OIKOS

Research article

Parasite-mediated changes in host traits alter food web dynamics

Ines Klemme, Tommi Perälä, Sami O. Lehtinen and Anna Kuparinen

Department of Biological and Environmental Science, University of Jyvaskyla, Jyvaskyla, Finland

Correspondence: Ines Klemme (ines.klemme@jyu.fi)

Oikos

2024: e10374

doi: 10.1111/oik.10374

Subject Editor: Ellen Decaestecker
Editor-in-Chief: Pedro Peres-Neto
Accepted 26 December 2023

Parasites commonly alter the phenotype of their hosts, thereby influencing competitive and consumer–resource interactions. This could trigger a cascade effect on the dynamics of biological communities, but the role of parasites in ecosystem processes is poorly understood. In this study, we investigate how parasite-induced trait modifications shape the dynamics of a complex lake food web using an allometric trophic network model (ATN). We simulated infections of stage-structured fish host populations via increased maintenance costs and predation risk. Our results show that host trait modifications can significantly impact host demography, with stage-specific biomass declines up to 60%. However, less severely affected host stages buffered these effects and sustained the population. Importantly, host biomass decline altered the dynamics of species interactions and these effects cascaded through the entire community, with biomass changes observed at all trophic levels. Our findings emphasize the importance of incorporating both indirect parasite effects and host life history in ecological network studies for more realistic simulations of community dynamics.

Key words: allometric trophic network (ATN), food web, indirect effect, parasitism, population stage structure, trait-mediated indirect interaction

Introduction

Parasites are often neglected in ecological network research, because they are small, have a cryptic lifestyle, and are difficult to study (Marcogliese and Cone 1997, Lafferty et al. 2008, Preston et al. 2016). Increasing evidence, however, underscores the influential roles that parasites can assume in shaping ecosystem structure and function (Lafferty et al. 2006, 2008, Amundsen et al. 2009). For example, parasites can indirectly alter consumer–resource and competitive interactions by modifying host phenotype (Preston et al. 2016, Buck 2019). Such interaction modifications can exceed the impact of direct trophic interactions (Wootton 1994, Preisser et al. 2005), but are a common gap in food web studies (Bolker et al. 2003, Kéfi et al. 2012, Ohgushi et al. 2012, Terry et al. 2017).

Parasite-induced changes in host phenotype can arise before and during infection. Under infection risk, susceptible hosts often adopt avoidance strategies similar to anti-predator adaptations in prey (Brown et al. 1999, Moore 2002). Unlike predators,

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parasites can also modify their victim's phenotype during consumption, because infections are rarely lethal (Buck and Ripple 2017, Buck 2019). For example, parasites elicit immune responses and tissue repair, diverting host energy away from production. This can cause physiological changes related to growth, development, and reproduction (Sheldon and Verhulst 1996, Lochmiller and Deerenberg 2000). Further, host vulnerability to predation often increases during infection either via behavioural manipulations that facilitate parasite transmission (Moore 2002, Lefèvre et al. 2009, Poulin 2010) or via side effects that make the host weak or more conspicuous (Moore 2002, Poulin 2010).

Empirical studies have shown that parasite-induced trait modifications can have large-scale community effects. For example, trematode infections alter grazing rates of herbivorous aquatic snails, which in turn changes algal community composition (Wood et al. 2007, Bernot and Lamberti 2008). Further, horsehair worms manipulate their cricket hosts to enter freshwater habitats, where they provide an important prey subsidy for predatory fish (Sato et al. 2011). This decreases predation pressure on benthic aquatic invertebrates and consequently reduces benthic algae biomass and increases leaf litter decomposition (Sato et al. 2012). In theoretical community studies, earlier attempts to incorporate interaction modifications by parasites have been limited to simple three- or four-species trophic networks. These studies have shown that changes in predator attack rate or in prey susceptibility to predation can destabilise the dynamics of predator–prey communities (Fenton and Rands 2006) and alter coexistence outcomes among competing species (Hatcher et al. 2014, Prosnier et al. 2018). However, to improve our understanding of the community-wide consequences of such parasite effects requires incorporating them into more complex and realistic food-web models.

Here, we used an allometric trophic network (ATN) model to simulate how infection-induced changes in host phenotype affect the dynamics of a complex food web in a well-studied lake ecosystem (Boit et al. 2012, Kuparinen et al. 2016). The model comprises 30 functional guilds of bacteria, phytoplankton, zooplankton, and fish, the latter of which are subdivided into five developmental stages to more realistically capture the diversity and complexity of the system (Kuparinen et al. 2016). We did not add parasites as a new functional guild, but simulated infections in two fish host species with different feeding ecology via trait modifications. Specifically, we assumed that infections increase host maintenance costs and predation risk and incorporated these changes in the maintenance cost function of the host and the functional response of the host's predators. Both trait changes were functions of infection intensity (the number of parasites in the host), which was parametrized using empirical infection data of a common macroparasite. The data showed the typical pattern of increasing infection prevalence and intensity with host age (Hudson and Dobson 1995), which translates into increasing trait changes with age. We examined the effects of parasite-induced trait modifications on the dynamics of all 30 functional guilds to determine their potential 1) to alter host demography, 2) to induce interaction modifications and consequently 3) trophic cascades.

