Animal Behaviour 172 (2021) 171–182

Contents lists available at ScienceDirect

Animal Behaviour

journal homepage: www.elsevier.com/locate/anbehav

Parasitism and host social behaviour: a meta-analysis of insights derived from social network analysis

Léa Briard [a, b, *], Vanessa O. Ezenwa [a, c]

[a] Odum School of Ecology, University of Georgia, Athens, GA, U.S.A.
[b] Research Centre on Animal Cognition (CRCA), Centre for Integrative Biology (CBI), Toulouse University, CNRS, UPS, Toulouse, France
[c] Department of Infectious Diseases, College of Veterinary Medicine, University of Georgia, Athens, GA, U.S.A.

ARTICLE INFO

Article history:
Received 23 April 2020
Initial acceptance 17 June 2020
Final acceptance 25 September 2020
Available online 8 January 2021
MS. number: 20-00284R

Keywords:
animal behaviour
contact
disease ecology
metaregression analysis
parasite transmission
proximity
sociality

Social behaviour is a key component of animal behaviour that facilitates the spread of parasites. Traditionally, group size has been used as a primary metric for quantifying the impact of social behaviour on parasite transmission; however, with the emergence of social network analysis an increasing number of studies are using this more nuanced tool to study links between social behaviour and parasite infection. In this study, we synthesized insights derived from empirical studies on social networks and parasites using a meta-analytical approach. We analysed 210 associations between parasite burden and individual level network metrics extracted from 18 published articles. Overall, we found a positive effect of social behaviour, measured by social network metrics, on parasite infection at the individual level, with no evidence of publication bias. The magnitude of the mean effect size for associations between social network metrics and measures of parasitism was nearly twice as large as that observed for group size in previous meta-analyses. However, there was significant heterogeneity in effect sizes across studies, and this pattern could not be explained by either host traits, parasite traits or the social network metric examined. We discuss potential reasons for this unexplained heterogeneity, such as possible mismatches between focal host social behaviour and focal parasite biology as well as methodological considerations. We also suggest future research directions that can help fill gaps that remain in our understanding of the drivers of variation in interactions between social hosts and parasites.

Social behaviour is a key component of animal behaviour that facilitates the spread of parasites. Consequently, questions about the impact of host social behaviour on infectious disease transmission are of considerable importance to epidemiologists and disease ecologists (Altizer et al., 2003). Moreover, given the potent selection pressure that increased parasite transmission imposes on social hosts, behavioural ecologists also have an abiding interest in the role parasites play in the evolution of social behaviour (Alexander, 1974; Loehle, 1995). This convergence of interests has given rise to decades of research on the links between social behaviour and parasite transmission in animal populations (reviewed in Altizer et al., 2003); yet many unanswered questions remain. These questions include when and how social behaviour is most likely to influence parasite transmission and what forms of social behaviour are most strongly shaped by parasitism.

Group size is by far the most widely used metric for capturing the effects of social behaviour on parasite transmission. This measure provides an intuitive proxy for the number of social contacts an 'average' group member experiences, and a sizeable body of work on group size–parasitism relationships shows that group size is an important predictor of parasite transmission under a range of circumstances (Cote & Poulin, 1995; Patterson & Ruckstuhl, 2013; Rifkin, Nunn, & Garamszegi, 2012), although the strength of this association often varies depending on the measure of parasitism used (e.g. abundance, prevalence versus richness; Rifkin et al., 2012) and the transmission mode of the parasite (e.g. mobile versus nonmobile; Cote & Poulin, 1995). This body of work also reveals that group size does not adequately capture many important nuances of animal social organization relevant to parasite transmission (Ezenwa, 2004; Griffin & Nunn, 2012). For example, individuals within a group can vary in the degree to which they engage in social interactions, and some individuals may change groups frequently, shuffling and reshuffling the number and/or identity of group mates. In both cases, group size may fail to capture the real quantity of interest, i.e. the

* Correspondence: L. Briard, Research Centre on Animal Cognition (CRCA – CBI), UMR 5169, CNRS, Door 220, Bat 4R3, Université Paul Sabatier, 118 route de Narbonne, 31062 Toulouse, France.
E-mail address: drleabriard@gmail.com (L. Briard).

https://doi.org/10.1016/j.anbehav.2020.11.010
0003-3472/

number or frequency of distinct social contacts. Recently, the limitations of group size as a metric of social contact, along with advances in computing systems, have helped spur the rise of social network analysis (SNA) as a tool for probing the links between social behaviour and parasite transmission in wild animal systems (Craft, 2015; Wey, Blumstein, Shen, & Jordán, 2008). Social networks, and the metrics derived from them, provide a nuanced view of animal social behaviour (Farine & Whitehead, 2015; Wey et al., 2008), and in the past decade SNA has been applied to understanding patterns of parasitism in a growing number of animals (for a review see Godfrey, 2013; White, Forester, & Craft, 2017; Fig. 1). Thus, an opportunity now exists to synthesize the current literature on SNA and parasitism to identify general patterns that are emerging from this new approach.

A social network connects individuals (nodes) to one another based on social interactions (edges). Interactions used to construct social networks can range from associations such as membership in the same group to direct interactions such as grooming or aggression. Ultimately, SNA provides quantitative measurements that capture fine-scale heterogeneities in individual social relationships, and for researchers interested in the links between social behaviour and parasitism these measures can be used to explore both the health implications of social behaviour and its consequences for parasite transmission. The ability of SNA to capture key complexities in animal social behaviour, often hypothesized to have distinct consequences for parasite infection, is another advantage of this approach. For example, recent SNA studies show that different types of social connectivity can have different implications for infection in the same host–parasite system (e.g. strength [intensity of connections] versus closeness [a node's 'distance' from other nodes] for *Strongyloides* worms in spider monkeys, *Ateles hybridus* [Rimbach et al., 2015]). Likewise, more subtle differences in social behaviour can affect risk of infection in distinct ways. For instance, in tuataras, *Sphenodon punctatus*, the strength of connections directed towards an individual by females was associated with parasitic mite, *Neotrombicula* spp., burdens, whereas the strength of male connections was not (Fig. 1), while in meerkats, *Suricata suricatta*, the number of connections passing through an individual

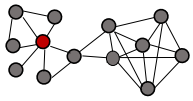
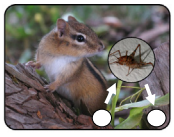
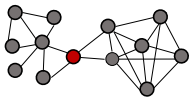
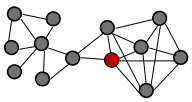
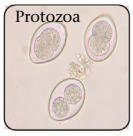
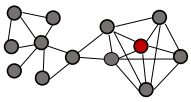
Metric	Definition	Parasite type	Host: Linked to parasite transmission	Host: NOT linked to parasite transmission	Source
Degree 	The number of individuals a focal node is connected to (Whitehead, 2008)				Grear et al. (2013) Chipmunk - helminth transmitted by nonclose transmission vs helminth transmitted by intermediate host
Strength 	The sum of edge weights directly connected to a focal individual (Whitehead, 2008)				Godfrey et al. (2010) Tuatara - *Neotrombicula* spp.; social connectivity based on contacts initiated by female vs male hosts
Betweenness 	The number of shortest paths between all pairs of individuals that flow through a focal individual (Krause et al., 2015)				Drewe (2009) Meerkat - *Mycobacterium bovis*; social connectivity based on aggression vs grooming contact
Closeness 	The shortest path between an individual and all other individuals in a network (Krause et al., 2015)				Williams et al. (2017) Grant's gazelle - *Eimeria* spp.vs VanderWaal et al. (2013) Ground squirrel - *Cryptosporidium* spp.
Eigenvector centrality 	How well-connected, both directly and indirectly, a focal node is (Whitehead, 2008)				MacIntosh et al. (2012) Japanese macaque - *Strongyloides fuelleborni* vs *Trichuris trichiura*

Figure 1. Examples of common social network metrics used to quantify social interactions in animals and relationships with infection by a specific parasite type. For each metric - parasite pair, examples show a host for which a significant versus nonsignificant correlation between social connectivity and parasite infection was found. For the network illustrations, the width of an edge represents the weight (i.e. see 'strength' network), the larger the edge the higher the weight. The red node in each network illustration highlights the node with the highest value for the described metric. Definitions are from Whitehead, 2008 and Krause, James, Franks, & Croft, 2015. Helminth image available from Wikimedia Commons under a CC BY-SA 2.5 licence. Chipmunk image by Gilles Gonthier available on Flickr under a CC BY 2.0 licence. Camel cricket image by Audrey R. Hoff available on Flickr under a CC BY-NC-ND 2.0 licence. *Sphenodon punctatus* image available from Wikimedia Commons under a public domain licence. Bacteria image from the NIH image Gallery available from Flickr under a CC BY-NC 2.0 licence. Badger image by Tony C.C. Gray available from Flickr under a CC BY-NC-ND 2.0 licence. Fighting meerkat image by Tambako 2014, available from Flickr under a CC BY-ND 2.0 licence. Grooming meerkat image by Beth Wilson (Mirsasha), available from Flickr under a CC BY-NC 2.0 licence. Protozoa image by Joel Mills under a CC BY-SA 3.0 licence. Grant's gazelle image from Charles J. Sharp under a CC BY-SA 3.0 licence. Ground Squirrel image by K. Schneider, available from Flickr under a CC BY-NC 2.0 licence. *Cryptosporidium* image by the Centers for Disease Control and Prevention under a CC BY-SA 4.0 licence. *Strongyloides* sp. egg image by Roland Yao Wa Kouassi, Scott William McGraw, Patrick Kouassi Yao, Ahmed Abou-Bacar, Julie Brunet, Bernard Pesson, Bassirou Bonfoh, Eliezer Kouakou N'goran and Ermanno Candolfi, available from Wikimedia Commons under a CC BY 4.0 licence. *Macaca fuscata* image courtesy of Christof Neumann.

[betweenness] was linked to tuberculosis, *Mycobacterium bovis*, infection only when these connections were based on aggression and not grooming (Fig. 1). Understanding these types of nuances is crucial for identifying specific aspects of social behaviour that differentially incur parasite-associated costs or that are most influential in fuelling transmission.

