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Author for correspondence:
Shweta Bansal
e-mail: shweta.bansal@georgetown.edu

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Pathogen transmission modes determine contact network structure, altering other pathogen characteristics

Melissa Collier[1], Gregory F. Albery[1,2], Grant C. McDonald[3] and Shweta Bansal[1]

[1]Department of Biology, Georgetown University, Washington, DC, USA
[2]Leibniz Institute of Freshwater Ecology and Inland Fisheries, Berlin, Germany
[3]Department of Ecology, University of Veterinary Medicine Budapest, Budapest, Hungary

MC, 0000-0003-0266-9878; GFA, 0000-0001-6260-2662; SB, 0000-0002-1740-5421

Pathogen traits can vary greatly and heavily impact the ability of a pathogen to persist in a population. Although this variation is fundamental to disease ecology, little is known about the evolutionary pressures that drive these differences, particularly where they interact with host behaviour. We hypothesized that host behaviours relevant to different transmission routes give rise to differences in contact network structure, constraining the space over which pathogen traits can evolve to maximize fitness. Our analysis of 232 contact networks across mammals, birds, reptiles, amphibians, arthropods, fish and molluscs found that contact network topology varies by contact type, most notably in networks that are representative of fluid-exchange transmission. Using infectious disease model simulations, we showed that these differences in network structure suggest pathogens transmitted through fluid-exchange contact types will need traits associated with high transmissibility to successfully proliferate, compared to pathogens that transmit through other types of contact. These findings were supported through a review of known traits of pathogens that transmit in humans. Our work demonstrates that contact network structure may drive the evolution of compensatory pathogen traits according to transmission strategy, providing essential context for understanding pathogen evolution and ecology.

1. Introduction

Pathogens vary in a range of important characteristics including transmission mode, infectivity and duration of infection, many of which determine epidemiological characteristics such as their ability to persist in a population [1–5]. Although this diversity in pathogen traits is fundamental to disease ecology, we know little about the ecological factors driving the evolution of such traits; in particular, it is unclear how transmission ecology determines the evolution of pathogen characteristics.

Pathogens are spread by a range of different contact types facilitated by specific host behaviours such as respiration, physical contact or shared space use, which define different pathogen transmission modes [6]. In a contact network, the behaviour that defines its edges (i.e. a contact type) can be associated with different transmission modes (e.g. mating versus grooming versus spatial associations), and exhibit distinctive contact patterns [7–11]. For instance, when analysing contact types in mice (*Mus musculus*), researchers found that agonistic, grooming and sniffing events were associated with distinct network properties such as density, average path length and node centrality [12]. Such network properties can influence the transmission efficiency of pathogens, with downstream impacts on the evolution of their traits [13–19]. Furthermore, it is known that individual contact effort across contact types can be heterogeneous (a concept known as social fluidity), and can lead to the formation of weak

Table 1. The four transmission mode categories used to define our 232 contact networks based on the contact types present in our dataset, and the host behaviours that represent each type.

transmission mode	contact types in dataset	example host behaviours
fluid-exchange	fluid-exchange mating	mammal intromission
		bird cloacal transfer
		spermatophore transfer to genitals in insects
	trophallaxis	mouth to mouth food sharing
direct physical	dominance interactions	headbutting
		biting
		physical contests
	physical contact	grooming
		petting
		touching
	non-fluid-exchange mating	spermatophore transfer to not genital body part
		non-amplexus spawning
non-physical close	non-physical social interactions	group membership
		spatial proximity
		group foraging
	synchronous resource sharing	birds or bats using the same roost
		possums sharing the same den
		birds using the same feeders
		voles caught in the same traps
indirect	asynchronous resource sharing	tortoises using same burrow at different times
		birds building nests in same chamber at different times

ties [9]. These weak ties play an important role in defining network structure [18], but the extent to which they impact the evolution of pathogen traits remains unknown. Understanding variation in these characteristics is essential for understanding pathogen ecology, and therefore for developing control measures and testing hypotheses regarding their evolutionary origins.

Pathogens should evolve to maximize their fitness, principally described by their R_0 (basic reproduction number, i.e. the total number of new infections caused by one infection in a totally susceptible population) [20]. To persist in a population, a pathogen's R_0 must be greater than 1. A pathogen's R_0 depends on both the behaviour of its host population, and on its own transmissibility [1,21,22]. Host behaviours create the relevant contact that defines the path of transmission for a pathogen, while transmissibility represents the epidemiological characteristics (e.g. infectious duration, infection probability) that determine effective transmission upon a relevant contact. Consequently, host behaviour can affect R_0 which could drive the evolution of pathogen traits.

Associations between contact network structure and pathogen traits are well-supported by theory. For example, sexually transmitted pathogens such as gonorrhea (*Neisseria gonorrhoeae*) or herpes simplex virus rely on rare, dyadic transmission events, likely producing a sparse contact network; to compensate for this sparseness, they are thought to exhibit longer duration infections and higher infection probability respectively [1,5,23]. By contrast, tick-borne flaviviruses are only infectious for about 2–3 days in mammal hosts, but persist in tick populations due to their host's aggregated co-feeding behaviours and consequently high rates of contact [24]. Despite these kinds of anecdotal observations, there is no comparative or meta-analytic evidence to demonstrate the relationship between transmission routes and pathogen characteristics.

Thus, a critical gap in disease ecology is our understanding of how different contact types required for pathogen transmission routes might exhibit distinct contact network structure, and how they might alter the evolution of adaptive pathogen characteristics required to capitalize on these host networks. Thus, we sought to answer the following questions: (1) how does non-human contact network structure differ depending on the transmission mode associated with its contact type? (2) How does the resulting contact network structure affect a pathogen's ability to persist on that network? (3) How might these results be reflected in known pathogen traits? To address these questions, we conducted a quantitative analysis on 232 animal contact networks spanning eight taxonomic classes to investigate the impact of contact type on pathogen traits. First, we categorized networks into four different horizontal transmission mode categories based on their contact types (table 1). Next, we used a multivariate generalized linear mixed model (GLMM) to identify how network structure is predicted by its associated transmission mode category. We then mathematically examined how pathogen traits (i.e. critical transmissibility) may change in order to persist on these different contact networks and compare our results to current knowledge of pathogen traits. We provide practical evidence that contact network structure is influenced by contact types,

and that this structural variation causes differences in pathogen transmissibility thresholds that are reflective of our current knowledge of pathogen infection characteristics.

2. Methods

In this study, we used a GLMM to examine how contact types associated with different pathogen transmission modes predict eight different descriptors of network structure. We then calculated a pathogen's critical transmissibility (T_c) value on these different network types, or the value of transmissibility (T) necessary for a pathogen to persist on a network (basic reproduction number (R_0) > 1) where epidemics might occur. Finally, we collate published information on pathogen traits in humans (due to the lack of these data in non-human systems) that make up T_c (e.g. probability of infection, infectious period) and provide a preliminary comparison between these pathogen traits, their transmission routes, and our model predictions. Therefore, we aimed to provide evidence that transmission mode affects emergent contact networks, and therefore selects for specific pathogen traits to maximize transmission and persistence.

(a) Dataset

We compiled a dataset of animal contact networks where edges represent one of 12 different contact types, using the Animal Social Network Repository (ASNR) [25,26]. The ASNR is an open-source animal behaviour network library in which we have compiled network data from the available literature across eight animal taxonomic classes (Mammalia, Aves, Reptilia, Amphibia, Insecta, Arachnida, Actinopterygii and Cephalapoda). Contact types include group membership, non-physical social interactions, spatial proximity, foraging interactions, trophallaxis (mouth-to-mouth food sharing), synchronous and asynchronous resource sharing, agonistic behaviours, grooming, other physical contact or mating interactions. Our sample size for this study consisted of 232 contact networks from all eight taxonomic classes (electronic supplementary material, figure S1). Of these 232 networks, 181 had weighted edges determined by the duration, frequency or association probability (e.g. half-weight index) of the contact type. We assume that these networks were observed without the presence of a pathogen or active infection.

