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Epidemiological Consequences of Individual Centrality on Wild Chimpanzees

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ABSTRACT

Disease outbreaks are one of the key threats to great apes and other wildlife. Because the spread of some pathogens (e.g., respiratory viruses, sexually transmitted diseases, ectoparasites) are mediated by social interactions, there is a growing interest in understanding how social networks predict the chain of pathogen transmission. In this study, we built a party network from wild chimpanzees (*Pan troglodytes*), and used agent-based modeling to test: (i) whether individual attributes (sex, age) predict individual centrality (i.e., whether it is more or less socially connected); (ii) whether individual centrality affects an individual's role in the chain of pathogen transmission; and, (iii) whether the basic reproduction number (R_0) and infectious period modulate the influence of centrality on pathogen transmission. We show that sex and age predict individual centrality, with older males presenting many (degree centrality) and strong (strength centrality) relationships. As expected, males are more central than females within their network, and their centrality determines their probability of getting infected during simulated outbreaks. We then demonstrate that direct measures of social interaction (strength centrality), as well as eigenvector centrality, strongly predict disease dynamics in the chimpanzee community. Finally, we show that this predictive power depends on the pathogen's R_0 and infectious period: individual centrality was most predictive in simulations with the most transmissible pathogens and long-lasting diseases. These findings highlight the importance of considering animal social networks when investigating disease outbreaks.

1 | Introduction

Across the primate order, nearly 60% of species are threatened with extinction and all nonhuman great apes are either Endangered or Critically Endangered (Estrada et al. 2017; IUCN 2021). Diseases are currently considered one of the main threats to primate survival, along with various other

anthropogenic activities (Rizkalla, Blanco-Silva, and Gruver 2007; Köndgen et al. 2008; Almeida et al. 2012; Nunn and Gillespie 2016; Dunay et al. 2018; Strier et al. 2019; Dietz et al. 2019; Azevedo et al. 2021; Köster et al. 2022). Because many pathogens are transmitted via proximity or social contact, for example, respiratory viruses, sexually transmitted diseases, and ectoparasites, there is a growing interest

Abbreviations: AI, association index; GLM, generalized linear model; R_0 , basic reproduction number; SIR, susceptible—infected—recovered; SNA, social network analysis.

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Summary

- Older male Chimpanzees are the most central within their network, and their centrality determines their probability of getting infected during simulated outbreaks.
- Direct measures of social interaction (strength centrality), as well as eigenvector centrality, strongly predict disease dynamics in the chimpanzee community.
- This predictive power depends on the pathogen's R_0 and infectious period: individual centrality was most predictive in simulations with the most transmissible pathogens and long-lasting diseases.

in understanding how networks of interacting individuals predict chains of pathogen transmission.

In this context, social network analysis has emerged as an important tool to study the influence of animal behavior on disease dynamics. For example, the position of an individual or a group in a social network, whether more or less socially connected (social centrality), is known to affect pathogen richness and transmission in both observational (VanderWaal et al. 2014; Sandel et al. 2020; Deere et al. 2021; Torfs et al. 2021) and simulation studies (Romano et al. 2016; Webber and Vander Wal 2020; Whittier et al. 2022). However, there are different centrality metrics, and it is necessary to clarify their relative impact in predicting epidemics (Borgatti 2005; Christley et al. 2005). For instance, considering grooming networks of Japanese macaques (*Macaca fuscata*), direct measures of centrality such as strength centrality, that is, the intensity of social relationships, were strong predictors of an individual's probability of becoming infected during a simulated outbreak. In contrast, indirect measures such as betweenness centrality, that is, the relative importance of individuals in indirectly connecting other individuals, were better predictors of how quickly an infectious agent could spread through an entire group (Romano et al. 2016).

Understanding the influence of individual centrality on pathogen transmission is thus not straightforward and another layer of complexity may be introduced with how pathogen-specific characteristics might moderate the effects of host networks on disease dynamics. Such characteristics include R_0 , that is, the expected number of infections produced by a single case in a naive population, and infectious period, that is, the duration during which an infected individual is infectious. While the effect of R_0 on the predictive power of centrality metrics over the course of an outbreak has already been explored (Christley et al. 2005), to our knowledge, no study has focused on the effect of infectious period. Hence, there is an open question on the combined effects of the contagiousness (R_0) and infectious period of a disease agent on the magnitude of centrality effects.

