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ORIGINAL ARTICLE

Evolution of social behaviour in an infectious world: comparative analysis of social network structure versus parasite richness

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Abstract

Social networks capture the occurrence, direction and strength of pairwise interactions among individual animals in a group. Their structure has proven to be a key determinant of the patterns of parasite transmission and disease spread within groups. In contrast, little is known about how parasites themselves may have influenced the evolution of host social networks. Here, we use a comparative analysis among primate species to test the hypothesis that strong selective pressures exerted by parasites may have favoured the evolution of social network patterns that reduce the probability of infection. After controlling for variation in study effort and for host phylogeny, we find that social networks in primate species infected by multiple species of contagious (contact- or proximity-transmitted) parasites present slightly different topology than those in primates exposed to fewer parasites. In particular, we uncovered a positive relationship across primate species between parasite species richness and degree heterogeneity, that is, the coefficient of variation in number of social contacts per individual. While correlative evidence is insufficient to demonstrate causality, this result provides some support for our hypothesis on the link between host sociality and parasitism: host social networks shape patterns of intra-group parasite transmission, whereas selective pressures from parasites may in turn influence the structure of host social networks.

Significance statement

Patterns of interactions among animals within a group form a social network, which provides a map of the possible routes of parasite transmission and disease spread within the group. At the same time, it is possible that selective pressures from the parasites themselves have shaped animal social networks, with species experiencing strong pressures from parasites (i.e. infected by many species of contagious parasites) having evolved social networks that limit the risk of infection for individual group members. We provide correlative evidence for the possible evolutionary influence of parasitism on social network structure, using a comparison among primate species. Our study provides a reversed narrative to the usual one in which social network structure drives infection.

Keywords Primates · Social networks · Disease · Contact transmission · Phylogeny

Introduction

Social networks have increasingly been used to explain and predict the transmission of parasitic diseases among individuals within animal groups or populations (e.g. Drewe 2010; Fenner et al. 2011; MacIntosh et al. 2012; Springer et al. 2017). They serve to quantify heterogeneity in social interactions among individuals, revealing which individuals are involved in a disproportionate number of transmission events and which ones are only rarely involved in spreading disease (Godfrey 2013; Grear et al. 2013; White et al. 2017; Stockmaier et al. 2021). Social networks capture the ensemble of pairwise interactions among individuals in a group, accounting for the frequency, duration, intensity and directionality of interactions. They thus provide a map of possible transmission routes for contagious parasites, i.e. those transmitted directly by physical contact or close proximity via aerosols (White et al. 2017; Stockmaier et al. 2021). For example, social network analysis has revealed that in meerkats (*Suricata suricatta*), individuals that groom others the most are more likely

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to acquire tuberculosis (Drewe 2010), whereas in Japanese macaques (*Macaca fuscata*), females with central positions and many partners in grooming networks acquire a greater number of directly transmitted ectoparasites (Duboscq et al. 2016). Although these are observational rather than experimental studies, their results support a role of network structure in determining patterns of infection.

Beyond the position or participation of individuals in social activities, group-level properties of the social network can also influence the proportion of individuals that become infected or the severity of their infections (Briard and Ezenwa 2021). In particular, modularity, that is the fragmentation of the group into smaller subgroups of individuals that interact mostly with each other rather than with members of other subgroups, can impact disease epidemiology. In both empirical results and simulation models, increased modularity is associated with fewer and/or less severe infections by given parasite species (Sah et al. 2017; Sumner et al. 2018; Lucatelli et al. 2020). Modularity of larger groups is even associated with lower species richness of contagious parasites in primates (Griffin and Nunn 2012).

