# A comparative network approach to assess the social-ecological underpinnings of zoonotic outbreaks at human-wildlife interfaces

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**Abstract:**

Pandemics caused by wildlife-origin pathogens, like COVID-19, highlight the importance of understanding the ecology of zoonosis at human-wildlife interfaces. To-date, the relative effects of human-wildlife and wildlife-wildlife interactions on zoonotic outbreaks among wildlife populations remain unclear. In this study, we used social network analysis and epidemiological Susceptible Infected Recovered (SIR) models, to track zoonotic outbreaks through wild animals' social-ecological co-interactions with humans and their social grooming interactions with conspecifics, for 10 groups of macaques (*Macaca* spp.) living in (peri)urban environments across Asia. Outbreak sizes predicted by the SIR models were related to structural features of the social networks, and particular properties of individual animals' connectivity within those networks. Outbreak sizes were larger when the first-infected animal was highly central, in both types of networks. Across host-species, particularly for rhesus and bonnet macaques, the effects of network centrality on outbreak sizes were stronger through macaques' human co-interaction networks compared to grooming networks. Our findings, independent of pathogen-transmissibility, suggest that for wildlife populations in the Anthropocene, vulnerability to zoonotic outbreaks may outweigh the potential/perceived benefits of interacting with humans to procure anthropogenic food. From One Health perspectives, animals that consistently interact with humans (and their own conspecifics) across time and space are useful targets for disease-control.

**Key Words:**

Human-wildlife interactions, behavioral ecology, zoonosis, social network analysis, epidemiological models, nonhuman primates

**Main Text:**

*Introduction:*

The COVID-19 pandemic has highlighted the importance of understanding infectious disease transmission at human-wildlife interfaces (HWIs) - the places where wildlife and humans come into contact, in order to prevent future pandemics[1,2] . Global population expansion has increased spatial overlap and contact rates between humans and wildlife[3–6]. HWIs are now widely recognized as 'hotspots' for the transmission of (anthropo)zoonotic (humans to wildlife, and vice-versa) infectious diseases[7–9]. Despite this widespread recognition, there exists little quantitative, comparative research that unravels the *pathways* through which infectious agents may enter into and spread through wildlife (and human) populations at these locations. From an evolutionary perspective, such assessments can provide insights into how infectious disease risk influences, and is in-turn influenced by, (mal)adaptive responses in wildlife socioecology[10,11,12] and behavioral flexibility when it comes to taking risks in these challenging environments[12–14]. From a conservation and public health perspective, they are critical to identify "edge" wildlife[15], that is individual animals or species that range at HWIs, which may act as 'reservoirs' and potential 'superspreaders' of infectious agents into overlapping wildlife populations and human communities[7–9].

Research on disease transmission at HWIs has been largely hampered by methodological and conceptual limitations[16]. Traditional research on wildlife populations assumed that the probability of acquiring an infectious agent is equal across individuals within a defined area or cohort[17]. In reality, wild animals at HWIs may interact with both other animals and humans, may

do so to different extents across individuals, time, and space, and may form patterns of associations through such interactions that could influence the acquisition and transmitting infectious agents. Social Network Analysis (SNA) offer exciting avenues to captures such patterns of associations[18–22]. Improving on traditional epidemiological frameworks, SNA offers promising quantitative ways to allow for animals' tendencies to interact differently, and to varying extents, with different socio-ecological aspects of their environment (e.g., their conspecifics, other animals, humans), and assess how such non-random contact patterns and behavior can impact infectious disease transmission[18–22]. Yet while SNA-based epidemiological assessments have been increasingly implemented to evaluate disease transmission in both group-living wild animals and humans[18–23], HWIs have seldom been the foci of such assessments.

To-date, epidemiological studies that have implemented SNA have largely focused on animal-animal interactions, and often on single behavioral features that define such interactions (reviewed below). However, infectious agents at HWIs may spread differently through different types of interactions, for instance through networks of human-wildlife and wildlife-wildlife interactions. Some examples of wildlife-wildlife social networks that have been associated with increased risk of infectious agent transmission include affiliative contact associations (e.g., Tasmanian devils, *Sarcophilus harrisii*[24]; skinks, *Egernia stokesii*[25]; giraffes, *Giraffa camelopardalis*[26]), aggression (e.g., meerkats, *Suricata suricatta*[27]), social grooming (e.g., Japanese macaques, *Macaca fuscata*[28]; brown spider monkeys, *Ateles geoffrey*[29]), and contact-huddling (rhesus macaques, *M. mulatta*[30]). While interesting, such studies largely do not capture important complexity and variability in animal social systems, such as individuals potentially interacting in different ways with their conspecifics compared to with other, shared features of their environment (e.g., accessing and sharing space and resources, interspecies encounters with predators, prey, and humans)[18–22]. In particular, disease transmission among wildlife at HWIs may be driven by such multiple, potentially interplaying types of interactions, including inter-individual differences in animals' interactions with conspecifics[31,32], humans [2,7–9], and anthropogenic features like contaminated water, soil, human foods, livestock, and other feral mammals[20,33]. It is therefore crucial to assess zoonotic transmission through multiple (rather than single or specific) types of interactions and their resultant network connections at HWIs.

Another issue is that epidemiological assessments of zoonotic transmission at HWIs continue to be hampered by many ecological (e.g., trapping and sampling of wildlife) and logistical (e.g., tracking human behavior and long-term health indicators) constraints. When data is incomplete or unavailable, mathematical models offer critical insights into the occurrence of real-world epidemiological processes[34,35]. In this regard, network approaches have been extensively combined with epidemiological models of the 'Susceptible Infected Recovered (SIR)' type[10,31,36–38], to simulate disease transmission and its associated outcomes through human (reviewed in[31,36,37]) and (less so) nonhuman animal (individual empirical studies[10,38–41]) networks. SIR models are bottom-up, compartmental epidemiological models that simulate disease spread by causing entities (individuals) to move across 'susceptible', 'infected', and 'recovered' disease states. They do so at dynamic probabilities that, based on user specifications of model complexity, may depend on a combination of one or more pathogen-specific epidemiological variables (e.g., transmissibility, basic reproduction number: defined below), host contact patterns (e.g., spatial or social network connectedness), and host attributes (e.g., age-sex class) or intrinsic states (e.g., physiology, rates of recovery). To date, studies that have implemented SIR models on wildlife spatial and social networks have revealed strong associations between network connectedness of the first-infected individual and simulated

disease outcomes such as the times to saturation (i.e. when all individuals have been infected thereby leaving no other susceptible individuals) or extinction (i.e. when all individuals have recovered from the disease and no more individuals can be infected), and outbreak sizes (mean % of infected individuals) of pathogens[38,40,41]. To our knowledge, SIR models remain largely unimplemented at HWIs (see [41] for an exceptional study of Barbary macaques, *M. sylvanus*), and especially in contexts of human-wildlife interactions in (peri)urban ecological settings where contact between people and wildlife maybe highly frequent and vary across time and space.