Unraveling these interactions may also be relevant for designing conservation strategies that rely on identifying individuals that could be superspreaders (Lloyd-Smith et al. 2005; Snijders et al. 2017). For instance, network-based interventions were suggested to be effective methods for preventing or slowing outbreaks (Miller and Hyman 2007; Salathé et al. 2010;

Rushmore et al. 2014; Singh et al. 2021; Moradmand, Siامي, and Shafai 2021; Evans et al. 2023; Berec et al. 2023). When social network data are not available, it may be possible to consider interventions based on individual traits like sex and age that stand in for centrality (Sosa 2016), as has been suggested for chimpanzees (Rushmore et al. 2014) and humans (Evans et al. 2023). These individual traits can be assessed for an entire group in just a few hours compared to weeks for a social network, and are thus particularly valuable when planning an intervention on a wild, unhabituated, or unstudied group. However, because of intergroup variability in terms of social style, social tolerance, and sociodemographic factors (sex ratio, age ratio, group size) (Cronin et al. 2014; Borgeaud et al. 2016; van Leeuwen, Cronin, and Haun 2018; van Leeuwen et al. 2021), it remains unclear to what extent individual traits are generally good predictors of centrality. Owing to these challenges, it remains difficult to generalize about the requirements for network-based interventions.

In this study, we investigated: (i) whether individual traits can be used as proxies for individual centrality; (ii) how an individual's social centrality influences disease dynamics according to different centrality metrics (direct versus indirect), and the extent to which this influence can be attributed to centrality purely explained by individual traits; and, (iii) how pathogen-specific characteristics might moderate the effects of host networks on disease dynamics. We combined SNA and agent-based modeling to test the impacts of sociality on the transmission of various simulated pathogens using an empirical data set collected on wild chimpanzees. Our hypotheses were that: (i) *traits like age and sex are good predictors of an individual's position within the network (whether more or less central)*; (ii) *an individual's centrality (and its individual traits, per hypothesis (i)) affects its role in the chain of pathogen transmission, and direct and indirect metrics exhibit variable influences*; and, (iii) *the basic reproduction number (R_0) and infectious period modulate the influence of centrality on the transmission chain within a network*.

Based on previous findings, we expected that male chimpanzees would be more central in their network than females, as they are generally more gregarious (i.e., inclined to aggregate) (Itoh and Nishida 2007; Gilby and Wrangham 2008; Kanngiesser et al. 2011; Shimada and Sueur 2014). We predicted that central individuals would acquire a pathogen during simulations and spread this pathogen to a larger number of individuals in a shorter amount of time, as was observed in other primate species (Romano et al. 2016). We also predicted that this effect would be stronger when using direct measures of centrality than when using indirect measures (Christley et al. 2005; Rozins et al. 2018), as we worked with an unstructured chimpanzee party network where all possible dyadic associations may occur (i.e., every individual may associate with every other individual) (Shimada and Sueur 2014). In other words, direct connections (e.g., strength) are sufficient to allow whole-group transmission, so second-level connections (e.g., betweenness) should matter less. Further, we predicted that individual traits, as a proxy for individual centrality, would partially predict disease outcomes, as has been reported for chimpanzees previously (Rushmore et al. 2014). Finally, we predicted that an individual's centrality should be most influential in the chain of

transmission for pathogens with a high R_0 (Christley et al. 2005) and long infectious periods, as they lead to longer outbreaks (Cross et al. 2005), with the most central individuals driving the spread of the infectious agent. However, we expected centrality to be less informative once the pathogen consistently achieved complete transmission. With this study, in addition to the theoretical contributions, we hope to provide a framework that can be applied to species threatened by infectious disease and to support conservationists and wildlife managers in the decision-making process.

2 | Materials and Methods

2.1 | Study Species

We worked with a wild community of chimpanzees (*Pan troglodytes schweinfurthii*; called M group) in the Mahale Mountains National Park, Tanzania (Nishida 2011; Nakamura et al. 2015). Chimpanzees live in permanent social groups and present a fusion-fission social structure. They form subgroups called parties on a daily basis that can vary in size and composition from 1 day to another (Itoh and Nishida 2007). Chimpanzee group dynamics are characterized by female-biased dispersal (Pusey 1980; Walker and Pusey 2020). Social interactions and centrality in the social network can vary substantially among females, while males, on the other hand, are generally more gregarious (Gilby and Wrangham 2008, in Kibale National Park, Uganda).