However, just as the architecture of host social networks determines the opportunities for a parasite to be transmitted and the dynamics of disease within host groups, parasites may in turn alter the structure of host social networks, with consequences for other aspects of social behaviour. Acknowledging the reciprocal feedbacks between parasites and social network structure allows the explicit integration of the two-way causality linking them (Ezenwa et al. 2016; Hawley et al. 2021). Indeed, susceptible individuals within a group can alter their social behaviours in response to the threat of infection from parasitized individuals, with measurable consequences for network topology (Croft et al. 2011). In addition, infected individuals can experience changes in their own behaviour, either due to pathology or the manipulative effects of parasites, and end up occupying different network positions and altering network structure (Poulin 2018).

Although the effects of parasites on host social network structure are starting to receive some attention, the focus to date has been on the immediate and short-term responses of hosts to the presence of parasites and the adjustments made via behavioural plasticity. Parasites can also exert selective pressures acting over evolutionary time to shape host social behaviour (Hart and Hart 2018). In particular, the number of different parasite species exploiting a host species in present times may provide a good index of the strength of selective pressures that the host species has faced in past times (Bordes and Morand 2009). Do social networks in animal species infected by multiple parasite species show different intrinsic topology and general properties than networks in related species infected by fewer parasites? In this paper, we first present a brief rationale in support of the hypothesis that parasite richness may have shaped host social networks. We then test this hypothesis by conducting a comparative analysis across primate species, the taxon with by far the most species having been studied for their social network structure, to test the relationship between parasite species richness and various network metrics. Our analysis is correlative and therefore does not allow causal inference; our main purpose is to demonstrate the plausibility of an alternative narrative in which parasites affect network structure, rather than the opposite.

Rationale and predictions

The ideal measure of the overall selective pressures exerted by parasites on host behaviour would require data on parasite species richness, as well as data on the prevalence (proportion of individuals that are infected), transmission efficiency and virulence (fitness reduction resulting from infection) of each parasite species. Data on prevalence of some species are either lacking or so variable in space and time that obtaining a single representative measure for the species is impossible. Data on virulence are almost always lacking and would be contingent on an individual's age, nutritional status, etc. Therefore, we assume that the more parasite species infect a host species, the greater the combined selective pressure they exert (see Bordes and Morand 2009). We then predict that the selective pressure imposed by parasitism on host social behaviour is proportional to the number of parasite species infecting that host species that can be transmitted during social contacts. In other words, across related host species which exhibit social behaviour, parasite species richness should correlate with some basic social network properties.

The prediction concerns solely parasite species transmitted by physical contact or when individual hosts are in close proximity (hereafter 'contagious parasites'). These include microbes such as viruses, bacteria, fungi and protozoans, as well as arthropod ectoparasites like lice, mites and fleas. Many diseases caused by microbes include some with serious fitness impacts, such as Ebola haemorrhagic fever and tuberculosis (Nunn and Altizer 2006). Arthropods too cause fitness losses (Lehmann 1993) and appear to have played a key role in the evolution of grooming behaviour (Clayton and Cotgreave 1994; Mooring et al. 2004; Nunn and Altizer 2006), one of the most frequently investigated pairwise interactions in social network studies on primates (Sah et al. 2019).

Social networks consist of nodes, one for each individual in a group, connected by edges that capture the strength (i.e. weight) and/or direction of pairwise interactions between individuals; the absence of any interaction between a given pair of individual results in a missing edge. A wide range of

metrics have been used to characterise social networks (Sosa et al. 2020). Many of these capture very similar aspects of network structure and are correlated with one another, and not all of them are equally likely to affect parasite transmission among individuals. Also, only some of these metrics have been measured in a large number of primate social networks, making them relevant for comparative purposes. Based on relevance to parasite transmission and availability of data, and acknowledging possible non-independence among metrics, here we focus our predictions on the following six metrics (see Sah et al. 2019; Sosa et al. 2020 for full definitions): (i) Network density, or the proportion of all possible edges that actually occur, i.e. the proportion of realised edges out of all those possible among all pairs of individuals in a group; (ii) Network average degree, or the average number of edges per node; (iii) Degree heterogeneity, or the coefficient of variation in number of edges per node; (iv) Weighted clustering coefficient, or the average clustering of nodes measured as the fraction of possible three-way interactions through that node that actually occur, taking edge weights into account; (v) Newman modularity, which measures the extent of subdivision of the network into modules of strongly interacting nodes; and (vi) Group cohesion, or the proportion of all realised edges that occur within modules as opposed to between modules.