In light of the rapid rise in empirical SNA studies focused on parasitism, here we used a systematic meta-analysis approach to examine general insights about social behaviour and parasitism derived so far from SNA. Our first goal was to quantify the overall magnitude and direction of the effect of individual social behaviour on parasitism in animals as measured by SNA for comparison with the effect of group size, which has been explored in previous meta-analyses (Cote & Poulin, 1995; Patterson & Ruckstuhl, 2013; Rifkin et al., 2012). In addition, we were interested in identifying any emergent patterns related to the types of social connectivity or social behaviour most strongly associated with effects on parasitism. Finally, we were interested in attributes of the host (e.g. type of social organization) and parasite (e.g. measure of infection [e.g. abundance versus richness], transmission mode) that might help explain variation in social behaviour–parasitism relationships. We investigated these latter questions by asking whether overall associations between SNA-derived measurements of social behaviour and parasite infection depended on the following moderator variables: (1) SNA metric, (2) host behaviour used to build social networks, (3) host social organization, (4) the measure used to quantify parasite infection and (5) parasite transmission mode. While other studies have recently reviewed the status of SNA in infectious disease research (Craft, 2015; Godfrey, 2013; White et al., 2017) our meta-analytical approach provides a first quantitative analysis of observed patterns revealing key priorities for future work.

METHODS

Literature Search and Inclusion Criteria

Our search protocol was designed to identify as many articles as possible on SNA and parasite infection while minimizing sampling biases. We used four different databases (JSTOR, PubMed, CAB abstract and Web of Science) to obtain an initial pool of articles published as of December 2018 (see PRISMA diagram in Fig. A1). We searched for articles using the following search string: **TOPIC:** (social* network*) *NOT* **TOPIC:** (human) *AND* **TOPIC:** (disease* OR parasite* OR helminth* OR virus OR arthropod* OR prion OR protozo* OR bacte* OR infect* OR pathoge*) **Refined by: CATEGORIES:** (veterinary sciences OR multidisciplinary sciences OR zoology OR ecology OR evolutionary biology). Next, we inspected the abstracts and titles of each article ($N = 1304$) to exclude those that were likely to be unsuitable for the meta-analysis, for example because they were purely theoretical studies or review papers ($N = 1223$). We read all the remaining articles ($N = 81$) more closely to assess their suitability for analysis based on our inclusion criteria.

For an article to be included in our analysis, the study had to use social network metrics to investigate empirical links between social behaviour and some aspect of parasite infection. Since our goal was to uncover effects of social behaviour on the transmission of infectious agents, we excluded studies performed on commensal microorganisms or induced immune responses. We also excluded theoretical articles that simulated transmission of parasites on a social network and studies on livestock. Importantly, because our focus was on the link between individual level social behaviour and infection, we focused on studies where the level of analysis was the individual (versus group or population). Ultimately, a total of 18 articles met our inclusion criteria (Fig. A1; Adelman, Moyers, Farine, & Hawley, 2015; Balasubramaniam, Beisner, Vandeleest, Atwill, & McCowan, 2016; Corner, Pfeiffer, & Morris, 2003; Drewe, 2009; Duboscq, Romano, Sueur, & Macintosh, 2016; Fenner, Godfrey, & Bull, 2011; Friant, Ziegler, & Goldberg, 2016; Godfrey, Moore, Nelson, & Bull, 2010; Grear, Luong, & Hudson, 2013; Leu, Kappeler, & Bull, 2010; MacIntosh et al., 2012; Otterstatter & Thomson, 2007; Rimbach et al., 2015; VanderWaal, Atwill, Hooper, Buckle, & McCowan, 2013; VanderWaal et al., 2016; Weber et al., 2013; Williams, Worsley-Tonks, & Ezenwa, 2017; Wohlfiel, Leu, Godfrey, & Bull, 2013).

Effect Size Calculation

From each of these 18 articles, we extracted measures of the relationship between a social network predictor and parasite response (i.e. effect sizes) for use in our meta-analysis. As a measure of effect size, we used correlation-based r values between social network metrics and parasite responses. If a study did not report an r value, we extracted test statistics (e.g. χ^2, F or t) and P values from the text and then converted these to effect size r values following methods described in Rosenthal and DiMatteo (2001). For four studies, we contacted authors for more detailed information (e.g. when 'nonsignificant' was provided in the text in place of a specific P value). For statistical models with multiple predictor variables or random effects, we converted the reported P value to a standard normal deviate Z score and used the sample size to obtain r following Bentz, Becker, and Navara (2016). We assigned a negative value to effect sizes for which the social network metric was negatively correlated with parasite infection. Directional effect sizes were then converted to Fisher's Z to normalize the distribution (Rosenthal & DiMatteo, 2001). We used the R package metafor (Viechtbauer, 2010) for r-to-Z effect size conversions (escalc function). For three articles that compared the same social network metric (based on the same behaviour) with the same parasite response for the same individuals across multiple seasons or years without reporting a global analysis, we averaged the effect sizes over all seasons/years. For five articles that performed analyses on distinct groups or populations, we considered each group or population to be an independent study unit. Overall, we extracted 210 effect sizes from 18 articles encompassing 23 study units.

Selection of Random Factors and Moderators

The first goal of our meta-analysis was to estimate the general effect of social behaviour on parasite infection at the individual level. To do this, we needed to account for the random effect of focal hosts and parasites in our analysis, so we extracted taxonomic information for both hosts and parasites for each effect size recorded. For hosts, all studies reported species level taxonomic information allowing us to include host species identity as a random factor in the analysis. For parasites, a majority of studies lacked species level taxonomic information, so we aggregated the parasite data into the following higher-level classifications, designated as 'parasite type': arthropod, bacteria, helminth, protozoa and multiple (i.e. more than two different categories of parasites pooled together). We used parasite type as the parasite random effect in our models to account for parasite taxonomy.

A second goal of our meta-analysis was to investigate attributes of the host and parasite that might influence the relationship between social behaviour and parasite transmission. To do this, we identified a number of host- and parasite-related variables as potential moderators of the social behaviour–parasite relationship and extracted information on each for every effect size recorded. The moderators included: (1) the social network metric used to quantify social behaviour (e.g. degree, strength; see Fig. 1 for

definitions), (2) the host behaviour used to build the social network (e.g. grooming versus number of neighbours), hereafter called social behaviour measure, (3) the host social organization, (4) the measure used to quantify parasitism (e.g. parasite richness versus abundance), hereafter called infection measure, and (5) the transmission mode of the parasite.

Given the wide variety of social network metrics used across studies, we classified all metrics into five broad categories: degree, strength, betweenness, closeness and eigenvector centrality (Fig. 1). Similarly, given the variety of behaviours used to build social networks, we classified these into three broad categories: contact, distance and space use. Contact refers to any interaction involving physical contact between two individuals (e.g. aggression, grooming or any other form of contact). Distance refers to social relationships measured by metric or topological distances (e.g. metres or nearest neighbours). Space use refers to social interactions measured by overlapping habitat use (e.g. territory overlap, sequential trap use). We classified host social organization as solitary-but-social, fission–fusion or stable. Solitary-but-social characterizes species that are predominantly solitary but exhibit some aspect of sociality (shared refuge use, brief associations). Fission–fusion characterizes species that live in groups, but for whom group composition is fluid in terms of size or membership. Stable refers to species living in groups that experience little or no change in group composition or size.

The different infection measures reported across studies were abundance, presence/absence, richness and risk of infection. Abundance was defined as the number of parasites per individual with an abundance of zero indicating the absence of infection; presence/absence was a binary variable referring to the presence (parasite abundance ≥ 1) or absence (parasite abundance $= 0$) of infection; richness was defined as the number of parasite taxa recorded per individual and was zero if no parasites were detected; and risk of infection was defined as the probability that an individual became infected during a specified study period. Finally, parasite transmission mode was classified as close, nonclose, intermediate host, vector or multiple following (Lindenfors et al., 2007; Pedersen, Altizer, Poss, Cunningham, & Nunn, 2005). Close transmission refers to highly contagious parasites spread by close proximity (e.g. via respiratory droplets) or direct physical contact (e.g. biting, scratching, mating) such as the bacterium *Mycobacterium bovis* [causative agent of bovine tuberculosis] in badgers, *Meles meles* (Weber et al., 2013). Nonclose transmission refers to parasites spread by soil, water, faeces, fomites or other forms of environmental contamination, such as the protozoan coccidia in Grant's gazelles, *Nanger granti* (Williams et al., 2017). Intermediate host refers to parasites with a transmission cycle involving an intermediate host (e.g. insect or gastropod) and trophic transmission, such as the helminth *Rictularia halli* in chipmunks, *Tamias striatus* (Grear et al., 2013). Vector refers to parasites whose transmission relies on a biting arthropod (e.g. mosquito or tick), such as the protozoan *Hepatozoon tuatarae* in tuataras (Godfrey et al., 2010). Finally, the multiple category was assigned to parasites with multiple transmission modes or to effect sizes that were derived from analyses in which parasites with different transmission modes were aggregated.