(b) Defining and characterizing contact networks

For each network in our dataset, nodes represented an individual animal and edges represented a contact type between two animals. Based on the contact type, we divided our dataset into four different transmission mode categories (table 1). We focus on four transmission modes that our sample represents well: fluid-exchange, direct physical, non-physical close and indirect. We define each transmission mode category as follows:

1. Fluid-exchange contact: host interactions that result in the exchange of bodily fluids. This includes sexual contact such as cloacal transfer, intromission and spermataphore transfer, as well as direct food-sharing interactions such as trophallaxis.
2. Direct physical contact: interactions of physical touch that include grooming, agonistic host behaviours (e.g. head-butting, fighting), and other physical social contact.
3. Non-physical close contact: close spatial proximity in which face-to-face contact, or respiratory droplet exchange, could occur. This includes group memberships, or spatial proximity.
4. Indirect contact: asynchronous resource-sharing interactions. Indirect contact is unique in that individuals do not need to be using the resource at the same time to be connected in the network.

We note that our non-physical, physical and fluid-exchange categories have an inherent nested structure with non-physical the broadest category, and fluid exchange the most selective (electronic supplementary material, figure S2). In other words, pathogens that transmit on non-physical networks (i.e. respiratory droplet pathogens such as SARS-CoV-2) can also transmit on direct physical and fluid-exchange networks, but fluid-exchange transmitted pathogens (e.g. sexually transmitted pathogens such as gonorrhea (*Neisseria gonorrhoeae*)) can only transmit on fluid-exchange networks. We manage this nested structure by classifying each empirical network into the most specific category possible using the definitions above.

For each network, we calculated the following eight network metrics that are known to influence infection dynamics and social structure, ignoring edge weights (table 2): total network density, degree heterogeneity, degree assortativity, average clustering coefficient, average betweenness centrality, network diameter, fragmentation and subgroup cohesion. Fragmentation (i.e. the number of communities in each network), was estimated using the Louvain method [27] and the remaining network metrics were calculated using the NetworkX package in Python (https://networkx.github.io/).

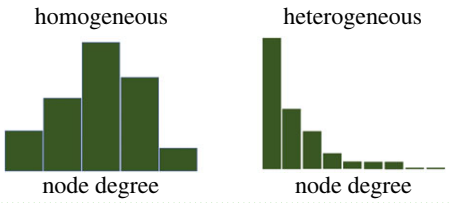
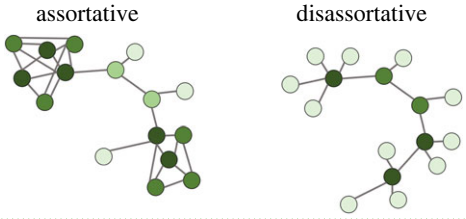
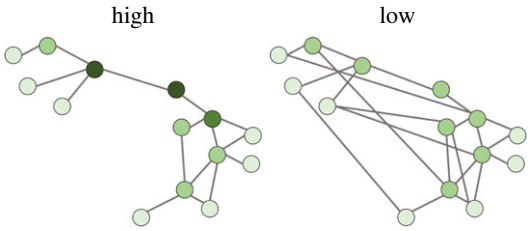
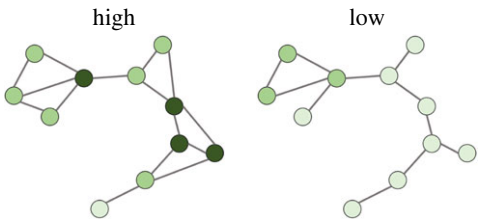
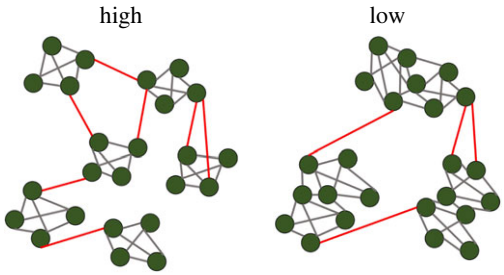
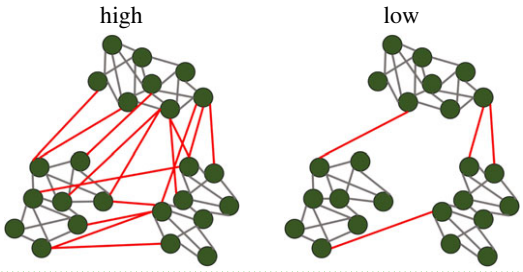
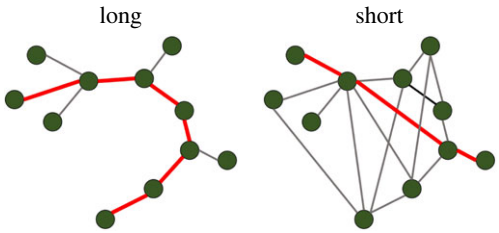
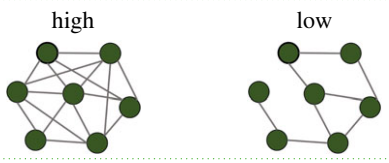
(c) Identifying how network structure depends on transmission mode

To examine how contact network structure differs depending on its associated transmission mode category, we fitted a multivariate GLMM using the MCMCglmm package in R [28], where the eight network metrics (table 2) made up our multivariate response, and the associated transmission mode category was our predictor variable.

We also controlled for the effect of network size on these metrics by including the number of nodes as a predictor. Edge weight type (weighted versus unweighted) was also included to control for data sampling design and edge weighting criteria. As the spatial scale of data collection has been shown to influence network structure [18], we also included sampling scale as a predictor. Studies that collected data on captive animal populations (where all nodes and interactions are theoretically known) were labelled as captive sampling. Studies that focused data collection on specific social groups were categorized as social sampling, and those that focused data collection on all individuals within a fixed spatial boundary were labelled as spatial sampling. Since the social system of an animal species is also shown to influence network structure [18], we included species social structure (relatively solitary, gregarious and socially hierarchical defined in electronic supplementary material, table S1 based on [18]) as a predictor. Finally, we controlled for repeated measurements within studies by including study ID as a random effect in the analysis. We were unable to include a random effect for taxonomic class or use a phylogenetically controlled model, because there was an unbalanced representation of different taxa across the four transmission mode categories that made these effects difficult to fit successfully (electronic supplementary material, figure S1).

All response variables were continuous; to encourage proper model fitting we log-transformed then centred them (by subtracting the mean) and then scaled to unit variances (by dividing by the standard deviation). We ran one MCMC chain for 10 500 iterations, with a thinning interval of 10 after burn-in of 500 with uninformative priors. Non-physical contact transmission was the intercept factor level for the transmission mode category fixed effect. For each response variable, if the effect sizes of the three remaining transmission mode categories overlapped with

Table 2. The eight network metric response variables used in the multivariate GLMM: degree heterogeneity, degree assortativity, average betweenness centrality, average clustering coefficient, fragmentation, cohesion and network diameter.

network metric	definition	visualization
degree heterogeneity	the coefficient of variation (CV) in the frequency distribution of each node's number of contacts (also known as the degree distribution). Equal to the network's mean degree divided by the standard deviation	
degree assortativity	the tendency of contacts to have a similar degree (darker node colour indicates higher degree). A disassortative network has high degree individuals associating with low-degree individuals. An assortative network has high-degree individuals forming social bonds with each other	
average betweeness centrality	the tendency of nodes to occupy a central position within the social network (darker node colour indicates a more central position)	
average clustering coefficient	the tendency for a set of three individuals to be interconnected (represented by triangles), indicating the propensity of an individual's social partners to interact with each other (darker node colour indicates higher clustering value)	
fragmentation	the number of subgroups, within a network (grey edges are within subgroups and red edges are among subgroups)	
cohesion	cohesion is the tendency of individuals to interact with members of their own subgroups (grey edges) compared to members of other subgroups (red edges)	
network diameter	a measure of the longest of all the shortest paths lengths between pairs of nodes in a network. Shown is an example of a network with long network diameter of 6, and a similar network with shorter network diameter of 4, indicated by red coloured edges	
network density	the proportion of existing edges to possible edges in a network	

zero then it was considered not different from non-physical contact. To examine differences between the remaining three modes, we observed the proportional overlap between their effect sizes across all 1000 iterations, multiplied by 2 per a two-tailed test; if it was less than 0.05, then the response variables were considered different between the transmission mode categories.

