Do Animals Living in Larger Groups Experience Greater Parasitism? A Meta-Analysis

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ABSTRACT: Parasitism is widely viewed as the primary cost of sociality and a constraint on group size, yet studies report varied associations between group size and parasitism. Using the largest database of its kind, we performed a meta-analysis of 69 studies of the relationship between group size and parasite risk, as measured by parasitism and immune defenses. We predicted a positive correlation between group size and parasitism with organisms that show contagious and environmental transmission and a negative correlation for searching parasites, parasitoids, and possibly vector-borne parasites (on the basis of the encounter-dilution effect). Overall, we found a positive effect of group size ($r = 0.187$) that varied in magnitude across transmission modes and measures of parasite risk, with only weak indications of publication bias. Among different groups of hosts, we found a stronger relationship between group size and parasite risk in birds than in mammals, which may be driven by ecological and social factors. A metaregression showed that effect sizes increased with maximum group size. Phylogenetic meta-analyses revealed no evidence for phylogenetic signal in the strength of the group size–parasitism relationship. We conclude that group size is a weak predictor of parasite risk except in species that live in large aggregations, such as colonial birds, in which effect sizes are larger.

Keywords: sociality, group size, parasitism, phylogenetic meta-analysis, phylogeny.

Introduction

Levels of infestation and infection by parasites differ among social groups, populations, and species, and researchers have long been interested in understanding the factors that account for variation in parasitism (Freeland 1979; Brown and Brown 1986; Poulin 1995; Nunn and Altizer 2006). A prominent hypothesis offered to explain this variation postulates a link between parasitism and gregariousness (Freeland 1979; Møller et al. 1993; Loehle

1995; Altizer et al. 2003). Living in a larger group may increase parasitism through several mechanisms. For example, group size has been used as a proxy for local density, which favors the establishment of infections (Anderson and May 1982). Animals living in larger groups may also interact more with conspecifics, resulting in more opportunities for parasite transmission (e.g., grooming; Dunbar 1991). Larger groups may receive and produce more migrants that carry infectious disease (Brown and Brown 2004), and larger groups may attract more vectors, resulting in a greater proportion of individuals being infected with vector-borne parasites (i.e., higher prevalence; Davies et al. 1991; Tella 2002; Nunn and Heymann 2005). Finally, individuals in larger groups often have greater day ranges or use their ranges more intensively and may therefore experience greater infection risk from parasites that are transmitted through feces (e.g., intestinal protozoa; Freeland 1979; Nunn and Dokey 2006; Nunn et al. 2011).

In some cases, however, larger groups may also protect individuals from parasites. For example, the encounter-dilution effect may protect individuals living in a larger group from attack by mobile parasites. This effect arises when the probability that a searching parasite detects a group fails to increase proportionally with group size and the searching parasite does not attack proportionally more hosts in a larger group (Mooring and Hart 1992). Under these circumstances, prey in groups may experience "safety in numbers" from attacks by these searching parasites and the infectious agents that they vector.

On the basis of these theoretical arguments, we expect to find associations between group size and levels of parasite infection that depend on transmission mode. In the case of socially transmitted infectious agents, individuals in larger groups are predicted to harbor greater numbers of parasites, and larger groups are likely to harbor more distinct parasites at a higher prevalence. In addition, evolutionary increases in sociality may favor stronger immune defenses by advantaging individuals who are better able

to resist infection. However, evidence for these predicted relationships is mixed. Ezenwa (2004) found that the prevalence of intestinal parasites in species of African artiodactyls increased with group size, and Whiteman and Parker (2004) demonstrated higher individual infestations of lice (*Colpocephalum turbinatum*) in larger groups of Galapagos hawks (*Buteo galapagoensis*). In addition, a recent study of primate molecular evolution found some evidence for positive selection on genes related to immunity in species living in larger groups (Wlasiuk et al. 2010). Conversely, Arnold and Lichtenstein (1993) failed to find significantly higher levels of ectoparasite infection in an intraspecific study of alpine marmots (*Marmota marmota*) living in larger groups, whereas in comparative studies of primates, group size showed no associations with estimates that reflected the efficiency of the immune system (Nunn et al. 2000; Nunn 2002; Semple et al. 2002).

From these and other studies, two important questions arise. First, on average, do measures of parasitism and immune defenses covary with group size? Second, what accounts for the varying findings across studies that involve different hosts and parasites? To investigate these questions, we performed a meta-analysis of previous studies of the association between group size and parasite risk, which refers to both measures of parasitism and immune defenses. Unlike traditional literature reviews, meta-analysis provides a quantitative approach to these questions (Arnqvist and Wooster 1995; Gates 2002; Borenstein et al. 2009), in which the outcome of each study is converted to an "effect size" and an overall effect size is calculated across studies, weighted by the precision of the study, with a confidence interval (CI) to reflect the precision of the estimate. The similarity of effect sizes across different studies can be estimated, as can the influences of covariates on the effect size.

In a previous meta-analysis, Côté and Poulin (1995) examined six studies of parasite prevalence and 15 studies of parasite intensity (the number of parasites per infected host or nesting site). The prevalence studies focused on contagious parasites, whereas the intensity studies included 10 contagious and five mobile parasites. Their meta-analysis showed a significant positive association between group size and parasite prevalence and a nonsignificant positive association between group size and parasite intensity. They also reanalyzed the intensity data after separating studies into mobile and nonmobile parasites, predicting that living in a larger group would reduce attack by mobile parasites through the encounter-dilution effect. Côté and Poulin (1995) found support for this prediction and suggested that individuals benefit from grouping to avoid attack from mobile parasites, such as biting flies, in a manner similar to the effects of grouping in reducing individual predation risk (Krause and Ruxton 2002).

