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Global drivers of parasitism in freshwater plankton communities

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Abstract

Zooplankton and phytoplankton communities play host to a wide diversity of parasites, which have been found to play a significant role in a number of ecosystem processes, such as facilitating energy transfer and promoting species succession through altering interspecific competition. Yet we know little about the mechanisms that drive parasite dynamics in aquatic ecosystems. Recent mathematical models have shown how habitat can shape parasite dynamics through influencing the efficacy of parasite transmission; however, these predictions have yet to be tested at larger ecological scales. Here, we present a comparative analysis of parasitism in planktonic communities, assembling data from a range of host and parasite taxa, habitat types, and geographic regions. Our results suggest that the prominent depth-prevalence relationship observed in studies on *Daphnia* in temperate lakes of North America is applicable to a wide range of aquatic habitats, hosts, and parasites; however, differences in transmission strategies between parasites can lead to considerable variation in parasite dynamics. Observational studies which incorporate a diversity of habitat types will be important in uncovering the mechanisms which underlie this relationship. In particular, more experimental work on transmission stage survivability and infectivity in aquatic environments will be necessary before we can make accurate predictive models of parasite spread in these ecosystems.

Microparasites are a common component of freshwater plankton communities and their diversity, particularly in larger host taxa, e.g., *Daphnia* or *Asterionella*, can be remarkably high (Sparrow 1960; Green 1974; Ebert 2005; Kagami et al. 2007). *Daphnia* represents one of the most commonly investigated hosts, with studies documenting anywhere from 16 to 20 epiparasitic and endoparasitic taxa infecting a single population or metapopulation (Stirnadel and Ebert 1997; Ebert et al. 2001). The full extent of this diversity has only recently begun to be appreciated through advances in molecular systematics, particularly of those parasites belonging to the Phyla Oomycota and Chytridiomycota (Lefevre et al. 2007; Wolinska et al. 2009). Although there exists a large number of taxa capable of infecting plankton species, most populations coexist with only a small subset of their potential parasites (Ebert et al. 2001) and some populations appear not to harbour any parasites at all. In Lake Brienz (Switzerland), for instance, a comprehensive survey over 2 yr found no parasites within its *Daphnia* population, despite infections occurring in the surrounding lakes (Wolinska et al. 2007). Similar landscape scale studies (e.g., Gaiser and Bachmann 1993; Schoebel et al. 2013; Goren and Ben-Ami 2013) have found that the distributions of plankton parasites are not strongly dependent on their host range, but point to a more complex relationship than that predicted by simple epidemiological models.

A number of ecological factors have been proposed to explain the variation in the presence or absence of specific parasite taxa as well as changes in parasite prevalence and duration of epidemics among lakes. Hall et al. (2010) outlined three overlapping routes in which among-lakes differences may affect rates of parasitism. These include differences in habitat quality, through its influence on growth and survival of both the host and parasite, changes in community structure (e.g., competitors, predators) and physical habitat differences that could promote the transfer of free-living (transmission) parasite stages. Transmission to a new host is a fundamental challenge for parasites and the success of transmission stages is a key determinant of parasite fitness (Anderson and May 1979). In the aquatic environment, parasites infect new hosts using either immobile or mobile transmission stages. Immobile stages drift in the water column until they are ingested by a susceptible host and can remain infective for months, especially at cold temperatures (Decaestecker et al. 2004), while mobile stages actively swim through the water column in search of a host and are generally shorter lived (Sparrow 1960). Parasite transmission stages are highly vulnerable; grazing, temperature, irradiance, and UV radiation have all been implicated as

Additional Supporting Information may be found in the online version of this article

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factors which can significantly affect their survival (*see* reviews by Pietrock and Marcogliese 2003 and Gleason and Lilje 2009).

Within lakes, annual and seasonal parasite dynamics are common and are thought to be driven by environmental variables which can affect transmission stage survival as well as encounter rates with susceptible hosts (Cáceres et al. 2006). These seasonal drivers include factors such as temperature and lake stratification as well as host density (Doggett and Porter 1996; Perez-Martinez et al. 2001; Vale et al. 2008). For instance, in Lake Maarseveen (The Netherlands), the severity of infection of the diatom *Asterionella* by a chytrid fungus was shown to be lower in years with mild winters, which reduced *Asterionella* densities, suggesting a tight coupling between host and parasite densities at the lake scale (Ibelings et al. 2011). A similar relationship was not evident in *Daphnia* parasitized by the fungus *Metschnikowia bicuspidata* in lakes in southern Michigan (U.S.A.), where interactions between lake morphology and late summer cooling resulted in large fluctuations in maximum prevalence irrespective of host density (Cáceres et al. 2006). Relationships between seasonal changes in host density and parasite prevalence, intensity, and diversity have been shown to be quite variable and may be modulated by differences in parasite life history strategies (e.g., Cote and Poulin 1995; Morand and Poulin 1998; Arneberg 2001).

Ecological patterns are dependent on the spatial and temporal scale at which they are viewed, and patterns in parasite prevalence are no different (e.g., Duffy et al. 2010; Thieltges et al. 2013). Combining studies from a number of different regions can be informative because, although rates of parasitism are proximately driven by local environmental conditions, ultimately those factors may be correlated over large spatial scales (Byers et al. 2008). Using a cross-continental dataset of trematode infections in marine gastropods, Thieltges et al. (2013) were able to establish a strong positive relationship between local prevalence and the geographical distribution of trematodes. In addition, they were able to identify the type of definitive host (fish vs. bird) as a significant driver of this relationship, pointing to a stronger role of definitive host dispersal than local environmental factors in shaping the biogeographic distributions of these parasites. More studies such as these are needed to resolve the role of local and regional processes in explaining global patterns in parasite distribution and prevalence.