Within the ASNR, several studies provided more than one network for the study species or population. To avoid biasing our sample towards these studies, we randomly selected a maximum of 15 networks from each study ($n = 232$). To ensure that our results were not affected by this random subsampling, we reran our model 1000 times, each time choosing a different sample of networks from each study with more than 15 networks. We then chose a random estimate from each model run and computed an average of model estimates across the 1000 different subsamples.

(i) Investigating the role of weak ties

To determine if weak ties (i.e. low edge weights) might drive contact network structure across transmission modes, we recalculated all eight network metrics for each network in our dataset when the lowest 5%, 10% and 15% of weighted edges were dropped from the network. We then reran our GLMM to see if differences in network metrics among our different transmission mode categories still hold true when low-weight edges are no longer accounted for.

(d) Characterizing critical transmission thresholds

We assume that our dataset consists of contact networks constructed in the absence of pathogens or active infections. Therefore, to examine how network structure affects a pathogen's ability to persist on a network, we must model the way a pathogen spreads on these networks using mathematical models.

We sought to identify a pathogen's critical transmissibility (T_c) on contact networks based on transmission mode. For a pathogen to persist in a network, its basic reproduction number (R_0) must be greater than 1, and R_0 depends on both a pathogen's T and the contact patterns of the network it travels on. Therefore, for each network, we sought T_c for which $T_c(\text{contact}) = R_0 > 1$. To estimate T_c, we considered the impact of contact network structure by calculating R_0 using Monte Carlo simulations of a susceptible–infected–recovered (SIR) model of infection spread through each network. We ignored edge weights because the impact of interaction weight (e.g. contact duration or frequency) on infection spread is not well understood generally. We used an SIR percolation simulation model [29], where each outbreak was initiated by infecting a randomly chosen individual in the network. For the first generation of the simulation, the individual is given an opportunity to infect all its contacts, with transmissibility T, and then recover. This process is then repeated for each infected node, until no infections remain in the network. For each network, we simulated 250 disease outbreaks and of those classified large-scale epidemics as those where at least 10% of the population is infected. We repeated this for each T value in the range (0.01–0.8), and recorded the first T value for which at least 10% of the outbreaks were large-scale epidemics. Percolation theory suggests that our expectation for the probability of having a large-scale epidemic should match the expected size of a large-scale epidemic [29]. This reported T value is our estimate of the network's critical transmissibility T_c.

Past studies suggest that degree (i.e. density) and degree heterogeneity are the most important aspects of network structure affecting how a pathogen will transmit across a network [30]. In order to test this, we considered two control scenarios where we (1) isolated the effect of homogeneous degree (i.e. all individuals have the same number of contacts) on T_c and (2) isolated the effect of heterogeneous degree (i.e. on average, individuals have the number of contacts as scenario 1, but individual degree varies around this mean) on T_c. Therefore, we sought T_c for which $T_c\langle k_e\rangle = R_0 > 1$, where $\langle k_e\rangle$ is the average excess degree of the network. The excess degree is the potential number of contacts an individual can infect after they have been infected by one of their contacts, and on average this value is larger for networks with degree heterogeneity than for homogeneous degree networks [22].

For the first control scenario, we considered a pathogen's T_c in a homogeneous degree network, in which the average excess degree, $\langle k_e\rangle$, is:

$$\langle k_e\rangle = \langle k\rangle - 1, \tag{2.1}$$

where $\langle k\rangle$ is the average degree of the network. For the second control scenario, we considered a network with degree heterogeneity, thus the average excess degree is:

$$\langle k_e\rangle = \frac{\langle k^2\rangle - \langle k\rangle}{\langle k\rangle}. \tag{2.2}$$

We consider these to be 'control' scenarios because equations (2.1) and (2.2) allow us to consider *only* the two network metrics of interest (density and degree heterogeneity), while our simulation method will inherently take in to account all aspects of network structure (centrality, clustering, etc.). By isolating how homogeneous and heterogeneous degree effects T_c, and comparing these results to our full network structure simulation method, we can elucidate how much of the variation in T_c may be attributed to the degree (density) and degree heterogeneity of a network.

We compared the T_c values for each transmission mode category of contact networks (non-physical close, direct physical, fluid-exchange, indirect) within each scenario using a one-way ANOVA, and pairwise t-tests with a Tukey HSD family-wise error-rate correction.

(e) Examining diversity of empirical pathogen characteristics

To examine how our results are reflected in known pathogen traits, we considered the transmissibility of a pathogen (T) as a function of its infectious duration (G) and its probability of infection (β) [21,22]:

$$T = \frac{\beta}{\beta + (1/G)}, \tag{2.3}$$

To provide context for the covarying characteristics of known pathogens, we examine known β and G values (infection characteristics) for a small set of well-characterized pathogens to see how they compare to our findings. Because data on pathogen traits in non-human animals is limited, we instead focus on these traits in common human pathogens. We use two systematic reviews on the natural histories of pathogens for common diseases in preschools [31,32] to summarize data on pathogens that use each transmission mode. We only included the pathogens in our summary if the source of the data was from a well-designed study, using the levels of evidence of I and II provided in the first review [31] (electronic supplementary material, table S2). If there were no data, or the source of the data was poor (levels of evidence of III or IV) for the pathogen's infectious period (G), we instead used the shedding periods, defined as the period of time during which an individual excretes the pathogen; the shedding period can be used to estimate the duration of infectiousness when there is lack of direct evidence [31]. If there were no or poor data for the shedding period, then it was not included in this summary. We then verified the data for these pathogens using the second review [32].