Material and methods

ATN food web model

Our model system is Lake Constance (47°N, 9°E), one of the largest lakes in central Europe, for which the ATN model has been parameterized and validated (Boit et al. 2012, Kuparinen et al. 2016). This complex food web contains 133 feeding links among six producer guilds (mainly algae), 24 consumer guilds (bacterivores, herbivores, omnivores, and carnivores) and detritus (Fig. 1, Supporting information). The two fish species in the model, the piscivorous Eurasian perch *Perca fluviatilis* and the planktivorous European whitefish *Coregonus lavaretus*, are two of the commercially most important fish species in Lake Constance. We divided the fish into five life-history stages, including larvae and juveniles (aged zero and one year), and adults (aged two, three and ≥ four years) (Kuparinen et al. 2016). While larvae and juveniles are subject to predation by adult perch, adult perch and whitefish have no predators in this model. In the wild, predation on adults does occur, but perch and whitefish adhere to the common pattern for teleost fish, with juveniles being significantly more vulnerable to predation (Sogard 1997).

The biomass (carbon density) dynamics are modelled using a system of ordinary differential equations (Supporting information). During a 90-day annual growth season, producer dynamics are described by their intrinsic growth and loss to herbivores. Consumer dynamics are described by gains from feeding, losses to predators, and maintenance metabolism. At the end of the growth season, fish biomass moves from one age class to the next, and adult fish reproduce by transforming a fraction of gained biomass into larval biomass for the next growth season. To prevent an unnatural accumulation of biomass in the guild comprising fish aged four years or older, a background mortality rate of 0.5/year is added. We expanded this previously established ATN model by incorporating infection of fish hosts via modifications of host parameters.

Parasitic infection

We model macroparasitic infections, such as those caused by nematodes, trematodes, and cestodes, because several macroparasitic characteristics suggest a high potential for host-trait modifications. First, infections are typically chronic, as macroparasites are long-lived and host defence against them is often incomplete (Anderson and May 1992). Additionally, many macroparasites have complex life cycles with trophic transmission between hosts (Anderson and May 1992), which often leads to host manipulation aiming at increased predation risk to facilitate transmission. This manipulation can also increase the predation risk to predators that are not part of the parasite's life cycle (Seppälä and Jokela 2008).

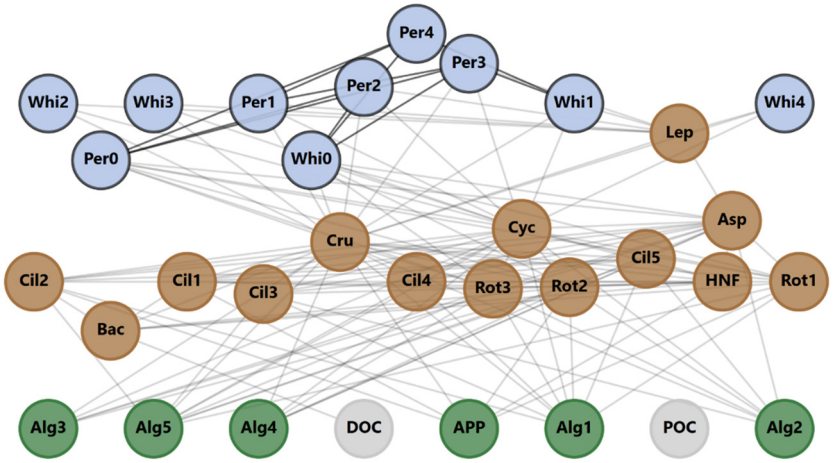

Figure 1. Structure of the simulated Lake Constance food web. Nodes describe functional guilds (labels correspond to those in the Supporting information) with connecting feeding interactions. Producers, consumers and fishes (perch and whitefish, each consisting of five age classes) are shown in green, brown and blue, respectively, and particulate and dissolved organic carbon (POC and DOC) in grey. The vertical position is determined by the guild's prey-averaged trophic position. Parasites were not included as functional guild, but infection was modelled via modifications of host traits. Specifically, guilds with a black border are susceptible to infection and infection increases the energetic maintenance costs of the host. Infection also increased predation susceptibility and black feeding interactions represent those altered by infection.

Other common traits of macroparasites are important to consider for the study. Unlike microparasites, macroparasites typically do not directly replicate within their host, and thus infection intensity only increases with new infection events. Due to their chronic nature, macroparasite infections therefore often accumulate with age (Hudson and Dobson 1995), leading to an increase in the magnitude of infection-induced host changes (Dobson 1988).

We parametrized our model with empirically collected age-specific infection prevalence and intensity data (Supporting information) of the tapeworm *Triaenophorus nodulosus* infecting Eurasian perch in Lake Constance (Molzen 2005). The observed range of infection prevalence and intensity is typical for *T. nodulosus* infecting perch in Lake Constance (Dieterich and Eckmann 2000, Brinker and Hamers 2007) and other systems (Brinker and Hamers 2007, Morley and Lewis 2017, Borvinskaya et al. 2019). Tapeworms commonly affect the physiology and behaviour of their intermediate fish hosts (Scholz et al. 2021), making this an ideal system to study infection-induced changes in host traits. *Triaenophorus nodulosus* has a complex life cycle with trophic transmission, using copepods as the first intermediate host, perch and other freshwater fish as the second intermediate host, and pike *Esox lucius* as the final host (Scholz et al. 2021). Note, however, that our purpose was not to model the entire parasite life cycle, but to isolate the impact of parasite-mediated host modifications. Thus, we focused on the second intermediate host, i.e. perch and whitefish. Whitefish has not been described as host for *T. nodulus*, but is commonly infected with a closely related tapeworm *T. crassus*, for which similar age-specific infection prevalence and intensity have been documented (Amundsen and Kristoffersen 1990). Simulating infection for both fish species allowed us to include hosts with a different feeding ecology.