The primary objective of this study was to examine large-scale patterns of parasitism in planktonic communities and how these patterns vary among different hosts and parasite species. To do this, a large dataset of prevalence data was compiled from a number of studies representing a diverse range of hosts, parasite types, and waterbodies. Our first objective was to examine within-lake patterns of parasite prevalence; e.g., relationships with host density, seasonality, and annual variation in maximum prevalence. We expected that parasites with mobile transmission stages would show a stronger relationship with host density relative to parasites with immobile transmission stages. Our second objective was to examine the relative contribution of among-waterbody (e.g., size and trophic status) and within-waterbody factors to variation in maximum parasite prevalence and how this may differ relative to parasite life history strategies using methods similar to those of Duffy et al. (2010). We expected to see higher parasite prevalence in shallower waterbodies due to a reduction in barriers to parasite transmission (e.g., parasite spores lost to the hypolimnion). By revealing patterns in prevalence across such a broad spatial scale and encompassing a diversity of parasites and hosts, we offer general insights into the most likely drivers of parasite prevalence in planktonic organisms.

Methods

We searched Web of Science and Aquatic Sciences and Fisheries Abstract Database (and the references cited therein) for studies that examined the influence of parasitism on zooplankton and phytoplankton populations. All relevant combinations of the search term "parasite" with one host specific term (zooplankton, phytoplankton, cladoceran, copepod, rotifer, algae, cyanobacteria, diatom, and dinoflagellate) and one parasite specific term (epibiont, bacteria, protozoa, fungi, microsporidia, chytrid) were used across multiple searches. We included studies considering either internal or external parasites, including epibionts which do not feed directly on the host, but use the surface of the host as a living environment. Although they do not cause direct harm, epibionts can have effects due to increased drag, respiration, and/or predation (Willey et al. 1990; Allen et al. 1993). They also utilize zooplankton hosts in the same manner, occupying their plankton host for growth and reproduction, spreading throughout the population by way of free-living transmission stages (Threlkeld et al. 1993). After elimination of irrelevant hits, the searches yielded 103 studies. These studies were examined individually, of which 66 met our inclusion criterion (the reporting of maximum prevalence, summarized below).

We compiled estimates of parasite prevalence from plankton samples that were used in the linear mixed models described below. Prevalence estimates were reported in a number of ways, typically the number of infected individuals of a specified host taxon in a single sample (or a number of pooled samples), often with a minimum number of hosts examined per sample. We only included studies that allowed us to estimate maximum prevalence, either by collecting multiple samples over the course of a season or a year or by reporting maximum prevalence directly (e.g., Canter and Lund 1953; Duffy et al. 2010). Most studies sampled the same waterbody over multiple years and, therefore, we have more than one estimate of maximum prevalence for these

Table 1. Description of predictor variables used in the linear mixed models

Variable	Description
Parasite descriptors	
Transmission	Immobile, Mobile
Parasite Taxa	Epibiont, *Pasteuria, Spirobacillus, Caullerya*, Microsporidia, *Metschnikowia*, Chytrid, Oomycete
Host descriptors	
Host Taxa	Cladoceran, Copepod, Cyanobacteria, Diatom, Algae, Dinoflagellate
Lake descriptors	
Trophic status	Eutrophic, Mesotrophic, Oligotrophic
Maximum depth	Continuous
Surface area	Continuous
Temporal descriptors	
Season	Spring, Summer, Autumn, Winter
Year	Nested random effect

waterbodies. Maximum prevalence was chosen as it can represent how successful a parasite is in occupying all the available habitat patches (i.e., hosts) available, it is a commonly reported metric, and it can be compared between studies. We also compiled data on the strength of the relationship between parasite prevalence and host density from a set of studies that provided a measure of this relationship in the form of a correlation coefficient (r) or provided sufficient data to calculate this coefficient. Some of the studies provided data only in figure format. In these cases, we used digitizing software (GetData Graph Digitizer ver. 2.24; http://www.getdata-graph-digitizer.com/index.php) to obtain both parasite prevalence and host density estimates.

For each prevalence estimate from a plankton sample, parasite and host taxonomy were recorded to the lowest taxonomic level provided by the study. For zooplankton hosts, taxa were grouped into cladocerans or copepods. Phytoplankton hosts were categorized as cyanobacteria, diatoms, dinoflagellates, or algae (encompassing green, brown, and golden algae). Parasite taxa were grouped into eight categories (Table 1) with the goal of keeping taxa separate when large differences in virulence or ecology are known (e.g., among bacterial taxa) while grouping taxa into appropriate taxonomic units to maintain adequate sample sizes, particularly when virulence differences are negligible or not published (e.g., microsporidians). Due to the large number of taxonomic groups present among epibiont taxa, they were not subdivided further. We also grouped parasites based on their transmission mode; those with immobile infectious stages (represented by *Spirobacillus, Pasteuria, Caullerya, Metschnikowia*, microsporidians, and diatom epibionts) and those with mobile infectious stages (represented by chytrids, oomycetes, all other epibionts).