Human-nonhuman primate interfaces are well-suited to address the above gaps. Beyond sharing close evolutionary histories with humans[42,43], several nonhuman primate (hereafter NHP) taxa share social-ecological relationships with humans and anthropogenic factors. While some have done so for longer periods of their evolutionary history due to their ecological flexibility (e.g., Chacma baboons, *Papio ursinus*[44]; macaques, *Macaca* spp.[45–47]; plains langurs, *Semnopithecus* spp.[48]), others have more recently been exposed to anthropogenic factors through activities like tourism and habitat encroachment (e.g., chimpanzees, *Pan troglodytes*[49]; mountain gorillas, *Gorilla gorilla beringei*[50]). Unsurprisingly, human-primate interfaces are 'hotspots' for zoonosis and disease emergence[7,8,16,51–53] meaning that these phenomena consistently and repeatedly occur in such places, and share many diseases with humans including malaria, measles, HIV/AIDS, Herpes B, and tuberculosis (reviewed in [8]). For example, a recent study also revealed that all apes as well as African and Asian primates are vulnerable to infection from SARS-CoV-2[54]. The genus *Macaca* are among the most ecologically and behaviorally flexible of all nonhuman primates[46,55,56]. In the wild, many macaque species, particularly rhesus macaques, long-tailed macaques (*M. fascicularis*), and bonnet macaques (*M. radiata*), are considered 'edge' wildlife species that form 'synanthropic' associations[57] with humans across a variety of anthropogenic landscapes (e.g. cities, temples, parks, fields) where they experience highly spatiotemporally variant overlap and interactions with humans[46,55,56,58,59]. Influenced by both their evolutionary histories and their adaptive responses to socioecological[60,61] and anthropogenic factors[58], macaques also show marked inter- and intra-specific variation in social behavior with their conspecifics and (consequently) social networks[12,58,61,62]. Many epidemiological studies have documented evidence of human-to-macaque disease transmission (and vice-versa), including vector-borne malaria parasites, respiratory viruses, and gastrointestinal enteric bacteria and protozoa (reviewed in [16,63]). Yet there is little knowledge of the social-ecological pathways of zoonotic transmission at human-macaque, or indeed other human-primate, interfaces.

We used network approaches combined with SIR models to evaluate the dynamics of zoonotic transmission and outbreaks at multiple human-macaque interfaces in India and Malaysia. For multiple groups and species of macaques living in varying degrees of human impact, we simulated and quantitatively evaluated the relative vulnerability (versus resistance) of wildlife to zoonotic outbreaks through their social-ecological interactions with humans, and their social interactions with conspecifics. To capture patterns of associations in macaques' social-ecological interactions with humans, we constructed networks of macaques' (nodes) shared tendencies to jointly engage in risk-taking or 'co-interacting' with humans (edges), defined as two or more animals that exchanged contact- or non-contact (within three meters) behaviors with humans at the same time and location in the context of anthropogenic spaces[64]. To use network terminology, we treated bimodal social-ecological human-macaque interactions as effectively unimodal (hereafter) 'co-interaction networks' (as has been done previously for host-parasite sharing[65]). To capture patterns of associations of macaque-macaque social interactions, we constructed social 'grooming networks' that linked macaques based on the proportions of time

they spent engaging in grooming their conspecifics. In a previous study, we revealed that macaques' grooming relationships did not predict their tendencies to co-interact with people[64]. In other words, we established a premise to expect that co-interaction networks and grooming networks may offer different, somewhat independent pathways for the transmission of zoonotic agents[64].

Independent of pathogen transmissibility, defined here as the probability of pathogen transmission from an infected individual to a susceptible individual during its infectious period ([38], see Methods for more details), here we examined the impact of the behavioral ecology of wildlife host-species at HWIs on zoonotic outbreaks. *Specifically, we examined the effects of hosts' interaction- or network-type (social-ecological co-interactions with humans, versus grooming of conspecifics), host-species (rhesus, long-tailed, and bonnet macaque), and their interactions with the network connectedness or (hereafter) centrality of the first-infected macaque, on zoonotic transmission and outbreak sizes as predicted by epidemiological models.* As in previous research implementing SIR models on animal networks[38,41], we predicted that the connectedness or (hereafter) centrality of the first-infected macaque, irrespective of host-species and network-type, will be positively correlated to disease outbreak sizes. We also examined whether the magnitude of this effect was different across network-type (co-interaction versus grooming) for each host-species, and across host-species (bonnet macaques versus long-tailed macaques versus rhesus macaques) for each network-type. Rhesus and long-tailed macaques, compared to bonnet macaques, are more geographically widespread and ecologically flexible, and overlap more with humans and anthropogenic environments[46,66]. Yet they typically show more nepotistic (than bonnet macaques) social systems, with individuals preferring to engage more with just specific subsets of group conspecifics than with others[62]. Given these differences, we tested the following predictions. Across network-type for each host-species, we predicted that the co-interaction network centrality of first-infected macaques would have a stronger effect on outbreak sizes than grooming network centrality for rhesus macaques and long-tailed macaques, but that bonnet macaques would show the opposite effect. Across host-species for each network type, we predicted that the effect of co-interaction network centrality of first-infected macaques on outbreak sizes would be higher for rhesus macaques and long-tailed macaques compared to bonnet macaques, but that the reverse would be true (bonnet macaques > rhesus and long-tailed macaques) for the effects of grooming network centrality on outbreak sizes.

We also examined the effects of sociodemographic (sex, dominance rank) characteristics of the first-infected macaque on outbreak sizes. Since females and high-ranking individuals form the core of macaque grooming networks[62,67], we predicted that outbreak sizes through grooming networks would be higher when the first-infected individuals were females (versus males) and higher-ranking (versus lower-ranking) individuals. On the other hand, given the exploratory and increased risk-taking behavior of males resulting in their being more well-connected in co-interaction networks compared to females[60, 64], we predicted that outbreak sizes through co-interaction networks would be higher when the first-infected individuals are males (versus females). Finally, we also explored whether the overall anthropogenic exposure of first-infected macaques, specifically their frequencies of interactions with humans, and time spent foraging on anthropogenic food, influenced zoonotic outbreak sizes.

*Results:*

*Construction of macaques' co-interaction networks and social grooming networks:*

For each of 10 groups of three macaque species observed for between 11-18 months in (peri)urban environments in India and Malaysia (details in [64]; Supplementary Table 1; Supplementary Figure 1), we collected and analyzed demographic and behavioral data on human-macaque interactions, macaque activity budgets, and macaque-macaque social behavior. From this data, we constructed weighted, undirected human co-interaction networks[64]. In these, the nodes were individual, pre-identified macaques, and the weighted edges represented the frequencies with which animals jointly engaged in taking risks in anthropogenic environments, i.e. co-interacted with one or more humans within the same time-frame and anthropogenic space[64]. We also constructed weighted, undirected social grooming networks in which individual monkeys (nodes) were linked based on their frequencies of engaging in social grooming interactions (edges) with their conspecifics[64].