Expanding the ATN model with parasite effects

We simulate parasite effects on host maintenance cost or vulnerability to predation, or both. First, infection increases the host's maintenance respiration coefficient $f_{m,i}$, i.e. the rate at which carbon is respired by maintenance of basic bodily functions. This additional expended energy or biomass loss can be considered as reduced per capita growth or reproduction, but also as increased mortality due to infection. We assume that the relative increase in the maintenance cost for a fish guild i is directly proportional to the average number of parasites infecting the individuals of that age class, μ_i, as well as to the proportion of individuals infected, ϕ_i. We also assume that the maximum relative increase in the maintenance cost of the infected biomass in fish guild i is β_{max} and it is realized in guilds for which the average infection intensity exceeds the number of parasites assumed to cause the maximal effect, i.e. $\mu_i \geq \mu_{max}$. Mathematically, the maintenance respiration coefficient for the fish guilds is expressed as:

$$f_{m,i} = \delta_{m,i} f_m^0 \quad (1)$$

where:

$$\delta_{m,i} = 1 + \beta_{max} \min\left(1, \frac{\mu_i}{\mu_{max}}\right) \times \phi_i \quad (2)$$

is the scaling factor and $f_m^0 = 0.1$ is the baseline maintenance respiration coefficient representing the uninfected state. For a complete description of the maintenance loss term in the ATN model, see the Supporting information. Previous studies on fish reported a 25–35% increase in basal metabolic rate in response to infections with various macroparasites

(Östlund-Nilsson et al. 2005, Voutilainen et al. 2008, Grutter et al. 2011, Binning et al. 2013, Hvas and Bui 2022). Here, the maximum increase, β_{max}, was either 10, 25, or 50%. The average infection intensity at which this maximum increase was reached was chosen to be $\mu_{max} = 5.5$ in all three scenarios, which was close to the maximum average infection intensity in the data. The realized scaling factors for the maintenance respiration coefficients for each fish guild are shown in the Supporting information.

Second, infection increases the host's predation risk by impairing its predator avoidance or ability to escape. Infected hosts are often more susceptible to predation, for example due to weakness, morphological impairments, reduced predator recognition skills, loss of fear, increased hunger, or decreased competitive ability (Moore 2002, Poulin 2010). Empirical studies have shown that infected hosts are typically 4–15 times, but up to 30 times more likely to be eaten than their uninfected conspecifics (Lafferty et al. 2008, Cézilly et al. 2010). To simulate increased predation risk, we modified the predator's functional response (Supporting information) in two ways: either via decreasing the predator interference term to simulate a reduced refuge searching rate of the host, or via increasing the prey biomass dependent terms to simulate increased capture rates of the host.

To link predator avoidance with the predator interference term of the functional response, we adopted the interpretation of Geritz and Gyllenberg (2012), who showed mechanistically how refuge seeking behaviour leads to predator dependence in the denominator of the DeAngelis–Beddington functional response (in ATN models often called 'intraspecific predator interference'). We applied it to our hybrid DeAngelis–Beddington–Holling type III functional response (Supporting information) and assumed that it is proportional to the prey refuge searching rate. Thus, changes in the prey refuge searching rate can be implemented by scaling the predator interference parameter d_{ij}^0 by the same scaling factor that is used to scale the refuge searching rate. We adopted a similar idea of a saturating and linearly increasing parasite effect as for the increased maintenance cost, and mathematically the increased predation risk is modelled as:

$$d_{ij} = \delta_{d,j} d_{ij}^0 \tag{3}$$

where:

$$\delta_{d,j} = 1 - \gamma_{max} \min\left(1, \frac{\mu_j}{\mu_{max}}\right) \phi_j \tag{4}$$

is the scaling factor, γ_{max} is the factor by which the refuge searching rate is reduced at the maximum effect, which we chose either as 50, 90 or 99%, and d_{ij}^0 is the baseline predator interference representing the uninfected state. The realized scaling factors for the intraspecific predator interference parameter are shown in the Supporting information.

To simulate a lowered ability to escape from predators, we modified the prey biomass dependent terms in the functional response. We divided each host age class based on infection intensity and multiplied the prey term in the functional response by a coefficient Λ_j. This coefficient accounts for the overall increase in the predation risk of guild j by its predators, given the scaling factors associated with infection intensity and the infection distribution in that guild. We assume that host capture rate increases with infection intensity, modelled by a linearly increasing and saturating scaling factor $\lambda(\mu)$. Mathematically, the increased predation risk is modelled as:

$$\lambda(\mu) = 1 + \lambda_{max} \min\left(1, \frac{\mu}{\mu_{max}}\right) \tag{5}$$

where 1 is the baseline scaling corresponding to uninfected prey and λ_{max} occurs when the individual host is infected by μ_{max} parasites. In other words, the scaling factor towards infected hosts is capped at $1 + \lambda_{max}$ even when they are infected by more than μ_{max} parasites. The maximum increase in capture rate was either 100, 250 or 500%.