Our general hypothesis is that strong selective pressures exerted by parasites (high parasite species richness) have favoured the evolution of social network patterns that reduce the probability of infection. We expect that fewer, weaker or more variable interactions among individuals, or between different modules, would reduce transmission of contagious parasites. Therefore, we predict negative interspecific relationships among primates between parasite species richness and Network density, Network average degree and Weighted clustering coefficients and a positive relationship between parasite species richness and both Degree heterogeneity and Newman modularity (Fig. 1). The expected relationships between parasite species richness and Group cohesion is not clear, as greater cohesion would minimise network-wide transmission, but enhance transmission probability within modules or small subsets of individuals.

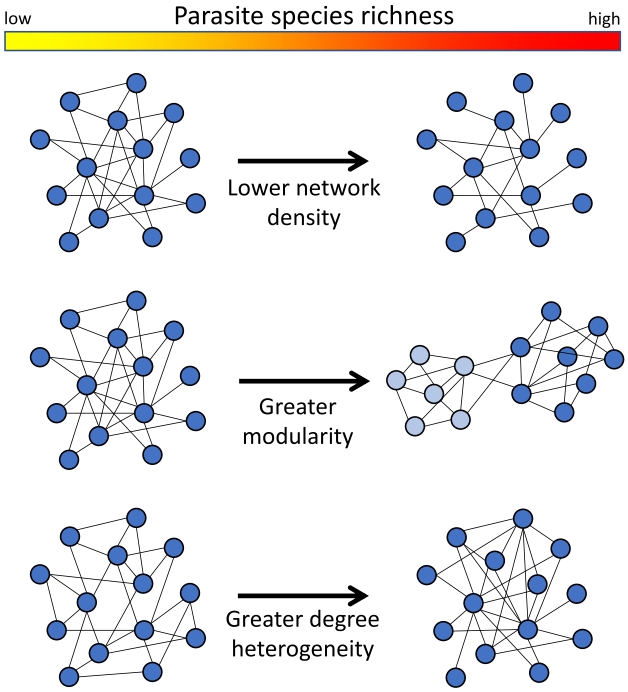

Fig. 1 Schematic illustration of some of the predicted changes in host social network structure as a function of increasing species richness of contagious parasites. Network density, i.e. the proportion of all possible edges that actually occur, might decrease as host individuals limit their number of interacting partners (top). Alternatively, without any change in network density, the network might become increasingly modular, i.e. subdivided into modules of strongly interacting individuals, to limit exposure to infection from individuals in different modules (middle). Finally, degree heterogeneity, i.e. the coefficient of variation in number of edges per node, might increase without any change in network density (bottom)

Methods

Dataset

We compiled data for this study from two public databases: the Global Mammal Parasite Database (GMPD), a comprehensive list of mammalian pathogens assembled from the scientific literature (Stephens et al. 2017), and the Animal Social Network Repository (ASNR), a multi-species repository of social network data (Sah et al. 2019). For the GMPD, we firstly selected all hosts that were primates and pooled all parasite species (including viruses, bacteria and eukaryotes) classified in the database as transmitted by 'close contact', to generate the species richness of contagious parasites for each host species. In the ASNR repository of social network data, network information on different primate species is based on different types of interaction type: dominance, grooming, physical contact or spatial proximity. Ideally, all networks would be based on the same interactions; however, here we assume that correlations exist among interactions, such that greater frequency of grooming means greater frequency of physical contacts, greater average proximity between individuals, etc. For three primate species, data were available for networks based on two interaction types (Table 1). Choosing one over the other, for instance, using values associated with the interaction presumably involving the greatest amount of physical contact (e.g. grooming chosen over spatial proximity), made no difference to the results; therefore, we consistently use the highest metric value of the two available in the analyses presented here. Finally, for