Statistical Analysis

All statistical analyses were performed in R version 3.6.1 (R Core Team 2019). We used random-effects models (REMs) to test the overall relationship between social behaviour measured by social network metrics and parasitism. Before estimating the average effect size and heterogeneity among effect sizes, we identified the best random effects structure for our REM by fitting different models using restricted maximum likelihood estimation (function rma.mv, R package metafor, Viechtbauer, 2010), and then comparing these models using likelihood ratio tests (Zuur 2009; Pinheiro & Bates 2000). To do this, first, we ran an intercept-only model without random effects with the following structure: rma.mv ($y_i \sim 1$, v_i) with y_i the observed effect sizes and v_i the corresponding sampling variance. Next, we included as random effects either observation nested within study, host species or parasite type in separate REMs to account for study pseudoreplication, host phylogeny and parasite taxonomy, respectively. These three random effects significantly improved the intercept-only model (likelihood ratio test, study/observation: $\chi^2_1 = 220.05$, $P < 0.0001$; host species: $\chi^2_1 = 202.24$, $P < 0.0001$; parasite type: $\chi^2_1 = 5.70$, $P = 0.017$). Therefore, we ran REMs with combinations of study/observation and host species or study/observation and parasite type, and both of these bivariate models performed significantly better than the single REMs (bivariate versus single; host species: $\chi^2_1 = 26.26$, $P < 0.0001$; parasite type: $\chi^2_1 = 222.41$, $P \leq 0.001$). Finally, we ran a REM with all three random factors and this multivariate model performed significantly better than the bivariate REMs including host species and study/observation ($\chi^2_1 = 5.46$, $P = 0.02$) or parasite type and study/observation ($\chi^2_1 = 6.85$, $P = 0.009$). We therefore retained the REM model with all three random factors for our meta-analysis. To test for the presence of significant effect size heterogeneity in the final REM, we used Cochran's *Q*, a nonparametric test of interobservation variability (Cochran 1954). We estimated the percentage of true heterogeneity using the I^2 statistic (Borenstein, Hedges, Higgins, & Rothstein, 2009), and then partitioned the total I^2 into the relative contribution of each random factor ($I^2_{species}$, I^2_{study}, $I^2_{observation}$) following Nakagawa and Santos (2012). $I^2 = 25$, 50 and 75% are considered as low, moderate and high contributions, respectively (Higgins, Thompson, Deeks, & Altman, 2003).

We tested how host- and parasite-related moderators (social network metric, social behaviour measure, host social organization, infection measure and transmission mode) affected the relationship between host social behaviour and parasite infection using univariate mixed-effects models (MEMs). As recommended by Zuur (2009), because these MEMs had similar random-effect structures but differed in their fixed-effect specification, we fitted these models using maximum likelihood and compared them using the Akaike information criterion (AIC) corrected for small sample size (AICc, Burnham & Anderson, 2002). Next, we refitted the models using restricted maximum likelihood to obtain unbiased estimates of variance components and then tested whether each moderator explained significant effect size heterogeneity using Cochran's *Q*. To quantify the variation in effect size explained per moderator, we calculated the proportional reduction in the summed variance components from each MEM compared with the summed variance components of the REM, equivalent to a pseudo-R^2 value (Bentz et al., 2016). We also reran each MEM without the intercept to calculate whether each moderator level differed from zero, enabling us to discern how the distribution of effect sizes differed by host social organization, infection measure, parasite transmission mode, etc. When the mean effect size for a given level of a moderator does not differ significantly from zero, this indicates a relatively weak influence of social behaviour on parasitism for effects characterized by this level of the moderator.

We assumed correlations of zero among effect sizes (function impute_covariance_matrix, R package ClubSandwich, Pustejovsky, 2019) and dealt with possible nonindependence among effect sizes derived from the same groups of individuals by calculating robust standard errors (R package metafor, Vietchbauer, 2010). For all models, mean effect sizes are presented with 95% confidence

intervals; *P* values and test statistics are based on robust tests and confidence intervals of the model coefficients. Results were back-transformed from Fisher's *Z* to *r* for easier interpretation (function fisherz2r, R package psych, Revelle, 2019).

Controlling for Phylogenetic Signal

Closely related host species may have similar relationships between social behaviour and parasite infection, and therefore might show similar effect sizes, so we performed phylogenetic versions of the REM and univariate MEMs to deal with the potential phylogenetic dependence of host effect sizes. To do this, we calculated a mean effect size for each species by weighting each observation by the corresponding sample size. We then estimated the phylogenetic signal in the distribution of effect sizes by estimating Pagel's λ (Molina-Venegas & Rodríguez, 2017; Pagel, 1999). We compared Pagel's model against two alternatives, a model with no phylogenetic signal ($\lambda = 0$) and a Brownian motion model with complete phylogenetic signal ($\lambda = 1$) using likelihood ratio tests (R package geiger, function fitContinuous, Harmon, Weir, Brock, Glor, & Challenger, 2008). The maximum-likelihood estimate of λ was very close to 0 ($\lambda < 0.00007$) and likelihood ratio tests suggested this estimate did not differ from the no phylogenetic signal model ($\chi^2_1 = 0$, $P = 0.99$), but we could also not reject the Brownian motion model ($\chi^2_1 = 3.65$, $P = 0.057$). As such, we accounted for phylogenetic nonindependence in our analyses by specifying the covariance structure of the host species random effect using the correlation matrix of our phylogeny (function vcv.phylo, R package geiger), which is equivalent to a phylogenetic meta-analysis. While it would have been interesting to also investigate the effect of parasite phylogeny, parasites were not always identified to species level. Moreover, our understanding of evolutionary relationships among parasites is less complete than for hosts.

Model Sensitivity

The sensitivity of meta-analytical studies is vulnerable to outliers and influential data points. As such, we evaluated the sensitivity of our analyses by comparing fitted models with and without effect sizes that we defined as influential outliers. Influential outliers are often defined as effect sizes with leverage greater than two times the average value and standardized residual values exceeding 3.0 (Aguinis, Gottfredson, & Joo, 2013; Stevens, 1984; Viechtbauer & Cheung, 2010). However, none of our observations met both criteria (top-right corner of Fig. A2a), although some effect sizes deviated greatly from the mean. For this reason, we explored an alternative measure of outliers, Cook's distance (D_i), which indicates the relative influence of each effect size on the summary estimate. A standard rule of thumb is that D_i values greater than three times the mean D_i may be potential outliers. Fifteen effect sizes exceeded this threshold (see Fig. A2b), so we ran exploratory analyses after their exclusion as a sensitivity check. We saw no substantial changes in the summary estimate of the REM or the results of the moderators, except for the smaller, but still significant mean effect sizes, and the ranking order of the univariate MEMs. These secondary analyses are available in the Appendix.

Publication Bias

We also tested for publication bias, the preferential publication of significant over nonsignificant results or large over small effect sizes. We used an extension of the Egger's regression test (Egger, Smith, Schneider, & Minder, 1997) for multivariate/multilevel models by adding a measure of precision (i.e. the square-root of the sampling variance) as a moderator in our REM with study/observation, host species and parasite type included as random effects. When the intercept of the Egger's regression test deviates significantly from zero, the overall relationship between precision and effect size is considered asymmetrical, and therefore, biased (Sterne & Egger, 2005). We considered analyses to be biased if the intercept differed from zero at $P = 0.10$ (as in Egger et al., 1997).

RESULTS

General Patterns

From 23 study units (representing 18 articles) included in our meta-analysis, we extracted 210 effect sizes on 16 host species (Table 1). We found no evidence of publication bias among studies reporting associations between social network metrics and measures of parasite infection (extended Egger's regression: mean $(r) \pm SE = -0.42 \pm 0.52$, $t = 0.81$, $P = 0.433$). However, a majority of studies focused on vertebrates, especially mammals, with very few birds and invertebrates represented (*N* species = 16; mammals, $N = 11$; reptiles, $N = 3$; birds, $N = 1$; insects, $N = 1$). In terms of parasite type, fewer studies used arthropods (*N* studies = 6, *N* observations = 17), protozoa (*N* studies = 7, *N* observations = 28) or pathogenic bacteria (*N* studies = 7, *N* observations = 49) as parasite models; while half of the observations focused on helminths (*N* studies = 9, *N* observations = 108). Characteristics of the focal hosts and parasites and study design elements were also unbalanced across studies. In terms of social network metric used, more than half of the studies focused on strength (*N* studies = 19) and degree (*N* studies = 13). The behaviours used to build networks were fairly evenly distributed, as was the type of social organization of the focal hosts. In contrast, infection measures were highly unbalanced, with over half of studies quantifying parasite presence or absence (*N* studies = 16), and relatively few reporting richness (*N* studies = 2) or risk of infection (*N* studies = 3). Furthermore, most studies reported on parasites with multiple transmission modes (*N* studies = 10) or parasites transmitted via nonclose contact (e.g. transmission via faecal contamination; *N* studies = 14), while fewer studies included parasites transmitted via close contact, vectors or intermediate hosts (*N* studies ≤ 2).

Overall Effect of Social Behaviour on Parasitism

Our REM with study/observation, host species and parasite type as random effects showed a significant and positive effect of social behaviour measured by social network metrics on parasite infection at the individual level ($t = 2.98$, $P = 0.009$; Table 1, Fig. 2). Because we could not distinguish between models with ($\lambda = 1$) and without ($\lambda = 0$) a phylogenetic signal, we report results from both nonphylogenetically and phylogenetically controlled REMs. Controlling for host phylogeny improved the fit of the REM (nonphylogenetic REM: AICc = 58.34; phylogenetic REM: AICc= 52.84), and increased the point estimate of *r* ($t = 3.33$, $P = 0.029$; Table 1, Fig. 2).

The variance components for the study level, the parasite level and the observation level random effects made a small contribution to the total variance in effect size (Table 1). The host species random effect, on the other hand, made a much larger contribution to the variance in effect size (Table 1). Finally, although there was a significant relationship between social behaviour and parasitism across studies, there was a high degree of heterogeneity among effect sizes ($Q_{209} = 616.66$; Table 1, Fig. 2), suggesting that differences between studies might contribute to this variation.

Table 1
Results from nonphylogenetic and phylogenetic multilevel random-effects models (no moderator variables included)

Type	*N* obs	*N* studies	*N* host species	Mean (*r*)	Lower CI (2.5%)	Upper CI (97.5%)	$I^2_{species}$(%)	I^2_{study}(%)	I^2_{obs}(%)	$I^2_{parasite}$(%)	I^2_{total}(%)	Pagel's λ
Nonphylogenetic REM	210	23	16	0.221	0.064	0.367	56.14	13.85	6.87	8.14	84.99	–
Phylogenetic REM	210	23	16	0.317	0.055	0.539	74.83	6.77	4.68	3.96	90.25	0.00007

Mean (*r*) and 95% confidence interval (CI) are presented along with the number (*N*) of observations, studies and host species, the ratio of true heterogeneity among effect sizes (I^2_{total}) and the contribution from each random factor ($I^2_{species}$, I^2_{study}, I^2_{obs}, $I^2_{parasite}$) and the phylogenetic signal index (Pagel's λ).