General waterbody descriptors (maximum and mean depth, surface area, stratification, hydroperiod, trophic status, geographic coordinates) were also recorded (*see* Supporting Information Table A1). When these variables were not given in the original study, they were obtained from online databases (e.g., North Temperate Lakes Long Term Ecological Research database) or other published studies. An important objective of this study was to look for general characteristics that would explain differences in parasite prevalence among waterbodies. After some preliminary analyses, we decided to focus on three waterbody descriptors (Table 1); maximum depth, surface area, and trophic status, which are commonly recorded in limnological studies and have been hypothesized to affect parasite prevalence. We focused on predictor variables that would remain relatively constant over time, and so water chemistry variables were not included. Geographic coordinates were not included in the analyses because there was distinct clustering in the distribution of the data (Fig. 1). As our dataset included data from both the Northern Hemisphere (NH) and Southern Hemispheres (SH), sampling month as a predictor was not informative. Therefore, the month in which maximum prevalence was observed was classified into 1 of 4 seasons; (1) Spring; March to May (NH) or September to November (SH), (2) Summer; June to August (NH) or December to February (SH), (3) Autumn; September to November (NH) or March to May (SH), and (4) Winter; December to February (NH) or June to August (SH).

Data analysis

Due to the spatial and temporal nonindependence within the dataset (several samples representing the same host-parasite pairs, and multiple years per waterbody), we used hierarchical (multilevel) linear mixed effect models (LME) to account for this (described in more detail in the Supporting Information). Our first objective was to examine the relative contribution of among-lake and within-lake factors to variation in maximum parasite prevalence. We created an LME model with an intercept-only fixed effect term and parasite group and waterbody identity as nested random factors to quantify this variation at multiple levels: among parasite types, among waterbodies within each parasite type, and within each waterbody (residual variation, which can represent variation among hosts or among year within a waterbody). As not every waterbody had multi-year data and multihost data, we adopted a dual modeling approach. First, to look at within waterbody differences in prevalence due to year effects, only waterbodies with multi-year data were used in the LME. Second, this was repeated for waterbodies represented by multiple hosts. Variance at each scale was converted to a percentage of the total variance to allow for comparison between the different subsets of the data.

We looked at specific waterbody factors as drivers of differences in maximum prevalence after accounting for the nested structure of the data. To accomplish this, maximum

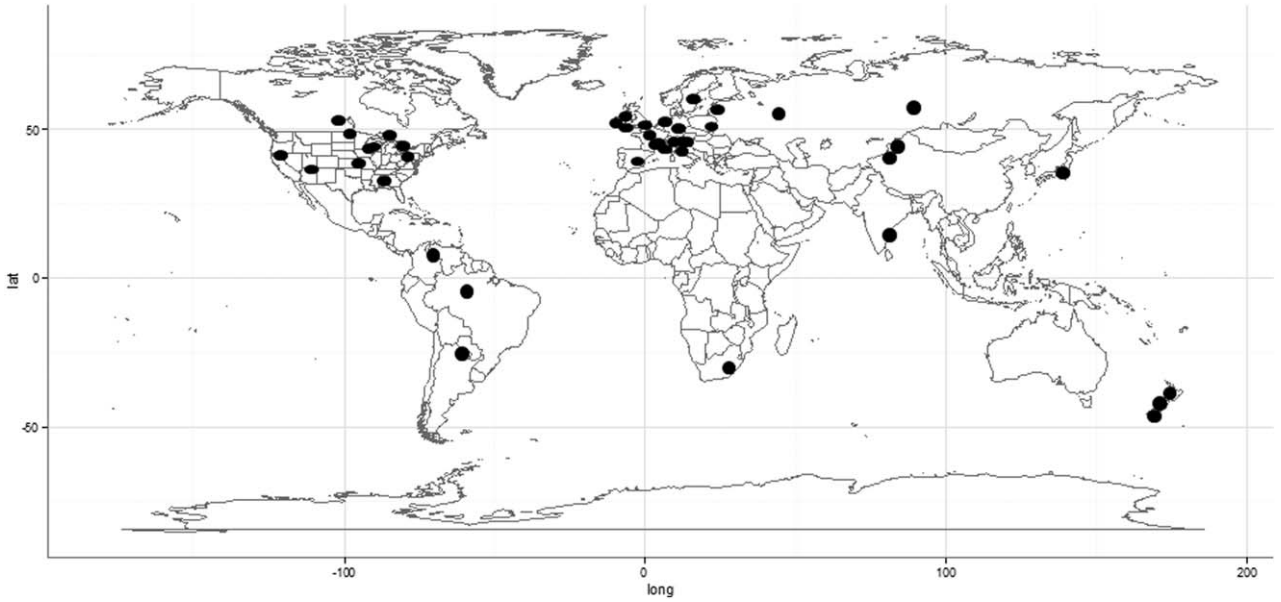

Fig. 1. The distribution of the lakes represented by the 66 studies used in the analyses. With the exception of the United Kingdom (13 studies) and the U.S.A. (18 studies), the remaining 20 countries were represented by less than then 4 studies.

depth, surface area, and trophic status were included as fixed factors in a LME on the full dataset. Parasite group and waterbody remained as random factors. Both random and fixed effects were tested by single term deletion with subsequent model comparison. The depleted model was then compared with the full model using an *F*-test of the likelihood ratios for the LME's. Restricted maximum likelihood was used to compare nested models in which only the random effects differed and maximum likelihood was used when comparing nested models where the fixed effects differed (Pinheiro and Bates 2000). We also included the Pearson product moment correlation coefficient (*r*) between host density and parasite prevalence in a subsequent set of LME's to determine if any of the waterbody factors could explain the variation in this relationship. Prior to analysis, effect sizes were transformed using the Fisher's *Z* transformation to account for differences in sample sizes. Not enough studies reported lake surface area, and, therefore, this variable was not included.