Table 1 Data for 18 primate species on contagious parasite species richness, type of social network studied, six metrics of social network structure and study effort (number of records on each primate species in Web of Science). Note: data on two different types of interaction networks are available for three primate species

Species	Parasite species richness	Interaction type	Network density	Degree heterogeneity	Network average degree	Weighted clustering coefficient	Newman modularity	Group cohesion	Study effort
Alouatta guariba	2	Grooming	1	0	4	0.332	0	1	114
Ateles geoffroyi	1	Grooming	0.429	0.355	6	0.089	0.18	0.46666667	447
Ateles hybridus	1	Physical contact	0.515	0.481	8.235	0.092	0.076	0.41430051	26
Brachyteles arachnoides	3	Spatial proximity	0.723	0.314	15.182	0.115	0.045	0.54490365	163
Sapajus apella	3	Grooming	0.652	0.227	7.167	0.075	0.087	0.60462304	130
Colobus guereza	11	Grooming	0.643	0.192	4.5	0.098	0.11	0.61111111	205
Erythrocebus patas	6	Grooming	0.287	0.484	5.158	0.074	0.29	0.57141691	344
Macaca arctoides	2	Grooming	0.667	0.223	12	0.074	0.052	0.42982456	384
Macaca fuscata	9	Dominance	0.617	0.197	37.645	0.141	0.096	0.44130438	1531
Macaca fuscata	9	Grooming	0.286	0.48	5.714	0.029	0.205	0.4833575	1531
Macaca mulatta	14	Grooming	0.6055	0.27	12.7055	0.056	0.0935	0.52646072	9933
Macaca mulatta	14	Physical contact	1	0	27	0.099	0	1	9933
Macaca radiata	1	Grooming	0.742	0.182	11.125	0.148	0.087	0.64044944	526
Macaca tonkeana	1	Physical contact	0.603	0.168	14.48	0.066	0.093	0.4640884	149
Pan paniscus	3	Grooming	0.322	0.348	5.789	0.061	0.254	0.52731587	1170
Pan troglodytes	30	Grooming	0.373	0.562	8.583	0.083	0.137	0.55341955	6236
Papio cynocephalus	22	Grooming	0.31235714	0.52817857	2.51246429	0.10167857	0.242	0.66309772	876
Papio cynocephalus	22	Spatial proximity	0.36603448	0.49375862	2.81303448	0.18896552	0.20144828	0.6146914	876
Papio papio	10	Grooming	0.323	0.393	7.76	0.031	0.198	0.48453608	633
Saguinus fuscicollis	3	Grooming	0.952	0.079	5.714	0.078	0	1.00005	432
Saguinus mystax	1	Grooming	1	0	5	0.301	0	1	186

a few primate species, a range of values were given for particular metrics instead of a single value; in these cases, we took the mid-point of the range, i.e. averaged the minimum and maximum values. This was the only way of obtaining a single species-level value. In the end, we obtained matching network data and parasite species richness data for 18 primate species (Table 1).

Data analysis

All analyses were conducted in R (v. 4.0.0, R Core Team 2020). Firstly, we downloaded a phylogenetic consensus tree comprising all primate species included in our dataset, from the Timetree online database (Kumar et al. 2017). Most species traits are phylogenetically conserved;

therefore correcting for phylogeny in the comparative analysis accounts to some extent for traits often correlated with parasite species richness (e.g. body size; Kamiya et al. 2014) and traits associated with complex social behaviours (e.g. relative brain size; Reader and Laland 2002; Ghazanfar and Santos 2004). For each of the 6 network metrics, we tested the extent to which they showed phylogenetic conservatism by calculating their phylogenetic signal based on Pagel's lambda (λ) using the *phylosig* function in the *phytools* package (Revell 2012) by mapping the metric values for each primate species onto the phylogeny. Values of λ close to 1 indicate that species resemblance is constrained by phylogeny, i.e. that closely related species display more similar network metric values than distant relatives.

