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Bottom–up regulation of parasite population densities in freshwater ecosystems

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Theory predicts the bottom–up coupling of resource and consumer densities, and epidemiological models make the same prediction for host–parasite interactions. Empirical evidence that spatial variation in local host density drives parasite population density remains scarce, however. We test the coupling of consumer (parasite) and resource (host) populations using data from 310 populations of metazoan parasites infecting invertebrates and fish in New Zealand lakes, spanning a range of transmission modes. Both parasite density (no. parasites per m^2) and intensity of infection (no. parasites per infected hosts) were quantified for each parasite population, and related to host density, spatial variability in host density and transmission mode (egg ingestion, contact transmission or trophic transmission). The results show that dense and temporally stable host populations are exploited by denser and more stable parasite populations. For parasites with multi-host cycles, density of the 'source' host did not matter: only density of the current host affected parasite density at a given life stage. For contact-transmitted parasites, intensity of infection decreased with increasing host density. Our results support the strong bottom–up coupling of consumer and resource densities, but also suggest that intraspecific competition among parasites may be weaker when hosts are abundant: high host density promotes greater parasite population density, but also reduces the number of conspecific parasites per individual host.

As pointed out by May (1999), what determines population density remains a mostly unanswered question and a challenge for 21st century ecology. In principle, the local abundance of a consumer should be driven in large part by the local abundance of its resource, in a bottom–up fashion (Yang et al. 2010, McCann 2012). Given a small number of limiting resources, variability in resource density at different sites should predict spatial variation in density of a focal consumer species. Yet examples of the tight coupling in the field between resource and consumer densities for predators and herbivores remain scarce (Adler 1998, Roth 2003, Karanth et al. 2004). The multiplicity of resources used by generalist consumers can weaken the coupling of their population density with that of any given resource species (Stenseth et al. 1997, Murdoch et al. 2002), which may explain the limited empirical evidence for that coupling.

Host–parasite interactions should be particularly good resource–consumer models to test the bottom–up control of consumer density. The host not only provides nutrients but also represents the sole habitat of a parasite at a given life stage, and most parasites exhibit some degree of host specificity, i.e. they are resource specialists. At a community level, the bottom–up control of parasite diversity is evident from the strong relationship between local host species richness and parasite species richness (Kamiya et al. 2014). The same bottom–up regulation should apply to the density of focal parasite species. Epidemiological theory is founded on the relationship between host density and parasite transmission success: for any parasite, there is a threshold host density necessary for parasite persistence, and parasite population size increases with host density beyond that threshold (Anderson and May 1978, 1979, May and Anderson 1979, Diekmann and Heesterbeek 2000). Several studies demonstrate the driving role of host density in epidemics of microparasites such as bacterial and viral pathogens (Roberts 1996, Grenfell and Bolker 1998, Rhodes et al. 1998, Packer et al. 1999). Empirical demonstrations involving metazoan macroparasites have generally focused on the link between host density and infection levels experienced by individual hosts, quantified as prevalence or intensity of infection (Arneberg et al. 1998, Arneberg 2001, Bagge et al. 2004).

In contrast, there are only a few studies asking what factors affect the density of parasites, in the traditional ecological sense of numbers of individuals per unit surface area. Perhaps this is because it seems obvious that the density of parasites will increase with host density. Hechinger et al. (2008) did this for trematodes in fishes, though they focused on the effect of fishing on parasite populations. The best study so far is that of Sonnenholzner et al. (2011), which describes how the density of parasitic snails increases with the density of their sea urchin host across sites, underscoring a strong bottom–up effect for this host–parasite association. Interestingly, the abundance of parasites per urchin did not increase with urchin density, suggesting that bottom–up processes that favored the parasite population as a whole did not translate into increased infection risk per host. Only few

other studies (Kuris et al. 2008, Hechinger et al. 2011) have measured multiple parasite species in terms of their density, yet there has been little effort to look at the relationship between the densities of hosts and parasite across many parasite species in the same system. Such evidence is needed to better understand the generality of bottom–up processes in consumer–resource dynamics and what drives local parasite density across localities.

Here, we use data from multiple host and metazoan parasite species from an extensive sampling of four freshwater food webs to address four general hypotheses about the coupling of host and parasite densities. First, we test whether the density of parasite populations covaries spatially with that of their host species, as predicted by the hypothesis that consumer densities and resource densities are tightly coupled. Second, we hypothesize that the temporal stability of host density, and not just its mean value, also favours denser and more stable parasite populations (Altizer et al. 2006). Localities characterised by stable host resources should allow the accumulation of greater numbers of parasites, whereas those where host resources fluctuate widely should experience regular parasite population crashes. Fluctuating resources exert selective pressures on parasites and other types of consumers to adapt their life histories or exploitation strategies (Tannerfeldt and Angerbjörn 1998, Krasnov et al. 2006); here, we will test the prediction that fluctuating host resources are also associated with lower parasite population densities.