As we also wanted to examine the potential for differential responses by parasites with immobile vs. mobile transmission stages, we achieved this by splitting the data into two sets according to transmission mode (immobile, mobile) and re-running the models to highlight the differences between these two modes of infection. For all analyses we used the lmer program (lme4 package; Bates and Sarkar 2006) in the R system for statistical computing (R Development Core Team 2013). As most studies did not provide sample sizes (i.e., number of individual hosts examined per sample), providing only calculated prevalence values, we were unable to explore the underlying binomial sampling process that characterizes prevalence estimates, and, therefore, LME's using logit-transformed prevalence data and assuming a normal distribution were used throughout.

Results

A total of 66 studies published between 1948 and 2013 were included, which represented plankton populations from 118 waterbodies from across the globe, although temperate lakes of the U.S.A. and Great Britain were disproportionately represented (Fig. 1). Waterbody types ranged from small rockpools, alpine ponds, sewage lagoons, and farm ponds to hydroelectric reservoirs and large lakes. The ranges in physicochemical parameters are included in Supporting Information. A total of 45 unique parasite taxa were reported (Table 2), covering four Kingdoms (Bacteria, Protozoa, Chromista, and Fungi). Among zooplankton hosts, microsporidians, and oomycetes were the most commonly reported parasites in this dataset. A bias towards *Daphnia* hosts was evident (59% of host-parasite pairs). Among phytoplankton taxa, parasites consisted solely of chytrids (Phylum Chytridiomycota) and oomycetes (Phylum Oomycota). As hosts, diatoms, in particular *Asterionella*, were the most common taxa examined, although green and golden algae, cyanobacteria, and dinoflagellates were also represented. In total, the present analyses are based on 482 samples, each representing a different host-parasite-waterbody combination.

Within lake differences: Host density and seasonality

We obtained 94 correlation coefficients illustrating the linear relationship between host density and parasite prevalence for a number of different host-parasite pairs. The

Table 2. Summary of the parasite groups included in the analyses and their ranges in maximum prevalence. Epibiont taxa were not subdivided in the analyses. The list of hosts is not exhaustive but includes those with enough data to calculate maximum prevalence

Parasite Type	Phylum (Phyla)	Genera	Hosts	Prevalence
Zooplankton Epibionts				
Ellobiopsids	Myzozoa	*Ellobiopsis*	Copepods	17–25.5%
Ciliates	Ciliophora	*Tokophyra, Epistylis, Carchesium, Vortichella*	*Alona*, Copepods, *Daphnia, Diaphanosoma*	0.5–100%
Photoautotrophs	Ochrophyta, Euglenoza, Chlorophyta	*Synedra, Characidiopsis, Colacium, Amoebidium*	Calanoid, *Daphnia*	2.2–100%
Zooplankton Endoparasites				
Bacteria	Firmicutes	*Pasteuria*	*Daphnia*	0.1–100%
	Proteobacteria	*Spirobacillus*	*Daphnia*	0.19–12%
Protozoans	Choanozoa	*Caullerya*	*Daphnia*	2.8–50%
	Amobozoa	*Pansporella*	*Sida, Simocephalus*	2.2–5.4%
Dinoflagellates	Myzozoa	Class Syndinea	Calanoids	2.5–26%
Microsporidia	Microsporidia	*Glugoides, Thelohania, Larssonia, Binucleata, Octosporea, Amblyospora, Hyalinocyst*	Cyclopoids, *Daphnia*, Rotifers	0.04–100%
Yeast Fungi	Ascomycota	*Metschnikowia*	*Daphnia*	0.18–41.6%
Chytrids	Chytridiomycota	*Agglomerata, Olpidium, Polycaryum*	*Daphnia*, Rotifera	0.5–41%
Oomycetes	Oomycota	*Aphanomyces, Blastulidium, Lagenidium, Pythium, Saprolegnia*	Calanoids, *Daphnia, Chydorus*, Rotifers	0.8–89%
Phytoplankton Endoparasites				
Chytrids	Chytridiomycota	*Chytriomyces, Chytridium, Rhizidium Rhizophydium, Rhizosiphon, Zygorhizidium*	Algae, Cyanobacteria, Diatoms, Dinoflagellate	1.4–100%
Oomycetes	Oomycota	*Aphanomycopsis*	Dinoflagellate	80–87%

relationship varied considerably, from highly significant (in both the positive and negative direction) to no relationship. The mean correlation coefficient across all relationships was positive ($\bar{r} = 0{\cdot}15$). The weighted-mean effect size, which accounted for differences in sample size, was $Z_r = 0.18$. The homogeneity of the weighted effect sizes among studies was tested with the Q test, which indicated heterogeneity across the studies (lakes) ($Q = 372.8$, $N = -93$, $p < 0.001$, critical $x^2 = 139.7$). This indicated significant differences among the relationships between host density and prevalence. We compared the responses of the two transmission modes (immobile vs. mobile) and found that for parasites with mobile transmission stages, typically chytrid fungi and their phytoplankton hosts, parasite prevalence was positively associated with host density ($Z_r = 0.27$, Fig. 2). For parasites with immobile transmission stages, particularly *Metschnikowia* and bacteria and protozoan parasites (e.g., *Caullerya, Pasteuria, Spirobacillus*) in *Daphnia*, prevalence was often negatively associated with host density ($Z_r = -0.26$, Fig. 2)