2.2 | Data Collection

Network data were collected as described in Shimada and Sueur (2014). MS collected behavioral data from habituated chimpanzees of M group from October 4 to November 3, 2010, and from August 25 to September 17, 2011 (21 and 17 observation days, respectively). At the time of data collection, the group was composed of 58 individuals, but only 52 were observed during both research periods. The group included 35 females (17 adults > 13 years old, 9 juveniles between 5 and 13 years old, and 9 dependent offspring ≤ 4 years old) and 17 males (9 adults > 14 years old, 7 juveniles between 5 and 14 years old, and 1 dependent offspring ≤ 4 years old). All 52 individuals were individually identified.

MS collected data on attendance of each group member in nomadic parties. Nomadic parties were defined according to Itoh and Nishida (2007, 89) as “loose aggregations in which members are not always in visible contact, but stay within acoustic range and roughly travel in the same direction on a given day.” During each observation day, a party was located and a single focal individual was selected and followed by MS and one research assistant for a period of 3 h. Focal observations were interrupted if the individual was no longer visible. After the focal follow was finished, an individual in another nomadic party was selected to avoid observation bias.

To estimate the tendency of each pair of individuals to join the same nomadic party, we created a party-level association index

(AI) following (Gilby and Wrangham 2008). In this context, the AI for individuals x and y was calculated as:

$$AI_{xy} = \frac{P_{xy}}{(P_x + P_y - P_{xy})}, \quad (1)$$

where P_{xy} is the number of nomadic parties observed that contained both x and y , P_x is the number of nomadic parties observed that contained x , and P_y is the number of nomadic parties observed that contained y . AI_{xy} takes a value between 0 and 1. A matrix of AIs was built using this method incorporating all possible dyads in the group using data aggregated from both research periods. This constitutes our observed chimpanzee network. Unlike Shimada and Sueur (2014), we worked directly with the weighted network as a disease may spread even during rare associations.

2.3 | Theoretical Model of Pathogen Transmission

We implemented a Susceptible—Infected—Recovered (SIR) model (Kermack and McKendrick 1927) of an infectious agent spreading along a social network using Netlogo 6.2.0 (Wilensky 1999). Built upon the models developed in Sueur, Petit, and Deneubourg (2009) and Romano et al. (2016), this model assumes that pathogen transmission is dependent on the social relationships between individuals. In the model, we input the observed chimpanzee network, which considers individual identities and their strength of social relationships.

To model the spread of a socially transmitted pathogen, we consider the probability of being infected nonsocially (λ) to be close to 0. At the beginning of the simulations, the probability of an individual being infected first is the same for all individuals. As soon as the first infected individual is randomly selected, the probability ψ_i for other individuals to become infected is no longer the same among individuals, but is dependent on their social relationships:

$$\psi_i = \lambda + C \sum_{k=1}^N r(k, i) \quad \text{with} \quad \frac{C \sum_{k=1}^N r(k, i)}{\lambda} = R_0, \quad (2)$$

where C is a mimetic coefficient or amplification process favoring infection, i is the individual at risk of being infected, N is the group size, and $r(k, i)$ is the weight of an edge linking a pair of individuals.

At each time step, a new individual's probability ψ_i of getting infected is computed for each susceptible individual who then has a chance to either move from “susceptible” to “infected” status or remain “susceptible” based on that probability. Model simulations operate on daily time steps, and each individual's status is updated just once per time step. To recover from the illness, the number of days an individual is infected should be equal to the infectious period, also known as the recovery time in the model interface. If the individual has been infected for a number of days less than the infectious period, then the number of days of being infected increases by 1. Individuals that are

recovered have permanent immunity against further infection and will no longer change status during the simulation. The simulation finishes when there are no more infected or susceptible individuals remaining.