Then, we used the primate phylogeny to build a series of simple phylogenetically corrected Bayesian multilevel models (MLM) (*brms* package; Bürkner 2017) to investigate the influence of parasite species richness on each of the 6 network metrics considered here. We chose those models as they are very efficient at sampling posterior distributions using the Hamiltonian Monte Carlo (HMC) method with the STAN algorithm. We used the primate species (N = 18) as an intercept group-level effect with a species covariance matrix (*ape* package; Paradis and Schlieps 2018) to account for stochastic effects of primate phylogeny on the results (see *Estimating Phylogenetic Multilevel Models with brms* vignette for more details: https://cran.r-project.org/web/packages/brms/vignettes/brms_phylogenetics.html). Because study effort on any host species influences its known parasite species richness (Walther et al. 1995), we included both study effort and contagious parasite species richness as the two predictors in all our models. Study effort was estimated as the total number of records mentioning the host species' Latin name in the Web of Science; this has been used previously as a measure of study effort in analyses using GMPD parasite data (e.g. Stephens et al. 2019). The following 5 response variables are, in principle, bounded between 0 and 1 and were modelled using a Beta distribution: Network density, Degree heterogeneity, Weighted clustering coefficient, Newman modularity and Group cohesion. To respect all assumptions of the Beta distribution, we slightly modified true values of 1 and 0 to 0.9999999999 and 0.0000000001, respectively. We chose a Gamma family distribution with a log link function for Network average degree as this metric is a strictly positive continuous response variable.

Each model was built with priors obtained with the *get_prior* function from the *brms* package and with 2 chains of 4000 iterations each (2000 for warmup, 2000 for sampling). We increased the *adapt_delta* function to 0.99 to lower the number of divergent transitions after warmup. We considered that parasite richness influenced a network metric if the 95% credible interval did not overlap with 0. We made sure every parameter in the model converged by checking the potential scale reduction factor on the split chains (Rhat) indicator (at convergence, Rhat is equal to one).

Results

The 18 primate species included in the analysis consist of 2 species of great apes, 9 species of Old World monkeys, and 7 species of New World monkeys (see Table 1). Among those primates, values of contagious parasite species richness ranged from 1 in a few species to 30 in chimpanzees, *Pan troglodytes*.

Overall, we show that some network metrics exhibit a strong phylogenetic signal, with Network density showing the strongest (λ = 0.879), followed by Degree heterogeneity (λ = 0.861) and Newman modularity (λ = 0.784) (see Fig. 2). In contrast, we observed weak phylogenetic signals for Network average degree (λ = 0.100), Weighted clustering coefficient (λ < 0.001) and Group cohesion (λ < 0.001).

We found that one out of the 6 investigated network metrics was related to parasite species richness when accounting for study effort (Fig. 3). Indeed, we show that parasite species richness positively covaries with Degree heterogeneity (effect size = 0.11; lower credible interval = 0.01, upper credible interval = 0.21). In contrast, we found no association (i.e. effect sizes overlap zero) between parasite species richness and Network density, Network average degree, Newman modularity, Group cohesion or Weighted clustering coefficient (see Fig. 3).

Discussion

Social network analysis has established itself as a powerful framework to study the spread of parasitic diseases within and among groups of animals (Godfrey 2013; Grear et al. 2013; White et al. 2017). Although it is now recognised that the structure of social networks affects parasite transmission, the reciprocal causal relationship has received much less attention. Can parasites possibly shape host social networks? The presence of infected individuals within a social group can lead to immediate behavioural adjustments and consequent changes in network structure (Croft et al. 2011; Poulin 2018; Stockmaier et al. 2021). Here, we looked for correlative evidence that sustained, long-term selective pressures from parasites might leave an evolutionary signature on host social network structure. Specifically, we tested the hypothesis that a high richness of contagious parasite species was correlated with social network structures in a manner that may reduce the probability of infection, using a comparative analysis among primate species. Our findings provide some limited support for this hypothesis and suggest that as with many other aspects of animal behaviour