Third, we also hypothesize that based on their transmission mode and how they infect their host, host density and its stability will have greater impact on the population density of certain parasites than others. In particular, we expect that host density will affect the success rate of parasites transmitted via free-living stages contacting hosts more strongly than that of parasites transmitted trophically (by ingestion of prey infected by juvenile parasite stages). Direct transmission is to a large degree determined by the frequency of contacts between hosts and parasites, a process dependent on host density (Diekmann and Heesterbeek 2000). In contrast, trophic transmission from a prey intermediate host to a predator definitive host is influenced by the level of aggregation of juvenile parasites in the prey population, manipulation of intermediate host phenotype by the parasite, individual predator behaviour, presence of non-host predators, and/or availability of alternative prey species (Lafferty 1992, 1999, Mouritsen and Poulin 2003, Kaldonski et al. 2008). Populations of trophically transmitted parasites are not expected to be regulated by host density (Arneberg 2001). Therefore, we expect a significant interaction between transmission mode and host density or stability in their effect on local parasite densities. Fourth, in parasites with complex life cycles, the population of a parasite at a particular life stage and in a particular host species all originates from the previous stage occupying a different host species. Thus, the density of parasites at a particular life stage might depend not only on the density of their current host, but also on that of their previous or 'source' host since the latter may affect the numbers of potential recruits to the next stage of the life cycle. Because we expect the present resource–consumer link to be stronger than that linking a consumer to the resources of its youth, we hypothesize that source host density will also affect the density of parasite populations, but to a lesser extent than the density of their current hosts.

In addition to these analyses of the determinants of total parasite population density, we also test the effect of host density and its temporal stability on a more traditional measure of parasite abundance, i.e. intensity of infection (mean number of individuals per infected host; sensu Margolis et al. 1982). This represents the mean infrapopulation size, that is, the mean size of groups of parasite individuals that can interact directly by virtue of sharing the same individual host. Infrapopulations within hosts are where intraspecific competition and density-dependence can be manifested through their effects on parasite growth, survival or reproduction (Poulin 2001). We therefore examine how host density influences the potential strength of intraspecific interactions within parasite populations.

Our tests of the above hypotheses span multiple taxa of hosts and metazoan parasites, and four different lake ecosystems. We therefore provide general assessments of our predictions that apply across localities and types of parasites. Our results represent a large-scale, replicated confirmation of the bottom–up control of consumer densities for resource specialists.

Methods

Field sampling and laboratory processing

We investigated the entire community of free-living and parasitic metazoans from the littoral zone of four lakes on the South Island of New Zealand. We chose small-to-medium sized lakes, mostly shallow, and at different altitudes and distances from the coast (for name, location and characteristics of each lake, see Supplementary Material Appendix 1 Fig. A1 and Table A1). In each lake, we sampled four square areas (15×15 m) with one side of the square along the shore, distant by 123 to 2250 m from each other and selected to represent all habitat types (substrate, macrophytes, riparian vegetation, etc.) present within each lake. This gave us 16 study sites (4 lakes $\times$ 4 sampling sites per lake). Each site was sampled in three seasons (September 2012, January and May 2013) to assess temporal changes in the communities, and on each occasion we sampled all fish, benthic and demersal invertebrates, plankton and all metazoan parasites on or within these organisms.

Fish were sampled using a combination of gear types following a standardized protocol to achieve estimates that represented as accurately as possible actual fish diversity and density (full details in Supplementary Material Appendix 1). Two fyke nets were set overnight along the edges of the sampling area, perpendicular to the shore, and two 15 m long multi-mesh gillnets were deployed in the same place during the day. These were used to capture all fish swimming in and out of the area, i.e. both residents and visitors to the area. In addition, a standard, fine-mesh purse seine net was dragged across the whole area to capture small and/or sedentary resident fish not captured by passive gear like fyke nets or gillnets. All fish caught were identified to species, counted, measured, and a subsample was returned to the laboratory for dissection. In each site and in each season, six samples

of benthic invertebrates, distributed haphazardly across the sampling area, were taken using a standard Surber sampler net with a 0.1-m² horizontal metal frame fitted with a 250-µm mesh collecting net. In addition, six samples of demersal invertebrates, living on or near the substrate but not captured in Surber nets, were sampled using a rectangular dip net (30 cm wide and 22 cm high opening) with a 250-µm mesh net; each sample consisted of a fast, 2-m long sweep of the net along the lake bottom without dredging the substrate. All invertebrate samples were preserved in ethanol for later identification, counting and dissection. Plankton samples were also collected but as no metazoan parasites were found in planktonic organisms, they are not considered here. Also, birds were not sampled as part of this study, and therefore any parasite adult stages using bird hosts are not included in the present analyses.