To obtain the overall increase in predation risk of resource guild j by its predators, we sum over all possible $\lambda(\mu)$ values, associated with μ parasites in the host body, multiplied by the corresponding infection prevalence. Thus, we arrive at:

$$\Lambda_j = \sum_{\mu=0}^{\mu_j^{max}} \lambda(\mu) \varphi_j(\mu) \tag{6}$$

where μ_j^{max} is the highest feasible parasite intensity of guild j and $\varphi_j(\mu)$ describes the proportion of guild j individuals infected by μ parasites. Due to the lack of empirical data about the distribution of infection intensities in the population, a hypothetical distribution was derived from the empirically obtained mean and standard deviation estimates. The distributions for the parasite infections for each fish guild susceptible to predation are shown in the Supporting information.

After incorporating these changes to the ATN model, we simulated a 100-year period with and without parasitism. This was sufficient time for all model scenarios to reach an equilibrium state, where the biomass of all guilds ceased fluctuating, which was after 16 years in the absence of parasitism and after 14–22 years with parasitism. The simulations were conducted with either none, one or both fish species susceptible to infection. In all fish species scenarios with parasitism, infection resulted in increased host maintenance costs or increased predation susceptibility, or both. We compared biomasses in the final year and at the end of the growth season (before fish reproduction) between the scenarios with and without parasitism.

Results

Increased host maintenance costs

Infection-induced increase in the energetic maintenance costs of fish hosts resulted in a declined host biomass, particularly in the oldest age classes that had the highest infection prevalence and intensity. As the maintenance costs rose from a maximum of 10–50%, host biomass decreased further. This resulted in modifications of species interactions involving the hosts, with cascading effects that varied based on the host species susceptible to infection.

When piscivorous perch were susceptible to infection, the biomass of the top predators – adult perch aged two, three or ≥ four years – decreased by 3–38% (Fig. 2, left). Because adult perch are cannibalistic, this led to a reduction in predation pressure on larval and juvenile perch, i.e. aged zero and one year. However, the biomass of young perch showed only minor changes, because the relaxed consumption pressure was offset by the parasite effects and a reduced reproduction of adult perch (per capita biomass allocated to reproduction was on average reduced by 2–8%). Adult perch also feed on larval and juvenile whitefish. In addition, the resource profile of perch aged zero to three years also includes the main resource of whitefish, i.e. crustaceans ('Cru', 'Lep', and Cyc' in Fig. 2). Thus, the overall decline in perch biomass reduced both predation and competition pressure for whitefish and consequently increased their biomass. The biomass of crustaceans remained stable, however, because the decreased consumption by perch and increased consumption by whitefish cancelled each other out. In consequence, lower trophic levels showed only small changes in biomass.

When planktivorous whitefish were susceptible to infection, the biomass of all whitefish age classes declined up to 32%, which increased the biomass of their main resource, i.e. crustaceans (Fig. 2, middle). This interaction modification cascaded further down the food web, but only with weak effects (biomass change < 1%). Additionally, changes in crustacean biomass led to bottom–up effects, observed as higher resource densities for perch aged between zero and three years. However, the net effect on perch biomass was low, because the lower biomasses of whitefish reduced reproduction of ≥ 4 year old and exclusively piscivorous perch by 0.6–3.1% and increased the risk of predation for young perch (biomass gain of older from young perch increased by 0.2–1.7%).