In the 15 years since Côté and Poulin (1995) published their meta-analysis, disease ecology has developed significantly as a field of research (Gulland 1995; Hudson et al. 2002; Collinge and Ray 2006; Nunn and Altizer 2006). As a consequence, new questions have arisen about the mechanisms that drive associations between parasitism and sociality (Wilson et al. 2003; Bonds et al. 2005), and many new studies have been conducted to assess the links between parasite risk and group size, including both field and comparative research. Simultaneously, meta-analysis techniques have grown in sophistication, flexibility, and implementation. Methods are now available, for example, to quantify the degree to which nonsignificant findings are underreported and correct for this bias (i.e., publication bias; Borenstein et al. 2009). It is also possible to conduct meta-analyses in a phylogenetic context by assessing whether phylogenetic relatedness accounts for variation in effect sizes and, if so, to take phylogeny into account in the meta-analysis (Adams 2008; Lajeunesse 2009). These theoretical and methodological advances might be highly relevant for understanding the relationship between group size and parasite risk and, specifically, for investigating factors that explain variable findings across individual studies of different host species and parasites.

In this article, we reexamine the association between group size and parasite risk using an expanded database of 69 individual studies (compared to Côté and Poulin's 15 studies) and significantly improved meta-analysis techniques, including the ability to examine publication bias, incorporate phylogeny, and examine how additional factors influence variation in effect sizes. Specifically, we test whether studies of group size and parasitism show a positive effect size overall. We then investigate whether transmission mode accounts for heterogeneity in effect size and direction by testing the following specific predictions: (i) contagious parasites would show an overall positive association with group size, because animals in a larger group are expected to have contact with more conspecifics; (ii) searching parasites and parasitoids would show a negative association, in accordance with predictions from encounter-dilution effects (Mooring and Hart 1992; Côté and Poulin 1995); (iii) if encounter-dilution effects hold for the searching vectors, we also expect a negative association between group size and parasitism for vector-transmitted infections (otherwise, we expect to find a positive association; e.g., Davies et al. 1991; Nunn and Heymann 2005); (iv) environmentally transmitted parasite infections would increase with group size, based on the observation that larger groups have larger territories and may thus be exposed to more infectious stages of parasites, and individuals in larger groups may also be exposed to more conspecific waste products from more individuals than those in smaller groups.

In addition to these specific predictions, we also considered whether outcomes differed between host taxa, between intraspecific and comparative studies, and according to the measure of disease risk used (including different measures of parasitism). We also tested whether studies of larger aggregations, such as colonially breeding birds, showed stronger effect sizes, as might be expected if the effect size varies with group size itself. Finally, we considered the role of host phylogeny in two ways: we compared effect sizes among the major phylogenetic lineages in our database (mammals, birds, lizards, ray-finned fish, insects, and arachnids), and we conducted a phylogenetic meta-analysis by examining effect sizes at the host species level. Our data set spans a wide range of host species, and given the large difference in host ecology and life history between animal classes, different patterns may be found in different evolutionary lineages.

Material and Methods

Database: Literature Search

We obtained data through two independent systematic searches of online databases and through solicitations of pointers to published articles and unpublished data. In 2006, C. L. Nunn and L. Z. Garamszegi conducted a preliminary data search in Web of Science from 1975 to 2006 and also solicited pointers to the association between group size and parasitism on major listservs in behavior, ecology, and evolution and in major journals and newsletters of organizations in these fields. This data search was initiated through a National Evolutionary Analysis and Synthesis Center Working Group on Meta-Analyses (led by M. Noor and M. Servedio). In 2010, J. L. Rifkin searched JSTOR, Google Scholar, and ProQuest.

The searches uncovered a wide variety of approaches to investigating the links between group size and parasite risk, including experimental, observational, and comparative studies. We included studies if they contained statistical tests relating parasitism to group size or the effects of parasites on hosts, along with explicit details on the direction of the effect and sample size. When necessary statistical details were missing, authors were contacted for additional information. In five cases, studies included raw data, and from these we calculated the correlation between group size and the measure of parasite risk using a Spearman's or a Pearson's correlation in R (R Development Core Team 2010). When multiple results were available from a single study, we included all of them in a complete raw data set and used the procedures described below to combine effects in different ways.

Combining the results of these searches, a total of 69 articles were identified, with publication dates ranging from 1976 to 2010. These articles yielded 344 separate effect sizes. Sample sizes ranged from 2 to 500 social groups or species (depending on whether the study was conducted at an intraspecific or interspecific level). Our data set included approximately seven phyla of parasites (including one virus and several fungi of uncertain taxonomic placement). Among these, nine taxonomic classes were identified.

Database

We entered all statistical tests of the relationship between parasitism and group size from each study into a database. For each effect size entered, we included the identity of the study, the approach taken (intraspecific studies of groups within a species or comparative studies of different species), the host species, the host class, the parasite species, the transmission mode, whether the study was observational or experimental, maximum group size, and the response measure of parasitism.

Statistics were converted to effect sizes in the form of Hedges's g (Hedges 1981) in the program Comprehensive Meta-analysis (CMA; Borenstein 2010), which uses conventional formulas for effect size transformation (e.g., Cohen 1988). Hedges's g, a measure of bias-corrected standardized mean difference, can be used to compare binary, continuous, and correlational effect sizes in the same statistical framework and thus was appropriate for our diverse data set. Hedges's g follows a noncentral t distribution (Hedges 1981), and in our data set, it ranged from -4.65 to 4.61 with a mean at 0.48. All effect sizes were converted within CMA from Hedges's g to a correlation (r) for reporting. Because F statistics were not accepted directly by CMA, all F statistics were converted to P values from the F statistic and degrees of freedom using GraphPad P Value Calculator (http://www.graphpad.com/quickcalcs/pvalue1.cfm; accessed April 2010) and were entered into CMA using "p-value and sample size for" the relevant type of test (either correlation or independent groups). The P values in the form of "$P < x$" (e.g., "$P < .05$") were entered as "$P = x$," which risks underestimating but not exaggerating effect sizes. Nonparametric statistics were entered into CMA and treated in the same manner as parametric statistics. In all cases, we required an effect direction to include a study in the meta-analysis; increases in parasitism with increasing group size were entered as positive effects.