In the laboratory, all individuals were identified to species and counted, after which a subsample of each species was dissected carefully following a standardised protocol for parasite recovery and identification (see Supplementary Material Appendix 1 for full details).

All data used in the following analyses are available from the Dryad Digital Repository (<http://dx.doi.org/10.5061/dryad.427v8>) (Lagrue and Poulin 2015).

Host and parasite variables

Distinct life stages of parasites with complex life cycles exploit completely different host species and infect them via different transmission routes; for example, in trematodes, the second intermediate host is infected by contact with free-swimming cercariae, and the definitive host by ingestion of infected prey. As our goal is to link parasite density with host density to determine if the latter affects epidemiological processes such as transmission success, host density must represent the resources available to a particular set of transmission stages. Therefore, different parasite life stages are treated here as separate 'parasite populations'. Each entry in the dataset is a population corresponding to a unique parasite life stage–by–site combination, though parasites not occurring in at least two sites per lake were excluded.

For each population of either free-living or parasite species, we calculated density (individuals per m²) as a measure of local population size. Given the inherent difficulties associated with density estimates of mobile organisms like fish (and to a lesser extent invertebrates), our estimates are to some degree measures of relative density. Parasites are rarely quantified this way (but see Hechinger et al. 2008, Sonnenholzner et al. 2011); instead, parasite populations are usually quantified as individuals per host rather than per surface area. However, density is not only a relevant population-level measure for metazoan macroparasites, but it also provides a common metric for all free-living and parasite species.

In the case of trematodes in their snail first intermediate host, we did not count each individual redia or sporocyst as separate individual parasites, since these are the product of clonal multiplication and not separate infection events. Except for rare cases of multiple infections, all rediae or sporocysts have the same genotype and are issued from the same larva hatched from a single egg. Their numbers within a host do not reflect transmission processes and are unrelated to host density, and therefore the densities of these life stages were measured as the number of infected snails per m². In contrast, although trematodes at subsequent life stages (e.g. metacercariae) also include several genetically identical individuals, each of these reaches its host via a separate infection event, and the density of their 'population' is therefore more directly linked to the availability of host resources (host density).

For each site and each sampling period, we first averaged the density values of each host or parasite population across all samples. Then, we calculated both the mean density per site across all three sampling periods, and the coefficient of variation (CV; standard deviation divided by the mean) of density across sampling periods. The former provides a measure of local population size, whereas the CV provides an index of the temporal stability of a population across seasons: the higher the CV, the less stable the population size, i.e. the more it fluctuates over time.

In most cases, a parasite population (as defined above) exploited a single host species. However, in cases where the parasites used two or more host species, the main host was defined as the species harbouring the most parasite individuals. If the second most-important host species harboured at least a quarter of the number of parasites found in the main host, it was combined with the main host to calculate mean host density, and CV, for that parasite population. In the rare cases where the third most-important host species harboured at least a quarter of the number of parasites found in the second most-important host species, it was also included in calculations of mean host density and its CV.

For all parasite populations, except for trematodes in their snail first intermediate host, we also calculated the mean intensity of infection, or the mean number of individuals per infected host (Margolis et al. 1982). It was calculated across all individuals from all samples obtained from a site, with parasites using two or more host species treated as above, i.e. secondary hosts were only included in the calculations if they met the above criteria. In our analyses, we only included data from parasite populations in which estimates of mean intensity of infection were based on at least six infected host individuals; in that subset, preliminary analyses indicated that mean intensity values did not correlate significantly with number of infected hosts.

For all helminths with complex life cycles, the population of one life stage in a host species originates from an 'upstream' population at an earlier life stage in a different host species (hereafter, the source host). For the subset of trematode, nematode and acanthocephalan species where it was possible, we also calculated mean source host density and its CV, as above. To avoid the circularity imposed by the nature of parasite life cycles, we only considered linkages in the egg-to-adult direction, and not the connection between density of hosts harbouring adults and that of juvenile parasites in their first or only intermediate host.