*Impact of the centrality of first-infected individuals by network-type and host-species on disease outbreak sizes:*

Using the SIR model simulations, we examined the impact of network-type, host-species, the network centrality of randomly selected first-infected macaque, and the interactions between them, on mean outbreak sizes. We also examined the effects of the first-infected individuals' sociodemographic characteristics (sex, dominance rank), and overall exposure to anthropogenic factors (frequency of interactions with humans, time spent foraging on anthropogenic food), on mean outbreak sizes. Outbreak sizes were calculated as the mean % of infected individuals within a macaque group at the end of each epidemiological SIR model simulation run marked by either disease extinction or saturation[40] (see Introduction and Methods below for definitions). For each macaque group and network-type, we ran 5000 SIR model simulations, 500 simulations for each of 10 artificially introduced pathogens that were allowed to infect a randomly-chosen 'first-infected' macaque, and whose transmissibility $\tau$ ranged from 0.05 (lowest) to 0.50 (highest) in increments of 0.05. Transmissibility values and ranges were selected based on the corresponding values for pathogens of average basic reproduction numbers ($R_0$) that ranged from 'low' (=1.6) to 'high' (=14.0) in accordance with the human literature[38,40] (see Methods for details). Simulated outbreak sizes were then averaged for each macaque for each network-type from across all its first-infected simulation runs irrespective of pathogen-specific $\tau$. As measures of network centrality, we calculated, for both co-interaction networks and grooming networks, individuals' strength centrality (the number and sum of its direct connections or edge-weights[69,70]), but also its betweenness centrality (the tendency for an individual to inter-link or 'bridge' different parts of a network[19,71,72]), and eigenvector centrality (the number and strength of an individuals' direct and secondary network connections[73–75]) centrality measures (see Methods for more detailed definitions), all of which may influence disease transmission and outbreak sizes[19].

To test our predictions, we used Generalized Linear Mixed Models (GLMMs) with macaque group ID within host-species entered a random effect and implementing a corrected Akaike Information selection Criterion (AICc)[76,77] to identify a single best-fit model from each set. To ensure our findings were not impacted by inter-dependencies in network measures across individuals, we calculated permuted p values for each predictor in our best-fit models, implementing a permutation-based 'null-model' approach that used a post-network node-swapping procedure[78–80]. In support of our prediction, we found that across network-types and

host-species, the strength centrality of the first-infected macaque, which better predicted outbreak sizes than betweenness centrality or eigenvector centrality (model 1 in Supplementary Tables 2A-C, 3A-B), was significantly, positively correlated to mean outbreak size (Tables 1 and 2; Figures 1 and 2). In other words, disease-causing agents generally infected more individuals if they entered into a population by first infecting central or more well-connected individuals.

Moreover, the magnitude of these effects of first-infected macaque centrality on outbreak sizes varied across network types and species, although not always in the predicted directions. For a given host-species but across the two different types of networks, we found a significant interaction between network-type and strength centrality for rhesus macaques and bonnet macaques, but not for long-tailed macaques (Table 1; Figure 1). As predicted, rhesus macaques showed a significantly stronger effect of the mean centrality of first-infected individuals on outbreak sizes through their co-interaction networks compared to their grooming networks (Table 1; Figure 1). In other words, disease-causing agents were likely to infect more individuals if they entered into a population by first infecting monkeys that were more central in human co-interaction networks, compared to by first infecting monkeys that were more central in grooming networks. Contrary to our predictions, bonnet macaques also showed the same (rather than the opposite) effect as rhesus macaques, although the magnitude of difference was somewhat lesser than for rhesus (Table 1; Figure 1). Finally, although the centrality of first-infected macaques within their co-interaction networks once again showed an overall greater effect on outbreak sizes than the centrality of macaques within their grooming networks for long-tailed macaques, this difference was not significant (Table 1; Figure 1). Moreover, long-tailed macaques also seemed to show separate groupings within each network-type (Figure 1). In other words, they seem to show intra-specific differences in the effects of the network centrality of macaques within each network-type on outbreak sizes (Discussion).

For a given network-type but across host-species, we found a significant interaction between species and strength centrality for both co-interaction networks and grooming networks (Table 2). For co-interaction networks, rhesus macaques showed the strongest effect of strength centrality on outbreak sizes as predicted. Contrary to our predictions, bonnet macaques fell within the range of rhesus macaques, and long-tailed macaques showed a significantly lower effect than both rhesus and bonnet macaques (Table 2; Figure2). For grooming networks, the differences were in the directions we predicted – bonnet macaques showed the strongest effects of strength centrality on outbreak sizes, followed by long-tailed macaques, and finally rhesus macaques that showed a significantly lower effect compared to bonnet macaques (Table 2; Figure2). For all three species, the magnitude of the effects of strength centrality on outbreak sizes was markedly greater for co-interaction networks compared to grooming networks (Figure 2). In other words, across host-species, the infection of macaques that were central in their co-interaction networks led to consistently higher disease outbreaks (more individuals infected) than the infection of macaques that were central in their grooming networks.

For grooming networks, but not for co-interaction networks, we also found a significant effect of sex and dominance rank of the first-infected individual on mean outbreak sizes – disease outbreak sizes were higher when first-infected macaques within grooming networks were females compared to males, and higher-ranking compared to lower-ranking individuals (Table 2). However, the magnitude of these effects were much lower than those of the strength centrality of first-infected macaques (Table 2). Finally, the overall anthropogenic exposure of first-infected macaques, i.e. their frequencies of interactions with humans and times spent foraging on human foods, had no impact on disease outbreak sizes (Table 1, 2).

**Table 1: Standardized model coefficients from the best-fit GLMMs (model 1 from Supplementary Tables 2A, 2B and 2C) of network centrality by network-type (co-interaction vs grooming) for a given host-species. The models examined the effects of the attributes of the first-infected 'patient-zero' macaque, including an interaction between their network strength centrality and network type, on mean outbreak sizes (proportion of infected macaques) across SIR model simulations run for pathogens of 'low' to 'high' transmissibility. In each model, we included macaque animal ID (repeated measure across network-type) nested within group ID as random effects, to account for intraspecific variation.**

| | Model Coefficients | | |
|---|---|---|---|
| **Predictor** | **Bonnet macaques** | **Long-tailed macaques** | **Rhesus macaques** |
| (Intercept) | 1.20* | 0.95* | 0.64* |
| Sex (males vs females) | -0.07* | -0.01 | -0.03 |
| Rank percentile | 0.01 | 0.05 | 0.04 |
| Network (grooming vs co-interaction) | -0.10** | 0.12** | -0.21** |
| Network strength (co-interaction) | 0.37** | 0.22** | 0.91** |
| Network strength (grooming) | 0.18** | 0.16** | 0.28** |
| Frequency of interactions with humans | 0.04 | 0.03 | 0.02 |
| Foraging on anthropogenic food | 0.04 | 0.01 | -0.04 |
| Network strength (grooming vs co-interaction) | -0.19** | -0.06 | -0.63** |

*p < 0.05; **p < 0.01

Note: p values were calculated after re-running the GLMMs using network centrality measures generated from 1000 post-network randomizations or node-swappings conducted on the co-interaction network and the grooming network.