We coded effect sizes as intraspecific when they focused on different-sized groups of a single host species and as comparative when multiple host species were compared. Transmission modes were assigned as follows: in studies of a single parasite or a small group of similar parasites, parasites were given nonexclusive binary scores (0 or 1, representing "no" and "yes") for five transmission char-

acteristics: searching, contagious, environmentally transmitted, vector-borne, and parasitoid. Searching parasites were defined as mobile parasites that could travel between aggregations of hosts; contagious parasites required close contact to transfer between hosts and were generally restricted to within-group transmission; environmentally transmitted parasites were defined as parasites acquired from the host's abiotic environment (typically through fecal-oral transmission and from contaminated water, ground, or food); vector-borne parasites were deposited in or on their host by a mobile vector of a different species and often undergo a developmental stage in the vector; parasitoids were defined as organisms that spend their larval stage inside a host, often resulting in host death, and transmit their young to new hosts as mobile adults. Many parasites were classified into more than one overlapping category. For example, a parasite that could be transmitted by fecal contamination or direct contact would be coded as environmental and contagious. However, because we defined parasitoids as the overlap between searching and vector-borne parasites, no parasite was classified into more than one of these three categories. The binary (0 and 1) scores for these transmission categories were used to group effect sizes by parasite category for the analysis and to calculate mean effect sizes separately for each parasite category. Comparative studies of immunity received scores of 0 for all five categories and thus were not included in meta-analyses that investigated the effect of transmission modes. Similarly, when studies examined collections of parasites with variable or uncertain transmission modes, these were given a score of 0 for all categories.

Response measures included parasite abundance, intensity, prevalence, richness, and immune measures. Abundance was defined as the mean number of parasites per individual; intensity was defined as the number of parasites per infected individual; prevalence was defined as the proportion of individuals infected in a group; richness was defined as the number of parasite species in a group or individual. Several proxy measures for immune response were grouped, included swelling responses, organ sizes, and white blood cell counts, which are generally assumed to reflect the efficiency of the immune system. Summary statistics for each study and the full data set are deposited in Dryad.

Analysis

We performed all traditional meta-analysis tests in CMA (Borenstein 2010). We used a random-effects model for all tests across studies, because we expected variability in the effects being measured among different species of hosts and parasites.

For our analyses of transmission mode, response measure, and species-specific effects, effect sizes were not combined within studies, because many articles included multiple effect sizes from different measures of parasitism and transmission modes. Combining all effect sizes within a study would therefore have obscured differences and reduced sample size. We ran separate random-effects meta-analyses on effect sizes that were not combined by study, grouping by transmission mode and response measure. However, more than one effect size relying on the same individuals was often available within a study, which risks a degree of pseudoreplication (for example, an animal more heavily parasitized with one parasite might have features that make it more susceptible to other parasites). To avoid such problems, we also repeated our analyses by using "study" as the level of analysis. In this additional analysis, effect sizes were combined to give a single entry per study. This approach revealed qualitatively similar results (see the zip file, available online). Finally, we generated species-specific effect sizes for our phylogenetic meta-analysis.

We also conducted metaregression analyses to assess the degree to which group size itself influences the association between sociality and parasitism; such an influence might occur, for example, if larger effect sizes are found in studies of larger social groups. In metaregression, the predictor variables are continuous rather than discretely varying. To determine the influence of group size, we entered numerical estimates of maximum group sizes from source articles. Studies often measured sociality using different units, such as nests rather than individuals, and on occasion reported minimum estimates of some large aggregations. Given these limitations, we assumed that values provided by authors reflect true differences in effective group size and that any error caused by the imprecise estimation of "group size" causes statistical noise rather than systematic bias toward finding a pattern where none exists; given that the size of colonially breeding aggregations will tend to be underestimated using these criteria, the latter assumption is reasonable. We performed a metaregression of Fisher's Z effect sizes combined within studies on log-transformed maximum group size data (note that log transformation also reduces the problems caused by different group size units).

All effect sizes are reported as correlation coefficients (r) with 95% CIs. In the ecological literature, $r = 0.1$ is generally considered a small effect, $r = 0.3$ is considered a medium effect, and $r = 0.5$ is considered a strong effect (Cohen 1988). Sample sizes were too small to determine whether experimental studies differed from observational studies in their findings. Additional details are provided in the zip file.
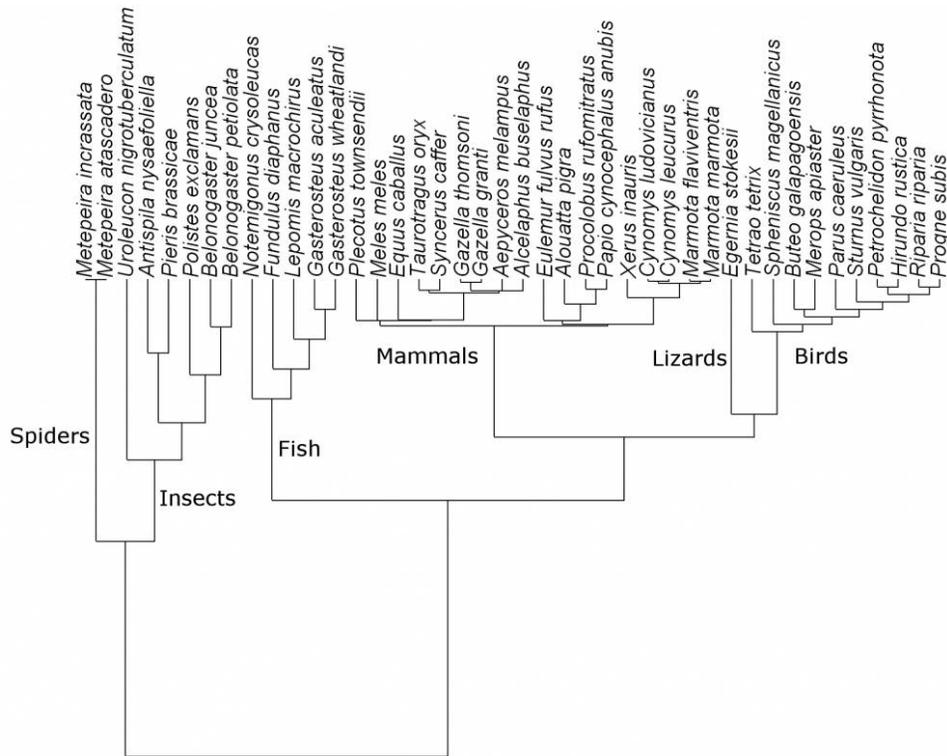
Figure 1: Phylogenetic tree of the species included in the phylogenetic meta-analysis. Branch lengths reflect divergence times.