Finally, each parasite population was classified based on its transmission mode, i.e. the infection process by which it accumulated within its current hosts. There were three transmission modes: 1) egg ingestion, whereby parasite eggs are eaten by the hosts, e.g. nematodes and acanthocephalans in their intermediate hosts or some trematodes in their first intermediate hosts; 2) contact, involving a free-swimming

infective stage contacting the host's external surface, as in ectoparasitic mites or the miracidial and cercarial transmission stages of trematodes; and 3) trophic transmission, in which a definitive host acquires packets of juvenile parasites each time it eats infected prey, a common transmission route for helminths toward their final host. A fourth group of parasites were excluded from the analyses: these include a few trematode species whose asexually-produced infective stages (cercariae), instead of leaving the snail first intermediate host to seek another host, remain in it to encyst as metacercariae. Their numbers reflect within-host multiplication processes independent of local host density, and they are therefore inappropriate for the present analyses.

Statistical analysis

The three measures of parasite populations used as response variables in the following analyses, i.e. parasite density per site, its CV and mean intensity per host, were only weakly correlated with each other (density versus CV: $n = 310$, $R^2 = 0.014$; density versus intensity: $n = 129$, $R^2 = 0.048$; CV density versus intensity: $n = 192$, $R^2 = 0.081$). They are therefore somewhat independent of each other and will be considered separately.

Parasite density per site, the CV of parasite density, and mean intensity per host were log-transformed and then analysed using three separate mixed-effects models with Gaussian error structure implemented in JMP ver. 11.0 (SAS Inst.). Our main goal was to test the effect of host density and CV in host density on local parasite population density, its stability, and mean infection intensity, and to test whether these effects differed among parasites with different transmission modes. Therefore, host density, CV in host density (both log-transformed) and transmission mode (egg ingestion, contact transmission or trophic transmission) were included as fixed factors in the models. The two-way interactions between transmission mode and host density, and between transmission mode and CV in host density, were also included in the original model, but dropped from the models in which they were not significant and did not improve model fit.

Lake and sampling site (nested within lake) were included as random factors in the models. This accounts for idiosyncrasies of particular lakes and for the non-independence and correlated structures in the data arising from the fact that multiple data points come from the same site. In addition, parasite species and their higher taxon (trematodes, cestodes, nematodes, acanthocephalans or mites), as well as the main host species and its higher taxon (snail, leech, crustacean, insect or fish), were also included as nested random factors to account for any phylogenetic influences. We calculated the proportion of the total variance unexplained by the fixed effects that could be accounted for by each random effect (Nakagawa and Schielzeth 2013).

Finally, the above mixed-effects models with parasite density per site and its CV as response variables were repeated, with the same predictors, using the subset of the data for which it had been possible to obtain mean density and CV for the source host species. In this case, mean source host density and its CV (both log-transformed) were included as additional fixed factors.

Results

The dataset consisted of information on 310 parasite populations (Table 1), as defined in the Methods, representing 30 distinct parasite species, the majority of which were trematodes, infecting 17 host species (Supplementary material Appendix 1 Table A2). The densities of these parasite populations ranged from less than 1 to over 14 000 individuals m^{-2}. Across the 310 parasite populations, untransformed values of CV in density ranged from 0.22 to 4.24.

Parasite population density and its CV

Spatial variation in local parasite density was significantly affected by both the density and the stability (inverse of CV) of local host density (Table 2). Parasite populations achieved higher densities where host densities were higher (Fig. 1) and, to a much lesser extent, where host densities were more stable over time, i.e. showed a lower CV (Supplementary material Appendix 1 Fig. A2). Although parasites transmitted trophically and those acquired by egg ingestion form distinct clusters because of different host densities (Fig. 1), the host density versus parasite density relationship is significant within each group of parasites (contact transmission: $R^2 = 0.329$, $p < 0.0001$; egg ingestion: $R^2 = 0.342$, $p < 0.0001$; trophic transmission: $R^2 = 0.101$, $p = 0.0018$). Parasite densities were generally higher, for a given host density, for parasites transmitted by contact than for those transmitted either trophically or by egg ingestion (Table 2, Fig. 1). In addition, there was a weak but significant interaction between transmission mode and host density (Table 2). This interaction is apparent when considering the host density versus parasite density relationships across sites within the same lake for each parasite species and life stage: the individual correlations are slightly stronger for contact-transmitted parasites (median = 0.877) and weaker for trophically transmitted parasites (median = 0.737; Fig. 2). Finally, the identity of the parasite species and host higher taxon involved were the only random factors to explain a substantial portion of the remaining variance in parasite population densities;

Table 1. Distribution of the 310 parasite populations considered here among parasite and host taxa and transmission modes.