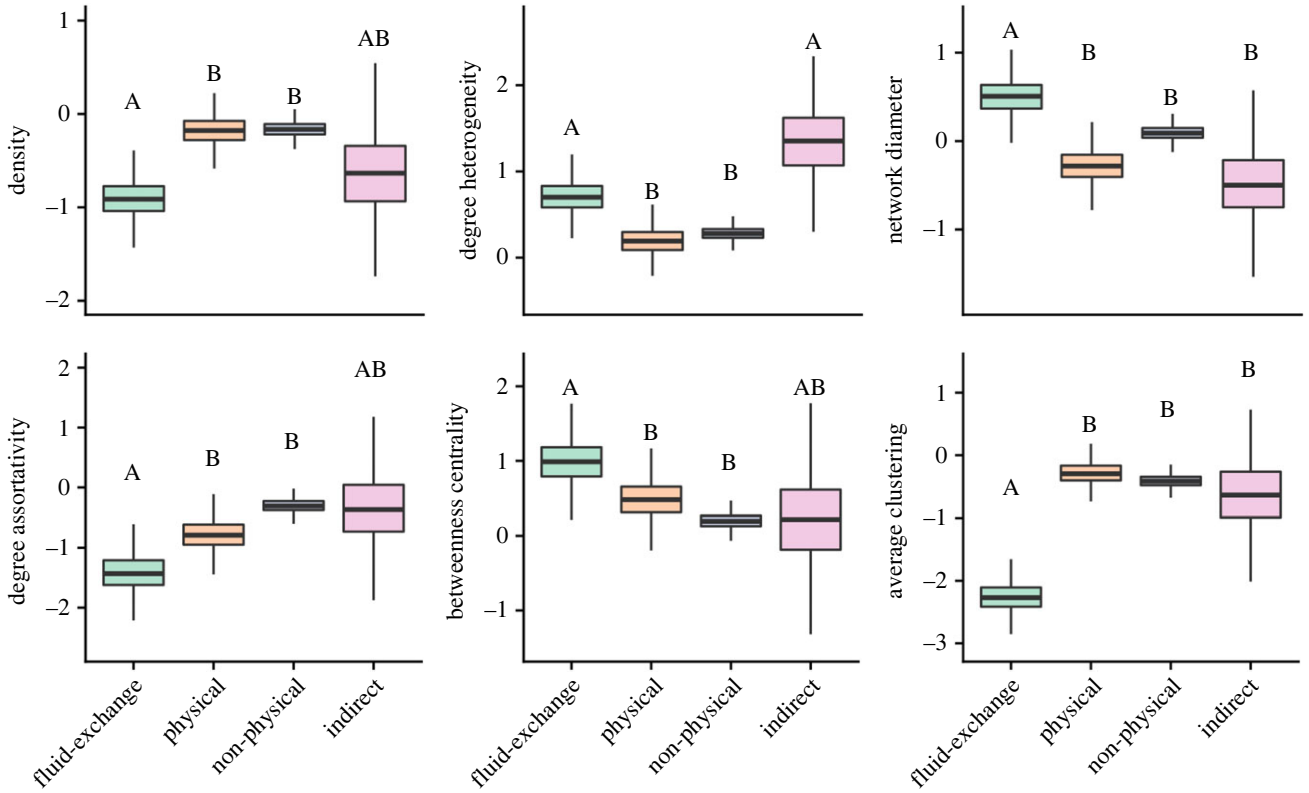


Figure 1. The predicted distributions of six of the eight network metrics for each transmission mode category, based on the results of the GLMM. Letters represent significant differences between transmission mode categories. Results for fragmentation and cohesion were not different among transmission mode categories and are therefore not included in this figure. (Online version in colour.)

Since these reviews did not contain any sexually transmitted pathogens, we took the 10 sexually transmitted pathogens listed on the CDC website (www.cdc.gov/std) and examined the literature for their natural histories. We found studies that estimated both the probability of infection (β) and infectious period (G) of four of the 10 pathogens (syphilis [33,34], gonorrhea [35,36], chlamydia [37,38] and trichomoniasis [39,40]).

3. Results

(a) Network structure is dependent on host behaviour type

We examined how network structure was predicted by the pathogen transmission mode represented by specific contact types using a multivariate GLMM. We summarized network structure with eight topological characteristics (table 2), and all the metrics except subgroup cohesion and fragmentation differed among transmission mode categories. The predicted distributions for the six remaining network metrics by transmission mode category are summarized in figure 1 and all other effect sizes from our model are in electronic supplementary material, tables S3–S6.

Fluid-exchange contact networks differed from physical and non-physical contact networks in all six network metrics and differed from indirect contact networks for clustering and network diameter. Indirect contact networks only differed from physical and non-physical networks in their degree heterogeneity. Physical and non-physical networks could not be differentiated by the network metrics we tested. This suggests that fluid-exchange contact types create the most unique contact patterns.

Physical and non-physical contact networks had higher density values and shorter network diameters than fluid-exchange networks, and lower degree heterogeneity than both fluid-exchange and indirect contact networks. This indicates that physical and non-physical contact types create networks that are more connected, with less variation in each individual's number of contacts.

Fluid-exchange contact networks also had lower degree assortativity than physical and non-physical contact networks. That is, high-degree individuals in fluid-exchange contact networks tend to be connected to low-degree individuals, whereas high-degree individuals in physical and non-physical contact networks are more likely to be connected to other high degree individuals. Indirect contact networks did not differ in their degree assortativity from any other contact network. Fluid-exchange contact networks also had lower average clustering values than all other contact networks.

Finally, fluid-exchange contact networks have higher betweenness centrality values than physical and non-physical contact networks. This means that these networks tend to have more 'bridge nodes', or nodes that connect different communities together: this is despite the fact that the number of communities (fragmentation) and the cohesiveness of those communities do not differ among contact networks.

(i) Weak ties

To examine the role of weak interactions (i.e. low edge weights) in determining unique network structure, we reran the GLMM on our dataset after dropping the lowest 5%, 10% or 15% of weighted edges in each network. We found that the significant

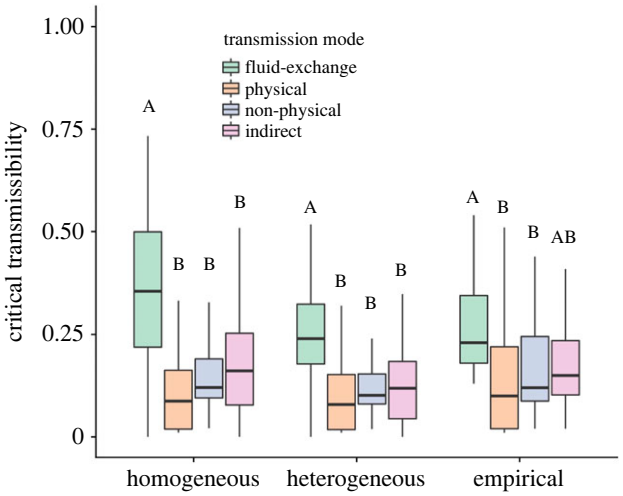


Figure 2. The critical transmissibility values (T_c) needed for a pathogen to persist ($R_0 > 1$) on networks in each transmission mode category. T_c was estimated empirically using SIR simulations on each network to account for the effect of full network structure. T_c was then calculated in two control scenarios that examined the effects of homogeneous degree (equation (2.1)) and heterogeneous degree (equation (2.2)). Colours represent the different transmission mode categories, and letters indicate significant differences within each method based on a one-way ANOVA and Tukey's HSD. (Online version in colour.)

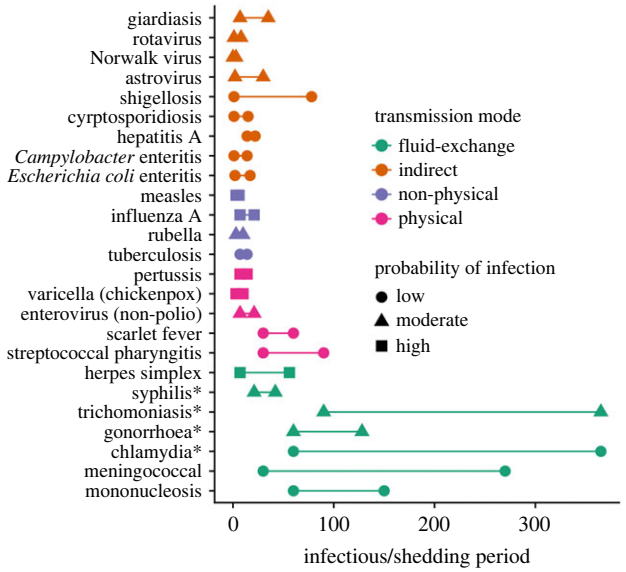


Figure 3. The typical infectious periods of 25 different human pathogens from two literature reviews [31,32] (pathogens with asterisks came from alternative sources [33–40]). Colour denotes the transmission mode category of each pathogen. Shapes indicate the pathogen's probability of infecting a host given contact. (Online version in colour.)

differences in average clustering and degree assortativity in networks across the four transmission mode categories are maintained as we filter low-weight edges. However, as edges are filtered, there are no longer any differences in density and degree heterogeneity (electronic supplementary material, figure S3) among transmission mode categories. In other words, without low weight edges, contact networks in each transmission mode category become more similar in their number and variation of contacts.

(b) Pathogen transmissibility must be higher for contact networks with lower connectivity

We demonstrated the effect of full contact structure on critical transmissibility values (T_c) in networks for each of our four transmission mode categories. We then specifically examined the effect of network density (average number of contacts) and degree heterogeneity (variation in number of contacts) on T_c in two different control scenarios.

We find that generally pathogens needed significantly higher critical transmissibility values on fluid-exchange contact networks than on physical, non-physical, and indirect transmission contact networks (figure 2). By comparing our empirical simulation scenario to the two control scenarios, we find that fluid-exchange and indirect contact networks are more vulnerable to disease invasion (i.e. lower T_c) than expected based on their average connectivity (figure 2). This result is consistent with network epidemiology theory, which predicts that higher degree heterogeneity (as we find in fluid-exchange and indirect contact networks) make disease invasion more likely. For physical and non-physical contact networks, on the other hand, the critical transmissibility is comparable in all three scenarios, suggesting that the average network connectivity (or network density) is sufficient to predict disease invasion in such networks.