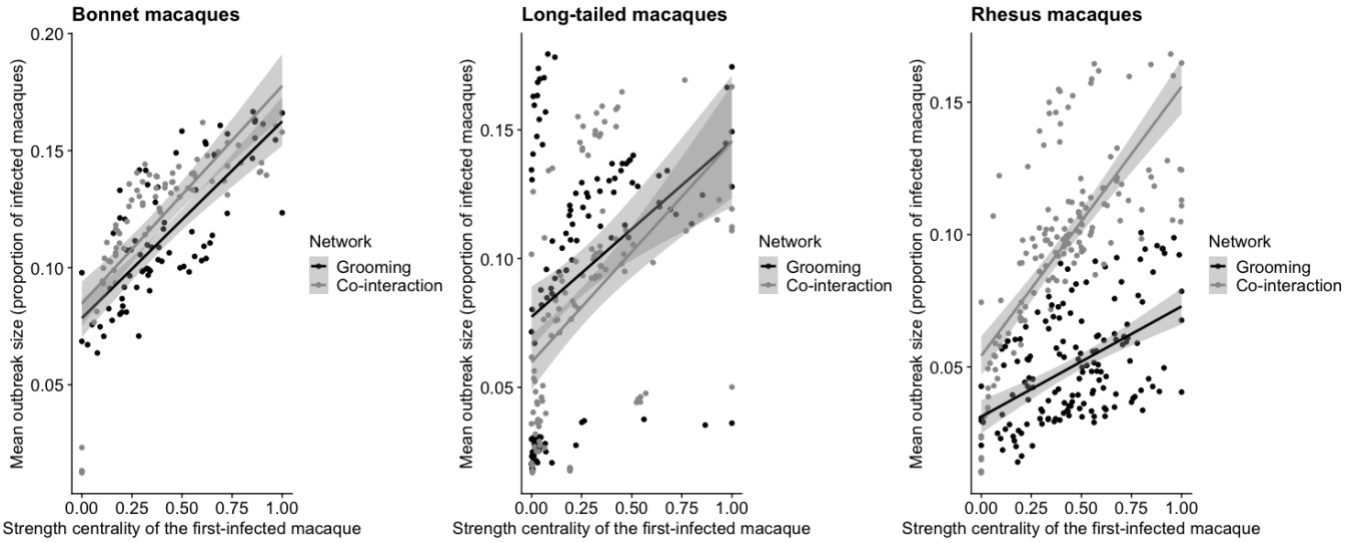


**Figure 1: Scatterplots showing positive correlations between the strength centrality of first-infected macaques by network-type for each host-species. For rhesus macaques and bonnet macaques, the effect of strength centrality on outbreak sizes was significantly stronger (greater slopes) through macaques' co-interaction networks compared to grooming networks. For long-tailed macaques, although the effects were stronger for co-interaction networks compared to grooming networks, the difference was not significant. Moreover, the plot for long-tailed macaques shows separate groupings within each network-type, suggesting possible inter-group variation in these effects (see Discussion).**

**Table 2: Standardized model coefficients from the best-fit GLMMs (model 1 in Supplementary Table 3A and 3B) of network centrality by species (bonnet vs long-tailed vs rhesus macaques) for a given network-type. The models examined the effects of the attributes of the first-infected 'patient-zero' macaque, including an interaction between their network strength centrality and species ID, on mean outbreak sizes (proportion of infected macaques) across SIR model simulations run for pathogens of 'low' to 'high' transmissibility. In each model, we included macaque group ID as a random effect, to account for intraspecific variation.**

| | Model Coefficients | |
|---|---|---|
| **Predictor** | **Co-interaction networks** | **Grooming networks** |
| **(Intercept)** | 1.20** | 1.19** |
| **Sex (males vs females)** | -0.03 | -0.05* |
| **Rank percentile** | 0.02 | 0.03* |
| **Species (long-tailed vs bonnet)** | -0.27 | -0.14 |
| **Species (rhesus vs bonnet)** | -0.23 | -0.65 |
| **Species (long-tailed vs rhesus)** | -0.04 | 0.50 |
| **Strength (bonnet)** | 0.49** | 0.25** |
| **Strength (long-tailed)** | 0.38** | 0.21** |
| **Strength (rhesus)** | 0.48** | 0.17** |
| **Frequency of human-macaque interactions** | 0.00 | 0.01 |
| **Foraging on anthropogenic food** | 0.02 | 0.01 |
| **Strength (long-tailed vs bonnet)** | -0.11* | -0.04 |
| **Strength (rhesus vs bonnet)** | -0.01 | -0.08* |
| **Strength (long-tailed vs rhesus)** | 0.10* | 0.04 |

*$p < 0.05$; **$p < 0.01$

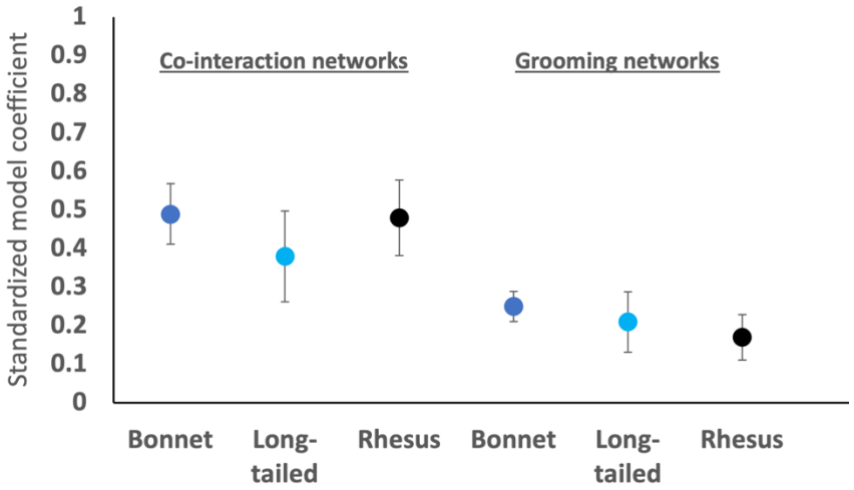



**Figure 2: Plots of standardized model-coefficients (Y-axis; values from Table 2) to show the difference in effects of the strength centrality of first-infected macaques on disease outbreak sizes through co-interaction networks and grooming networks. Coefficients for the same host-species are colored the same (dark blue = bonnet macaques; light blue = long-tailed macaques; black = rhesus macaques). Error bars represent 95% confidence intervals for each coefficient. All coefficients plotted show a significant, positive relationship (values > 0) between the centrality of the first-infected macaque and disease outbreak size. For all three host-species, these coefficients were greater for co-interaction networks compared to grooming networks, with these differences reaching significance for bonnet macaques and rhesus macaques but not long-tailed macaques.**

*Discussion:*

Although HWIs are now globally recognized as 'hotspots' for zoonotic transmission and disease emergence, there is little understanding of the underlying social, ecological and environmental factors that influence disease transmission and outbreaks at HWIs. Here we addressed this critical gap in the scientific literature -- adopting a comparative, network-based approach, we showed that disease outbreaks among wild primate populations living in (peri)urban anthropogenic environments maybe more strongly, or at least as strongly, influenced by animals' social-ecological interactions with humans as they are by animals' social interactions with conspecifics. We found that wild macaques were at least as (if not more) vulnerable to high disease outbreak sizes through their tendencies to interact with humans as through their tendencies to interact with other macaques. Specifically, disease outbreak sizes were positively predicted by the connectedness or centrality of the first-infected macaque within both their human co-interaction networks and its social grooming networks. This finding builds on previous studies during the past decade or so, that have implemented network approaches to epidemiologically model the vulnerability of wildlife populations to zoonotic outbreaks through wildlife-wildlife rather than human-wildlife spatial overlap or social interactions (e.g. meerkats, *Suricata suricatta*[81]; European badgers, *Meles meles*[82]; chimpanzees[40]; barbary macaques[41]; interspecies comparative studies[10,38]; studies implementing simulated networks[39,83]).