		Transmission mode		
Parasite taxon	Host taxon	Egg ingestion	Contact	Trophic
Acanthocephalans	crustaceans	5	–	–
	fish	–	–	6
Nematodes	crustaceans	3	–	–
	fish	–	–	34
Cestodes	fish	–	–	4
Trematodes	snails	35	85	–
	leeches	–	3	–
	crustaceans	–	23	–
	insects	–	6	–
	fish	–	52	50
Mites	insects	–	4	–

Table 2. Results of the mixed-effects model with (a) mean parasite density and (b) the coefficient of variation (CV) in parasite density per parasite population as response variables, showing the effects of the main predictors and the proportion of the remaining variance accounted for by the random factors. * trophic transmission is included in the intercept.

Fixed factors	Estimate	Std error	*t*-value	p	Random factors	% variance
(a) Mean parasite density						
Intercept*	−1.2142	0.7485	1.62	0.1515	Site [lake]	0.37
Log host density	0.9118	0.0732	12.46	<0.0001	Lake	1.35
Log CV host density	−0.5235	0.1701	3.08	0.0024	Parasite species [higher taxon]	24.06
Transmission mode (contact)	0.4299	0.1765	2.44	0.0154	Parasite higher taxon	8.36
Transmission mode (egg ingestion)	−0.1434	0.3327	0.43	0.6667	Host species [higher host taxon]	9.87
Transmission (contact) × host density	0.1983	0.0788	2.52	0.0124	Host higher taxon	47.14
Transmission (egg ingestion) × host density	−0.1357	0.1155	1.18	0.2409		
(b) CV in parasite density						
Intercept*	0.4177	0.1565	2.67	0.0517	Site [lake]	0.23
Log host density	−0.0948	0.0168	5.63	<0.0001	Lake	1.31
Log CV host density	0.3462	0.0447	7.74	<0.0001	Parasite species [higher taxon]	3.04
Transmission mode (contact)	−0.0255	0.0160	1.59	0.1123	Parasite higher taxon	4.36
Transmission mode (egg ingestion)	−0.0008	0.0265	0.03	0.9743	Host species [higher host taxon]	5.64
					Host higher taxon	69.25

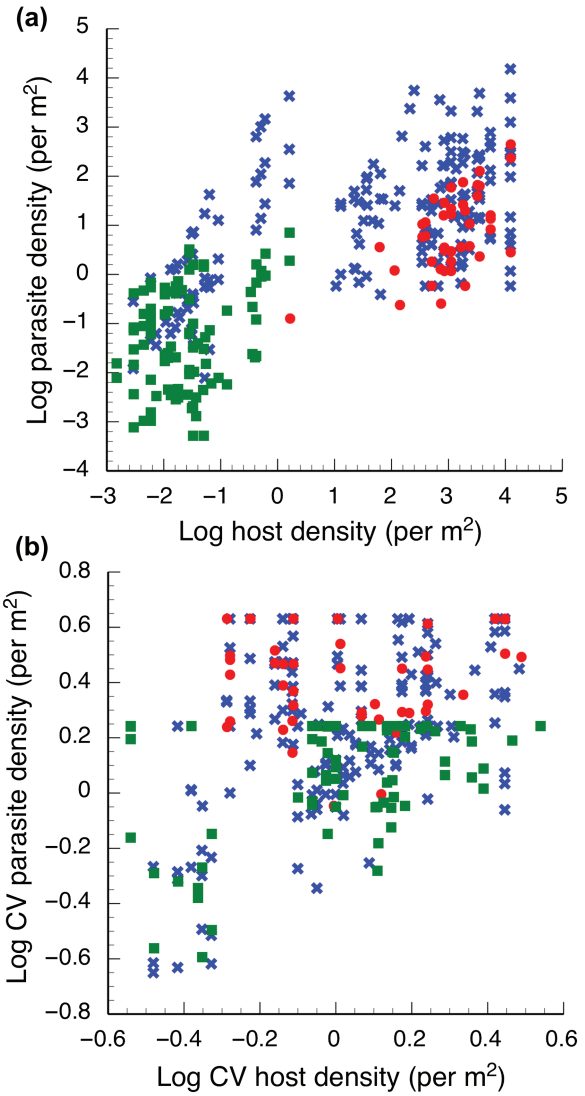

Figure 1. Mean local parasite density as a function of host density (a), and coefficient of variation (CV) in local parasite density as a function of CV in host density calculated over a year (b), across 310 parasite populations from freshwater ecosystems. Parasites transmitted by contact (blue crosses), egg ingestion (red circles) or trophically (green squares) are shown by different symbols.

notably, lake identity and site within lake explained less than 2% of the variance (Table 2).