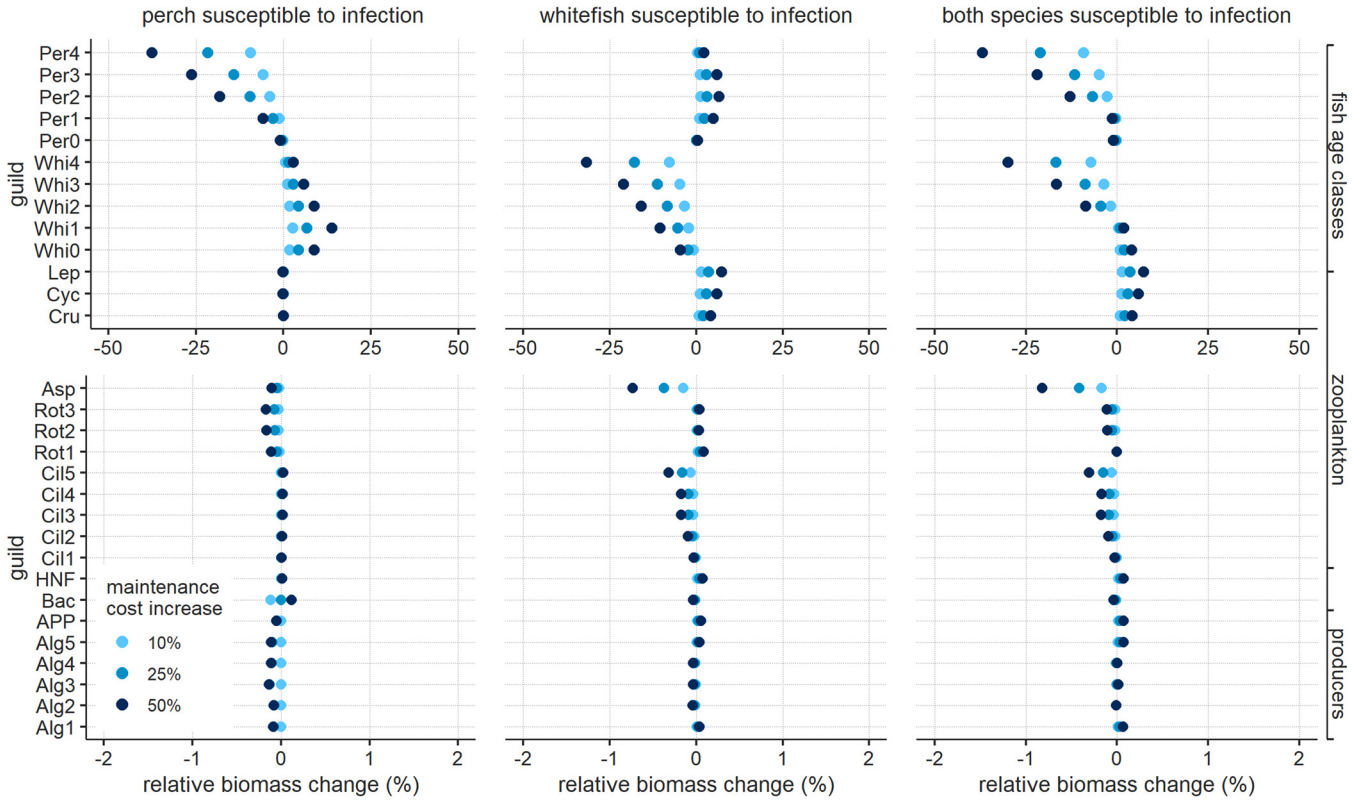

Figure 2. Impact of increased fish host maintenance costs on the biomass of 30 functional guilds in a lake food web. The figure displays the percentage changes in the total biomass of the guilds, relative to the absence of parasitism, for three scenarios based on which fish species was susceptible to infection: perch ('Per', five age classes: 0 = zero-year-old, 1 = one-year-old, 2 = two-year-old, 3 = three-year-old, 4 = ≥ four-year-old, left), whitefish ('Whi', middle), and both species (right). Each scenario considered three maximum increases in maintenance costs (10, 25, and 50%). The upper and lower rows of the figure show the guilds divided by the magnitude of biomass change (note different horizontal axis scales).

When both perch and whitefish were simultaneously susceptible to infection, a combination of the aforementioned effects were observed (Fig. 2, right). Across all scenarios, the net effect of increased host maintenance costs was a slightly reduced food web productivity, i.e. the total biomass decreased by 0.2–1.9%.

Increased host predation susceptibility

Increased predation susceptibility, simulated through a decline in the hosts' capability of seeking refuge from predators, significantly decreased host biomass. Only hosts aged zero and one year were vulnerable to predation, but the largest biomass declines were observed among hosts aged one- to three-years. As the refuge search rate decreased by a maximum of 50–99%, host biomass continued to decline, leading to modifications of species interactions and cascading effects throughout the entire food web.

When piscivorous perch were susceptible to infection, juvenile biomass (one year old) declined by 20–45% (Fig. 3, left). This resulted in low recruitment, and thus the parasite effect was also evident among older age classes that are not vulnerable to predation. However, the decline in biomass became smaller in each ascending age class, because low perch density relaxed intraspecific competition and young infected perch were more accessible as prey. In consequence, the biomass of the oldest perch age class was almost unchanged compared to levels without parasitism. The relaxed competition also increased perch reproduction and, subsequently, larval biomass of perch. Total perch biomass, however, remained below levels without parasitism, which relaxed competition and predation pressure for whitefish. This again increased whitefish biomass and led to their increased consumption of crustaceans. The net biomass change of crustaceans was nevertheless slightly positive (1–4%) due to strongly reduced predation by perch. This change in crustacean biomass had cascading effects on the biomass of lower trophic levels (change < 3%).

When planktivorous whitefish were susceptible to infection, patterns in host biomass change resembled those of perch (Fig. 3, middle). Perch experienced an increase in biomass due to more accessible infected whitefish prey and higher abundance of perch juveniles and crustaceans. However, the increased cannibalism dampened perch biomass growth. Crustacean biomass increased by 2–13%, which caused cascading effects in all lower trophic levels.