Sources of Heterogeneity: Effect of Moderators

To understand the potential drivers of heterogeneity in effect sizes, we included five host- and parasite-related moderators in our models: social network metric, host social behaviour measure and social organization, infection measure and parasite transmission mode. Using a model comparison approach, we found that almost none of the moderators contributed substantially to explaining the observed heterogeneity in effect sizes across studies, in both nonphylogenetic and phylogenetic MEMs (Table 2). The only case in which a moderator emerged as potentially influential was in the nonphylogenetic MEM including host social organization. This model explained 27% of the variance in effect size and was the top-ranked MEM in the nonphylogenetic model comparison. Solitary-but-social species had relatively larger positive effect sizes (nonphylogenetic MEM without intercept: mean [95%CI], $r = 0.44$ [0.05–0.84], $P = 0.032$; Fig. 3) compared to species with stable or fission–fusion social organization. Thus, for solitary-but-social species, the effect of social behaviour on parasitism seemed to be larger than for any other type of social organization. However, the Cochran's Q test was not significant ($Q_M = 1.983$, $P = 0.177$). Furthermore, the intercept-only models could not be excluded from the top-ranked REMs, for both the phylogenetic and the nonphylogenetic comparisons (ΔAICc < 2; Table 2).

Although no moderator explained significant variation in effect sizes, there were clear trends for some moderators where effect sizes appeared to depend on specific host or parasite traits. For example, parasite infection measured by presence/absence tended to be more strongly associated with social behaviour (i.e. larger effect sizes with a 95% CI not overlapping zero) than infection measured using richness, abundance or risk of infection (Fig. 3). Similarly, eigenvector centrality and strength were two social network metrics that did not overlap with zero suggesting a stronger association with parasitism than for betweenness, closeness or degree (Fig. 3). Likewise, for host social behaviour measure, contact and distance did not overlap with zero for the phylogenetic MEM, suggesting a stronger association with parasitism than for space use (Fig. 3).

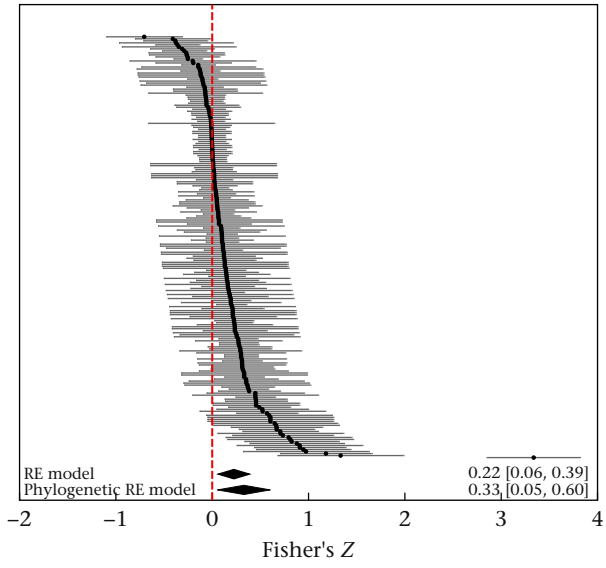


Figure 2. Forest plot of effect sizes (dots) and confidence intervals (bars) for each observation and the mean effect size (Fisher's Z) and 95 % confidence intervals for the nonphylogenetically controlled and phylogenetically controlled random-effects models (REMs; diamonds). The red dashed line is a zero-effect line (Fisher's Z = 0).

DISCUSSION

Animals are exposed to a variety of parasites that can impact their survival and reproduction. Social behaviour plays a critical role in parasite transmission and SNA can help reveal important and nuanced ways in which social behaviour shapes infection risk (Drewe, 2009; VanderWaal et al., 2016). Given the growing number of studies on the relationship between social network position and parasite infection in wild animal systems, our goal was to identify general patterns emerging from this body of work. In particular, we used a meta-analytical approach to quantify consistencies and inconsistencies among study findings (Nakagawa & Poulin, 2012). Overall, we found that across all studies, social behaviour, quantified using common social network metrics, was significantly and positively associated with parasite infection. However, current studies are biased towards certain groups of hosts (e.g. mammals) and parasites (e.g. helminths), and there is a large degree of heterogeneity in effect sizes among studies. Interestingly, none of the moderator variables associated with host or parasite traits that we examined significantly explained this heterogeneity, indicating that there are important gaps in our current understanding of the factors that influence the strength of relationships between social network position and parasitism. Based on these patterns, we suggest three strategies that might help advance our understanding of social networks and parasite transmission, including accounting for the alignment between host social behaviour and parasite transmission mode, diversifying the host–parasite systems studied and approaching social network–parasite studies in a more systematic manner. Addressing all three of these issues may be crucial for understanding heterogeneity in reported patterns and for designing future studies.

Our primary observation that, across studies, social behaviour quantified by SNA is positively associated with parasite infection suggests that individuals engaging in more social interactions, encountering a higher diversity of contacts, or sharing space with more conspecifics, experience greater rates of parasite infection. Although there was significant heterogeneity in effect sizes, this result held in both nonphylogenetic and phylogenetic models that included parasite and host identity as random effects, suggesting a general and robust pattern across host and parasite taxa. These findings are consistent with the results from meta-analyses focused on group size as a proxy of social behaviour (Cote & Poulin, 1995; Patterson & Ruckstuhl, 2013; Rifkin et al., 2012). However, the magnitude of the mean effect size estimated across studies in our analysis (nonphylogenetic and phylogenetic: $r = 0.22$ and 0.33) was

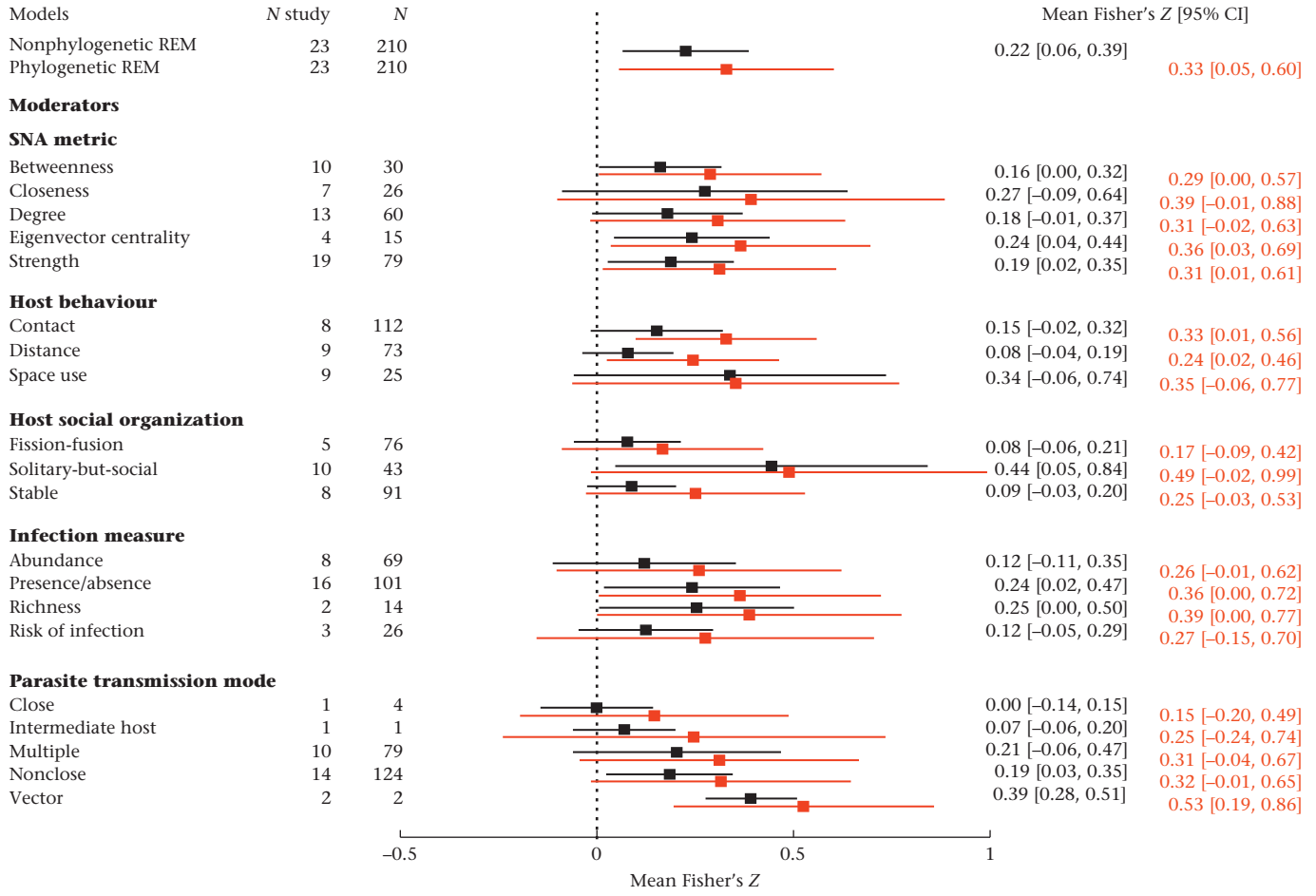


Figure 3. Mean ± 95% confidence interval effect size (Fisher's Z) for each level of five moderators. Results for the nonphylogenetic models (black) and phylogenetic models (red) are presented. Each moderator was analysed using a separate meta-regression. Nonphylogenetic and phylogenetic meta-analytic means (N = 210) were calculated without including any moderator.

nearly twice as large as the mean effect size estimated across studies focused on group size (nonphylogenetic and phylogenetic: $r = 0.1$ and 0.19; Rifkin et al., 2012). The larger mean effect size in our study could reflect the higher degree of subtlety in social behaviour captured by social network metrics compared to group size. This finding supports the prevailing opinion that SNA provides a more sensitive tool for understanding links between social behaviour and parasite infection.