(c) The structure of a contact network can influence the infection characteristics of associated pathogens

We compiled peer-reviewed data from common human pathogens as life-history data on pathogens in non-human animals was extremely limited. We visualized two of the characteristics that define a pathogen's transmissibility: its infectious period and its infection probability (figure 3).

First, we showed that pathogens that transmit on indirect contact networks (e.g. food/waterborne, faecal–oral) have relatively short infectious periods, and varied between low to moderate infection probability. Next, we found pathogens that transmit on fluid-exchange contact networks (e.g. sex, saliva) have the longest infectious periods. Of those, the two pathogens (herpes, syphilis) that tend to have shorter infectious periods (despite intermittent infectivity over long periods) but alternatively have moderate to high infectivity. These results suggest that fluid-exchange pathogens do in fact have higher T_c values than other pathogens, and they tend to increase their T_c by extending their infectious periods over increasing their infection probability. Finally, we see that pathogens that transmit on physical and non-physical contact networks have some of the shortest infectious periods and a range of infection probabilities, but most pathogens with high infectivity are associated with these transmission modes.

4. Discussion

Our study shows that networks characteristic of different pathogen transmission modes differ in terms of their structure. We then go on to demonstrate that differences in network structure will affect the transmissibility required for a pathogen to successfully proliferate. Finally, we suggest these network structures likely impact the evolution of a focal pathogen's infection characteristics, supported by a review of human pathogen traits.

(a) Differences among contact networks and their implications for pathogen spread

Most notably, compared to physical and non-physical contact networks, fluid-exchange networks were less dense and had more heterogeneous degree values, with greater diameters, reduced clustering and greater disassortativity by degree. They also had more bridge nodes than all other network types. That is, these networks tend to be more poorly connected, and with greater skew in nodes' importance in the network. This selection of network traits likely arises through a combination of mechanisms, linked to the fact that many of these networks were based on sexual interactions.

First, the infrequency of copulation events will drive low network density, with correlated increases in the diameter of the network and the prevalence of bridge connections; second, the fact that all networks solely included male–female copulation events likely increases clustering and reduces the tendency for assortative mating; and finally, the common nature of polygyny and overdispersed mating events (i.e. few individuals monopolizing sexual resources) will drive greater degree heterogeneity, as well as driving disassortativity. These latter traits agree with our understanding of the overdispersed nature of sexual interaction networks [41–43]. The fluid-exchange category also included trophallaxis networks, which are also generally highly heterogeneous; in ants, more than 50% of trophallaxis interactions may come from less than 25% of individuals [44]. While the majority of the trophallaxis networks represented in our dataset are indeed in ants, many other species such as birds and mammals also partake in trophallaxis, usually in the form of parental care [45]; these unique parent–offspring interactions would also likely result in sparse, heterogeneous and highly fragmented networks.

These traits could have a selection of important consequences for transmission of pathogens through fluid-exchange networks. First, less dense networks are likely to provide fewer transmission opportunities for fluid-borne pathogens [46], while larger diameters will inhibit the spread of an outbreak. Second, degree heterogeneity is known to be a key driver of sexually transmitted pathogen risk in human contact networks [16,47]. Where contact numbers are highly heterogeneous, superspreaders can lead to rapid, explosive outbreaks, allowing pathogens to persist [21]. Indeed, past work has shown that HIV, a sexually transmitted pathogen, can exploit this contact heterogeneity to attain sufficient transmissibility, and other sexually transmitted pathogens likely do the same [48]. However, previous work on contact networks has shown that network density and individual variation in contact are negatively correlated [49]. Our results support these findings, as we found fluid-exchange and indirect contact networks have lower densities and higher-degree heterogeneity compared to physical and non-physical contact networks. This suggests that while having high-degree heterogeneity might make a network more vulnerable to explosive outbreaks [18,21], this may trade off with lower overall transmission probabilities [49]. Third, low clustering values might be beneficial for pathogens, especially given the low densities, as a pathogen may be less likely to get stuck in cliques that might form among individuals [50]. Fourth, the common nature of bridge nodes in fluid-exchange networks might be especially important when considering control measures for pathogens on these networks. Previous work has shown that pathogens with high transmissibility are able to persist in socially fragmented networks because bridge nodes allow for transmission among communities [30]. Therefore, the removal of these nodes will prevent or slow pathogen spread through a population [47]. While both physical and fluid-exchange transmission require relatively close contact, controlling disease spread by identifying bridge nodes might be more powerful for fluid-exchange pathogens than for others.

Notably, indirect contact networks had a more heterogeneous degree distribution than physical and non-physical contact networks, meaning there is more between-individual variation in asynchronous resource sharing compared to that in close proximity or physical touch. Past research has demonstrated this phenomenon in many solitary desert species that share burrows asynchronously, and while there are many ecological factors that might drive these individual preferences for resource-use patterns (such as differences in sex, age and environment) [30,51], we still do not have a full mechanistic understanding of them. It is possible that this pattern will drive greater heterogeneity in infection with indirectly transmitted pathogens.

(b) Network structure and the pressures on pathogen characteristics

Pathogens that transmit on indirect contact networks (e.g. food/waterborne, faecal–oral) seem to have relatively short infectious periods, and vary between low to moderate infection probabilities (figure 3). However, we found many aspects of indirect contact network structure were not different from fluid-exchange networks (i.e. low network densities and high degree heterogeneity) (figure 1). These results contradict expectations that these indirect networks should be highly connected since individuals need to have only used the same space at some point in time to be connected. For example, many individuals sharing the same sanitation facilities through time such as on airplanes [52], cruise ships [53] and hotels [54] can cause recurring outbreaks of Norwalk virus, a common faecal–oral pathogen; as there is no need to physically contact an individual to be infected by them, these contact networks are known to be extremely highly connected [55]. There are several possible explanations for our surprising finding: first, our results may be driven by the fact that relatively solitary species (which have low connectivity due to their social structure [30]) are highly represented in our indirect networks sample. Additionally, these networks often involve territorial species; resource sharing (both synchronous and asynchronous) is likely minimized in species that hold territories (e.g. [56]). Moreover, territoriality can be sex specific in that males very rarely use the same space and resources even asynchronously but females can move freely between male territories, which can result in a sex-specific degree heterogeneity in some species (e.g. [57,58]). Indeed, our GLMM showed a high amount of variation in the effect sizes of indirect contact networks and we found that pathogens using indirect contact networks do not need high T_c values to persist on these networks. This suggests that a species' social system strongly influences the structure of indirect contact networks. Including additional species with other social systems may increase the average connectivity of indirect contact networks, which would be more representative of their associated pathogen characteristics. This paucity of variation in social systems is a common problem in meta-analyses of social

network structure, and ongoing data collection may help to ameliorate this difficulty in the future.

Our simulations revealed that contact network structure should motivate the evolution of higher transmissibility for fluid-exchange pathogens to persist (figure 2); a prediction that was supported by our literature review (figure 3). This supports what we know about the host behaviours involved in bodily fluid exchange; individuals usually have long temporal gaps in between fluid-exchange events compared to other potential disease transmission host behaviours [1]. Therefore, pathogens would benefit more from longer infectious periods giving them more time to spread.

(c) The role of weak ties in defining relevant disease-spreading contact

Previous studies have shown that structural differences between networks are primarily driven by 'weak ties' that are disproportionately lower in intensity, frequency, or duration than other contacts [18]. When we eliminated weak ties, we found that differences between transmission mode categories persisted for some structural features (e.g. average clustering and degree assortativity), but others (density and degree heterogeneity) were lost; in other words, removing the weak ties from contact networks makes them more similar to each other in their number and variation of contacts. Given that the structural features of density and degree heterogeneity were most different among fluid-exchange networks, this finding suggests that not only do individuals tend to vary in the number of fluid-exchange contacts they have compared to their other types of contacts, but individuals also vary more in the strength of connections between their different fluid-exchange contacts, compared to their other types of contacts. We hypothesize that this might be the crucial difference between pathogens that spread via fluid exchange and others, but additional data would be required to confirm this hypothesis.