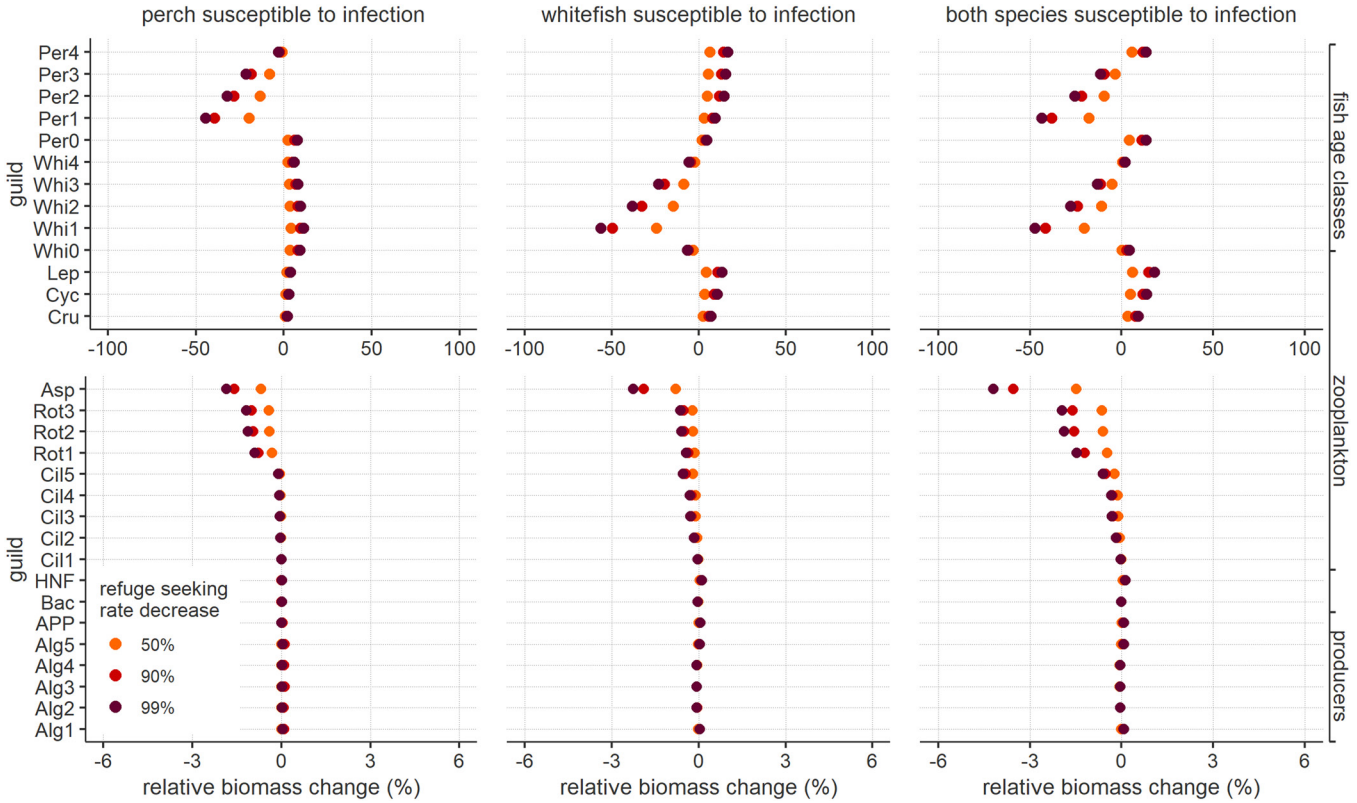

Figure 3. Impact of elevated host vulnerability to predation due to lower refuge search rate on the biomass of 30 functional guilds in a lake food web. The figure displays the percentage changes in the total biomass of the guilds, relative to the absence of parasitism, for three scenarios based on which fish species was susceptible to infection: perch ('Per', five age classes: 0 = zero-year-old, 1 = one-year-old, 2 = two-year-old, 3 = three-year-old, 4 = ≥ four-year-old, left), whitefish ('Whi', middle), and both species (right). For each scenario, three maximum decreases of the host refuge search rate were modelled (50, 90, and 99%). The upper and lower rows of the figure show the guilds divided by the magnitude of biomass change (note different horizontal axis scales).

When both fish species were simultaneously susceptible to infection, a combination of the aforementioned effects was again observed (Fig. 3, right). Notably, the increase in crustacean biomass of 3–18% caused large cascading effects in lower trophic levels. Across all species scenarios, the total biomass of the food web decreased by 0.2–1.0% due to increased host susceptibility to predation.

As an alternative to adjusting host refuge search rate to simulate an increase in predation risk, we also used an approach that adjusted the host capture rate. The patterns of biomass change with this approach were similar to those in Fig. 3 (Supporting information).

Simultaneously increased host maintenance costs and predation susceptibility

When host maintenance costs and predation susceptibility were both altered by the infection, the largest host biomass declines (up to 61%) and cascading effects were observed (Fig. 4). The total loss of biomass in the food web was also greatest (0.3–2.6%).

Discussion

Parasites are ubiquitous components of most, if not all ecosystems, but their influence on ecosystem dynamics remains largely unexplored. Using a food web model, we show that infection-induced changes in host phenotype can substantially reduce the biomass of host populations and alter their age structure. These changes can affect the abundance of species interacting with the host and cause abundance cascades. Additionally, our study highlights how complex networks can buffer such parasite effects despite changes in community structure and reduced productivity.

Lethal infections are known to regulate host populations by causing age-specific mortality (Jones et al. 2008, Campbell et al. 2018). We show that parasite-induced trait changes, often resulting from persistent sublethal infections, can also impact host demography by age-specific abundance changes, depending on the host trait affected by the infection. When infection increased host maintenance costs, the strongest decline in host biomass was observed in the oldest, reproducing age classes. This is because the simulated infection