We looked at five different host- and parasite-related moderator variables that we predicted would explain the observed heterogeneity in effect sizes and were surprised to find little to no support for a general effect of these variables on the relationship between host social behaviour and parasite infection. Host social organization was the only moderator that emerged as potentially influential. The effect of social behaviour on parasite infection tended to be stronger in solitary-but-social species compared to species living in stable social groups. A recent study by Sah, Leu, Cross, Hudson, and Bansal (2017) found that across 666 social networks analysed, solitary species showed the most variation in numbers of social partners, which could explain the slightly stronger relationship between social behaviour and infection that we observed in solitary animals. However, this result should be interpreted with caution since the level of heterogeneity among host social organization types was not significant and an intercept-only model fitted the data equally well. Interestingly, we found no specific patterns related to host social behaviours used to construct networks (e.g. contact versus space use) or network metrics (e.g. degree versus closeness). Although within each category, some levels of behaviour (e.g. contact, distance) or social network metric (e.g. eigenvector centrality, strength) had larger mean effect sizes than other levels (Fig. 3), these differences were not significant (Table 2). Similarly, there was no impact of infection measure or parasite transmission mode on the observed heterogeneity in effect sizes. This is in contrast to three meta-analyses using group size as a proxy of social behaviour which found that parasite richness had a weaker association with group size than other measures of parasitism (prevalence, intensity, abundance, Patterson & Ruckstuhl, 2013; Rifkin et al., 2012), and that parasite transmission mode affected the direction and magnitude of the relationship between parasitism and group size (Cote & Poulin, 1995; Patterson & Ruckstuhl, 2013; Rifkin et al., 2012). One of these studies also found that certain classes of hosts (birds) had larger mean effect sizes (Rifkin et al., 2012).

The absence of strong patterns related to host and parasite moderators in our analysis could be due to the high specificity of the host–parasite–social connectivity interaction (Godfrey, 2013; White et al., 2017). Social behaviour can both facilitate and impede parasite transmission depending on the parasite's mode of transmission. For instance, in primates, allogrooming plays an important hygienic function against ectoparasites, in addition to its acknowledged social function, but this behaviour can also increase the transmission of other pathogens (Nunn & Altizer, 2006;

Table 2
Univariate rankings of nonphylogenetic and phylogenetic mixed-effects models (MEMs) predicting effect size for the relationship between social behaviour (measured by social network metrics) and parasite infection for the full data set ($N = 23$ studies, 210 effect sizes)

Moderators	k	$I^2_{species}$	I^2_{study}	I^2_{obs}	$I^2_{parasite}$	I^2_{total}	Q_M	df	P value	AICc	ΔAICc	w_i	R^2
Nonphylogenetic models													
Host social organization	3	39.56	18.11	8.65	14.16	80.49	1.983	2	0.177	56.53	0	0.65	0.27
Intercept (REM)	1	56.14	13.85	6.87	8.13	84.99	8.879	1	0.009	58.34	1.81	0.26	0.00
Social behaviour measure	3	54.47	14.66	7.3	8.04	84.47	0.914	2	0.425	61.61	5.08	0.05	0.03
Social network metric	5	56.93	13.16	6.59	8.1	84.79	0.815	4	0.541	63.56	7.03	0.02	0.00
Infection measure	4	60.59	10.98	7.99	4.85	84.41	0.565	3	0.649	64.57	8.04	0.01	0.03
Parasite transmission mode	5	57.53	10.40	7.44	9.04	84.41	4.740	4	0.018	65.16	8.63	0.01	0.03
Phylogenetic models													
Intercept (REM)	1	74.83	6.77	4.69	3.96	90.25	7.103	1	0.018	52.84	0	0.64	0.00
Host social organization	3	62.12	11.03	6.07	7.28	86.50	1.145	2	0.348	54.38	1.54	0.30	0.14
Social behaviour measure	3	76.4	6	4.58	3.75	90.73	0.545	2	0.593	59.13	6.29	0.03	0.01
Infection measure	4	77.94	4.75	5.16	2.37	90.22	0.264	3	0.850	60.19	7.35	0.02	0.01
Social network metric	5	74.83	7.18	4.43	3.90	90.33	0.747	4	0.580	60.42	7.58	0.02	0.01
Parasite transmission mode	5	76.00	4.74	4.84	4.79	90.37	2.323	4	0.121	61.88	90.37	0.01	0.02

Each model contained only one moderator (host social organization, social behaviour measure, social network metric, infection measure or parasite transmission mode) and the intercept. Competing models are ranked by AICc. Also listed are the number of model coefficients (k), the ratio of true heterogeneity among effect sizes (I^2_{total}) and the contribution from each random factor ($I^2_{species}$, I^2_{study}, I^2_{obs}, $I^2_{parasite}$), tests of moderator significance (Cochran's QM), Akaike weights (wi) and the pseudo-R2 statistic for each MEM.

Poirotte et al., 2017). Two studies included in our meta-analysis illustrate this nuance. In Japanese macaques, *Macaca fuscata*, the same social network measure, number of grooming partners or in-degree, was negatively correlated with lice load (Duboscq et al., 2016), but was not correlated with gastrointestinal helminth burden (MacIntosh et al., 2012). These types of patterns suggest that the degree to which the social network metric matches focal parasite biology can contribute to variation in effect size direction and magnitude.

Indeed, the level of congruence between a particular social behaviour or social network metric and the transmission mode of a focal parasite is among the most important determinants of the efficacy with which SNA can be used to understand parasite transmission (Craft, 2015). For example, a social network based on spatial proximity might be less likely to represent the dynamics of a highly contagious parasite transmitted by direct contact, compared to one transmitted by indirect contact (i.e. parasites that require an intermediate host or vector for transmission). The reciprocal is also true for contact-based networks and indirectly transmitted parasites. In support of this idea, when add N rule between behaviour and transmission mode. matching to reclassify each observation in our meta-analysis as either a host–parasite match (e.g. contact-based network associated with a parasite transmitted by direct contact; Fig. A3) or mismatch (e.g. proximity-based network and a parasite transmitted by direct contact; Fig. A3), we found that observations classified as 'matches' had higher effect sizes (median = 0.20, $N = 70$) than those classified as 'mismatches' (median = 0.11, $N = 61$). Moreover, the distribution of effect sizes from these two populations were significantly different (one-tailed Mann–Whitney U test: $U = 2541.5$, $P = 0.03$), implying that studies are more likely to find a strong relationship between host social networks and parasite burden when the social network metric matches parasite biology. We also looked at the relative proportion of 'match' observations present for different moderator variables and found that the highest proportion of matches (0.8) was associated with the solitary-but-social category of the host social organization variable, the only moderator for which there was some support for its involvement in explaining effect size variation across studies. Thus, mismatched host–parasite pairings probably contributed to the poor performance of our moderators in explaining heterogeneity across studies. More generally, our findings reinforce the idea that social networks should be carefully constructed based on the biology of both the host and the parasite under consideration. Importantly, as the body of literature on social networks and parasitism grows, future meta-analyses can help generate new hypotheses about the drivers of variation in sociality–parasitism relationships by evaluating the explanatory power of meaningfully paired host and parasite traits.

Our analysis also revealed the need to diversify both the host and parasite taxa studied in the context of social networks and infection. While there was no evidence of publication bias with respect to effect size magnitude, the majority of observations were on mammalian hosts and helminth parasites. However, because the parasite costs of social behaviour can vary greatly depending on host social organization, host social network structure and parasite transmissibility (Sah, Leu, et al., 2017; Sah, Mann, & Bansal, 2017), the observed magnitude of the positive relationship between network position and infection status might depend on the diversity of host–parasite systems examined. In fact, we saw that host species made a significant and large contribution to the observed heterogeneity in effect sizes across studies (Table 1), which suggests that characteristics of the focal host species play a key role in determining the relationship between social behaviour and infection status. Although we saw very little effect of parasite taxa on observed patterns (Table 1), this could be because parasites were often not identified to species level (ca. 50% of observations) and we had to group them into broad taxonomic categories (i.e. bacteria, arthropods, etc.). Thus, a more refined understanding of the biology and taxonomy of parasites is likely also to contribute to a better understanding of the complex relationship between sociality and parasitism. Finally, inclusion of a greater diversity of host–parasite systems might confirm some of the trends that we observed in our analysis. For example, for our parasite transmission mode moderator, nonclose contact appeared to have a weak influence on the relationship between host social behaviour and parasite infection; however, nearly 60% of observations focused on parasites transmitted by nonclose contact, while another 38% focused on parasites with multiple transmission modes (see Fig. 3). This extreme bias, where only 2% of observations involved all three of the other key modes of transmission combined (close, intermediate host, vector), probably limited our ability to uncover any potential influential role of parasite transmission mode. Focusing future empirical research on the understudied parasite and host groups we identify in this study will help advance our understanding of the sources of heterogeneity in host social behaviour–parasite transmission interactions.

Methodological differences represent another potential source of heterogeneity in effect sizes observed across SNA–parasite studies. First, compared to group size, SNA offers a far greater diversity of ways in which social behaviour can be measured, making generalizations across host–parasite systems challenging. Thus, a feature that represents the strength of SNA, as it offers a greater level of precision and realism (White et al., 2017), can also be its weakness. For instance, within our data set, the metric 'degree' was used in eight different forms (e.g. degree, in-degree, out-degree, weighted degree, etc.) and calculated based on at least nine different types of behaviours (e.g. physical contact, nearest neighbour, refuge sharing, etc.). The field of SNA therefore has the potential to suffer from what Simmons, Nelson, and Simonsohn (2011) described as the researcher's degree of freedom, a phenomenon that arises when researchers must rely on arbitrary methodological decisions, which in this context can happen at multiple stages, including what behaviour to record, at what frequency, what type of social network to use (e.g. weighted, directed, etc.) and what metric (e.g. degree, in-degree, degree with infected individuals, etc.).

A second methodological source of heterogeneity may be differences in how SNA studies test hypotheses. Social network data are nonindependent (for an individual to have n edges in a network requires n other individuals to have at least one edge) and thus violate the assumptions of data independence in parametric statistics (Croft, Madden, Franks, & James, 2011; James, Croft, & Krause, 2009). Consequently, statistical procedures, such as randomization or simulations, are useful for robust effect size estimation (Croft et al., 2011; Farine, 2017; Farine & Aplin, 2019; James et al., 2009). However, these tools have not yet gained widespread usage in the SNA–parasitism literature. For example, of the 18 publications used in our meta-analysis, only 44% ($N = 8$) accounted for statistical nonindependence. Thus, differing statistical approaches may have increased the noise in our effect size data set, limiting our ability to detect sources of heterogeneity. Interestingly, we found no particular temporal trend in our data set indicating that the use of randomization or simulation-based methods is increasing over time in SNA–parasite studies.