Variation across parasite populations in their temporal stability (inverse of CV) was significantly driven by both the density and the stability of local host populations (Table 2). The higher the mean host density, the more stable the parasite population density. And the more stable the host density, the more stable the parasite density over time (i.e. positive relationship between CVs of host and parasite densities; Fig. 1). Transmission mode had no direct influence on parasite population stability, nor did it interact with host density measures (interactions were excluded from the final model).

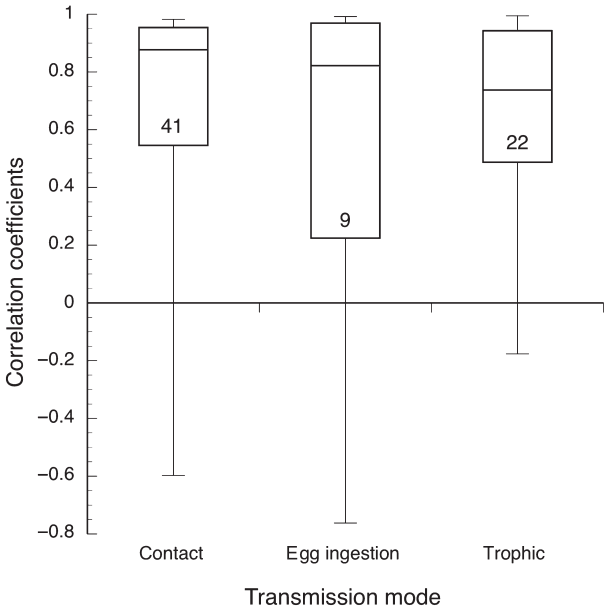

Figure 2. Boxplot showing the distribution of correlation coefficients between mean host density and parasite density, calculated for each set of 3–4 parasite populations from the same species, same life stage and same lake. The correlations are shown separately for parasites with different transmission modes; the line extends from the 10th to the 90th percentile, the box comprises the interquartile range, and the line through the box corresponds to the median value. Numbers of correlation coefficients in each group are given in the boxes.

As before, lake identity and site within lake explained very little of the remaining variance, and only the host higher taxon involved accounted for a substantial portion of that variance (Table 2).

Data on the mean density and CV in density of the source host species were available for 116 of the 310 parasite populations, none of which were transmitted via egg ingestion. Across this subset of 116 parasite populations, those with high mean densities tend to have a source host with high density, but only when all parasites are pooled and not among those with the same transmission mode (Supplementary material Appendix 1 Fig. A3). However, when source host density and its CV were added as fixed factors to the above models, i.e. when population measures of the source host and current host were allowed to 'compete' as predictors, only the density of the current host emerged as a significant predictor of parasite population density (Supplementary material Appendix 1 Table A3). Similarly, in the model explaining variation across parasite populations in their temporal CV in density, only the CV of the current host's population density was a significant predictor (Supplementary material Appendix 1 Table A4).

Parasite intensity of infection

Data on the mean intensity of infection were available for 129 of the 310 parasite populations, none of which were transmitted via egg ingestion. Variation across parasite populations in mean intensity of infection per host was significantly affected by the density of local host populations (Table 3). The higher the mean host density, the lower the infection intensity (Fig. 3). There was a significant interaction between transmission mode and host density, such that the effect of the latter was mostly restricted to contact-transmitted parasites. As before, lake identity and site within lake explained very little of the remaining variance, and only the identity of parasite and host taxa accounted for a substantial portion of that variance (Table 3).

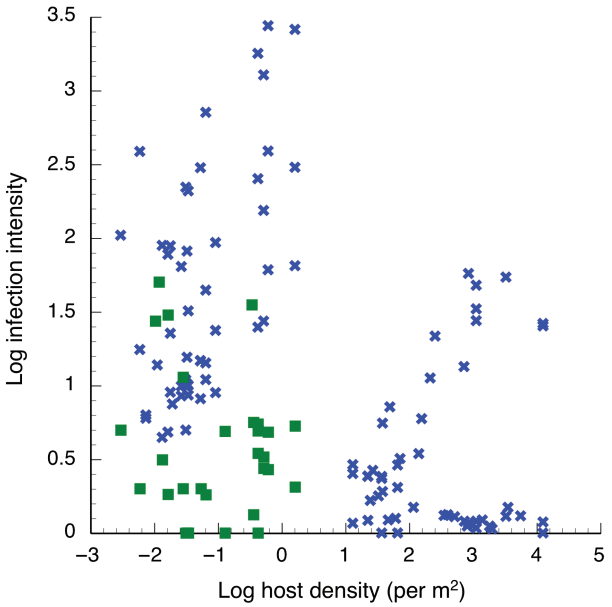

Figure 3. Mean intensity of infection (no. parasite individuals per infected host) as a function of host density, across 129 parasite populations from freshwater ecosystems. Parasites transmitted by contact (blue crosses) or trophically (green squares) are shown by different symbols.

