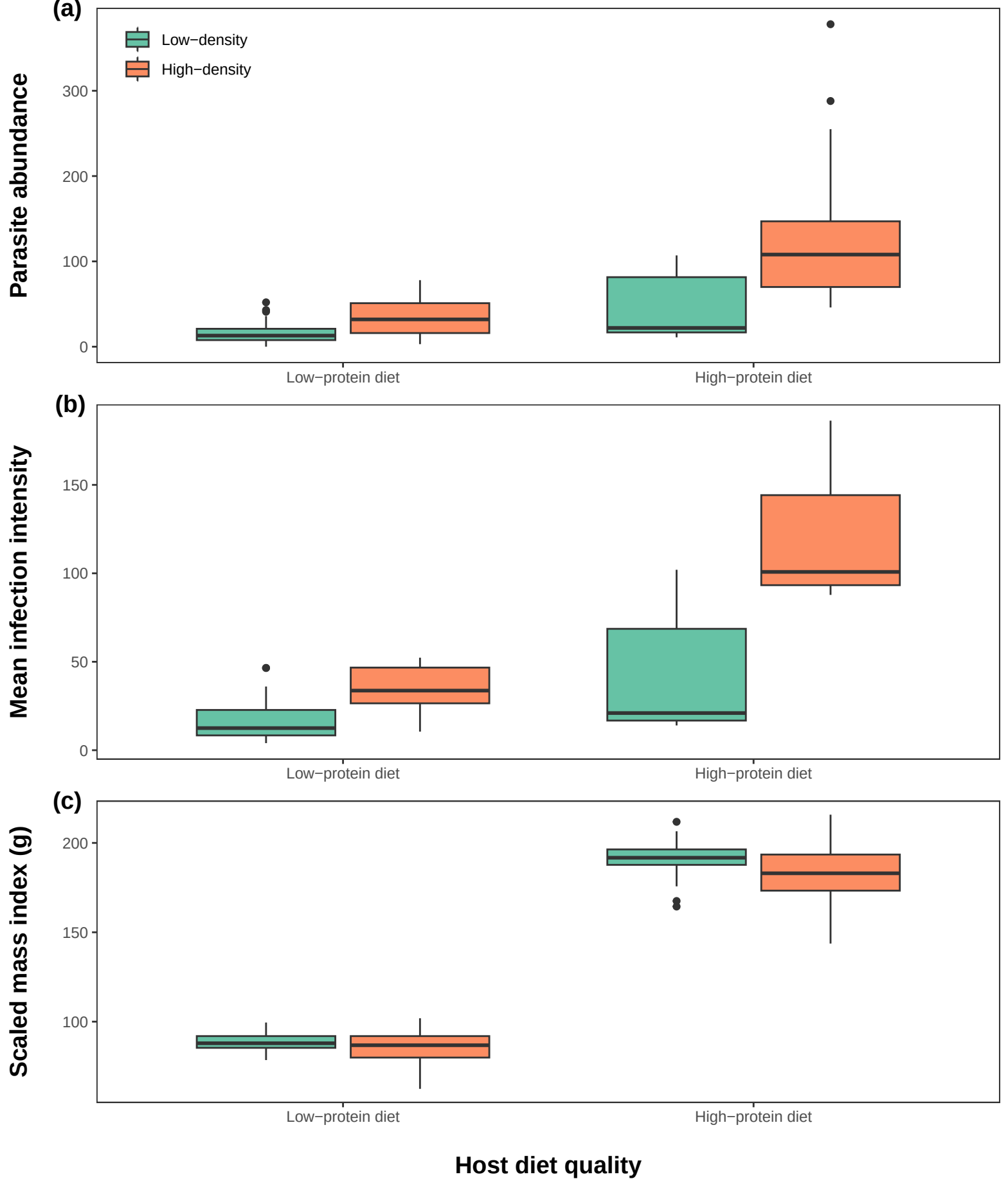
Figure 3. Variation of tolerance expressed as slope of host condition (SMI = scaled mass index, g) against the parasite abundance for host diet (A) and host density (B) treatments. The tick