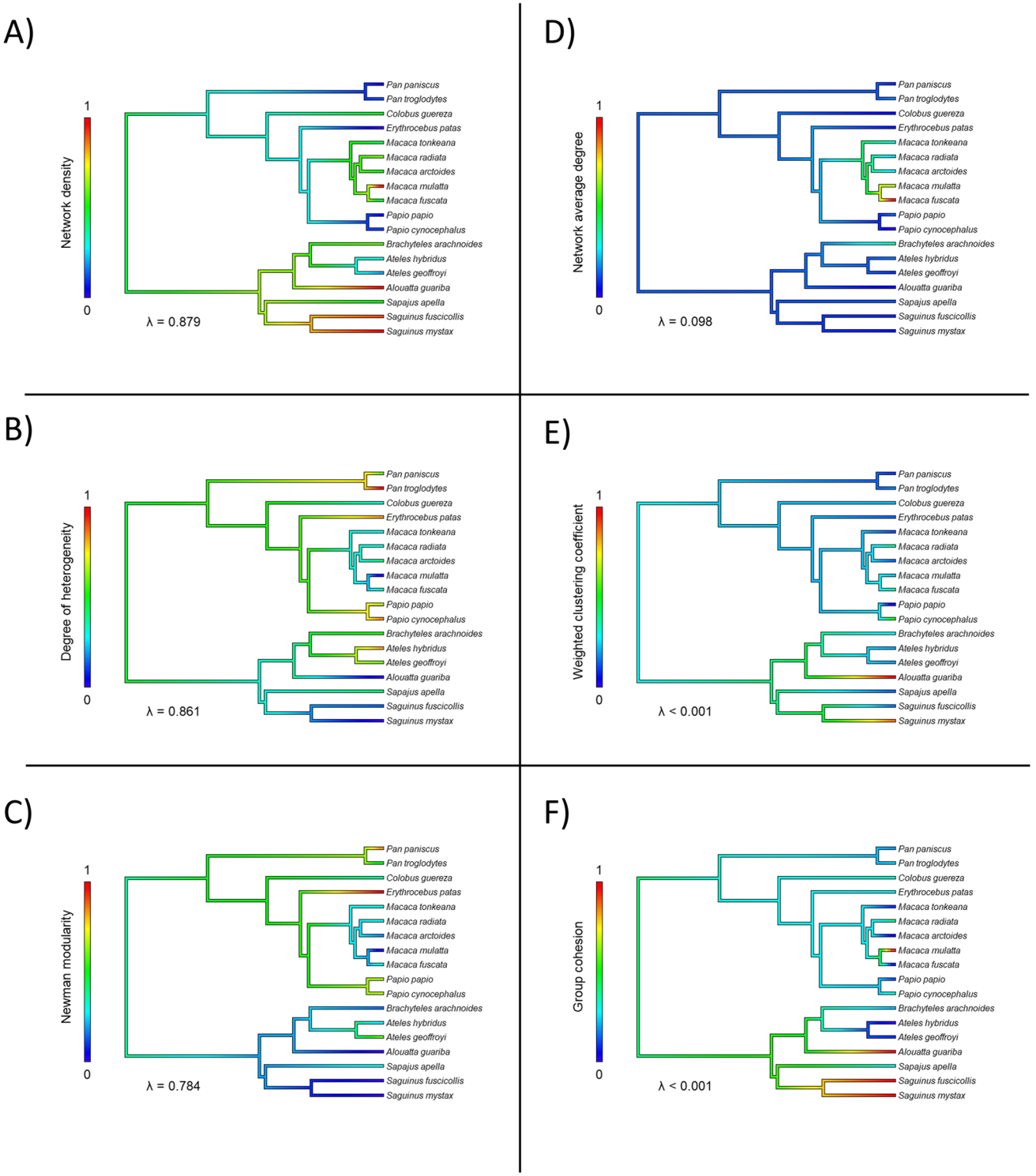

Fig. 2 Phylogenetic distribution of network metric values for **A** Network density, **B** Degree heterogeneity, **C** Newman modularity, **D** Network average degree, **E** Weighted clustering coefficient, and **F** Group cohesion, among 18 primate species. Also shown is Pagel's λ for each metric