Discussion

Our results provide general support, across multiple species and lake ecosystems, for the direct coupling of parasite density with host density. They are consistent with past work showing that host density drives parasite density (Sonnenholzner et al. 2011), as predicted by basic logic and theory. Also, as Sonnenholzner et al. (2011), we did not find that mean infection intensity increases with host density. In principle, if infective stages are finite, they must be shared among downstream hosts, potentially leading to a negative association between downstream host density and mean intensity in downstream hosts. Where our study differs from past efforts is in showing that a large number of parasite populations and species share this pattern. Specifically, we found that: 1) host populations occurring at high densities and/or, to a lesser extent, those with temporally stable densities, harboured denser and more stable parasite populations; 2) host density affected the population density of parasites transmitted via free-living stages (i.e. through direct contact with hosts) more strongly than that of parasites transmitted trophically; 3) in parasites with complex life cycles, the density of the current host had a much greater impact on parasite density than the density of the source host; and 4) contact-transmitted parasites achieved lower intensity of infection per host when host population density was high.

The only previous test of the parasite density versus host density relationship that we are aware of in which parasite population size was measured per surface area is that by Sonnenholzner et al. (2011), who found that densities of parasitic snails were positively related to those of sea urchin

Table 3. Results of the mixed-effects model with the mean intensity of infection per parasite population as the response variable, showing the effects of the main predictors and the proportion of the remaining variance accounted for by the random factors. *trophic transmission is included in the intercept.

Fixed factors	Estimate	Std error	*t*-value	p	Random factors	% variance
Intercept*	0.7262	0.3101	2.34	0.0377	Site [lake]	0.20
Log host density	−0.1145	0.0525	2.18	0.0313	Lake	4.07
Log CV host density	−0.0772	0.1615	0.48	0.6336	Parasite species [higher taxon]	61.02
Transmission mode (contact)	−0.0041	0.2177	0.02	0.9853	Parasite higher taxon	4.46
Transmission × host density	0.0935	0.0447	2.09	0.0388	Host species [higher host taxon]	3.49
Transmission × CV host density	−0.4924	0.1510	3.26	0.0015	Host higher taxon	19.25

hosts. Although intuitively obvious, this relationship is not inevitable. Parasite density, as measured here, depends not only on host density, but also on the proportion of hosts that are infected and the mean number of parasite individuals per host. High host density could simply result in a dilution of parasite individuals among multiple resource patches, and not necessarily lead to higher parasite population size. Such a dilution seems to occur in the freshwater systems we studied, where, at least in the case of contact-transmitted parasites, higher host densities lead to lower intensity of infection. This may in part be due to constraints imposed by host body size on maximum numbers of parasites per host, since host species occurring at higher densities also tend to be small-bodied (Blackburn et al. 1990). Even so, the pattern suggests that intraspecific competition among parasites, which occurs within infected individual hosts and is proportional to the number of co-occurring conspecific parasites (Shostak and Scott 1993, Churcher et al. 2005), may be reduced at high host densities.

Density is only one component of host population size; the other key one is its stability over time. Host density can fluctuate seasonally due to recruitment, mortality or migration, with consequences for the availability of resources for parasites (White et al. 1996, Altizer et al. 2006). We found that the more stable host populations, i.e. those with a low coefficient of variation (CV) in density, harboured not only more stable parasite populations, but also denser parasite populations. On the one hand, parasite populations, if not exerting significant fitness reductions in their hosts, may be regulated by host population density. Both juvenile and adult stages of metazoan parasites can generally survive for several months, even years, allowing for their accumulation over time if the host population remains stable. On the other hand, the coupling between host and parasite populations may result from the negative impact of parasites on their hosts, leading to parasite-mediated control of host populations and coupled population cycles on short or long time scales (Hudson et al. 1998, Hall et al. 2011). Only experimental manipulations could establish the direction of causality. However, the parasite species we studied range from benign gut parasites to more virulent parasites capable of host castration or growth impairment. It is therefore likely that some of the cases of coupled host–parasite densities reflect bottom–up regulation of consumer densities, whereas others reflect top–down control of host resources.