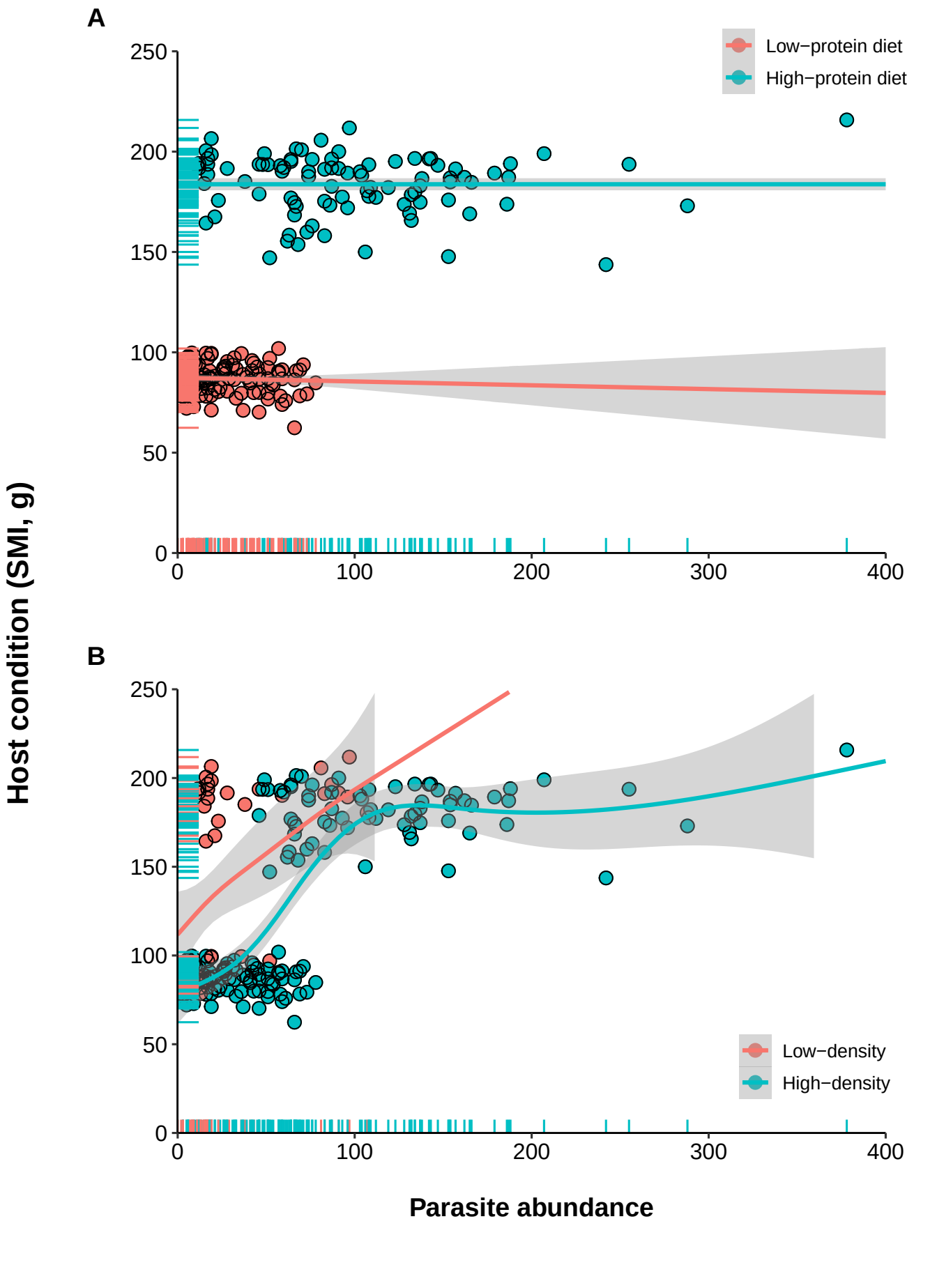
marks along the axes represent individual data points. The incorporated lines show GAM as smoothing function, and the grey shade represents the 95% point-wise confidence limits of the GAM estimate.


(a)
Tilapia fish farm
Fish experimental facility
Low-protein diet (Lettuce)
High-protein diet (Stick feed)
Uninfected target host (THL)
Infected donor host (DHH)
Uninfected target host (THH)


(b)
Tank 500 L
0.77 h
0.91 ø
THL
DHH
THH
Mesh
T1
T2
T3
T4
THL and THH at low density
THL and THH at high density
THL at low density
THH at high density
THH at low density


(c)
High-protein diet
Low-protein diet
High-density
Low-density
High-density
Low-density
Host diet quality
Parasite load in target hosts
Day 1
Parasite exposure
Day 20
More tolerant
Intermediate
Less tolerant
Tolerance to parasite


(a)
Low−density
High−density
Parasite abundance
300
200
100
0
Low−protein diet
High−protein diet
(b)
Mean infection intensity
150
100
50
0
Low−protein diet
High−protein diet
(c)
Scaled mass index (g)
200
150
100
Low−protein diet
High−protein diet
Host diet quality