Publication Bias

Publication bias is a serious concern for meta-analyses, because the published literature may have an overabundance of significant results (Palmer 1999; Møller and Jennions 2001; Jennions and Møller 2002). Additional tests can be used to assess publication bias, and using data imputation procedures, "missing studies" can be added to the meta-analysis. Publication bias was assessed using three methods: funnel plot (Light and Pillemer 1984; Light et al. 1994), which plots the effect size and precision of each study to examine asymmetry in their distribution around the mean; rank correlation (Begg and Mazumdar 1994), which is effectively a mathematical formulation of the funnel plot; and trim and fill (Duval and Tweedie 2000*a*, 2000*b*), which hypothetically reproduces the potentially missing studies that are needed to achieve a symmetrical funnel plot. By imputing these missing studies, it is possible to determine whether adding their results to the analysis would substantially change the overall estimate of the effect size.

Phylogenetic Meta-Analysis

Phylogeny is important for a meta-analysis that involves multiple species, because more closely related species may show similar effect sizes. To deal with the possible phylogenetic dependence of species' effect sizes, we also conducted phylogenetic meta-analyses using a method developed by Adams (2008) and extended by Lajeunesse (2009). As with phylogenetic comparative methods, phylogenetic meta-analysis incorporates nonindependence that arises because of shared ancestry into the error term of a generalized linear model by using the phylogenetic covariance among taxa. Traditional (nonphylogenetic) and phylogenetic meta-analyses can be compared using model selection procedures on the basis of Akaike Information Criterion (AIC), thus providing a way to assess whether it is important to include phylogeny in the meta-analysis (Lajeunesse 2009).

We assembled an ultrametric tree in R package APE 2.7.1 (Paradis et al. 2004) for the 42 individual host species in the database (fig. 1). Branch lengths and topology came from Tree of Life (Maddison et al. 2007) and TimeTree (Hedges et al. 2006) for deep structure and clade-specific phylogenies for tips (birds: Sheldon et al. 2005; Hackett et al. 2008; mammals: Bininda-Emonds 2007; ray-finned fishes: Li et al. 2007; insects: Kjer 2004; Simon et al. 2009; see the zip file for the tree in Newick format). When divergence times were not available, nodes were distributed

evenly from the time of the last known branching event (14 of 40 nodes on the tree). To measure phylogenetic signal in the distribution of effect size, we estimated Pagel's λ (Pagel 1994) using the R packages APE (Paradis et al. 2004) and GEIGER (Harmon et al. 2008).

By using effect sizes averaged by species from the intraspecific tests, we performed both a traditional and a phylogenetic meta-analysis using Lajeunesse's (2009) method in the program PhyloMeta (Lajeunesse 2011) via an additional script to run PhyloMeta in R (Chamberlain 2011). Because PhyloMeta requires a single effect size per species, we obtained a combined effect size for each species by entering host species as a covariate into CMA and used these species-specific Fisher's Z effect sizes and variances in the phylogenetic meta-analysis. We assessed the importance of phylogenetic correction in the meta-analysis using the AIC model in PhyloMeta. Although it might be interesting to simultaneously investigate phylogenetic effects relative to parasite phylogeny, we currently lack an approach that can take into account two phylogenies at the same time. In addition, our knowledge of evolutionary relationships is more complete for hosts than for parasites.

Results

Overall Effect and Publication Bias

When samples were averaged by study, 19 studies showed negative associations and 50 studies showed positive associations between group size and parasite risk (fig. 2). The mean effect size across all data averaged by study (r) was 0.187 (95% CI, 0.127–0.246, $n = 69$) and differed significantly from a null effect ($Z = 6.01$, $P < .0001$). We found significant heterogeneity in effect sizes across studies ($Q = 1,059.86$, df $= 68$, $P < .0001$).