This heterogeneity in how individuals distribute their contact effort is known as 'social fluidity', where higher social fluidity suggests a higher prevalence of weak ties [9]. Past work has shown that non-physical contact networks (e.g. spatial association) have smaller values of social fluidity than fluid-exchange networks (e.g. trophallaxis), suggesting that social fluidity and weak ties are especially relevant when considering disease transmission potential in these networks [9]. In some instances, not considering a very brief or infrequent contact as 'relevant' for disease transmission might make sense, such as for pathogens that propagate on networks with low social fluidity (e.g. flu). However, for pathogens that are spread via fluid exchange, we found that the density and degree heterogeneity of contact networks are important predictors for determining their transmissibility and traits. This suggests it is imperative to include short or infrequent fluid-exchange interactions when considering the definition of relevant contact for modelling the transmission of these pathogens. By not including these 'weak' contacts, it is likely that estimates of fluid-exchange pathogen spread would be inaccurate, and proposed control measures based on these estimates could be unreliable.

(d) Study limitations

Our study has some important limitations. First, we investigated non-human animal networks, while using pathogen traits in humans to understand the implications of network structure on infection characteristics. We would expect some aspects of human contact networks to differ from animal contact networks, particularly with fluid-exchange transmission. For example, human sexual networks have much higher clustering values than what we observed in our animal networks [59]. However, this could be due to the lack of recorded same-sex sexual host behaviours in non-human animal species; same-sex host behaviours including fluid exchange do occur in many non-human animals and are therefore likely underrepresented in our sample [60]. Regardless, even if true clustering values are higher than observed, we would expect this to *reduce* the R_0 of a pathogen in a network; this might further increase the transmissibility needed to persist [59], further supporting our current results. Overall, we found that network density and degree heterogeneity were the most important metrics when considering a pathogen's ability to persist on a network; we show that fluid-exchange contact networks have low densities and high degree of variation which holds true in human networks [1,16,47]. Future studies could further validate this work by exploring the contact structures of available human contact networks. Alternatively, future work could provide a better overview of the natural histories of pathogens in wildlife. While we would expect infection characteristics to be similar based on our network analysis, we suggest a more thorough review of these characteristics across different taxa.

Our available network datasets likewise restricted our ability to test and untangle some factors. First, because we had a lack of data on indirect contact networks in wildlife species, we were unable to investigate the evolution of traits of environmentally transmitted pathogens. We also did not have good representation of contact networks representative of each transmission mode category across all taxonomic classes, which reduced our ability to control for host taxonomy in our model. Future work may be able to ameliorate these difficulties by measuring indirect contact networks from species with gregarious and hierarchical social makeups, as well increasing the taxonomic sample size of each transmission mode category, for a better representation of contact networks across species and social systems.

Lastly, our work does not consider the impacts of pathogen-mediated changes to contact structure as caused by sickness behavioural changes due to host immune response or pathogen virulence, nor due to pathogen manipulation of host behaviour [15]. For example, rabies can cause increased aggression and biting in hosts, which could increase the number of edges in some physical contact networks [61]. By contrast, some pathogens cause 'sickness behaviours' in their hosts, such as bacterial pneumonia in kudu antelope (*Tragelaphus strepsiceros*), where individuals have been shown to develop fevers and reduce their daily activities by 60% [62]. In cases where infected individuals exhibit sickness behaviours like these, they may reduce their contact rates such that the subsequent contact networks will have fewer edges. We assume the networks in our dataset represent host behaviour in the absence of active infections, but recognize that such disease-mediated behaviour change can alter both network structure and realized pathogen characteristics (e.g. infectious periods can be effectively reduced via sickness behaviours). Future work must consider this critical feedback loop between contact structure and pathogen characteristics, in light of different pathogen transmission modes.

(e) Broader disease ecology implications

It is widely accepted in disease ecology that host behaviour drives pathogen spread, and we have demonstrated how this relationship is affected by the type of contact necessary for transmission. For epidemic dynamics, understanding transmission routes are also necessary as they determine how the density and structure of the population affect the rate at which the disease will spread. Typically, if disease spreads indirectly or through co-incident contact then the transmission rate is assumed to scale proportionally with population density (density-dependence), whereas if transmission requires close, intentional contacts then we expect social connectivity to determine the outcome (frequency-dependence). Classically, this scaling of transmission with population density has been based on the transmission route of the pathogen, with sexually transmitted diseases generally assumed to be transmitted in a frequency-dependent manner [59,63], and respiratory-transmitted disease expected to spread in a density-dependent manner. However, past work has demonstrated that the scaling relationship depends more explicitly on heterogeneity in contacts [9,64], with higher heterogeneity being associated with less density-dependence, rather than pathogen biology. Our work empirically links these two concepts by demonstrating that pathogen transmission mode is associated with contact structure, e.g. contact networks relevant to sexually transmitted diseases are more heterogeneous in contacts and thus are expected to be frequency-dependent. However, our *a priori* classification of behaviours by transmission mode may be obscuring structural variability that exists within modes. For example, Colman *et al*. [9] demonstrate that networks with aggressive contacts (e.g. head-butting) have low heterogeneity (thus suggesting density-dependence), while networks with bonding contacts (e.g. grooming) have high heterogeneity (thus suggesting frequency-dependence). In our work, we classified both of these contact types in the physical transmission category, thus limiting the ability to detect this variation in scaling within transmission modes. Future work would benefit from a characterization of transmission scaling based on contact network structure, rather than assumptions about pathogen transmission modes.

Additional variation in host behaviour can be attributed to social differences among and within species [18]. For example, the pace-of-life-history can explain variation in social relationships and across taxonomic scales. While we control for different social systems in our model, there is also potential for further network variation within these social systems and within species. For example, mating system dynamics and patterns of sexual promiscuity can vary widely within groups and populations of the same species, as a function of ecological variation and population sex ratios [43,65,66]. Moreover, two ungulate species Grevy's zebra (*Equus grevyi*) and onagers (*Equus hemionus khur*), which both have gregarious social systems, have been shown to have significantly different network structures, likely due to species traits that have evolved from inhabiting different environments [67]. Additionally, recent work has shown the importance of considering spatial components of individual behaviour (e.g. home ranges, landscape use) when modelling social networks and disease transmission [68] as they allow for more accurate model estimates, including a better inference of pathogen transmission modes [69]. We do expect there to be variation in host contact network structure related to traits and spatial behaviours, and suggest that future meta-analytic work that captures host heterogeneities and spatial structure will be necessary to better address this problem empirically.

Data accessibility. All networks used in this study can be found in the Animal Social Network Repository (https://github.com/bansallab/asnr) [25]. All code can be found at https://github.com/mac532/pathogen-transmission-contact-network.

The data are provided in the electronic supplementary material [70].

Authors' contributions. M.C.: conceptualization, data curation, formal analysis, investigation, methodology, visualization, writing—original draft; S.B.: conceptualization, funding acquisition, investigation, methodology, supervision, writing—review and editing; G.F.A.: formal analysis, methodology, supervision, validation, writing—review and editing; G.C.M.: data curation, resources, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