Plankton sampling was often restricted to the ice-free season in north temperate lakes (May through October), however, seasonal differences in prevalence among parasite groups were apparent (Fig. 3), with many parasite groups favoring a particular season. The bacterial parasites, *Spirobacillus* and *Pasteuria*, always reached their highest prevalence in summer. *Caullerya* and *Metschnikowia* peaked much later in the year, typically in the autumn, or in the case of

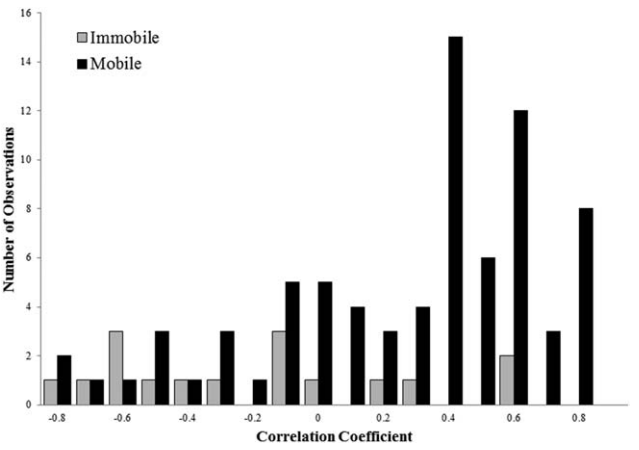

Fig. 2. Frequency distribution of the correlation coefficient between host density and intensity of parasite prevalence ($n = 94$). Parasites with immobile transmission stages (white bars) and those with mobile transmission stages (black bars) are displayed separately.

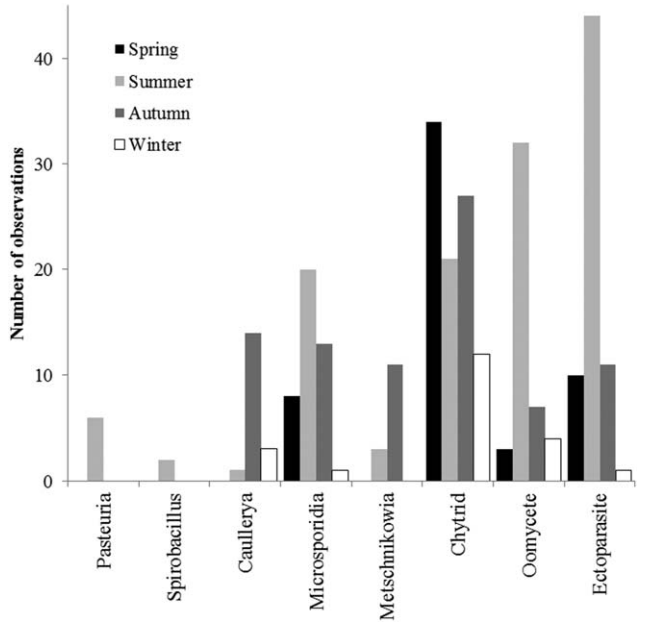

Fig. 3. Seasonal distribution of maximum prevalence observations for each parasite group.

Caullerya, into the winter. Both oomycetes and epibiont taxa often reached their highest infections rates in the summer. Microsporidians were seasonally variable, with maximum prevalence recorded in most seasons. Maximum prevalence of chytrid parasites occurred in every season and prevalence often peaked a number of times throughout the year. Seasonal patterns in prevalence appeared to be temporally stable, however, there was not enough data to examine long term seasonal trends.

Among lake differences: Parasites and hosts

Maximal prevalence was highly variable, both within and between parasite groups (Table 2).Variation between parasite groups was significant ($p < 0.01$), accounting for 30% of the total variance explained (Fig. 4). Among these groups, the highest maximum prevalence values were observed in zooplankton hosts infected by the bacterium *Pasteuria*, microsporidians and epibiotic taxa as well as chytrids infecting phytoplankton hosts. Infection levels by these parasites regularly reached 100% in their host population. Slightly more moderate infection rates were observed in hosts parasitized by *Metschnikowia* (41.6%) and *Caullerya* (50%). Oomycete infections were also high (89%), but only in copepods hosts. In contrast, maximum prevalence of the bacterium *Spirobacillus* was always low ($< 10\%$), with prevalence in most waterbodies never exceeding 1%. Variation among the different waterbodies within each parasite group was also significant ($p < 0.01$), accounting for 35% of the total variance explained. Residual variation, representing differences in prevalence within each waterbody, was 26%. This variation represents differences in maximum prevalence among host types and among different sampling years. We explored these differences further by restricting the dataset to only those waterbodies with data for multiple host species or multiple years. The variation represented by these two levels were similar (30% and 26%), both exceeding the variation among different parasite groups.

To compare the responses of the two transmission modes (immobile vs. mobile), we looked at the variation within each group separately (Fig. 4). Maximal prevalence of parasites with immobile transmission stages varied considerably among waterbodies within each parasite type. A smaller amount of the variance was represented by parasite group (19%) and within waterbody variation (15%). Among parasites with mobile infectious stages, the variance explained by waterbody identity was much lower (32.4%), but still significant, with most of the variation contained at the within-waterbody scale. There was no evidence that differences between parasite types contributed to the observed variation in maximal prevalence as its inclusion in the multi-level model was not significant.