A funnel plot of precision for effect sizes averaged by study suggested a small amount of publication bias (fig. 3). However, a rank correlation between precision and effect size, which was effectively a mathematical formulation of the funnel plot (Begg and Mazumdar 1994), was not statistically significant (Kendall's $\tau = 0.012$, $P = .440$; one-tailed without continuity correction). The trim-and-fill analysis of the random effects model imputed ten missing negative studies and reduced the point estimate of r to 0.141 (95% CI, 0.078–0.203). Effect sizes not combined within studies yielded similar results. Thus, we found weak evidence for publication bias, and overall evidence in support of a positive association between group size and parasite risk remained after correcting for this bias.
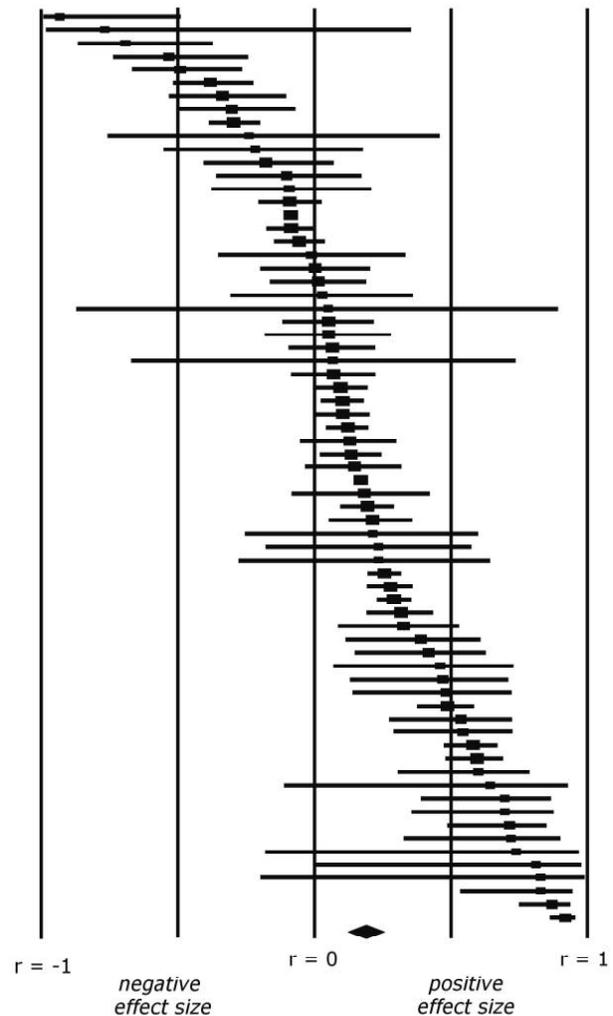

Figure 2: Forest plot of effect sizes (rectangles) and confidence intervals (bars) for each study and the effect averaged across all studies (diamond).

Sources of Heterogeneity: Transmission Mode, Response Measure, and Study Type

To assess sources of heterogeneity in effect sizes, we first examined the influence of parasite transmission mode using all effect sizes (resulting in a sample size of 344 effect sizes from 69 studies). We found consistent, positive effects of group size on parasitism for every transmission mode except searching, which had 95% CIs that clearly encompassed 0 (table 1). The results were consistent with our predictions for contagious and environmental parasites but not for our prediction of a negative relationship for mobile parasites: the correlation for searching parasites was slightly positive and was not significantly different

from 0, whereas it was significantly positive for parasitoids. Infection with vector-borne parasites also showed a positive association with group size. Qualitatively similar results were obtained when averaging effect sizes to the "study" level to control for possible effects of pseudoreplication (table A1, deposited in Dryad).

We also examined whether studies using different response measures produced different effect sizes (table 2). Analyses that used parasite abundance, intensity, and prevalence all showed a positive overall effect size varying between "small" and "intermediate" in magnitude (sensu Cohen 1988). Parasite abundance showed the strongest association with group size. By contrast, the effect size for studies of parasite richness was the smallest, with a relatively wide 95% CI that clearly included 0. Effect sizes measuring host immunity (of which 40 effect sizes from six studies were comparative and 11 effect sizes from three studies were intraspecific) also found that it related positively to group size, although the effect size was relatively small. Similar results were obtained when averaging effect sizes to the "study" level to control for possible effects of pseudoreplication, except that 95% CIs on effect size in studies using "intensity" encompassed 0 (table A2, available in the zip file).

We assessed whether intraspecific studies produced effect sizes that differed from those of comparative studies. Both intraspecific and comparative studies showed positive

Table 1: Averaged effect sizes, sorted by transmission and not averaged by study

		Test for null	
Transmission (*n*)	Correlation point estimate (95% CI)	*Z* value	*P*
Searching (32)	.046 (−.059 to .150)	.864	.388
Contagious (164)	.213 (.165–.260)	8.487	<.0001
Environmental (173)	.179 (.129–.228)	6.926	<.0001
Vector (42)	.396 (.320–.467)	9.429	<.0001
Parasitoid (48)	.222 (.146–.296)	5.608	<.0001

effect sizes overall (intraspecific: $r = 0.164$ [95% CI, 0.074–0.251], $n = 47$, $Z = 3.548$, $P < .0001$; comparative: $r = 0.258$ [95% CI, 0.107–0.371], $n = 20$, $Z = 4.092$, $P < .0001$; two studies that contained both intraspecific and comparative data were excluded). All types of response data (prevalence, abundance, intensity, richness, and immunity) were represented in both intraspecific and comparative studies. We found no significant heterogeneity of effect sizes between comparative and intraspecific studies ($Q = 1.64$, df = 2, $P = .440$).

Phylogenetic Variation and Meta-Analysis

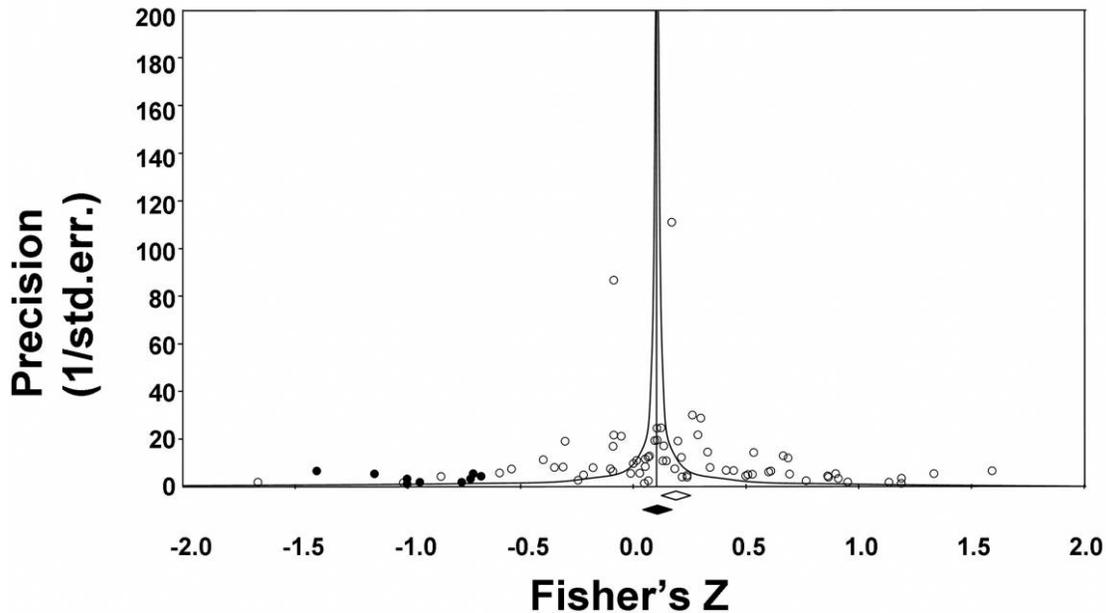Sample sizes varied considerably among taxonomic classes. For example, we located 28 intraspecific and interspecific