Among the most widespread, ecologically flexible of all mammals outside of the family Rodentia, wild macaques may live in dense populations in a variety of anthropogenic environments (e.g. urban, agricultural, forest-fragmented habitats) across South and South East Asia, where they frequently overlap and interact with people[66,84]. Thus, our finding that macaques' co-interactions with people make them especially highly vulnerable to disease outbreaks has implications for our understanding of contemporary evolution, and specifically of the behavioral flexibility of wild animals living in dynamic, varying (peri)urban environments. In a previous study, we showed that macaques that engaged in specific forms of social affiliation that were shorter in duration were also more likely to jointly take risks in anthropogenic environments by co-interacting with humans within the same time and space[64]. We speculated that such joint risk-taking would better enable wild primates to procure high-energy anthropogenic foods[64,68]. Here, our findings suggest that the benefits of procuring such foods may be offset by high zoonotic risk. Evaluating these costs-benefits tradeoffs on the fitness and survival of wildlife in human-impacted environments is important but may be complicated by the frequent trapping and re-location of animals in (peri)urban environments, that may hinder long-term studies on these populations. As such, our approaches in this study should encourage similar efforts on other wildlife populations that better distinguish between the (relative) effects of human-wildlife compared to wildlife-wildlife interactions on disease transmission and outbreaks (e.g. elephants (*Loxodonta africana*) in agricultural fields[14]; co-occurrence and space-use sharing of wild ungulates and livestock[18,85]; human provisioning of birds, raccoons (*Procyon lotor*) and red foxes (*Vulpes vulpes*) in urban environments[86]).

For all three species, we found that the centrality of macaques within their co-interaction networks consistently led to higher disease outbreak sizes compared to their centrality within grooming networks. In a previous study, we revealed that macaques' grooming relationships were not related to their tendencies to co-interact with people, which led us to speculate that the patterning and distribution of social contact with humans versus may offer potentially different 'pathways' for disease transmission[64]. Testing this speculation, here we reveal that co-interactions with humans may generally pose an even greater risk of diseases reaching higher outbreak sizes, than macaques' social interactions with conspecifics. This finding has major implications the consideration of how human-environmental interactions impact both human and environmental welfare and health outcomes in equal measure – a public health approach referred to as "One Health"[87,88]. To-date, research on disease transmission through wildlife populations has identified individual animals or populations that, in lieu of being more well-connected to other individuals and populations, may function as disease 'superspreaders'. Identification of 'superspreaders' is of paramount importance because epidemiologists and veterinary biologists often target them (as opposed to traditional mass vaccination[89]) for controlling the spread of endemic and emerging infectious disease[19,40,89]. Our findings suggest that macaques which are central in their co-interaction networks may function as potential intra- and inter-species (wildlife, humans, livestock) super spreaders[89]. This is because such individuals, in addition to being at increased zoonotic risk themselves and transmitting infectious agents to their conspecifics, are also more likely to inter-connect human populations with whom they interact across time and space and thereby pose a high risk of inter-species disease 'spill-over' into otherwise uninfected humans, livestock, and other animals[8,9]. Confirmation of this might await future studies at HWIs to evaluate variation in wildlife spatial and social interactions with humans and other anthropogenic factors as multi-modal networks[90,91], potentially including studies with wild animals, humans, and other anthropogenic factors are all nodes that are

interlinked based on their shared space-use or social interactions. Our findings indicate the importance of considering macaques that are central both within their co-interaction networks and their social networks as targets for disease prevention and control within these human-natural systems (e.g., vaccination, antimicrobial treatment)[40].

We found cross-species differences in the extent to which co-interaction networks more strongly predicted the sizes of disease outbreaks compared to grooming networks. As predicted, rhesus macaques were the most vulnerable to disease outbreaks through co-interaction networks, and the least vulnerable through their grooming networks. This highlights the importance of evaluating the relative effects of multiple (rather than single, as is often the case) single aspects of animal ecology on disease transmission[38,83]. Rhesus macaques, more so than the other two macaque species, may preferentially engage in affiliative behaviors such as grooming with close kin or allies[62,67]; the resultant sub-grouping of individuals within their social networks may potentially function as 'social bottlenecks' to disease transmission in this species[10,92]. Yet animals that show sub-divided social networks may nevertheless be vulnerable to outbreaks through other types of associations, and often in specific social-ecological contexts around human-provisioned food that may cause wild animals to aggregate together[86] and co-interact with people (as we have shown[64]).

Contrary to our predictions the effects of co-interaction networks on outbreak sizes in bonnet macaques were marginally greater (rather than lesser) than the effects of grooming networks, and were in fact within the range of rhesus macaques. One reason for this may be the spatial distribution of human-wildlife interactions in this population. Bonnet macaques are less geographically widespread and ecologically flexible compared to rhesus macaques[84]. Although the bonnet macaques in our study experienced markedly lower frequencies of interactions with humans compared to rhesus macaques and long-tailed macaques[12], these interactions were highly geospatially restricted to within specific areas or 'blocks' within their home-range. It is likely that such spatially dense social-ecological associations with people, through increasing the connectivity of macaques within their co-interaction networks, leads to a considerable increase in the risk of zoonotic outbreaks despite their relatively lower overall frequencies of interactions with humans. More generally, this finding suggests that zoonotic agents may enter into and rapidly spread even through populations of less ecologically flexible or socially gregarious wildlife that, despite interacting less frequently with humans or their conspecifics, may congregate within specific parts of their home-range around anthropogenic factors (e.g., contexts of food provisioning, crop-foraging, ecotourism activity)[86]. Our network approach, through capturing the spatiotemporal aspects of these human-wildlife interactions, provided a more accurate estimation of zoonotic risk in these populations than just their overall frequencies of interactions with humans. Aside from being the least ecologically flexible of the three species in this study, bonnet macaques are also the most vulnerable to human-impact[82], with many populations facing the imminent threat of local extinction. Thus, the identification and treatment of 'super spreader' individuals may be especially important in this population.

Contrary to our prediction, long-tailed macaques showed no differences in disease outbreak sizes across network-types. At least one explanation for this may be intra-specific variation, specifically between-group differences in their overall exposure to humans. We observed two groups of long-tailed macaques at a Hindu temple and popular tourist location within Kuala Lumpur, where the monkeys were exposed to dense human populations with whom they interacted highly frequently[58]. On the other hand, we observed two other groups in at a recreational park at the edge of the city bordering a fragmented forest area, where interactions

with humans were comparatively less frequent[58]. Moreover, long-tailed macaques also showed marked differences in their grooming behavior across these locations as a response to interactions with humans[58]. This explanation seems to be supported by the separate groupings for the relationships between network centrality and outbreak sizes for long-tailed macaques, even for the same network-type (Figure 1). A more comprehensive assessment of the disease vulnerability of these populations would require within-species, cross-group comparisons (analysis on-going for a future study).

Disease outbreak sizes through macaques' grooming networks were generally higher when the first-infected individuals were females compared to males, or when they were higher-ranking compared to lower-ranking individuals. Nevertheless, the effect sizes of sex and dominance rank on disease outbreaks were a lot weaker than the effects of individuals' network centrality. In many wildlife species, animals' sociodemographic attributes like their age, sex and dominance rank may influence their behavioral ecology, specifically their life-history strategies, social interactions, and their adaptive responses to changing (anthropogenic) environments[12,68]. It may therefore be important to evaluate the potentially interactive effects of such factors with animals' network connectedness on disease outbreaks.