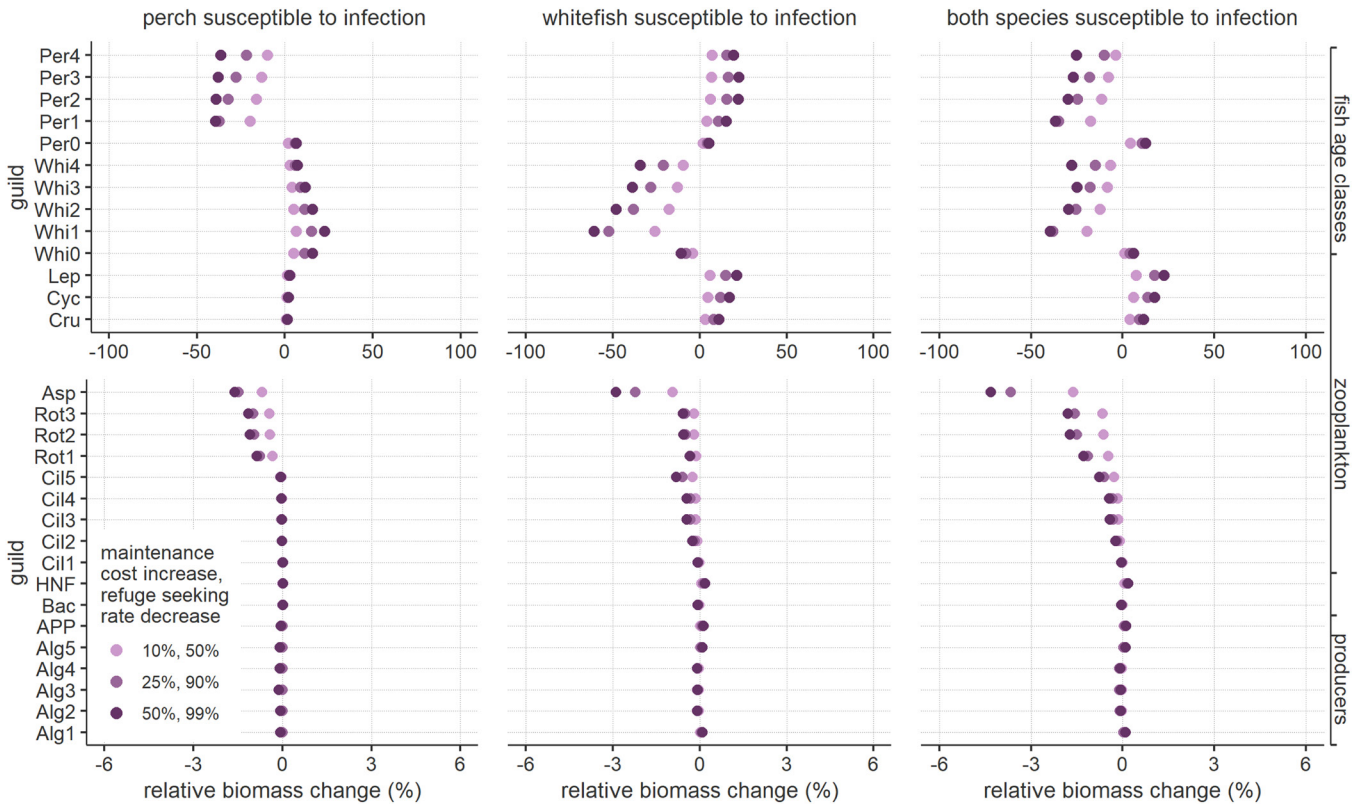

Figure 4. Impact of simultaneously increased host maintenance costs and predation susceptibility (reduced refuge search rate) on the total biomass of 30 functional guilds in a lake food web. The figure displays the percentage changes in the total biomass of the guilds, relative to the absence of parasitism, for three scenarios based on which fish species was susceptible to infection: perch ('Per', five age classes: 0 = zero-year-old, 1 = one-year-old, 2 = two-year-old, 3 = three-year-old, 4 = ≥four-year-old, left), whitefish ('Whi', middle), and both species (right). The upper and lower rows of the figure show the guilds divided by the magnitude of biomass change (note different horizontal axis scales). While each species scenario includes nine combinations of the magnitude of trait changes, only three combinations are shown here. All combinations can be found in the Supporting information.

accumulates with age and the magnitude of the associated host trait change increases with infection intensity, as is typical for macroparasites (Dobson 1988, Hudson and Dobson 1995). The decline in biomass in this case can be attributed to a reduced population growth rate due to increased energetic costs that divert resources from both growth and reproduction and elevate mortality rate. Conversely, when infection increased susceptibility to predation, the largest decline in host biomass was observed among middle age classes. This is because predation targeted juveniles, causing delayed effects due to lower recruitment to adult age classes, which gradually fade out. The decline in biomass in this case can be attributed to a slowed population growth rate due to higher loss to predation. When both traits were altered simultaneously, the largest decline in host biomass was again observed among middle age classes, as the impact of predation enhancement was greater overall.

Despite substantial host biomass declines, host populations persisted. This can be explained by compensatory responses in age classes that were less severely affected by parasitism or predation. When infection increased host maintenance costs, reproduction was reduced. However, larval biomasses remained nearly unchanged, because larvae have a lower parasite prevalence and intensity, and in addition experienced lower predation pressure or increased resource abundance due to the biomass decline in older age classes. This sustained the population from the base. When infection altered host predation susceptibility, the reduction in host abundance increased per capita resource availability, which relaxed intraspecific competition. The oldest age class then produced more fish larvae biomass than without parasitism, buffering the increased predation risk of juveniles. Similar responses have been observed in both empirical and theoretical studies, where high adult mortality or sterility due to infection led to increased juvenile biomass production, compensating, or even overcompensating the loss and therefore stabilizing populations (Ohlberger et al. 2011, Preston and Sauer 2020, Simon et al. 2022). Generally, the incorporation of within-guild structure to food webs has been identified as a mechanism promoting stability in complex networks (de Roos 2021). Thus, our results further highlight the importance of including host life history in studies exploring community effects of parasitism.