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References

1. Garnett GP, Bowden FJ. 2000 Epidemiology and control of curable sexually transmitted diseases: opportunities and problems. *Sex. Transm. Dis.* **27**, 588–599. (doi:10.1097/00007435-200011000-00007)
2. Kirchner JW, Roy BA. 2002 Evolutionary implications of host–pathogen specificity: fitness consequences of pathogen virulence traits. *Evol. Ecol. Res.* **4**, 27–48.
3. Day T. 2001 Parasite transmission modes and the evolution of virulence. *Evolution* **55**, 2389–2400.
4. Antonovics J. 2017 Transmission dynamics: critical questions and challenges. *Phil. Trans. R. Soc. B* **372**, 20160087. (doi:10.1098/rstb.2016.0087)
5. Robinson K, Cohen T, Colijn C. 2012 The dynamics of sexual contact networks: effects on disease spread and control. *Theor. Popul. Biol.* **81**, 89–96. (doi:10.1016/j.tpb.2011.12.009)
6. Antonovics J *et al.* 2017 The evolution of transmission mode. *Phil. Trans. R. Soc. B* **372**, 20160083. (doi:10.1098/rstb.2016.0083)
7. Leu ST, Kappeler PM, Bull CM. 2010 Refuge sharing network predicts ectoparasite load in a lizard. *Behav. Ecol. Sociobiol.* **64**, 1495–1503. (doi:10.1007/s00265-010-0964-6)
8. Leu ST, Sah P, Krzyszczyk E, Jacoby A-M, Mann J, Bansal S. 2020 Sex, synchrony, and skin contact: integrating multiple behaviors to assess pathogen transmission risk. *Behav. Ecol.* **31**, 651–660. (doi:10.1093/beheco/araa002)
9. Colman E, Colizza V, Hanks EM, Hughes DP, Bansal S. 2021 Social fluidity mobilizes contagion in human and animal populations. *Elife* **10**, e62177. (doi:10.7554/eLife.62177)
10. Romano V, Duboscq J, Sarabian C, Thomas E, Sueur C, MacIntosh AJJ. 2016 Modeling infection transmission in primate networks to predict centrality-based risk. *Am. J. Primatol.* **78**, 767–779. (doi:10.1002/ajp.22542)
11. Sah P, Otterstatter M, Leu ST, Leviyang S, Bansal S. 2021 Revealing mechanisms of infectious disease

spread through empirical contact networks. *PLoS Comput. Biol.* **17**, e1009604. (doi:10.1371/journal.pcbi.1009604)

12. So N, Franks B, Lim S, Curley JP. 2015 A social network approach reveals associations between mouse social dominance and brain gene expression. *PLoS ONE* **10**, e0134509. (doi:10.1371/journal.pone.0134509)
13. Ames GM, George DB, Hampson CP, Kanarek AR, McBee CD, Lockwood DR, Achter JD, Webb CT. 2011 Using network properties to predict disease dynamics on human contact networks. *Proc. R. Soc. B* **278**, 3544–3550. (doi:10.1098/rspb.2011.0290)
14. Bansal S, Meyers LA. 2012 The impact of past epidemics on future disease dynamics. *J. Theor. Biol.* **309**, 176–184. (doi:10.1016/j.jtbi.2012.06.012)
15. Bansal S, Read J, Pourbohloul B, Meyers LA. 2010 The dynamic nature of contact networks in infectious disease epidemiology. *J. Biol. Dyn.* **4**, 478–489. (doi:10.1080/17513758.2010.503376)
16. Kretzschmar M. 2000 Sexual network structure and sexually transmitted disease prevention: a modeling perspective. *Sex. Transm. Dis.* **27**, 627–635. (doi:10.1097/00007435-200011000-00011)
17. Mohr S, Deason M, Churakov M, Doherty T, Kao RR. 2018 Manipulation of contact network structure and the impact on foot-and-mouth disease transmission. *Prev. Vet. Med.* **157**, 8–18. (doi:10.1016/j.prevetmed.2018.05.006)
18. Sah P, Mann J, Bansal S. 2018 Disease implications of animal social network structure: a synthesis across social systems. *J. Anim. Ecol.* **87**, 546–558. (doi:10.1111/1365-2656.12786)
19. Woolhouse MEJ *et al.* 1997 Heterogeneities in the transmission of infectious agents: implications for the design of control programs. *Proc. Natl Acad. Sci. USA* **94**, 338–342. (doi:10.1073/pnas.94.1.338)
20. Anderson RM, May RM. 1982 Coevolution of hosts and parasites. *Parasitology* **85**, 411–426. (doi:10.1017/S0031182000055360)
21. Bansal S, Grenfell BT, Meyers LA. 2007 When individual behaviour matters: homogeneous and network models in epidemiology. *J. R. Soc. Interface.* **4**, 879–891. (doi:10.1098/rsif.2007.1100)
22. Meyers LA, Pourbohloul B, Newman MEJ, Skowronski DM, Brunham RC. 2005 Network theory and SARS: predicting outbreak diversity. *J. Theor. Biol.* **232**, 71–81. (doi:10.1016/j.jtbi.2004.07.026)
23. Platt R, Rice PA, McCormack WM. 1983 Risk of acquiring gonorrhea and prevalence of abnormal adnexal findings among women recently exposed to gonorrhea. *J. Am. Med. Assoc.* **250**, 3205–3209. (doi:10.1001/jama.1983.03340230057031)
24. Nonaka E, Ebel GD, Wearing HJ. 2010 Persistence of pathogens with short infectious periods in seasonal tick populations: the relative importance of three transmission routes. *PLoS ONE* **5**, e11745. (doi:10.1371/journal.pone.0011745)
25. Sah P, Collier M, Ali S, Mendez JD, Bansal S. 2020 Animal Social Network Repository (Version 2.0). See https://github.com/bansallab/asnr.
26. Sah P, Méndez JD, Bansal S. 2019 A multi-species repository of social networks. *Sci. Data.* **6**, 44. (doi:10.1038/s41597-019-0056-z)
27. Blondel VD, Guillaume JL, Lambiotte R, Lefebvre E. 2008 Fast unfolding of communities in large networks. *J. Stat. Mech: Theory Exp.* **2008**, P10008. (doi:10.1088/1742-5468/2008/10/P10008)
28. Hadfield J 2021 MCMCglmm Course Notes. See https://mran.microsoft.com/snapshot/2018-08-24/web/packages/MCMCglmm/vignettes/CourseNotes.pdf.
29. Newman MEJ. 2002 Spread of epidemic disease on networks. *Phys. Rev. E – Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.* **66**, 016128.
30. Sah P, Nussear KE, Esque TC, Aiello CM, Hudson PJ, Bansal S. 2016 Inferring social structure and its drivers from refuge use in the desert tortoise, a relatively solitary species. *Behav. Ecol. Sociobiol.* **70**, 1277–1289. (doi:10.1007/s00265-016-2136-9)
31. ECDC. 2016 Systematic review on the incubation and infectiousness/shedding period of communicable diseases in children. See https://www.ecdc.europa.eu/sites/default/files/media/en/publications/Publications/systematic-review-incubation-period-shedding-children.pdf.
32. Richardson M, Elliman D, Maguire H, Simpson J, Nicoll A. 2001 Evidence base of incubation periods, periods of infectiousness and exclusion policies for the control of communicable diseases in schools and preschools. *Pediatr. Infect. Dis. J.* **20**, 380–391. (doi:10.1097/00006454-200104000-00004)
33. Stamm LV. 2015 Syphilis: antibiotic treatment and resistance. *Epidemiol. Infect.* **143**, 1567–1574. (doi:10.1017/S0950268814002830)
34. Garnett GP, Aral SO, Hoyle DV, Cates W, Anderson RM. 1997 The natural history of syphilis: implications for the transmission dynamics and control of infection. *Sex. Transm. Dis.* **24**, 185–200. (doi:10.1097/00007435-199704000-00002)
35. Garnett GP. 2002 The geographical and temporal evolution of sexually transmitted disease epidemics. *Sex. Transm. Infect.* **78**, i14–i19. (doi:10.1136/sti.78.suppl_1.i14)
36. Stupiansky NW, Van Der Pol B, Williams JA, Weaver B, Taylor SE, Fortenberry JD. 2011 The natural history of incident gonococcal infection in adolescent women. *Sex. Transm. Dis.* **38**, 750–754. (doi:10.1097/OLQ.0b013e31820ff9a4)
37. Althaus CL, Heijne JCM, Low N. 2012 Towards more robust rstimates of the transmissibility of *Chlamydia trachomatis*. *Sex. Transm. Dis.* **39**, 402–404. (doi:10.1097/OLQ.0b013e318248a550)
38. Golden MR, Schillinger JA, Markowitz L, St. Louis ME. 2000 Duration of untreated genital infections with *Chlamydia trachomatis*: a review of the literature. *Sex. Transm. Dis.* **27**, 329–337. (doi:10.1097/00007435-200007000-00006)
39. Krieger JN, Verdon M, Siegel N, Holmes KK. 1993 Natural history of urogenital trichomoniasis in men. *J. Urol.* **149**, 1455–1458. (doi:10.1016/S0022-5347(17)36414-5)
40. Van Der Pol B, Williams JA, Orr DP, Batteiger BE, Fortenberry JD. 2005 Prevalence, incidence, natural history, and response to treatment of *Trichomonas vaginalis* infection among adolescent women. *J. Infect. Dis.* **192**, 2039–2044. (doi:10.1086/498217)
41. Janicke T, Häderer IK, Lajeunesse MJ, Anthes N. 2016 Evolutionary biology: Darwinian sex roles confirmed across the animal kingdom. *Sci. Adv.* **2**, 1–11. (doi:10.1126/sciadv.1500983)
42. Liljeros F, Edling CR, Amaral LAN, Stanley HE, Åberg Y. 2001 The web of human sexual contacts. *Nature* **411**, 907–908. (doi:10.1038/35082140)
43. Taylor ML, Price TAR, Wedell N. 2014 Polyandry in nature: a global analysis. *Trends Ecol. Evol.* **29**, 376–383. (doi:10.1016/j.tree.2014.04.005)
44. Bles O, Deneubourg JL, Nicolis SC. 2018 Food dissemination in ants: robustness of the trophallactic network against resource quality. *J. Exp. Biol.* **221**, 1–4.
45. Rosenblatt JS. 2003 Outline of the evolution of behavioral and nonbehavioral patterns of parental care among the vertebrates: critical characteristics of mammalian and avian parental behavior. *Scand. J. Psychol.* **44**, 265–271. (doi:10.1111/1467-9450.00344)
46. Bloodgood JM, Hornsby JS, Rutherford M, McFarland RG. 2017 The role of network density and betweenness centrality in diffusing new venture legitimacy: an epidemiological approach. *Int. Entrep. Manag. J.* **13**, 525–552. (doi:10.1007/s11365-016-0412-9)
47. Doherty IA, Adimora AA, Muth SQ, Serre ML, Leone PA, Miller WC. 2011 Comparison of sexual mixing patterns for syphilis in endemic and outbreak settings. *Sex. Transm. Dis.* **38**, 378–384. (doi:10.1097/OLQ.0b013e318203e2ef)
48. Kao RR. 2006 Evolution of pathogens towards low R0 in heterogeneous populations. *J. Theor. Biol.* **242**, 634–642. (doi:10.1016/j.jtbi.2006.04.003)
49. Davis S, Abbasi B, Shah S, Telfer S, Begon M. 2015 Spatial analyses of wildlife contact networks. *J. R Soc. Interface.* **12**, 20141004. (doi:10.1098/rsif.2014.1004)
50. Wey T, Blumstein DT, Shen W, Jordán F. 2008 Social network analysis of animal behaviour: a promising tool for the study of sociality. *Anim. Behav.* **75**, 333–344. (doi:10.1016/j.anbehav.2007.06.020)
51. Santos JWA, Lacey EA. 2011 Burrow sharing in the desert-adapted torch-tail spiny rat, *Trinomys yonenagae*. *J. Mammal.* **92**, 3–11. (doi:10.1644/09-MAMM-S-389.1)
52. Thornley CN, Emslie NA, Sprott TW, Greening GE, Rapana JP. 2011 Recurring norovirus transmission on an airplane. *Clin. Infect. Dis.* **53**, 515–520. (doi:10.1093/cid/cir465)
53. Isakbaeva ET *et al.* 2005 Norovirus transmission on cruise ship. *Emerg. Infect. Dis.* **11**, 154–157. (doi:10.3201/eid1101.040434)
54. Cheesbrough JS, Green J, Gallimore CI, Wright PA, Brown DWG. 2000 Widespread environmental contamination with Norwalk-like viruses (NLV) detected in a prolonged hotel outbreak of gastroenteritis. *Epidemiol. Infect.* **125**, 93–98. (doi:10.1017/S095026889900432X)
55. Rushton SP, Sanderson RA, Reid WDK, Shirley MDF, Harris JP, Hunter PR, O'Brien SJ. 2019 Transmission