Figure 3: Funnel plot of effect size (Fisher's *Z*) by precision (1/SE). The white circles represent included studies, and the black circles represent missing imputed studies. The white diamond represents overall effect size, and the black diamond represents the corrected effect size. std.err., standard error.

Table 2: Averaged effect sizes, sorted by response variable and not averaged by study

Response variable (*n*)	Correlation point estimate (95% CI)	Test for null	
		Z value	*P*
Abundance (65)	.275 (.199–.348)	6.875	<.0001
Immune (51)	.100 (.012–.186)	2.222	.026
Intensity (67)	.176 (.096–.251)	4.373	<.0001
Prevalence (130)	.217 (.161–.271)	7.475	<.0001
Richness (31)	.047 (−.063 to .157)	.843	.399

studies of mammals and 27 studies of birds, but only one study of arachnids and two studies of reptiles. Effect sizes displayed significant heterogeneity among classes (table 3, $Q = 30.60$, df = 5, $P < .0001$; both comparative and intraspecific studies). Studies of birds showed the largest mean effect size, which remained when the analysis was repeated excluding the cliff swallow *Petrochelidon (Hirundo) pyrrhonota*, the most common focal host species in studies of birds (9 of 27). Combining effect sizes first by class and then by study reduced the overall effect size from $r = 0.187$ to $r = 0.133$.

We also considered the subset of studies involving single species, which spanned 42 animal species in 25 families across 20 orders and 6 classes, for a total of 197 effect sizes in 49 articles. Overall values from intraspecific effect sizes grouped by species (but not combined by study) showed a small positive effect ($r = 0.039$ [95% CI, 0.014–0.063], $n = 197$ effect sizes, $Z = 3.098$, $P = .002$). A Q-test of heterogeneity in intraspecific studies grouped by class gave a significant result ($Q = 26.76$, df = 5, $P < .0001$). Among intraspecific studies, only research on birds yielded an overall effect size that differed significantly from 0 (birds: $r = 0.378$ [95% CI, 0.235–0.505], $n = 18$; test of null: $Z = 4.937$, $P < .0001$). Effect sizes from intraspecific studies of mammals were close to 0 overall ($r = 0.026$ [95% CI, −0.026 to −0.088], $n = 20$; test of null: $Z = 0.445$, $P = .656$).

Biases in studying particular parasite transmission modes and response measures may be partly responsible for these differences in effect sizes between the major vertebrate groups. A *G*-test of independence found significant differences between the transmission modes and response variables represented in studies of birds and mammals (transmission: $G = 25.81$, df = 3, $P < .0001$; response: $G = 26.52$, df = 4, $P < .0001$). Because of the disparity in results, we performed three additional analyses, including a separate analysis of the overall data with birds removed (table 3) and analyses of publication bias in studies involving birds and nonbirds. In a test of publication bias in studies involving birds, the funnel plot suggested several missing studies, and a rank correlation test for publication

bias had significant results (Kendall's $\tau = 0.302$, $P = .014$, one-tailed without continuity correction). A trim and fill analysis of the random effects model for only birds imputed six missing studies (a large number, given that only 27 studies were included in the sample) and reduced the point estimate of the correlation from 0.401 to 0.276 (95% CI, 0.147–0.395). We thus found more evidence for publication bias in studies involving birds than in the overall data set, but the medium effect size for birds remained even when we controlled for publication bias. In the subset of nonavian species, we found no evidence of publication bias with either a rank correlation test (Kendall's $\tau = 0.009$, $P = .465$, one-tailed without continuity correction) or a trim and fill analysis of the random effects model (0 missing studies). In the nonbirds, the mean effect size across studies was 0.049 (95% CI, −0.017 to 0.113), which did not differ significantly from 0 ($Z = 1.462$, $P = .144$).

We also investigated whether the relationship between sociality and parasitism is mediated by differences in group size, on the basis of the observation that maximum group sizes were generally larger in studies involving birds than in those involving mammals (fig. 4). Using a mixed-effect metaregression approach and estimates of maximum group size, we found a significant positive slope of 0.082 (95% CI, 0.011–0.153; $Z = 2.281$, $P = .023$), showing that effect sizes are generally more consistently positive in the largest aggregations (fig. 5). As expected, taxon-specific regressions gave different results (mammals: slope estimate = −0.002 [95% CI −0.230 to 0.226], $Z = −0.018$, $P = .986$; birds: slope estimate = 0.109 [95% CI, −0.005 to 0.223], $Z = 1.882$, $P = .060$). The large negative effect size at large group size in figure 5 is a study of immune function in penguins (Tella et al. 2001). When this study is excluded, the slope estimate for birds is 0.181 (95% CI, 0.075–0.286).