The consistently stronger effects of strength centrality compared to betweenness centrality or eigenvector centrality on outbreak sizes suggests that animals' direct connections played a greater role in disease transmission than their secondary connections. This finding is consistent with many previous studies (reviewed in [19,82]), but not so with others (e.g., betweenness as a stronger predictor of outbreaks across communities of humans[93] and chimpanzees[40]). Such differences in the role of direct versus indirect connections in disease transmission may depend on the host population, network-type, or more global aspects of networks[38,39,82]. These may be community modularity or the tendency of animals to form sub-groups[39], inter-individual *differences* in network connectedness or centrality[80], and the efficiency of the flow or transfer of information through networks[94]. Examining how these global aspects of macaques' co-interaction networks and grooming networks may impact infectious agent transmission and consequential disease outbreaks in these populations would be a critical next step.

Our results were independent of pathogen-specific transmissibility which, through influencing basic reproduction numbers ($R_0$ values), may strongly impact disease outbreaks. We chose to account for, rather than quantitatively evaluate, the effects of a suite of zoonotic respiratory pathogens of different transmissibility (e.g., influenza virus, measles virus, *Mycobacterium* spp., SARs-CoV-2)[38,40], that typically spread through social interactions and are capable of causing disease in both humans and in closely related, ecologically overlapping wild primates (e.g. chimpanzees, baboons, macaques)[1,253]. Pathogen transmissibility may interact with animal ecology in complicated ways to influence outbreak sizes. For instance, the effects of animal social interactions on disease outbreaks may diminish for pathogens of exceptionally high transmissibility which may reach high outbreak sizes irrespective of social connections (e.g., measles virus[17])[38,40]. On the other hand, other studies have revealed that social interactions have stronger effects on outbreak sizes for pathogens of intermediate compared to low or high transmissibility[82]. Given the current lack of disease parameters on these macaque populations, our pathogen transmissibility values were also based on the human epidemiological literature (similar to other epidemiological studies on wildlife populations[38,40,41]). Such inter-host and inter-pathogen differences would need to be considered while constructing more sophisticated but system-specific epidemiological models of disease transmission for these and other HWIs.

In conclusion, our findings suggest that in the Anthropocene, wild animals remain highly vulnerable to zoonotic outbreaks through their social-ecological interactions with humans, in addition to their social interactions with conspecifics. Even in ecologically flexible, (peri)urban wildlife, disease-related costs may likely outweigh the potential or perceived benefits of increased assess to anthropogenic food. From One Health perspectives, our network approaches and findings demonstrate the importance of considering animals that consistently co-interact with humans across time and space (rather than just those that frequently interact with humans or their conspecifics), as targets for disease control. This is critical for preventing disease outbreaks in wildlife, but also for preventing cross-species wildlife-to-human disease spill-over events which have the potential to trigger future global pandemics.

## Methods:

*Study Locations and Subjects:*

We observed 10 macaque groups representing three different species at human-primate interfaces across three locations in Asia – four groups of rhesus macaques in Shimla in Northern India ($31.05^0$N, $77.1^0$E) between July 2016 and February 2018, four groups of long-tailed macaques in Kuala Lumpur in Malaysia ($3.3^0$N, $101^0$E) between September 2016 and February 2018, and two groups of bonnet macaques in Thenmala in Southern India ($8.90^0$N, $77.10^0$E) between July 2017 and May 2018 (Supplementary Figure 1). All macaque groups were observed in (peri)urban environments, and their home-ranges overlapped with humans and anthropogenic settlements - e.g., Hindu temples (Shimla and Kuala Lumpur), recreational parks (outskirts of Kuala Lumpur, Thenmala), roadside areas (Thenmala, Shimla) – to varying extents[12,64,68]. Subjects were adult male and female macaques which were pre-identified during a two-month preliminary phase prior to data collection at each location. More details regarding the study locations, macaque group compositions and subjects, and observation efforts, may be found in our previous publications[12,6864] and in Supplementary Table 1.

*Data Collection:*

We collected behavioral and demographic data in a non-invasive manner using observation protocols that were standardized across observers within and across locations (details in [12,68,95]). All data were collected for five days a week, between 9:00 am and 5:00 pm. To record and spatiotemporally capture variation in human-macaque social-ecological interactions for the construction of co-interaction networks, we used an *event sampling* procedure[96,97]. For this we divided pre-identified parts of the home range of each macaque group in which human-macaque interactions were most likely to occur, into blocks of roughly equal area and observability[64]. We visited these blocks in a pre-identified, randomized order each day. Within a 10-minute sampling period, we recorded interactions between any pre-identified subject macaque and one or more humans that occurred within that block, in a sequential manner. Human-macaque interactions included all contact and non-contact behaviors initiated by macaques towards humans (e.g., approach, aggression, begging for food), or vice-versa (e.g. approach, aggression, provisioning with food) (more details in [12,68,95]). We undertook this sampling approach of visiting blocks at random in order to avoid over-sampling of human-macaque interactions in more (versus less) densely populated areas of macaques' home-ranges.

To record macaques' social behavior, and their overall anthropogenic exposure independent of spatiotemporal context, we used *focal animal sampling*[96]. For this we followed individual subjects in a pre-determined, randomized sequence for 10-minute durations. In a continuous manner, we recorded, within each focal session, instances of social grooming, and dyadic agonistic interactions that involved aggression (threat, lunge, chase, attack) that was followed by submission (avoidance, silent bared teeth, flee), between the focal animal and its group conspecifics. We also recorded interactions between the focal animal and one or more humans in a continuous manner (see above for definitions). Once every two minutes, we ceased recording continuous data to conduct a *point-time scan*[96] of the focal animal's main activity, i.e. one of resting, locomotion, socializing, interacting with a human, foraging on natural food, or foraging on anthropogenic food. More details regarding the data collection protocols and definitions of behaviors may be found in our previous publications[12,68,95].

We entered all data into Samsung Galaxy Tablets using customized data forms created in HanDBase® application (DDH software). From these we exported and tabulated all the data into MS Excel and MS Access databases daily. All observers within and across locations passed inter-observer reliably tests using Cohen's kappa (> 0.85)[98].

*Construction of Co-interaction Networks and Social Grooming Networks:*

From the human-macaque interactions collected using event sampling data, we constructed social-ecological co-interaction networks (Figure 3A). In these, nodes were individual macaques. Edges were based on the frequency with which pairs of macaques jointly engaged in interactions with humans at the same block and within the same event sampling session, per unit of event sampling observation time during which both members of the pair were present in the group and (thereby) observable[64]. Since our data were limited by lacking the identities of individual people, we refrained from constructing and analyzing bi-modal social-ecological networks with both in which both macaques and humans were considered as nodes (as in [99]). Instead, we "projected" interactions between humans and macaques into co-interaction networks, in which nodes were macaques and edges defined by their joint tendencies to interact with humans within the same time-frame and anthropogenic space. We thus subject these social-ecological interactions to SNA (similar to the conversion of primate-parasite bimodal networks to social networks in [65]).

We constructed macaque-macaque social grooming networks using the focal sampling data (Figure 3B). In these, we linked individual macaques (nodes) based on the frequency which they engaged in social grooming interactions per unit of total focal observation times (edges) calculated for each pair of macaques when both members of the pair were present in the group and (thereby) observable[12,64]. Our use of different types of data (event sampling versus focal sampling) to construct co-interaction networks and social grooming networks respectively, minimized the potentially confounding effects of data inter-dependencies and sampling bias on our networks[72].