routes of rare seasonal diseases: the case of norovirus infections. *Phil. Trans. R. Soc. B* **374**, 20180267. (doi:10.1098/rstb.2018.0267)
56. Stamps JA. 1990 The effect of contender pressure on territory size and overlap in seasonally territorial species. *Am. Nat.* **135**, 614–632. (doi:10.1086/285065)
57. Eikenaar C, Richardson DS, Brouwer L, Bristol R, Komdeur J. 2009 Experimental evaluation of sex differences in territory acquisition in a cooperatively breeding bird. *Behav. Ecol.* **20**, 207–214. (doi:10.1093/beheco/arn136)
58. Roth T, Sprau P, Schmidt R, Naguib M, Amrhein V. 2009 Sex-specific timing of mate searching and territory prospecting in the nightingale: nocturnal life of females. *Proc. R. Soc. B* **276**, 2045–2050. (doi:10.1098/rspb.2008.1726)
59. Szendrói B, Csányi G. 2004 Polynomial epidemics and clustering in contact networks. *Proc. R. Soc. B* **271**(Suppl. 5), S364–S366. (doi:10.1098/rsbl.2004.0188)
60. Monk JD, Giglio E, Kamath A, Lambert MR, McDonough CE. 2019 An alternative hypothesis for the evolution of same-sex sexual behaviour in animals. *Nat. Ecol. Evol.* **3**, 1622–1631. (doi:10.1038/s41559-019-1019-7)
61. Jackson AC. 2016 Diabolical effects of rabies encephalitis. *J. Neurovirol.* **22**, 8–13. (doi:10.1007/s13365-015-0351-1)
62. Hetem RS, Mitchell D, Maloney SK, Meyer LCR, Fick LG, Kerley GIH, Fuller A. 2008 Fever and sickness behavior during an opportunistic infection in a free-living antelope, the greater kudu (*Tragelaphus strepsiceros*). *Am. J. Physiol. – Regul. Integr. Comp. Physiol.* **294**, 246–254. (doi:10.1152/ajpregu.00570.2007)
63. Antonovics J, Boots M, Abbate J, Baker C, Mcfrederick Q, Panjeti V. 2011 Biology and evolution of sexual transmission. *Ann. NY Acad. Sci.* **1230**, 12–24. (doi:10.1111/j.1749-6632.2011.06127.x)
64. Ferrari M, Perkins SE, Pomeroy LW, Bjørnstad ON. 2011 Pathogens, social networks, and the paradox of transmission scaling. *Interdiscip. Perspect. Infect. Dis.* **2011**, 267049. (doi:10.1155/2011/267049)
65. Sih A, Montiglio P, Wey T, Fogarty S. 2017 Altered physical and social conditions produce rapidly reversible mating systems in water striders. *Behav. Ecol.* **28**, 632–639. (doi:10.1093/beheco/arx021)
66. Kappeler PM *et al.* 2022 Sex roles and sex ratios in animals. *Biol. Rev*. (doi:10.1111/brv.12915)
67. Sundaresan SR, Fischhoff IR, Dushoff J, Rubenstein DI. 2007 Network metrics reveal differences in social organization between two fission–fusion species, Grevy's zebra and onager. *Oecologia* **151**, 140–149. (doi:10.1007/s00442-006-0553-6)
68. Albery GF, Morris A, Morris S, Pemberton JM, Clutton-Brock TH, Nussey DH, Firth JA. 2021 Multiple spatial behaviours govern social network positions in a wild ungulate. *Ecol. Lett.* **24**, 676–686. (doi:10.1111/ele.13684)
69. Albery GF, Kirkpatrick L, Firth JA, Bansal S. 2021 Unifying spatial and social network analysis in disease ecology. *J. Anim. Ecol.* **90**, 45–61. (doi:10.1111/1365-2656.13356)
70. Collier M, Albery GF, McDonald GC, Bansal S. 2022 Pathogen transmission modes determine contact network structure, altering other pathogen characteristics. Figshare. (doi:10.6084/m9.figshare.c.6316518)