We tested for phylogenetic signal in the distribution of effect size by estimating Pagel's λ (Pagel 1994). We found no significant phylogenetic signal in the distribution of effect sizes relating parasite risk and group size within species; we estimated λ to be very close to 0 (λ < 0.001) and not significantly different from 0, by χ^2 test. Application of formal phylogenetic meta-analysis techniques confirmed this finding: traditional meta-analysis methods fit the data better than did the phylogenetic meta-analysis (traditional random-effects meta-analysis: AIC = 137.80; phylogenetic random-effects meta-analysis: AIC = 260.34; lower AIC indicates better fit). Although the model fit the data less well, the results of the phylogenetic meta-analysis were consistent with the traditional meta-analysis, with an overall weak positive effect when phylogeny was taken into account ($r = 0.1$ [95% CI, −0.005 to 0.208]). Because of the low support for the phylogenetic meta-analysis model, we conclude that the detailed analyses of

Table 3: Averaged effect sizes, sorted by host class, including both interspecific and intraspecific studies, effect sizes averaged within studies

		Test for null	
Host class (*n*)	Correlation point estimate (95% CI)	*Z* value	*P*
Actinoptergyote (4)	.032 (−.248 to .307)	.219	.827
Arachnid (1)	.067 (−.093 to .224)	.824	.410
Bird (27)	.401 (.295–.497)	6.901	<.0001
Bird, excluding *Petrochelidon (Hirundo) pyrrhonota* (18)	.322 (.194–.440)	4.755	<.0001
Insect (7)	.189 (−.123 to .467)	1.192	.233
Mammal (28)	.041 (−.016 to .128)	.934	.351
Reptile (2)	.024 (−.129 to .176)	.310	.757
Overall (69)	.133 (.076–.189)	4.565	<.0001
Overall, excluding birds (42)	.049 (−.017 to .113)	1.462	.144

moderator variables and publication bias in the traditional meta-analysis provide statistically valid results.

Discussion

For most parasite transmission modes, we predicted that larger groups would have more parasites because of the effects of higher local density, more intensive conspecific interaction, more intensive range use, and possibly higher attraction of vectors. However, the encounter-dilution effect may protect animals in larger groups from parasites in which mobile arthropods are responsible for transmission (searching parasites and parasitoids, and possibly vector-borne diseases). In a meta-analysis of 69 studies, we found a weak but positive overall association between group size and parasite risk. This association was robust to controls for publication bias across all studies. We found stronger evidence for publication bias in studies involving birds, but the positive association between group size and parasite risk in birds remained after controlling for this bias. The mean effect size relating group size to parasitism varied by transmission mode, and the results were consistent with our predictions that contagious and environmentally transmitted parasites show a positive association with group size.

Instead of the negative association predicted for parasites with moving transmitting agents, however, we found a positive relationship for vector-borne parasites and parasitoids and no association for searching parasites, which differs from previous findings using a smaller data set (Côté and Poulin 1995). Several factors could account for these unexpected results in tests of the encounter-dilution effect. First, larger groups may be easier for searching parasites to detect, and a strong increase in detection by parasites could result in higher per capita risk in larger groups (Mooring and Hart 1992; Côté and Poulin 1995; Nunn and Heymann 2005). Parasite satiety could also play a role:

some micropredators attack several members of a group, whereas others attach and feed until full. Finally, when searching arthropods occur at very high local densities, the encounter-dilution effect may not apply, because there would be many more parasites than potential victims; under these circumstances, variation in intensity may be detected, but not variation in prevalence.

Effect sizes varied by response variable, but were positive across all response variables, including studies of immunity, where we found a weak but nonzero association with group size. Effect sizes varied only slightly by study type. Evolutionary-genetic evidence supports our finding on immunity: a recent study found some evidence for positive selection on genes related to immunity in primate species living in larger groups (Wlasiuk et al. 2010).

The greatest difference in effect size appeared between the two best-studied groups of animals in our data set: mammals and birds. These groups were represented by similar numbers of studies but differed in overall effect size by an order of magnitude, with much stronger support for a positive effect size in birds. Several statistical and sampling issues could be responsible for this difference. First, some of the difference in effect size between birds and mammals may be attributable to different numbers of studies involving, for example, parasite abundance or contagious parasites in the two groups. Differences in the strength of the effect between classes, however, were greater in magnitude than any of the differences between transmission modes or response measures. Second, pseudo-replication introduced by shared ancestry may be a factor. However, despite the large difference between classes, a traditional meta-analysis fit the data better than a phylogenetic meta-analysis, and in support of this finding, the effect sizes failed to show evidence for phylogenetic signal. Strong phylogenetic signal in the data would indicate that closely related species have more similar effect sizes than do distantly related species, and such nonindependence of
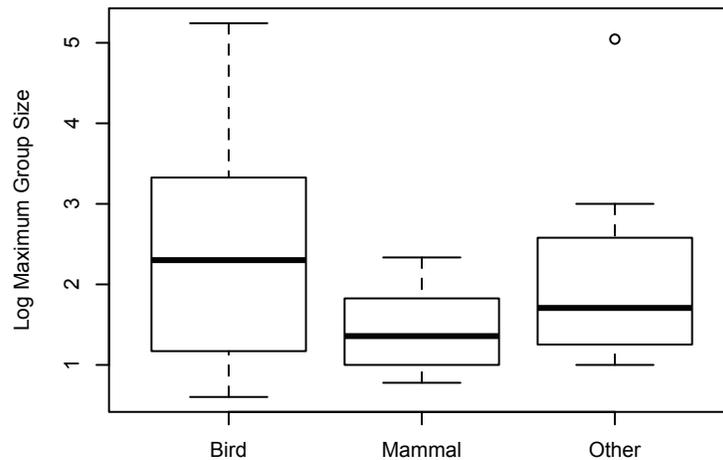
Figure 4: Maximum group size in taxonomic subsets of data. Plot shows $\log_{10}$-transformed maximum group size, which in some cases was based on proxies for group size, such as the number of active nests (see "Material and Methods"). Data are based on effect sizes averaged at the study level and include 27 studies of birds, 28 studies of mammals, and 14 studies of other taxonomic groups.