Conclusion

Our meta-analysis of relationships between host social behaviour, quantified by SNA, and parasite infection supports the hypothesis that social behaviour increases parasite infection risk at the individual level. Furthermore, the magnitude of the mean effect size suggests that SNA is better able to capture this relationship than is group size. However, the large degree of heterogeneity in effect sizes observed across studies and the absence of host and parasite traits that explained this variation highlights that important gaps remain in our understanding of the drivers of variation in interactions between social hosts and parasites. Appropriately pairing host behaviours and parasite characteristics, diversifying the host–parasite systems studied and the use of more systematic methods across studies are three strategies that might help the field build a more refined understanding of how host social behaviour and parasitism interact. Indeed, identifying the sources of the variability in social network–parasite studies is pivotal for generating new hypothesis to further our understanding of parasite transmission dynamics and the costs and benefits of social living.

Author Contributions

V.O.E. conceived the study. L.B. and V.O.E designed the study. L.B. collected the data and performed data analysis L.B. and V. O. E. wrote the paper. All authors gave final approval for publication.

Acknowledgments

We thank D. Vasquez, A. Williams and members of the Ezenwa lab at the University of Georgia for discussions during the initial stages of the study. We also thank Daniel Becker for advice and recommendations on the statistical analysis, and K. Balasubramaniam, J. Duboscq, S. Friant and K. VanderWaal for providing additional information necessary for the meta-analysis. L.B. was supported by the French–American Fulbright Scholar Program (Alsace Program 2016–2017).

References

Adelman, J. S., Moyers, S. C., Farine, D. R., & Hawley, D. M. (2015). Feeder use predicts both acquisition and transmission of a contagious pathogen in a North American songbird. *Proceedings of the Royal Society B: Biological Sciences, 282*(1815). https://doi.org/10.1098/rspb.2015.1429

Aguinis, H., Gottfredson, R. K., & Joo, H. (2013). Best-practice recommendations for defining, identifying, and handling outliers. *Organizational Research Methods, 16*(2), 270–301. https://doi.org/10.1177/1094428112470848

Alexander, R. D. (1974). The evolution of social behavior. *Annual Review of Ecology and Systematics, 5*(171), 325–383. https://doi.org/10.1111/j.1365-294X.2011.05232.x

Altizer, S., Nunn, C. L., Thrall, P. H., Gittleman, J. L., Antonovics, J., Cunningham, A. A., … Pulliam, J. R. C. (2003). Social organization and parasite risk in mammals: Integrating theory and empirical studies. *Annual Review of Ecology, Evolution and Systematics, 34*, 517–547. https://doi.org/10.1146/annurev.ecolsys.34.030102.151725

Balasubramaniam, K., Beisner, B., Vandeleest, J., Atwill, E. R., & McCowan, B. (2016). Social buffering and contact transmission: Network connections have beneficial and detrimental effects on Shigella infection risk among captive rhesus macaques. *PeerJ, 2016*(10), 1–26. https://doi.org/10.7717/peerj.2630

Bentz, A. B., Becker, D. J., & Navara, K. J. (2016). Evolutionary implications of interspecific variation in a maternal effect: A meta-analysis of yolk testosterone response to competition. *Royal Society Open Science, 3*(11). https://doi.org/10.1098/rsos.160499

Borenstein, M., Hedges, L. V., Higgins, J. P. T., & Rothstein, H. R. (2009). *Introduction to meta-analysis*. https://doi.org/10.1002/9780470743386. Retrieved from.

Burnham, K. P., & Anderson, D. B. (2002). *Model selection and multimodel inference: A pratical information-theoretic approach* (2nd ed.). New York, NY: Springer-Verlag https://www.springer.com/gb/book/9780387953649.

Cochran, W. G. (1954). The combination of estimates from different experiments. *Biometrics, 10*(1), 101–129. https://doi.org/10.2307/3001666

Corner, L. A. L., Pfeiffer, D. U., & Morris, R. S. (2003). Social-network analysis of Mycobacterium bovis transmission among captive brushtail possums (*Trichosurus vulpecula*). *Preventive Veterinary Medicine, 59*(3), 147–167. https://doi.org/10.1016/S0167-5877(03)00075-8

Cote, I. M., & Poulin, R. (1995). Parasitism and group size in social animals: A meta-analysis. *Behavioral Ecology, 6*(2), 159–165. https://doi.org/10.1093/beheco/6.2.159

Craft, M. E. (2015). Infectious disease transmission and contact networks in wildlife and livestock. *Philosophical Transactions of the Royal Society B: Biological Sciences, 370*(1669). https://doi.org/10.1098/rstb.2014.0107

Croft, D. P., Madden, J. R., Franks, D. W., & James, R. (2011). Hypothesis testing in animal social networks. *Trends in Ecology & Evolution, 26*(10), 502–507. https://doi.org/10.1016/j.tree.2011.05.012

Drewe, J. A. (2009). Who infects whom? Social networks and tuberculosis transmission in wild meerkats. *Proceedings of the Royal Society B: Biological Sciences, 277*(1681), 633–642. https://doi.org/10.1098/rspb.2009.1775

Duboscq, J., Romano, V., Sueur, C., & Macintosh, A. J. J. (2016). Network centrality and seasonality interact to predict lice load in a social primate. *Scientific Reports, 6*(April 2015), 1–13. https://doi.org/10.1038/srep22095

Egger, M., Smith, G. D., Schneider, M., & Minder, C. (1997). Bias in meta-analysis detected by a simple, graphical test. *British Medical Journal, 315*(7109), 629–634. https://doi.org/10.1136/bmj.316.7129.469

Ezenwa, V. O. (2004). Host social behavior and parasitic infection: A multifactorial approach. *Behavioral Ecology, 15*(3), 446–454. https://doi.org/10.1093/beheco/arh028

Farine, D. R. (2017). A guide to null models for animal social network analysis. *Methods in Ecology and Evolution, 8*(10), 1309–1320. https://doi.org/10.1111/2041-210X.12772

Farine, D. R., & Aplin, L. M. (2019). Spurious inference when comparing networks. *Proceedings of the National Academy of Sciences of the United States of America, 116*(34), 16674–16675. https://doi.org/10.1073/pnas.1900143116

Farine, D. R., & Whitehead, H. (2015). Constructing, conducting and interpreting animal social network analysis. *Journal of Animal Ecology, 84*(5), 1144–1163. https://doi.org/10.1111/1365-2656.12418

Fenner, A. L., Godfrey, S. S., & Bull, C. M. (2011). Using social networks to deduce whether residents or dispersers spread parasites in a lizard population. *Journal of Animal Ecology, 80*(4), 835–843. https://doi.org/10.1111/j.1365-2656.2011.01825.x

Friant, S., Ziegler, T. E., & Goldberg, T. L. (2016). Primate reinfection with gastrointestinal parasites: Behavioural and physiological predictors of parasite acquisition. *Animal Behaviour, 117*(8), 105–113. https://doi.org/10.1016/j.anbehav.2016.04.006

Godfrey, S. S. (2013). Networks and the ecology of parasite transmission: A framework for wildlife parasitology. *International Journal for Parasitology: Parasites and Wildlife, 2*(1), 235–245. https://doi.org/10.1016/j.ijppaw.2013.09.001

Godfrey, S. S., Moore, J. A., Nelson, N. J., & Bull, C. M. (2010). Social network structure and parasite infection patterns in a territorial reptile, the tuatara (*Sphenodon punctatus*). *International Journal for Parasitology, 40*(13), 1575–1585. https://doi.org/10.1016/j.ijpara.2010.06.002

Grear, D. A., Luong, L. T., & Hudson, P. J. (2013). Network transmission inference: Host behavior and parasite life cycle make social networks meaningful in disease ecology. *Ecological Applications, 23*(8), 1906–1914. https://doi.org/10.1890/13-0907.1

Griffin, R. H., & Nunn, C. L. (2012). Community structure and the spread of infectious disease in primate social networks. *Evolutionary Ecology, 26*(4), 779–800. https://doi.org/10.1007/s10682-011-9526-2

Harmon, L. J., Weir, J. T., Brock, C. D., Glor, R. E., & Challenger, W. (2008). GEIGER: Investigating evolutionary radiations. *Bioinformatics, 24*(1), 129–131. https://doi.org/10.1093/bioinformatics/btm538

Higgins, J. P. T., Thompson, S. G., Deeks, J. J., & Altman, D. G. (2003). Measuring inconsistency in meta-analyses. *Journal of Intelligent Information Systems, 327*(2), 159–184. https://doi.org/10.1007/s10844-006-2974-4

James, R., Croft, D. P., & Krause, J. (2009). Potential banana skins in animal social network analysis. *Behavioral Ecology and Sociobiology, 63*(7), 989–997. https://doi.org/10.1007/s00265-009-0742-5

Krause, J., James, R., Franks, D., & Croft, D. (Eds.). (2015). *Animal social networks*. Oxford, U.K.: Oxford University Press. https://doi.org/10.1093/acprof:oso/9780199679041.001.0001.