Among lake differences: Lake characteristics

Of the three predictor variables included in the LME as fixed factors (maximum depth, surface area, and trophic status), only maximum depth was found to be a significant predictor of maximum prevalence ($p = 0.009$), but only for parasites with immobile transmission stages (Table 3). Shallower waterbodies had higher maximum prevalence than deeper waterbodies (Fig. 5). This was most evident in zooplankton communities infected by microsporidians and the bacterial parasite *Pasteuria*. This effect was more striking in

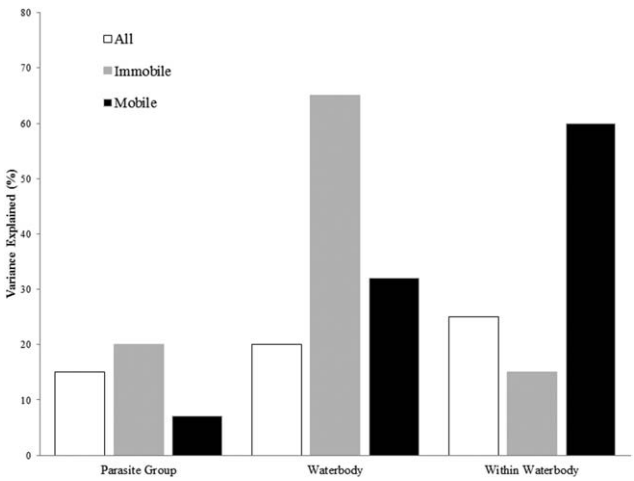

Fig. 4. Variance in maximum parasite prevalence as a percentage of total variance explained for each level in hierarchical linear mixed effect models: parasite group, waterbody identity (nested within parasite group) and within waterbody (residual) variation. Values are derived from hierarchical linear mixed effect models run for the entire dataset (white bars) and for each transmission mode, immobile (black bars) and mobile (gray bars) separately.

Table 3. Results of linear mixed-effect analyses investigating the determinants of (1) maximum prevalence and (2) correlation between host density and prevalence (Z_r) in planktonic host populations. Logit-transformed prevalence values were modeled as a function of three fixed effects: maximum depth, surface area, and trophic status index (TSI). Weighted mean-effect sizes (Z_r) between host density and parasite prevalence were modeled as a function of two fixed effects: maximum depth and TSI. Waterbody identity was treated as a random effect, nested within each of the eight parasite groups. Likelihood ratio (LR) tests were used to assess significance of the fixed effects. Significant effects are denoted with an asterisk ($p < 0.001$)

	All data		Immobile		Mobile	
Max prevalence	***t* Value**	**LRχ^2**	***t* Value**	**LRχ^2**	***t* Value**	**LRχ^2**
Fixed effects						
Maximum depth	−0.72	0.57	−3.95	13.57*	1.49	1.86
Surface area	0.16	0.04	0.92	1.09	−0.98	0.91
TSI - Eutrophic	−2.89	3.06	0.78		0.49	1.77
TSI - Mesotrophic	−2.30		−0.54		−0.04	
TSI - Oligotrophic	−2.37		−0.66		−0.03	
Random effects	Variance	SD	Variance	SD	Variance	SD
Parasite	0.09	0.30	0.04	0.21	0.16	0.07
Parasite/Waterbody	0.12	0.35	0.11	0.33	0.13	0.37
Correlation (Z_r)	*t* Value	LRχ^2	*t* Value	LRχ^2	*t* Value	LRχ^2
Fixed effects						
Maximum depth	−1.35	1.13	−0.27	1.40	−1.07	1.04
TSI - Eutrophic	−1.50	5.12	−0.94	1.22	−1.45	4.15
TSI - Mesotrophic	1.58		0.22		1.68	
TSI - Oligotrophic	0.69		0.06		0.98	
Random effects	Variance	SD	Variance	SD	Variance	SD
Parasite	0.07	0.26	<0.01	<0.01	0.01	0.01
Parasite/Waterbody	0.03	0.18	<0.01	<0.01	0.13	0.36

communities infected by *Pasteuria* (waterbodies < 4 m = 32–100%, waterbodies > 4 m = 0.2–18%) and microsporidians (waterbodies < 4 m = 3–100%, waterbodies > 4 m = 0.5–21.5%) Among those with mobile infectious stages (chytrids, oomycetes, epibionts), none of the predictor variables were able to explain the large variation in prevalence that occurred within these taxa.

We also included the weighted correlation coefficient (Z_r) as a response variable in a LME to determine if lake specific characteristics may influence the strength of this relationship. As lake area was missing from a number of these studies, we were only able to examine the effects of lake depth and lake trophic status. However, none of these variables showed a significant effect on the host density—prevalence relationship.

Discussion

Host-parasite dynamics are highly variable, with host populations exhibiting marked variation in prevalence across space and time. This variation in prevalence has been linked to differences in parasite virulence (Tack et al. 2012), host density (Arneberg 2001), host susceptibility (Ganz and Ebert 2010), and environmental variability through its effects on both parasite growth rate and host demography (Duncan et al. 2013). In this study, we found that the largest source of variance in maximum prevalence within our dataset was among different waterbodies, rather than among different parasite types, years, or hosts. Although local and regional differences in prevalence have been observed in other studies (e.g., Canter and Lund 1953; Ibelings et al. 2011; Wolinska et al. 2011), it has only been quantitatively addressed by Duffy et al. (2010). These authors demonstrated that differences in host susceptibility and habitat characteristics (among-lake differences) are responsible for the majority of the variation in their multi-parasite system. As the maximal infection prevalences of the various parasite species were correlated, at least for one host species, their results suggested that different parasite types may be responding similarly to the same lake characteristics. Although our results generally agree, the inclusion of a greater range of parasites revealed that the differences were not consistent among all parasites.