data would violate the underlying assumption of conventional meta-analysis when calculating the mean effect size over the entire data set. The absence of phylogenetic signal may reflect that variation in the relationship between parasite risk and group size is strongest at the level of classes rather than at lower taxonomic groupings, and intraspecific variation may have reduced the ability to detect phylogenetic signal (Ives et al. 2007). Third, the cause for the disparity between birds and mammals may be publication bias among studies of birds, which was stronger in the subset of avian species than in the overall data set. Publication bias is known to vary among laboratories and authors, notably among ecological studies of birds (Palmer 1999), and may also vary among study taxa. However, a statistical control for publication bias still revealed positive and statistically significant effect sizes for birds.

Ecological differences between birds and mammals could also explain the difference we found. The type of sociality varied, with many of the birds in our analysis being colonial breeders. Colonially breeding birds may be more heavily parasitized during the breeding season because of increased susceptibility resulting from breeding stress. Few studies have compared trends in parasitism between year-round colonial and seasonally colonial species, although Figuerola (2000) found that winter sociality but not breeding sociality affects feather mite prevalence, and Poiani (1992) observed that migratory pattern but not breeding sociality explained ectoparasite prevalence. Immune studies also offer evidence for the costs of colonial breeding: colonial breeders have been found to have larger spleens and bursae of Fabricius (Møller and Erritzoe 1996) and consistently stronger T cell and B cell responses

(Møller et al. 2001), compared with those of solitary breeders.

More generally, we found that effect sizes were larger in cases when individuals formed larger aggregations, and this pattern was particularly strong within birds. Specifically, we found a wider range of maximum group sizes in studies involving birds than in those involving mammals; for example, the largest mammalian group was a colony of prairie dogs numbering 216 individuals (Hoogland 1979), whereas data on birds included many studies with group sizes orders of magnitude larger than this (fig. 4). Indeed, our metaregression found an association between effect size and maximum group size (which in some cases was based on proxies for group size, such as active nests; see "Material and Methods"). This may indicate that, above a certain aggregation size, effects of group size on parasitism increase. The structure of interactions in groups may also have an influence on parasitism in large aggregations. A recent modeling article found that social group modularity increases with group size in primates and has a negative effect on parasite spread, which may counteract the effects of group size on parasite risk (Griffin and Nunn 2011). If avian breeding colonies are less socially structured than mammalian groups even at large group size, the former may exhibit a stronger relationship between group size and parasitism.

In addition, many colonial birds reuse colony sites and nests from year to year, which may result in greater accumulation of parasites within colonies. If colonial breeders are under more parasite pressure in breeding aggregations, comparing the parasite loads of the same population at their wintering and breeding grounds might
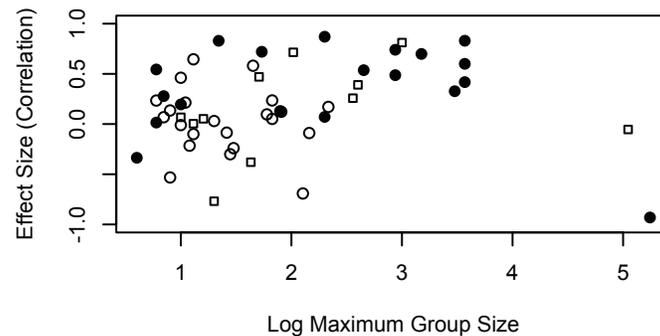
Figure 5: Effect size in relation to maximum group size. Plot shows effect sizes measured as correlation coefficients and $\log_{10}$-transformed maximum group size, which in some cases was based on proxies for group size, such as the number of active nests (see "Material and Methods"). Data are based on effect sizes averaged at the study level and include 27 studies of birds (black circles), 28 studies of mammals (white circles), and 14 studies of other taxonomic groups (squares).

help to explain this relationship. Controlling for type of sociality in a more focused analysis of birds may also be valuable, because questions of breeding aggregations and seasonality suggest that a simple count of hosts may not describe the magnitude of their group sizes adequately.

Finally, migration could play a role, because a greater proportion of the bird studies involved migratory species, compared with the mammal studies. Migration may increase the risk of parasitism through physiological stress and through exposure to diverse parasite fauna from more regions and host communities (Altizer et al. 2011). Migration may also enable animals to escape parasite risk, for example by reproducing or wintering in places with less parasite pressure (Alerstam et al. 2003; Altizer et al. 2011). In birds, migration is considered a risk factor for spreading pathogens (Hubalek 2004) and has been associated with higher transmission of West Nile virus (Zeller and Murgue 2001) and avian influenza virus (Garamszegi and Møller 2007). It is notable that the other taxonomic group with a strongly positive correlation between group size and parasite risk, insects, can also travel large distances in relation to their body size and form large aggregations, although our small sample size for insects limits our ability to draw conclusions.

We sought to investigate the effects of group size on parasite risk across a wide diversity of organisms and types of study, which inevitably resulted in the loss of some details when the results from multiple studies were placed in a common statistical framework. From this synthetic analysis, however, we found overall support for the existence of an association between parasite risk and group size. We were able to demonstrate that the association varies consistently across parasites with different transmission modes, response variables, group sizes, and host taxonomy. We suggest that a meta-analysis framework can

be used to explore the nuances of the relationships between sociality, parasitism, and other ecological factors. In the future, more targeted and complete sampling of the relationship between parasitism and group size across animals will help to reveal how this relationship varies among taxa, types of parasites, measures of parasite risk, and definitions of sociality. Theoretical models may also provide testable predictions for the dynamics of parasitism in relation to different host behaviors and parasite transmission modes.

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