A
B
Low−protein diet
High−protein diet
Low−density
High−density
Host condition (SMI, g)
Parasite abundance
0
50
100
150
200
250
0
100
200
300
400

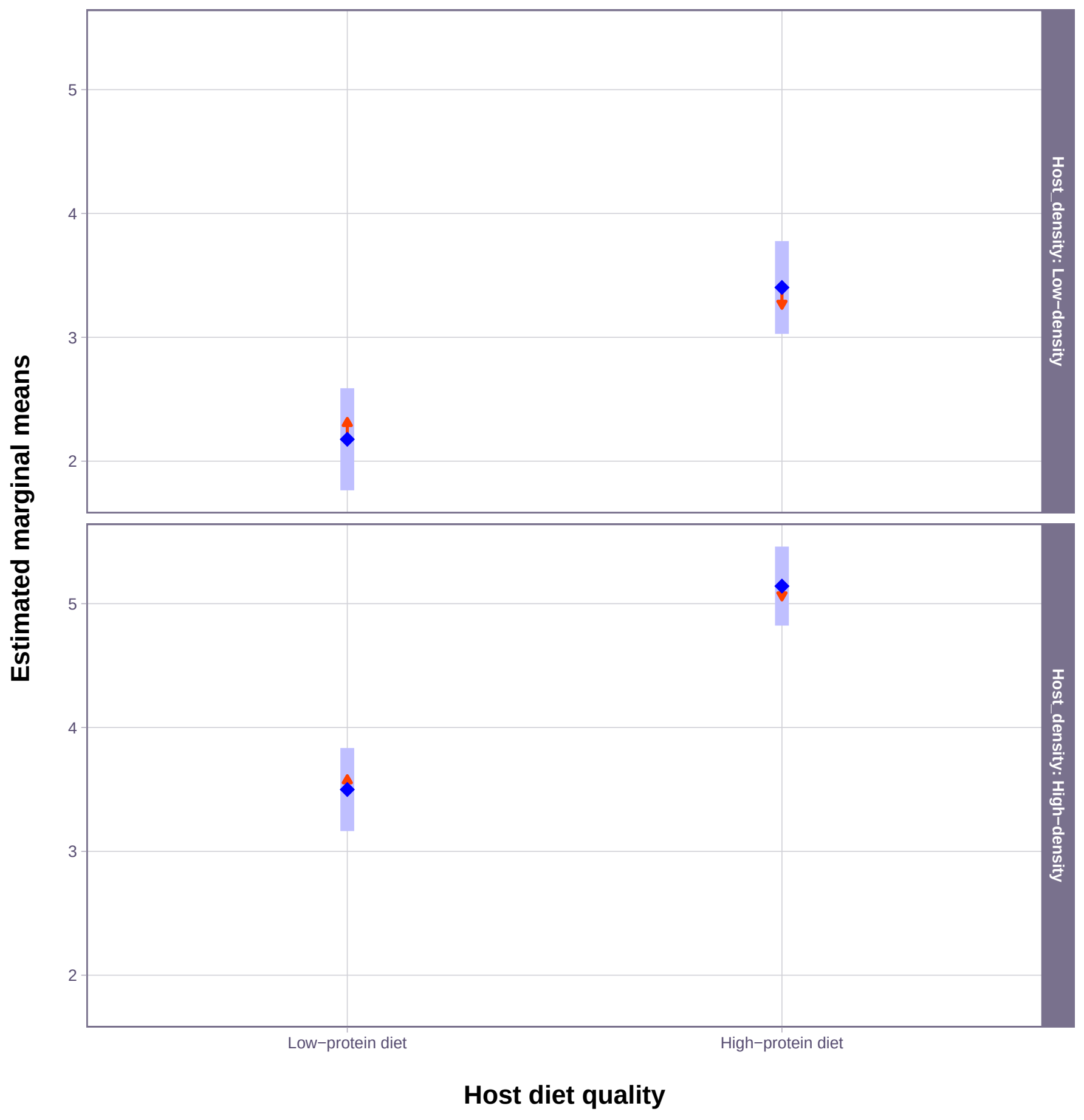
Supporting information

Figure legends

Figure S1. Estimated marginal mean values (EMMs) to visualize the post-hoc test between the treatments of the best GLMM model. The blue bars are confidence intervals for EMMs, and the red arrows are for the comparisons between them. Arrow of a mean that overlaps an arrow of another group, the difference is not significant, based on the adjustment setting (FDR-adjusted).