Density-dependent transmission has been shown in the laboratory for a number of *Daphnia* species (Ebert 1995; Bittner et al. 2002) but it is rarely shown in the field (Ebert 1995). We found density-dependent responses in prevalence for phytoplankton and their chytrid parasites, as well as epibionts and oomycete parasites of zooplankton. Yet there was

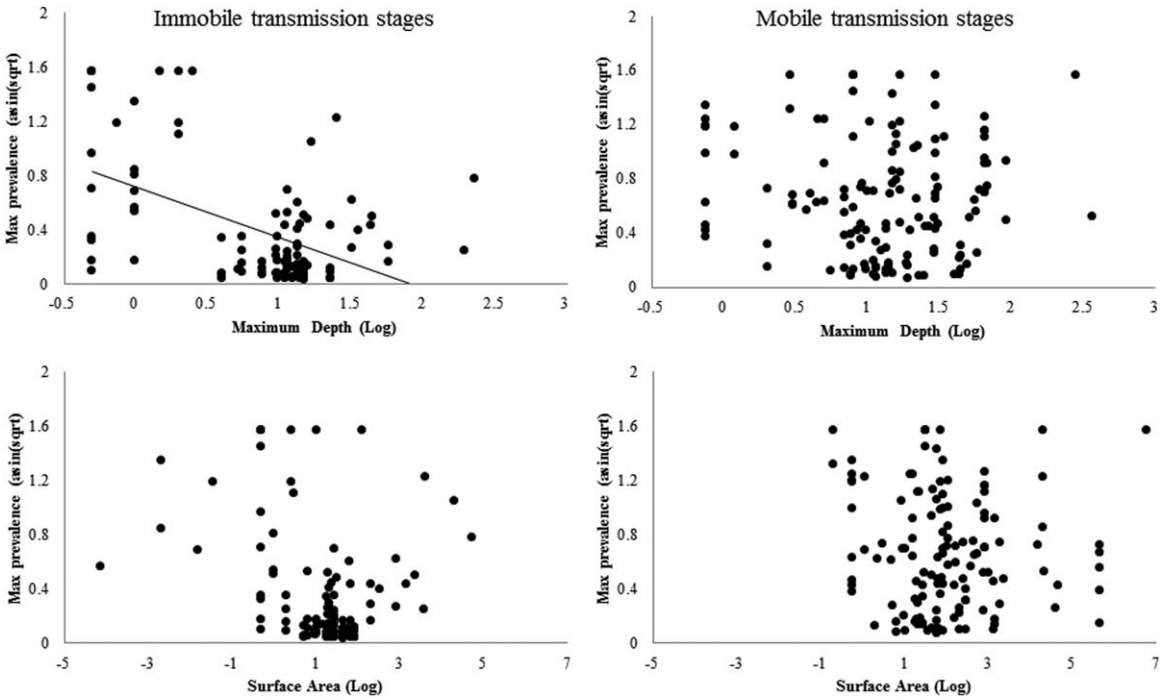

Fig. 5. Relationships between maximum depth and surface area (log transformed) in each waterbody and the logit transformed maximum prevalence for parasites of planktonic populations, shown separately for those with immobile and mobile transmission stages (*see* Table 2 for a full list of these parasite taxa).

no increase in parasite prevalence with higher host densities for those parasites with immobile stages. It may be that lake specific characteristics play a larger role in increasing host contact for parasites with immobile transmission stages (which are also long lived). Although we were unable to explore the role of lake mixing, temperature gradients, and lake size, Hall et al. (2010) found evidence for physical mechanisms in increasing host-parasite contact in a *Daphnia-Metschnikowia* system. Conversely, turbulence may reduce the ability of mobile stages to attach to their host (Kühn and Hofmann 1999), and, therefore, parasites with this transmission strategy may be responding primarily to factors that affect the density of their host.

The significant contribution of among-waterbody differences, rather than between different parasite types, to variation in maximum prevalence was particularly evident when analyses were restricted to parasites with immobile transmission stages. The production, dispersal, and infectivity of transmission stages are key factors in the spread of parasites in a population (Anderson and May 1979). Transmission stages are exposed to a number of abiotic and biotic factors that can influence their success by altering their survival or infectivity (Thieltges et al. 2008). In the aquatic environment, transmission stages of bacteria, protozoans, and some fungal parasites drift in the water column until they are ingested by a host and are dependent on the external environment to ensure host contact. Among-lake differences in transmission rates have been attributed to elevation, through its effect on temperature and, therefore, infectivity (Wolinska et al. 2007), fish community composition through its role in selective predation of parasitized individuals (Willey et al. 1993; Duffy et al. 2005), and hypolimnion size and stratification strength which affect the abundance of diluters (less competent hosts which reduce infection in focal hosts) and predators (Penczykowski et al. 2014). We found that the majority of variation in maximum prevalence among parasites with immobile transmission stages was between different waterbodies, indicating a prominent role for among-lake factors, such as thermal regimes, community composition, and lake morphology in shaping host-parasite dynamics for parasites exhibiting this transmission strategy.