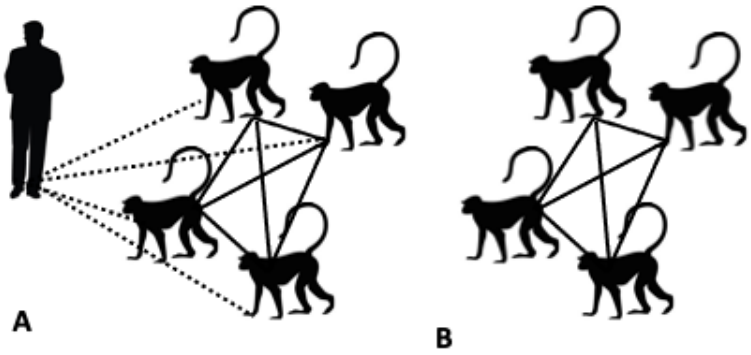



**Figure 3: Construction of macaques' (A) human co-interaction networks and (B) social grooming networks. Dotted lines represent macaques' interactions with humans within the same (10-minute) time-window and space, which defined the edges of the co-interaction networks.**

*Calculation of Network Measures:*

For each co-interaction network and grooming network, we calculated three measures of individual or node-level centrality. We calculated (1) weighted degree or strength centrality, i.e. the number and sum of the edge-weights of an individuals' direct network connections[69,70], (2) betweenness centrality, i.e. the proportion of shortest paths connecting each pair of nodes that pass through a particular node[19,71,72], and (3) eigenvector centrality as the number and strength of an individuals' direct and secondary network connections[73–75]. These centrality measures were selected based on the decision-trees pertaining to choosing appropriate network measures provided by [100]; they are among the most biologically relevant to modeling disease transmission pathways through animal networks[100]. Specifically, strength indicates an individuals' immediate susceptibility to acquiring infectious agents from infected conspecifics to whom they are directly connected. Betweenness indicates the tendency for an individual to function as a 'bridge' or a 'conduit' of disease spread. Eigenvector captures the reach of an individual within its network, and thereby its potential role in both acquiring and transmitting infectious agents to many other individuals (reviewed in [19100]). To account for cross-group differences in group size, we re-scaled centrality measures within each group into percentile values that ranged between 0 (least central individual) and 1 (most central individual).

*Macaque Sociodemographic Attributes and Overall Anthropogenic Exposure:*

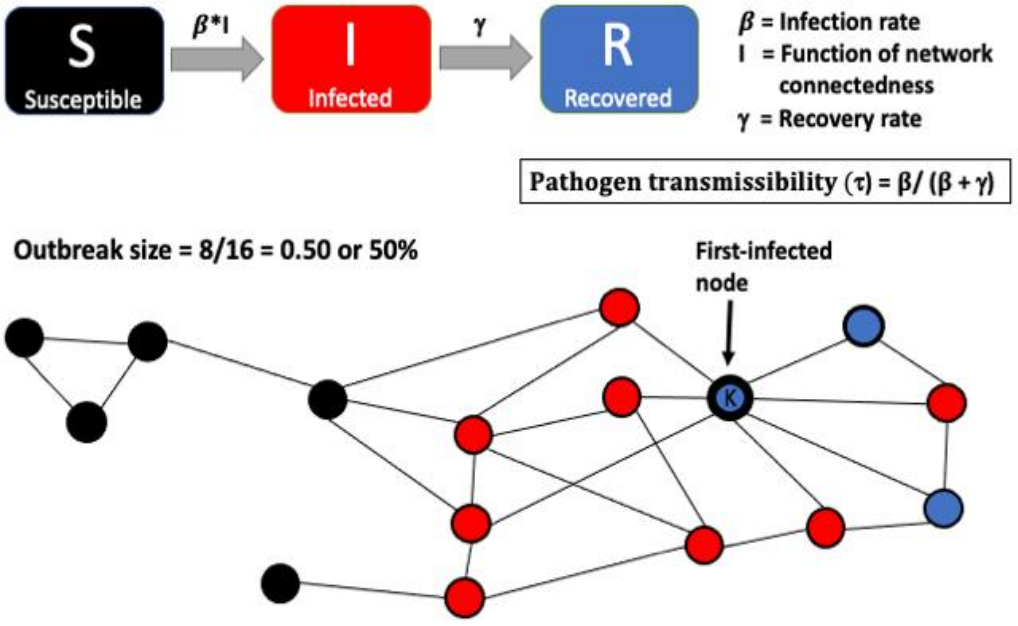
From the data on dyadic agonistic interactions with clear winners and losers, we calculated macaques' dominance ranks for each group, separately for male-male and female-female interactions, using the network-based Percolation and flow-conductance method (Package *Perc* in R[101]). Perc is a network-based ranking method that combines information from direct dominance interactions with information from multiple indirect dominance pathways (via common third parties) to quantify dyadic dominance relationships, and yield ordinal ranks from such relationships[101]. Aside from being successfully implemented in our previous studies (e.g. [30,58]), this method has been shown to yield animal rank orders that are highly consistent with those yielded by other, popularly used ranking methods in behavioral ecology, such as David's

score, I&SI ranks, and Elorating[102]. As with network centrality, we converted ordinal ranks of macaques within each group into percentile values that ranged between 0 (lowest-ranked individual) and 1 (highest-ranked individual). From the continuously collected focal sampling data, we calculated frequencies of human-macaque interactions per unit focal observation time. We also calculated, for each macaque, its time spent foraging on anthropogenic food as the ratio of the number of point-time scans in which it was foraging on anthropogenic food (Fa) to the total number of scans in which it was foraging on either anthropogenic food (Fa) or natural food (Fn), i.e. Fa/ (Fa + Fn).

*Zoonotic Disease Simulations:*

To simulate the spread of zoonotic agents of varying transmissibility ($\tau$) on macaques' co-interaction networks and grooming networks, we ran a series of Susceptible Infected Recovered (SIR) epidemiological models (using the *Epimdr* R package:[103]) (Figure 4A, B). We define '$\tau$' as a pathogen-specific characteristic, i.e. its probability of infecting a susceptible host within its infectious period[38,41]. $\tau$ is a function of the probability of pathogenic infection ($\beta$) and recovery rate ($\gamma$), and is calculated as $\beta/(\beta + \gamma)$[38]. For each network-type (human co-interaction, social grooming) and macaque group, we ran 5000 model simulations, 500 for each of 10 different values of $\tau$ ranging from 0.05 – 0.50 in increments of 0.05. These selections were based on the human literature that indicates that these values of $\tau$ correspond to zoonotic agents that range from low (e.g., influenza virus[104,105]), to moderate (e.g., respiratory pathogens like *Mycobacterium* spp. and SARS-CoV-2[38,106]), to high (e.g., measles[17]) contagiousness, and average basic reproduction numbers ($R_0$) of between 1.6 – 14.0[38,40]. We thus ran a total of 100,000 simulations (5000 per macaque group times 10 groups times two network-types). In each simulation, we deemed all macaques within a group to be initially 'susceptible', and then infected one individual (node) at random with an artificial zoonotic agent of a given $\tau$. A simulation proceeded using a discrete time, chain binomial method[38,107] that dynamically and temporally tracked the spread of infection through a weighted, undirected network through time (example in Figure 4B). In each simulation, animals were allowed to transition from 'susceptible' to 'infected' states, as a function of their network connections to individuals already in 'infected' states and the pathogen $\tau$ value. 'Infected' individuals were then allowed to transition into 'recovered' states at a fixed recovery rate ($\gamma$) of 0.2 that corresponds to an average infectious period of five days[38]. Each simulation was allowed to proceed until the disease proceeded to extinction when there were no remaining infected individuals in the network. At the end of each simulation, we calculated the disease outcome of 'mean outbreak size', as the average % of infected macaques (the number of 'infected' individuals divided by the total number of individuals) across all time-units of the simulation. We also extracted, for each simulation, the identity of the first-infected macaque 'k' (Figure 4A), and calculated an average of disease outbreak sizes from across all its first-infected simulation runs. We then matched this individual-level mean outbreak size with the sociodemographic characteristics, network centrality, and overall anthropogenic exposure of this (first-infected) individual.