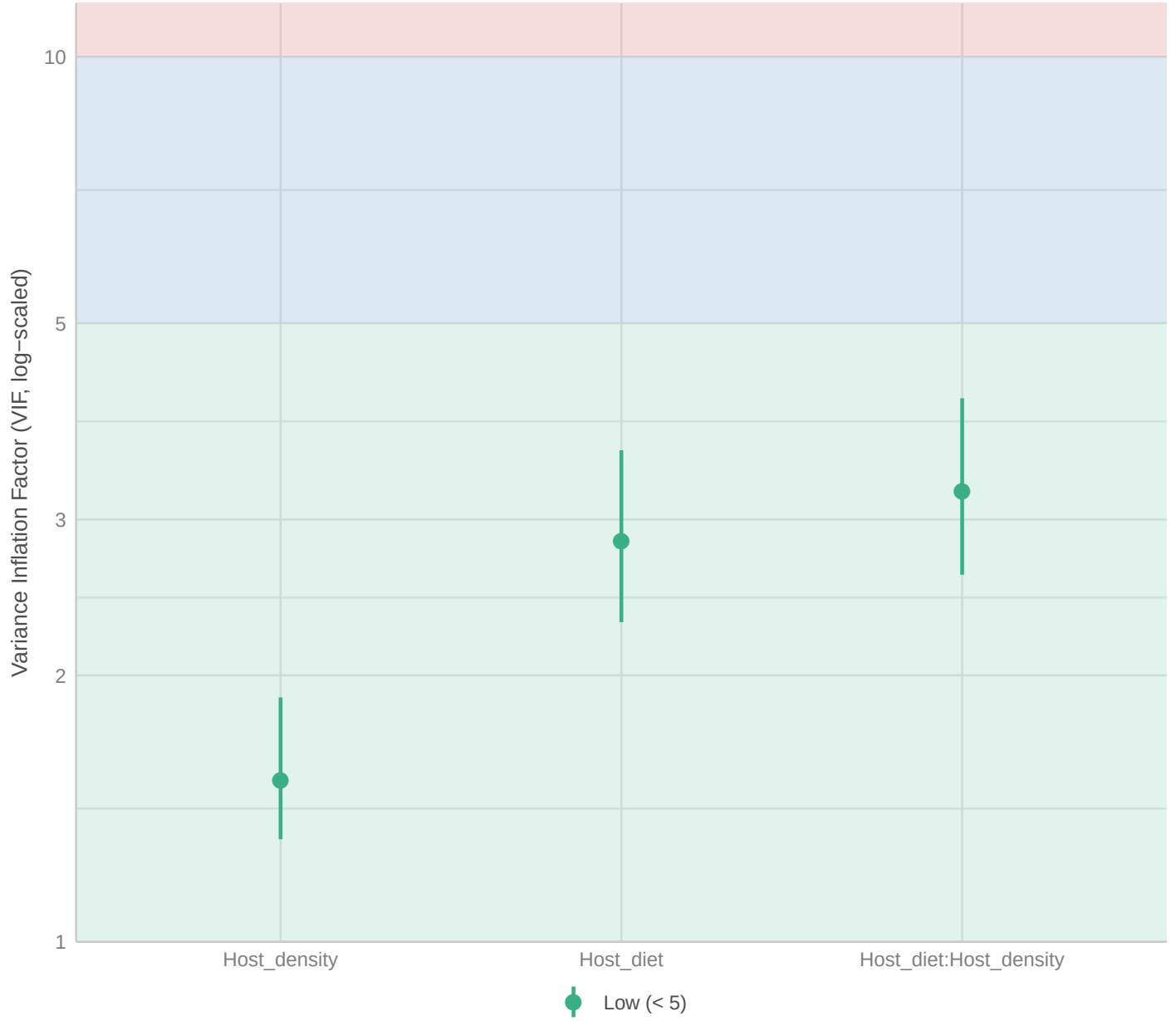
Figure S2. Variation inflation factor (VIF, log-scaled) for individual predictors and interactions. VIFs below five represent low multicollinearity among predictors.