To assess specific waterbody characteristics that may be responsible for differences in prevalence, we looked at two aspects of waterbody size, maximum depth and surface area, and trophic status, which is a general measure of habitat productivity. We found that prevalence increased with decreasing waterbody depth, although only among parasites with immobile transmission stages. A number of causal mechanisms have been offered to explain how waterbody morphology may alter host exposure to parasite transmission stages. For many parasites (e.g., *Spirobacillus, Metschnikowia*), infectious stages are released from dead hosts, congregating at the sediment-water interface after host decomposition (Thomas et al. 2011), therefore, requiring

turbulence to reconnect them with living hosts in the water column (Cáceres et al. 2006). Turbulence is affected by depth, surface area, and basin shape (Imboden and Wuest 1995). Sediments of shallow habitats are subject to more wind induced turbulence, ensuring transmission stages are in constant contact with hosts, and temperatures are higher, which may result in longer periods of infectivity and higher overall prevalence. In deeper habitats, which stratify for a portion of the year, transmission stages trapped in the sediment will only be suspended during lake turnover or mixing, which is thought to explain the seasonality of many parasite epidemics (Bittner et al. 2002; Cáceres et al. 2006). Mixing between nearshore and offshore areas also occur due to gravity currents resulting from temperature gradients. These currents are faster and stronger in waterbodies with steeply declining littoral zones (Hall et al. 2010). Although we were unable to examine the mechanisms responsible for the strong relationship between maximum prevalence and depth, the low prevalence of microsporidians and *Pasteuria* infections in deeper waterbodies was striking. Are there shallow habitats in which *Pasteuria* remains at low prevalence or alternatively, do *Pasteuria* infections reach host saturation in lakes?

Parasites with mobile transmission stages do not necessarily respond similarly to differences in morphology and mixing. For instance, chytrid infections in *Daphnia* reached peak prevalence under the ice (Johnson et al. 2006a), while oomycetes and epibionts often peak in the summer months (Dubovskaya et al. 2005; Wolinska et al. 2011), both periods of waterbody stability. For these parasites, turbulence can reduce prevalence by interrupting the chemosensory host-finding behavior of these mobile stages (Kühn and Hofmann 1999). However, mixing can resuspend resting spores of chytrids and oomycetes, and, therefore, may be more important for initiating, not maintaining, infections (Doggett and Porter 1996). Most evidence has shown that transmission of many of these parasites is significantly related to host density (Prasad et al. 1989; Møhlenberg and Kaas 1990; Chiavelli et al. 1993; Ibelings et al. 2011), and the short-lived nature of their transmission stages may explain the tight coupling between host density and prevalence. Among-lake differences may be manifested through factors that affect transmission success over short time scales, such as light levels (Johnson et al. 2006b), which affect motility and host recognition of transmission stages (Bruning 1991). Oomycete and chytrid taxa remain poorly studied in zooplankton populations, despite their high diversity (Wolinska et al. 2009) and potential for significant regulatory effects (Burns 1985; Johnson et al. 2006b).

Although we have demonstrated that the prominent depth-prevalence relationships seen in other systems extend to a variety of host-parasite combinations, we reveal that these relationships are not consistent across all parasite taxa. Parasites with mobile transmission stages appear to exhibit more within-lake variability, which may be explained by their strong relationship with host density and climatic variables that affect host succession (Ibelings et al. 2011). The smaller amount of variation explained by both host as well as year effects may result from our omission of zero prevalence reports due to few studies reporting an absence of parasitism. However, as Jovani and Tella (2006) point out, a zero prevalence is just as informative if calculated using appropriate sample sizes. By excluding zero prevalences we are omitting potential habitats with characteristics that prevent parasite establishment. For example, Ibelings et al. (2011) found that chytrid parasitism in *Asterionella* populations of Lake Maarseveen was temporally stable over a period of almost 30 years, occurring with high prevalence every single year. Yet, many other parasites (i.e., *Caullerya*; Schoebel et al. 2013) do not appear to be present every year. In addition to its exclusion of zero prevalence values, our dataset has other limitations. For instance, because most samples are clustered within a narrow latitudinal band in the northern temperate zone, it was not possible to test for latitudinal gradients in parasitism. Furthermore, our dataset did not include reports of parasitism of zooplankton by the juvenile stages of helminth parasites, like cestodes. However, the few available estimates of prevalence by these parasites in freshwater copepods indicate that they typically infect less than 1% of the host population (Marcogliese 1995), suggesting that they have little impact on host population dynamics.

Despite these limitations, our analysis nonetheless provides a clear and strong signal indicating that most of the variance among plankton populations in the prevalence of parasites is accounted by local factors associated with differences in the type of waterbody occupied. We have also identified one key factor (lake depth) influencing the prevalence of infection across various taxa of host and parasites, though only for those with immobile infectious stages. This supports the observation made by Cáceres et al (2014) that not all diseases seem to require mixing for epidemics to begin. Here, we build on that observation by establishing that transmission stage ecology is essential for understanding the host-parasite dynamics in aquatic systems. Our analysis sought global drivers of parasite infection in planktonic organisms; we uncovered one whose influence emerges above the strong idiosyncratic effects of local factors. In order to understand the role of parasites in aquatic food webs, particularly in influencing succession and altering energy flows (e.g., the Plankton Ecology Group model; Sommer et al. 2012), we need basic knowledge on the ecology of transmission stages, i.e., their longevity, their abundance in the environment, and how abundance of transmission stages actually relates to prevalence of infection in host populations. As a way forward, we recommend greater study of multi-parasites systems to determine how different parasites respond to the same local conditions.

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