(A)




(B)

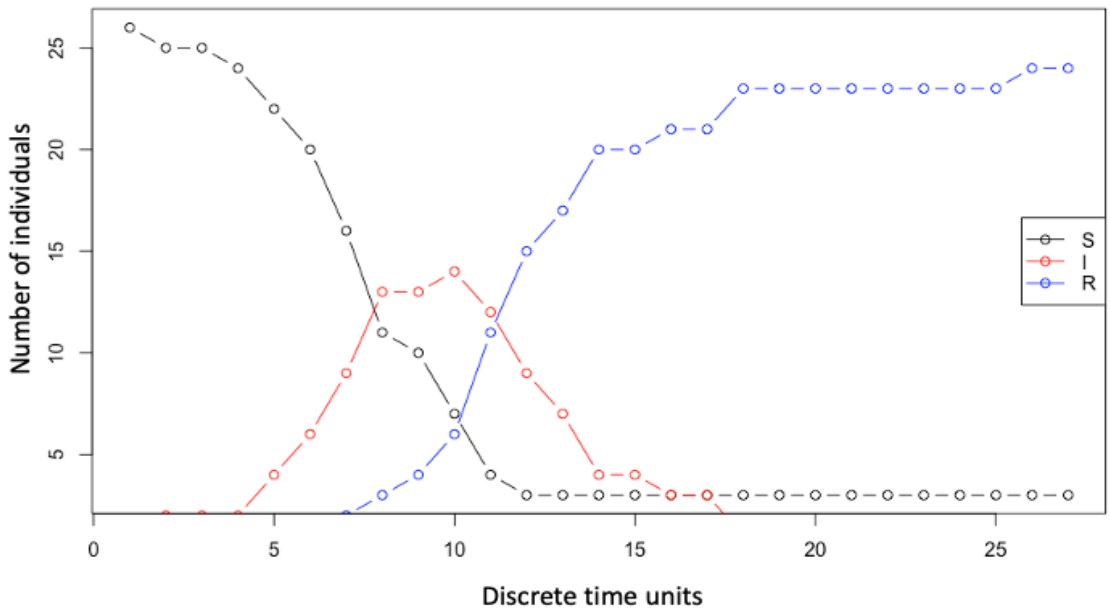



**Figure 4: A typical Susceptible Infected Recovered (SIR) model simulation of network-mediated disease transmission (A), and an output from a single discrete time-based SIR model simulation (B).**

*Statistical Analysis:*

We used General Linear Mixed Models (GLMMs) implementing a corrected Akaike Information Criterion (AICc)-based model-selection criterion (packages *Lme4*[108] and *MuMIn*[76,77] in R[109]), followed by 'null-model' post-network randomization or node-swapping tests[78–80], to test our predictions. In all GLMMs, we set mean outbreak size calculated at the level of the individual macaque through their co-interaction networks and/or grooming networks as the outcome variable. We used a Gaussian function since outcome variables did not deviate from a normal distribution (Shapiro Wilcoxon tests: $p > 0.05$ in each case). First, to examine the effect of the centrality of the first-infected macaque by network-type (co-interaction versus grooming) for a given host-species (bonnet or long-tailed or rhesus) on mean outbreak sizes, we ran three sets of three GLMMs each, one for each macaque species (details in Table 3A). In all models, we set the number of macaque subjects within the group (or 'effective group size') to be an offset variable, since group size can impact our outcome variable of mean outbreak sizes[38,39]. In all models, we also included 'animal ID' (a repeated measure for co-interaction networks and grooming networks) nested within macaque 'group ID' as a random effect to control for intraspecific variation. For each species, we ran three models, in each of which we included just one of the three different network measures of the centrality of the first-infected macaque, i.e. the strength, betweenness, or eigenvector, as a main effect. We favored this approach in order to avoid the confounding effects of potential inter-dependencies of network centrality measures[72]. In each of these three models, we also included an interaction term of network centrality by network-type (co-interaction versus grooming), to determine whether the magnitude of these effects were different for different types of interactions. In all models, we also included, as main effects, the sociodemographic attributes (sex, dominance rank) and the overall anthropogenic exposure (frequencies of interactions with humans, proportions of time spent foraging on anthropogenic food) of the first-infected macaque. From each model-set of three models, we identified a single best-fit model with a difference in AICc of at least 2 points or lower than the next best-fit model[77].

Second, to examine the effect of the centrality of the first-infected macaque by species (bonnet versus long-tailed versus rhesus) for a given network-type (co-interaction or grooming) on mean outbreak sizes, we ran two sets of three GLMMs each, one for each network-type (details in Table 3B). Once again, we set the number of macaque subjects to be an offset variable, and included 'group ID' as a random effect, in all the models. For each network-type, we once again ran three models, in each of which we included just one of the three different measures of the centrality of the first-infected macaque as a main effect. In each of these three models, we also included an interaction term of network centrality by host-species (bonnet versus long-tailed versus rhesus), to determine whether the magnitude of these effects were different for different species. Once again, we also included, as main effects, the sociodemographic attributes (sex, dominance rank) and the overall anthropogenic exposure (frequencies of interactions with humans, proportions of time spent foraging on anthropogenic food) of the first-infected macaque. From each model-set of three models, we identified a single best-fit model with a difference in AICc of at least 2 points or lower than the next best-fit model[77].

All GLMMs met the necessary assumptions of model validity (i.e., distribution of residuals, residuals plotted against fitted values[110]). All statistical tests were two-tailed, and we set the p values to attain statistical significance to be $< 0.05$.

**Table 3: Summary of GLMM sets to examine the impact of the centrality of the (A) the first-infected macaque by network-type for a given host-species, and (B) the first-infected macaque by species for a given network-type, on zoonotic outbreak sizes**

| (A) Effects of the first-infected macaque by network-type for a given species | | |
|---|---|---|
| **Bonnet macaques** | **Long-tailed macaques** | **Rhesus macaques** |
| 3 models (on 76 individuals repeated across two network-types) | 3 models (on 112 individuals repeated across two network-types) | 3 models (on 151 individuals repeated across two network-types) |

| (B) Effects of the first-infected macaque by species for a given network-type | |
|---|---|
| **Co-interaction networks** | **Grooming networks** |
| 3 models (on 339 individuals across three species) | 3 models (on 339 individuals across three species) |

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**Author Contributions:**

K.N.B (first- and corresponding-author), under the supervision of E. A. and B.M., took the lead in in the study design, supervision of data collection, and the conductance of data analysis and manuscript writing. N. A. provided assistance with designing the study and writing the manuscript. B.A.B. and E.B.M. were involved in the formulation of field data collection procedures and manuscript writing. P.M., S.S.K., and M.A. were all involved in the designing

and supervision of field-work (data collection), and participated in manuscript writing. E.A. and B.M. supervised the entire study.

**Competing Interests:**

The authors declare no competing interests.

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