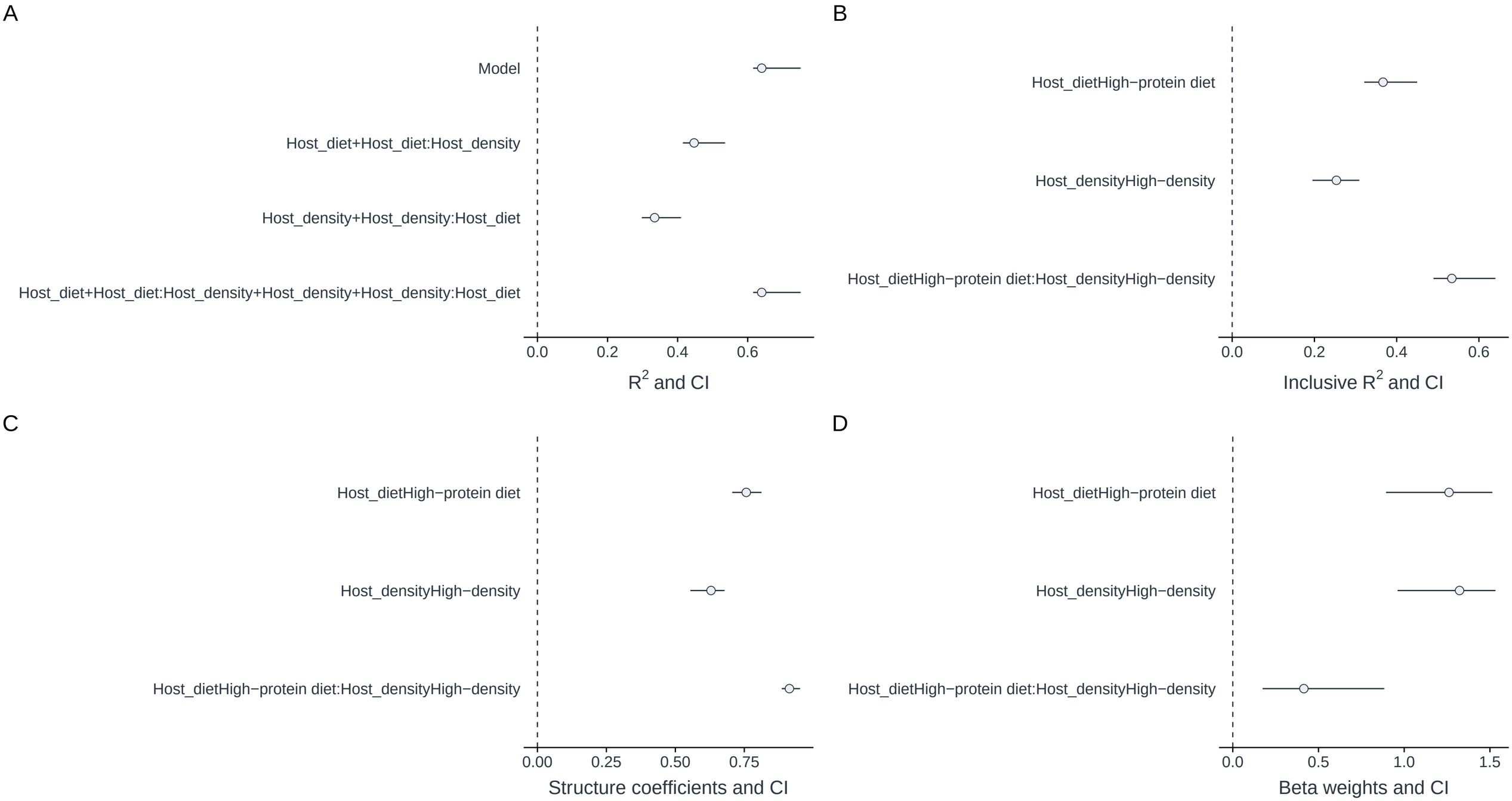
Figure S3. Comparison of part R^2 for individual predictors (A), inclusive R^2 (B), structure coefficients (C) and beta weights (D) for the best GLMM model. Statistical significance was assumed when 95% of confidence intervals did not overlap with zero.


Estimated marginal means
5
4
3
2
Host_density: Low−density
Host_density: High−density
Low−protein diet
High−protein diet
Host diet quality

Collinearity

High collinearity (VIF) may inflate parameter uncertainty




A
Model
Host_diet+Host_diet:Host_density
Host_density+Host_density:Host_diet
Host_diet+Host_diet:Host_density+Host_density+Host_density:Host_diet
0.0
0.2
0.4
0.6
R2 and CI
B
Host_dietHigh−protein diet
Host_densityHigh−density
Host_dietHigh−protein diet:Host_densityHigh−density
0.0
0.2
0.4
0.6
Inclusive R2 and CI
C
Host_dietHigh−protein diet
Host_densityHigh−density
Host_dietHigh−protein diet:Host_densityHigh−density
0.00
0.25
0.50
0.75
Structure coefficients and CI
D
Host_dietHigh−protein diet
Host_densityHigh−density
Host_dietHigh−protein diet:Host_densityHigh−density
0.0
0.5
1.0
1.5
Beta weights and CI