Despite compensatory mechanisms, overall host biomass declined due to parasitism, and this impacted interactions between the host and other species. First, host biomass decline relaxed predation pressure on the host's resource, resulting in the expected increase in resource biomass (Carpenter et al. 1985, Ripple et al. 2016). Additionally, it changed the resource availability for predators targeting the host (here, adult piscivorous perch). Although the energy flow from infected hosts to their predators can be expected to decrease in response to increased maintenance costs and to increase in response to increased vulnerability to predation, the net per capita gain of perch predators was negative in both cases when whitefish carried infections. This is because the increased consumption of infected whitefish under predation enhancement shifted the predator–prey biomass ratio to top-heavy, reducing per capita prey availability for perch. In consequence, predation enhancement can promote population oscillations that destabilize community dynamics, as shown in population dynamic models using simple predator–prey–parasite networks (Ives and Murray 1997, Fenton and Rands 2006). However, in more complex networks, where a predator and its prey are also often competitors (Arim and Marquet 2004), prey abundance decline can lead to competitive release for its predator. As a result, the reduced per capita gain from the infected prey (here whitefish) was compensated by increased gain from the resource shared with the prey (here crustaceans). Thus, the biomass of perch showed a low to moderate increase, relative to levels observed without parasitism. Consequently, changes in host traits propagated not only vertically to resources and consumers, but also horizontally to competitors, and including these competitive interactions buffered the decline in predator biomass.

The observed interaction modifications caused biomass changes across the entire food web. In some scenarios, cascading effects rippled down from the highest to the lowest trophic level. However, the parasite effect became progressively weaker towards lower trophic levels, a phenomenon commonly observed in trophic cascades (Shurin et al. 2002). This weakening can likely be attributed to the complexity of the network. Our study system includes a range of interactions, such as competition and cannibalism, as well as diverse prey, which can all buffer top–down effects (Borer et al. 2005). For example, copepods, daphnids, and large rotifers in our food web prey on 7–16 different guilds, including phytoplankton and zooplankton, which creates the potential for prey density compensation (Fox 2007). Additionally, phytoplankton in our model is abundant and highly productive, which also buffers top–down effects (Uusi-Heikkilä et al. 2022). However, the observed trophic cascades indicate that indirect effects of parasitism can have community-wide consequences. This was also observed in another study, where biomass changes induced by altered intrinsic growth rates of consumers, for example due to a disease outbreak or fade-out, negatively affected species at different trophic levels and sometimes led to quasi-extinctions (Selaković et al. 2022). Together, these studies suggest that parasites can be important elements of community dynamics and composition.

In this study, our focus was on modelling the indirect effects of parasites through changes in host phenotype in one specific life stage of the parasite. While this approach allowed us to isolate the distinct contributions of trait-mediated effects, it omits ecological feedbacks to the parasite via transmission events and changes in host biomass. Previous research integrating such dynamics in predator–prey–parasite models has suggested that intense predation on hosts may lead to a decline of parasite populations (Fenton and Rands 2006). While numerous examples of predation enhancement in natural systems (Moore 2002, Lefèvre et al. 2009, Poulin 2010) indicate that the strength of enhancement is optimized by natural selection, our model could be further expanded to

include parasite population dynamics. This would enable a more comprehensive investigation into the impact of parasites on food web dynamics.

This study highlights two important findings. First, a change in host phenotype caused by parasitic infection can significantly impact the dynamics of intra- and interspecific competition, as well as interactions between consumers and their resource, with cascading effects on the entire community. This suggests that indirect effects of parasitism are important elements of ecosystem dynamics. Second, despite the strong impact of parasitism, the host populations persisted. This is likely attributable to the complexity of the network, including different developmental stages of the host and their specific levels of infection, as well as various interactions between these stages and with other species. As understanding how ecosystems function is becoming increasingly important in addressing the effects of environmental change, it is crucial to include both parasite effects and host life history in ecological network studies for more realistic simulations of community dynamics.

Acknowledgements – We thank Anssi Karvonen for helpful comments on the manuscript.
Funding – This project was supported by the Academy of Finland (grant no. 317495 to AK and grant no. 350392 to SOL), Natural Sciences and Engineering Research Council of Canada (NSERC; Discovery grant no. RGPIN-2015-04249 to AK), and the European Research Council (ERC) under the European Union's Horizon 2020 research and innovation programme (grant no. 770884 to AK).

Author contributions

Ines Klemme: Conceptualization (equal); Formal analysis (equal); Visualization (lead); Writing – original draft (lead); Writing – review and editing (equal). **Tommi Perälä:** Conceptualization (equal); Formal analysis (equal); Methodology (lead); Software (lead); Visualization (supporting); Writing – original draft (supporting); Writing – review and editing (equal); **Sami O. Lehtinen:** Conceptualization (equal); Funding acquisition (supporting); Methodology (supporting); Software (supporting); Writing – original draft (supporting); Writing – review and editing (equal). **Anna Kuparinen:** Conceptualization (equal); Funding acquisition (lead); Writing – original draft (supporting); Writing – review and editing (equal).

Data availability statement

Data are available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.vx0k6djx7 (Perälä and Klemme 2024).

Supporting information

The Supporting information associated with this article is available with the online version.

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