The population density of parasites transmitted via free-living stages which contact hosts was more tightly coupled with host density than that of trophically-transmitted parasites. In aquatic environments, mobile infective stages incur severe losses and fail to reach their target host because of unfavourable abiotic conditions, predation by a wide range or organisms, contact with and attempted infection of unsuitable hosts, etc. (Pietrock and Marcogliese 2003, Thieltges et al. 2008, Johnson and Thieltges 2010). Our findings suggest that, all else being equal, a higher density of target hosts can overcome these transmission obstacles. In contrast, for trophically-transmitted parasites, a greater density of predatory definitive hosts is not as strongly coupled with parasite density in those hosts. The dynamics of transmission via predator–prey links may not always be tightly dependent on predator density because predators are often generalists, feeding on multiple prey species and not exclusively on those harbouring juvenile parasites. This is certainly the case with the fish host species in our dataset.

Intriguingly, whereas the population density of the parasite's current host significantly affects the parasite's own population, the density of the source host has no measurable influence. In parasites with complex life cycles, the individuals at one stage of the cycle originate from the previous stage inside a different host species; therefore, the availability of resources for the previous life stages should have knock-on effects downstream on the density of parasites in the next host. Although epidemiological models of parasites with multi-host life cycles include population densities of both the source and target hosts as key parameters (Dobson 1988), our data indicate that only the latter matters for parasite population density. There is nevertheless a statistical link between cohorts at different life stages, however. When we replaced the density of the source host by the density of the upstream parasite life stage as a predictor in our model, we found that there was a significant positive effect of the density of the upstream stage population on that of the downstream stage (Supplementary material Appendix 1 Table A5). For example, for any given trematode species, the higher the density of metacercariae in an area, the higher the density of the adult stage. Thus, the supply of parasite individuals from the preceding life stage, if not the density of the hosts they exploit, contributes to parasite population density downstream.

One aspect of our estimates of parasite density deserves a second look. In the case of trematodes in their snail first intermediate host, we did not consider each individual redia or sporocyst as separate individual parasites, since these are the product of clonal multiplication. We assumed all rediae or sporocysts were issued from the same larva hatched from a single egg, and we adopted the principle that one individual corresponds to one genotype. However, this is not always the case. In *Coitocaecum parvum*, the only trematode species in our dataset which has been studied using microsatellite genotyping, some snails harbour more than one genotype, for an average of 1.4 genotypes per infected snail (Lagrue et al. 2007). In *Maritrema novaezealandensis*, a relative of the trematode *M. poulini* included in our study, this average was 1.9 genotypes per infected snail (Keeney et al. 2007). Other trematode species in our dataset generally occur at lower prevalences than *C. parvum* and *M. poulini*, which should reduce the frequency at which multiple genotypes co-exist in the same snail host (Louhi et al. 2013). Using the above numbers as a correction for multiple genotypes per snail increases our density estimates for trematodes at that life cycle stage, but not very much. For instance, it results in a slight upward shift of several points in the upper-right corner of Fig. 1a, and has no impact on our general findings.

Our results expand a previous demonstration of a bottom–up control of parasite densities by host abundance to multiple parasite species. Perhaps more importantly, our findings also indicate that whereas parasite density increases with host density, the same is not true for intensity of infection. High host densities can cause the parasite population to become diluted among many host individuals, especially for contact-transmitted parasites, but nevertheless achieve higher overall density per surface area. Since

intraspecific competition among parasites for host resources occurs among individual parasites within the same host, the reduced intensities of infection associated with high host densities may allow higher per capita parasite growth and fecundity (Shostak and Scott 1993, Churcher et al. 2005). Thus, higher total parasite population size may not necessarily lead to more intense intraspecific competition.

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Supplementary material (available online as Appendix oik.02164 at <www.oikosjournal.org/appendix/oik-02164>). Appendix 1. Detailed Methods. Figure A1. Location of the 4 study lakes on the South Island of New Zealand. Figure A2. Mean local parasite density as a function of the coefficient of variation in host density calculated over a year, across 310 parasite populations. Figure A3. Mean local parasite density as a function of the local density of their source host species, across 116 parasite populations. Table A1. Geographical locations and characteristics of the four study lakes (South Island of New Zealand), and distance between sampling sites. Table A2. Parasite populations included in the present study. Table A3. Results of the mixed-effects model with mean parasite density per site as the response variable, including both population measures of the current host and the source host as predictors. Table A4. Results of the mixed-effects model with the coefficient of variation in parasite density per site as the response variable, including both population measures of the current host and the source host as predictors. Table A5. Results of the mixed-effects model with mean parasite density per site as the response variable, with both population measures of the current host and the density of the parasites' upstream stages (occurring in source hosts) as predictors.