Leu, S. T., Kappeler, P. M., & Bull, C. M. (2010). Refuge sharing network predicts ectoparasite load in a lizard. *Behavioral Ecology and Sociobiology, 64*(9), 1495–1503. https://doi.org/10.1007/s00265-010-0964-6

Lindenfors, P., Nunn, C. L., Jones, K. E., Cunningham, A. A., Sechrest, W., & Gittleman, J. L. (2007). Parasite species richness in carnivores: Effects of host body mass, latitude, geographical range and population density. *Global Ecology and Biogeography, 16*(4), 496–509. https://doi.org/10.1111/j.1466-8238.2006.00301.x

Loehle, C. (1995). Social barriers to pathogen transmission in wild animal populations. *Ecology, 76*(2), 326–335. https://doi.org/10.2307/1941192

MacIntosh, A. J. J., Jacobs, A., Garcia, C., Shimizu, K., Mouri, K., Huffman, M. A., & Hernandez, A. D. (2012). Monkeys in the middle: Parasite transmission through the social network of a wild primate. *PloS One, 7*(12), 15–21. https://doi.org/10.1371/journal.pone.0051144

Molina-Venegas, R., & Rodríguez, M. (2017). Revisiting phylogenetic signal; strong or negligible impacts of polytomies and branch length information? *BMC Evolutionary Biology, 17*(1), 1–10. https://doi.org/10.1186/s12862-017-0898-y

Nakagawa, S., & Poulin, R. (2012). Meta-analytic insights into evolutionary ecology: An introduction and synthesis. *Evolutionary Ecology, 26*(5), 1085–1099. https://doi.org/10.1007/s10682-012-9593-z

Nakagawa, S., & Santos, E. S. A. (2012). Methodological issues and advances in biological meta-analysis. *Evolutionary Ecology, 26*(5), 1253–1274. https://doi.org/10.1007/s10682-012-9555-5

Nunn, C. L., & Altizer, S. (2006). Host defenses: The immune system and behavioral counterstrategies. In C. L. Nunn, & S. Altizer (Eds.), *Infectious diseases in primates* (pp. 134–175). Oxford, U.K.: Oxford University Press. https://doi.org/10.1093/acprof:oso/9780198565857.003.0005.

Otterstatter, M. C., & Thomson, J. D. (2007). Contact networks and transmission of an intestinal pathogen in bumble bee (*Bombus impatiens*) colonies. *Oecologia, 154*(2), 411–421. https://doi.org/10.1007/s00442-007-0834-8

Pagel, M. (1999). Inferring the historical patterns of biological evolution. *Nature, 401*(6756), 877–884. Retrieved from http://cat.inist.fr/?aModele=afficheN&cpsidt=1178332.

Patterson, J. E. H., & Ruckstuhl, K. E. (2013). Parasite infection and host group size: A meta-analytical review. *Parasitology, 140*(7), 803–813. https://doi.org/10.1017/S0031182012002259

Pedersen, A. B., Altizer, S., Poss, M., Cunningham, A. A., & Nunn, C. L. (2005). Patterns of host specificity and transmission among parasites of wild primates. *International Journal for Parasitology, 35*(6), 647–657. https://doi.org/10.1016/j.ijpara.2005.01.005

Pinheiro, J. C., & Bates, D. M. (2000). Theory and computational methods for linear mixed-effects models. In J. C. Pinheiro, & D. M. Bates (Eds.), *Mixed-effects models in S and S-PLUS (pp. 57–96)*. New York, NY: Springer. https://doi.org/10.1007/0-387-22747-4 .

Poirotte, C., Massol, F., Herbert, A., Willaume, E., Bomo, P. M., Kappeler, P. M., & Charpentier, M. J. E. (2017). Mandrills use olfaction to socially avoid parasitized conspecifics. *Science Advances, 3*(4), Article e1601721. https://doi.org/10.1126/sciadv.1601721

Pustejovsky, J. (2019). *clubSandwich: Cluster-Robust (Sandwich) Variance Estimators with Small-Sample Corrections. R package version 0.3.5*. Retrieved from https://CRAN.R-project.org/package=clubSandwich.

Revelle, W. (2019). *psych: Procedures for Psychological, Psychometric, and Personality Research. R package version 1.9.12*. Retrieved from https://cran.r-project.org/package=psych.

Rifkin, J. L., Nunn, C. L., & Garamszegi, L. Z. (2012). Do animals living in larger groups experience greater parasitism? A meta-analysis. *The American Naturalist, 180*(1), 70–82. https://doi.org/10.1086/666081

Rimbach, R., Bisanzio, D., Galvis, N., Link, A., Di Fiore, A., & Gillespie, T. R. (2015). Brown spider monkeys (*Ateles hybridus*): A model for differentiating the role of social networks and physical contact on parasite transmission dynamics. *Philosophical Transactions of the Royal Society B: Biological Sciences, 370*(1669). https://doi.org/10.1098/rstb.2014.0110

Rosenthal, R., & DiMatteo, M. R. (2001). Meta-Analysis: Recent developments in quantitative methods for literature reviews. *Annual Review of Psychology, 52*(1), 59–82. https://doi.org/10.1146/annurev.psych.52.1.59

Sah, P., Leu, S. T., Cross, P. C., Hudson, P. J., & Bansal, S. (2017). Unraveling the disease consequences and mechanisms of modular structure in animal social networks. *Proceedings of the National Academy of Sciences of the United States of America, 114*(16), 4165–4170. https://doi.org/10.1073/pnas.1613616114

Sah, P., Mann, J., & Bansal, S. (2017). Disease implications of animal social network structure: A synthesis across social systems. *Journal of Animal Ecology, 87*(3), 546–558. https://doi.org/10.1101/106633

Simmons, J. P., Nelson, L. D., & Simonsohn, U. (2011). False-positive psychology: Undisclosed flexibility in data collection and analysis allows presenting anything as significant. *Psychological Science, 22*(11), 1359–1366. https://doi.org/10.1177/0956797611417632

Sterne, J. A. C., & Egger, M. (2005). Regression methods to detect publication and other bias in meta-analysis. In H. R. Rothstein, A. J. Sutton, & M. Borenstein (Eds.), *Publication bias in meta-analysis: Prevention, assessment and adjustments (pp. 99–110)*. Chichester, U.K: J. Wiley. https://doi.org/10.1002/0470870168.ch6.

Stevens, J. P. (1984). Outliers and influential data points in regression analysis. *Psychological Bulletin, 95*(2), 334–344. https://doi.org/10.1037/0033-2909.95.2.334

VanderWaal, K. L., Atwill, E. R., Hooper, S., Buckle, K., & McCowan, B. (2013). Network structure and prevalence of *Cryptosporidium* in Belding's ground squirrels. *Behavioral Ecology and Sociobiology, 67*(12), 1951–1959. https://doi.org/10.1007/s00265-013-1602-x

VanderWaal, K. L., Obanda, V., Omondi, G. P., McCowan, B., Wang, H., Fushing, H., & Isbell, L. A. (2016). The strength of weak ties and helminth parasitism in giraffe social networks. *Behavioral Ecology, 27*(4), 1190–1197. https://doi.org/10.1093/beheco/arw035

Viechtbauer, W. (2010). Conducting meta-analyses in R with the metafor. *Journal of Statistical Software, 36*(3), 1–48. https://www.jstatsoft.org/article/view/v036i03.

Viechtbauer, W., & Cheung, M. W.-L. (2010). Outlier and influence diagnostics for meta-analysis. *Research Synthesis Methods, 1*(2), 112–125. https://doi.org/10.1002/jrsm.11

Weber, N., Carter, S. P., Dall, S. R. X., Delahay, R. J., McDonald, J. L., Bearhop, S., & McDonald, R. A. (2013). Badger social networks correlate with tuberculosis infection. *Current Biology, 23*(20), R915–R916. https://doi.org/10.1016/j.cub.2013.09.011

Wey, T., Blumstein, D. T., Shen, W., & Jordán, F. (2008). Social network analysis of animal behaviour: A promising tool for the study of sociality. *Animal Behaviour, 75*(2), 333–344. https://doi.org/10.1016/j.anbehav.2007.06.020

White, L. A., Forester, J. D., & Craft, M. E. (2017). Using contact networks to explore mechanisms of parasite transmission in wildlife. *Biological Reviews, 92*(1), 389–409. https://doi.org/10.1111/brv.12236

Whitehead, H. (2008). *Analyzing animal societies*. Chicago, IL: University of Chicago Press. https://doi.org/10.7208/chicago/9780226895246.001.0001

Williams, A. E., Worsley-Tonks, K. E. L., & Ezenwa, V. O. (2017). Drivers and consequences of variation in individual social connectivity. *Animal Behaviour, 133*, 1–9. https://doi.org/10.1016/j.anbehav.2017.08.021

Wohlfiel, C. K., Leu, S. T., Godfrey, S. S., & Bull, C. M. (2013). Testing the robustness of transmission network models to predict ectoparasite loads. One lizard, two ticks and four years. *International Journal for Parasitology: Parasites and Wildlife, 2*(1), 271–277. https://doi.org/10.1016/j.ijppaw.2013.09.005

Zuur, A. F., Ieno, E. N., Walker, N. J., Saveliev, A. A., & Smith, G. M. (2009). Mixed effects modelling for nested data. In A. F. Zuur, E. N. Ieno, N. J. Walker, A. A. Saveliev, & G. M. Smith (Eds.), *Mixed effects models and extensions in ecology in R* (pp. 101–142). New York, NY: Springer. https://doi.org/10.1007/978-0-387-87458-6 .

Appendix: Model Sensitivity

None of our observations met the criteria to be deemed influential outliers (Fig. A2a). However, based on Cook's distances, we removed 15 values whose D_i values were greater than three times the mean D_i (Fig. A2b). We report below the results obtained from this reduced data set and highlight in bold the results that differ from the analysis with the full data set.