(Ezenwa et al. 2016; Hawley et al. 2021), coevolutionary feedbacks between parasitism and host sociality may involve two-way selective pressures. Our correlative evidence allows no causative inference; however, our main goal is to offer an alternative narrative to the one that dominates the literature in this area: wouldn't it be possible that parasitism influences social structure just as social structure affects parasite transmission?

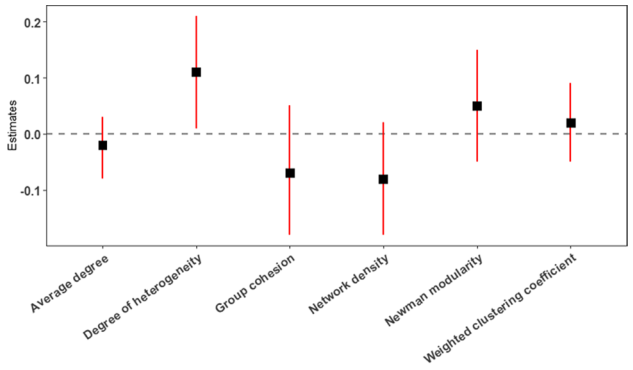

Fig. 3 Estimated effect size (and 95% credible interval) of contagious parasite species richness on six metrics of social network structure among 18 primate species, obtained while accounting for uneven study effort and phylogenetic relationships among species. Negative effects were predicted for Network density, Network average degree and Weighted clustering coefficients; positive effects were predicted for Degree heterogeneity and Newman modularity. No directional prediction was made for Group cohesion

Previous analyses considering parasite infections as a response variable and social network metrics as predictors have generally used individual-level measures of infection (infected or not, or parasite burden) by single parasite species. The exception is the study of Griffin and Nunn (2012), a comparative analysis across primate species in which they related network structural properties to parasite richness. Our analysis is thus the inverse of that by Griffin and Nunn (2012). Their results, based on the almost same subset of primate species and similar (though less up-to-date) parasite data, indicate that higher network modularity was associated with lower richness of contagious parasites. Instead of taking network structure as a predictor of parasite richness, in the present study we reversed the arrow of causality and considered parasite species richness as a predictor of network structure, through the selective pressures parasites exert on their hosts. We found no association between parasite richness and network modularity. Differences in analytical procedures may account for the contrasting results but also highlight the difficulties inherent with the use of correlational data to test causal hypotheses. Further complicating matters, network modularity tends to increase with group size (Nunn et al. 2015), with group size itself generally correlating intraspecifically with prevalence of some contagiously transmitted parasites (Côté and Poulin 1995; but see Lucatelli et al. 2020).

Nevertheless, of the 5 directional predictions we made, three were in the expected direction but non-significant, and only one had significant support in our analyses. We found a positive interspecific relationship among primates between parasite species richness and Degree heterogeneity and no significant association between parasite species richness and any of the other network metrics. The biological interpretation of these metrics and what they might mean in terms of infection avoidance requires caution (Sosa et al. 2020). Our results suggest that in primate species exploited by many species of contagious parasites, there is greater variance in the number of edges per individual, with some individuals involved in multiple pairwise interactions, and many other group members involved in very few. This individual-level heterogeneity may reflect a tendency for many individuals in species with many contagious parasites to restrict their interactions to a few necessary ones. In these species, selection may have favoured more plasticity in social strategies, with only strategies at both extremes of the interactivity spectrum yielding net benefits. On one hand, several group members may opt for low interactivity and avoid the cost of infection; on the other hand, a few group members opt for high interactivity, with the benefits outweighing the cost of infection. However, it is unclear what net impact variation in Degree heterogeneity would have on parasite transmission. Interestingly, the two species with the highest richness of contagious parasites, *Pan troglodytes* and *Papio cynocephalus* (Table 1), have markedly greater Degree heterogeneity than their closest relative with much fewer parasites (see Fig. 2B).