Random factor selection

We were left with **21** studies (**15** host species) for a total of **195** effect sizes. We ran an intercept-only model without random

Table A1
Univariate rankings of nonphylogenetic and phylogenetic mixed-effects models (MEMs) predicting effect size for the relationship between sociality (measured by social network metrics) and parasite infection for the reduced data set ($N = 21$ studies, 195 effect sizes)

Moderators	k	I^2_{total}	Q_M	df	P	AICc	ΔAICc	w_i	R^2
Nonphylogenetic models									
Host social organization	3	57.92	2.621	2	0.114	−63.22	0	0.58	0.3
Intercept (REM)	1	66.48	7.698	1	0.015	−60.96	2.26	0.19	0
Parasite transmission mode	5	67.47	3.89e^{15}	4	0	−60.37	2.85	0.14	0
Social behaviour measure	3	66.38	0.836	2	0.457	−58.02	5.20	0.04	0
Social network metric	5	67.00	0.992	4	0.455	−57.65	5.57	0.04	0
Infection measure	4	67.67	2.810	3	0.089	−54.75	8.47	0.01	0
Phylogenetic models									
Intercept (REM)	1	88.54	4.506	1	0.015	−61.28	0	0.70	0
Parasite transmission mode	4	89.19	693.36	4	<0.0001	−57.94	3.35	0.13	0
Host social organization	2	86.93	1.666	2	0.230	−57.57	3.71	0.11	0.13
Social behaviour measure	2	88.78	0.508	2	0.614	−54.63	6.66	0.03	0
Social network metric	4	89.13	1.023	4	0.441	−54.46	6.83	0.02	0
Infection measure	3	88.91	1.098	3	0.391	−52.21	9.07	0.01	0

Each model contained only one moderator (host social organization, social behaviour measure, social network metric, infection measure or parasite transmission mode) and the intercept. Competing models are ranked by AICc along with the number of model coefficients (k), the ratio of true heterogeneity among effect sizes (I^2_{total}); tests of moderator significance (Cochran's Q_M), Akaike weights (w_i) and the pseudo-R^2 statistic for each MEM. All values are those obtained after using the robust function with the host species as a cluster variable.

effects and compared with single random-effect models with either observation nested within study, host species identity or parasite taxonomy using likelihood ratio tests. The first two random effects significantly improved the intercept-only model (likelihood ratio test: study/observation: $\chi^2_1 = \mathbf{101.06}$, $\boldsymbol{P < 0.0001}$; host species: $\chi^2_1 = \mathbf{105.85}$, $\boldsymbol{P < 0.0001}$). The REM with parasite category as a random factor did not perform better than the model without any random factor ($\chi^2_1 = \mathbf{0}$, $\boldsymbol{P = 1}$). Therefore, we ran a REM with both study/observation and host species but it only performed significantly better than the REM with study/observation as a random effect (multiple versus single: study/observation: $\chi^2_1 = \mathbf{6.05}$, $\boldsymbol{P = 0.01}$; host species: $\chi^2_1 = \mathbf{1.26}$, $\boldsymbol{P = 0.53}$). We therefore retained the REM model including only host species as a random factor for our model sensitivity analysis.

Intercept-only multilevel random effect model

Our REM with study/observation, host species and parasite category as random effects showed a significant and positive effect of social behavior measured by social network metrics on parasite infection at the individual level (mean [95% CI], $\boldsymbol{r = 0.14}$ $\boldsymbol{[0.03 - 0.25]}$, $\boldsymbol{t = 2.78}$, $\boldsymbol{P = 0.015}$). Controlling for host phylogeny increased the point estimate of r to **0.24 (95% CI = 0.00 − 0.45, $t = 2.77$, $P = 0.05$)** but did not improve the fit of the REM (ΔAICc < 2; nonphylogenetic REM: AICc = **−61.02**; phylogenetic REM, AICc = **−61.35**). However, since we could not distinguish our model from a no-signal model ($\chi^2_1 = 0, P = 1$) or a Brownian model ($\chi^2_1 = 2.3, P = 0.13$), we chose to present the results from both the phylogenetic and nonphylogenetic models.

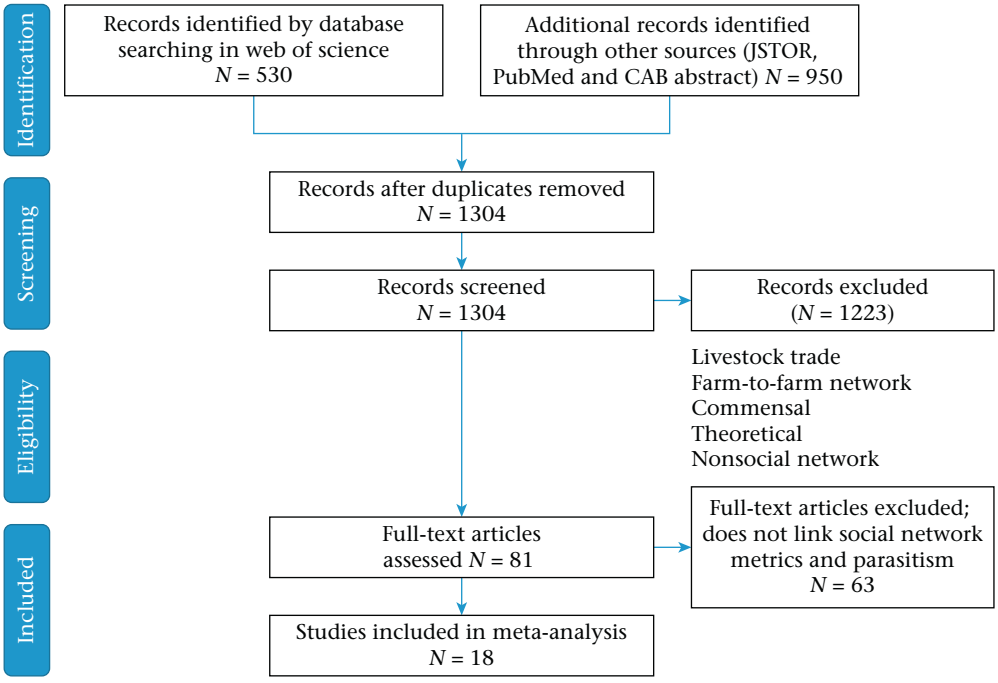


Figure A1. PRISMA diagram.

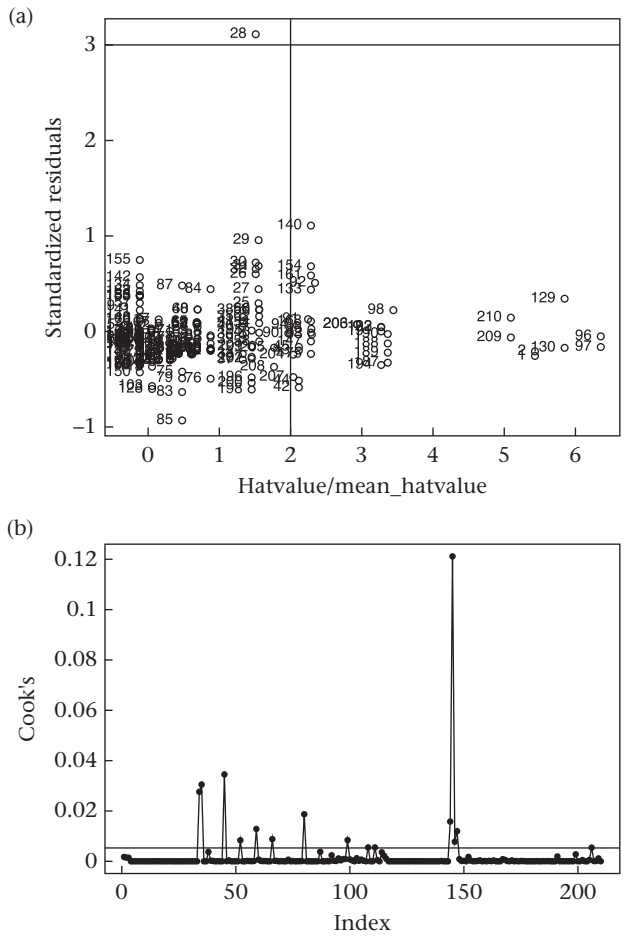


Figure A2. (a) Influential outliers (effect sizes with leverage greater than two times the average value and standardized residual values exceeding 3.0) and (b) Cook's distances, which indicate the relative influence of each effect size on the summary estimate. Distances greater than three times the mean (horizontal line) may be potential outliers. The number labelling each dot is the observation's unique identification number.

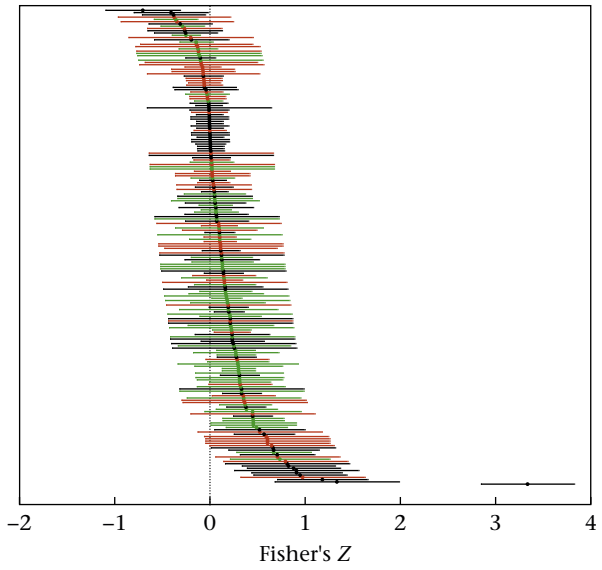


Figure A3. Caterpillar plot of observations classified by whether the focal host social behaviour and parasite transmission mode examined were matched versus mismatched. Forest plot of effect sizes (dots) and confidence intervals (bars) for each observation. Green dots and bars are observations that we classified as a match between host social behaviour and parasite transmission mode (e.g. direct contact-based network and a parasite transmitted by direct contact), while red dots and bars are observations that we considered to be a mismatch (e.g. a proximity-based network and a parasite transmitted by direct contact). Black dots and bars are observations for which the parasite transmission mode was categorized as 'multiple' and so were not classified.

There was a high degree of heterogeneity among effect sizes ($\boldsymbol{Q_{194} = 356.85, P < 0.0001}$; nonphylogenetically controlled REM: $\boldsymbol{I^2_{total} = 66.48\%}$; phylogenetically controlled REM: $\boldsymbol{I^2_{total} = 88.53\%}$), suggesting differences between studies might contribute to this variation.

Mixed-effect model selection

The large heterogeneity in our data warranted the inclusion of moderators in our models. Using a model comparison approach, we found that the only case in which a moderator emerged as potentially influential was in the nonphylogenetic MEM including host social organization. This model explained **30%** of the variation in effect size and was the top-ranked MEM in the nonphylogenetic model comparison. As in the analysis with the full data set, the solitary-but-social species had relatively larger positive effect sizes compared to stable or fission–fusion social organizations. However, the Cochran's *Q* test was not significant (Table A1). Contrary to the model comparison with the full data set, the intercept-only nonphylogenetic REM could be excluded from the top-ranked models (ΔAICc > 2; Table A1).