Multiple behaviours have evolved at the level of individual animals to minimise the risk of acquiring infections during interactions with other conspecifics (Hart 1994; Loehle 1995). When adopted by a substantial fraction of the population, these individual-level behaviours translate into particular features of social networks. Thus, selection on individual behaviours, in response to selective pressures from parasites, might possibly influence social interaction networks. Sociality comes with both benefits and costs, and selection should favour a social network structure that achieves the optimal compromise between these. For example, information and behavioural innovation spread through social networks, providing advantages to both recipients and originators (Firth 2020). Networks with distinct and pronounced modular structures may facilitate the spread of information and social learning while mitigating against the transmission of contagious parasites, compared to non-modular networks (Sah et al. 2017; Evans et al. 2020). However, a full understanding of how network structure evolves would require quantitative information on all selective pressures acting on animal sociality, and not just parasitism.

Interestingly, we found that some metrics of social network structure are clearly phylogenetically conserved among primates, whereas others are not. Exploring the reasons for these differences and their implications is beyond the scope of our study. However, this does suggest that some aspects of social networks may be more evolutionary malleable than others, making them better targets of selection under pressure from parasites or other potential costs or benefits of social behaviour.

Our results need to be interpreted with some caution for several reasons, however. The number of primate species included in the analysis was limited due to data availability. The different network metrics investigated are not fully independent of each other. Furthermore, the type of behavioural data on which these metrics are based, the way the data were collected (e.g. scan versus focal sampling, number of observation periods), the sizes of social groups that were observed and the algorithms used to compute the network metrics, all vary among original studies. Although we incorporated host phylogeny in our analysis and many of the host traits known to correlate with parasite species richness (Kamiya et al. 2014) and social behaviour are phylogenetically conserved, we did not explicitly include these traits as predictors. The same applies to the type of environment (e.g. dense forest versus open habitat, wild versus semi-captive populations) in which different primate species live, which may also shape their social networks. Furthermore, we included species-level values for both parasite species richness and social network metrics, without considering intraspecific and spatial variation. For instance, not all populations of any given primate species will harbour the full set of parasite species known to exploit that species. Finally, we could only use data on contemporary parasite species infecting primates, while various aspects of their social networks may have been shaped in the past by parasites no longer infecting them (Poulin et al. 2020). Alternatively, animal societies starting out with a particular social structure may have been intrinsically less likely to acquire certain parasite species over time. Nevertheless, our findings provide some limited support for our hypothesis that selective pressures from diverse contact-transmitted parasites might have shaped social network structure; the robustness and generality of this pattern will need validating as further comparative data become available, for primates as well as other taxonomic groups. For the purposes of this article, i.e. presenting an alternative way to look at the sociality versus disease relationship, we feel our analyses are suitable in spite of the above shortcomings, if only to generate discussion.

With data on an increasing number of animal social networks becoming available (Sah et al. 2019), and with their construction and analysis increasingly following comparable approaches (Farine and Whitehead 2015), further comparative studies on a broader range of species will soon become possible to further test the hypothesis that parasite richness is a driving factor in the structure of social networks. Future studies may even seek to incorporate data on parasite virulence to complement parasite richness and achieve a measure of parasite pressure more directly based on host fitness costs. With climate and habitat changes altering disease risk for many species, including primates (Chapman et al. 2005), we need to better understand how parasitic diseases shape their social structure in order to design appropriate conservation strategies.

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Author contribution RP conceived the study, AF compiled and analysed the data, and RP wrote the manuscript with input from AF.

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Data availability The full dataset is provided in Table 1.

Declarations

Ethics approval This research is based entirely on pre-existing data available in the public domain and did not involve any live animals.

Conflict of interest The authors declare no competing interests.

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