Finally, to determine how infectious a pathogen is, we set the basic reproduction number R_0 in the model interface. In simple terms, the higher the R_0 , the higher the average number of infected contacts per infected individuals. We tested multiple values of R_0 and of infectious period to simulate disease outbreaks caused by distinct pathogens. For R_0 we followed the values used in Rushmore et al. (2014), 0.7 being a low contagiousness pathogen, 1.5 being a mildly contagious pathogen such as influenza, 3 being a moderately contagious pathogen such as Ebola, and 10 being a highly contagious pathogen such as measles. For infectious period, we followed reports that infectious periods may range from a few days to 1 month for pathogens common to humans and great apes (Ekdahl et al. 1997; Weber, Weber, and Milligan 2001; Chowell et al. 2004; Rushmore et al. 2014; Scully et al. 2018; Negrey et al. 2019; Byrne et al. 2020; Morrison et al. 2021). We selected infectious periods of 3, 7, and 20 days for our simulations and ran 10,000 simulations for each combination of parameters (4 values of $R_0 \times 3$ values of infectious period). The source code is available in Zenodo: <https://zenodo.org/record/7377819>.

For each simulation, we stored the identity of each infected individual, its position in the order of infection, the latency of infection (since the previous and the first infection), and the day the simulation is at. We then computed global outcomes (the proportion of infected individuals and the duration of transmission events) and individual outcomes of the model (the probability of an individual i getting infected during the outbreak, the outbreak size and duration once individual i is the first to be infected). Details of each metric are given in the Supporting Information.

2.4 | Network Statistics

We used the package *igraph* version 1.2.6 (Csardi and Nepusz 2006) in R (R Core Team 2021) to calculate individual network centralities. Because our association matrix is symmetrical and nonbinary, we computed an undirected and weighted network.

We calculated node strength, betweenness, and eigenvector centralities, each of which has been previously suggested to influence pathogen transmission via divergent mechanisms (Sueur, Jacobs, et al. 2011; Romano et al. 2016; Silk et al. 2017; Snijders et al. 2017). The strength of a node is the sum of the weights of all edges connected to it in a weighted network (Barrat et al. 2004). It can be directed, having both in-strength and out-strength, but since our network is undirected, we used all the edges connected to each node as that individual's strength. The eigenvector centrality of a node sums the weights of all edges connecting it to others as well, but in this case is also weighted by the collective strengths of all nodes connected to that focal node (Bonacich 1987). Strength centrality allows us to determine how likely an individual is to infect or be infected

by others directly through repeated interactions; high strength increases opportunities for transmission to occur. Eigenvector centrality encompasses a broader likelihood of acquiring/transmitting an infection; individuals who are connected to others who themselves are well-connected are more likely to acquire and transmit infections. Both measures thus allow us to estimate the spreading potential of an individual in different ways. The betweenness centrality of a node refers to the number of shortest paths between two other nodes that pass through that focal node (Freeman 1978). The shortest path can either be a count or a sum of edge weights, which reflect betweenness for unweighted and weighted networks, respectively. We used a weighted method in our study, and because the edge weights are interpreted as distances, we computed the multiplicative inverse of the edge weights so that the higher is the AI between two individuals, the shorter is the distance between them. This measure is important for identifying key individuals that facilitate transmission between different subgroups in a network.

Given the nature of our networks (a party network where all individuals are connected to others), we explored the need for a cut-off to remove the least important connections in the network. We removed the 10% to 90% weakest connections at 10% steps, and for each cut-off value, we computed the correlation between the centrality metrics of the original network and those of the trimmed network. We found that removing interactions had nearly no effect on the individuals' strength and eigenvector centrality (Supporting Information S1: Figure S1). And to observe a substantial effect on the betweenness, we had to remove more than 50% of the connections (Supporting Information S1: Figure S1) which would deprive the pathogens of many potential routes of transmission within the network. For this reason, we chose to use the full network in all analyses. This decision is also supported by previous studies showing that removing weak ties tends to homogenize the number of connections in networks (Collier et al. 2022), whereas heterogeneity plays an important role in pathogen spread (May, Gupta, and McLean 2001; Kiss, Green, and Kao 2006).

2.5 | Statistical Analysis

To test whether individual traits (sex and age) predict individual centrality, we constructed a generalized linear model (glm) for each metric (i.e., eigenvector, betweenness and strength) as they are not independent from one another. One may expect an inverse U-shaped relationship between age and centrality with a peak in prime adulthood (between 20 and 35 years old) and a decline in old age (> 35 years old) (Watts 2018), which could be analyzed by grouping individuals into age classes. However, because such a pattern was not observed in our data set (Supporting Information S1: Figure S2), we decided to use age as a continuous variable in the analyses. We ran each glm according to the distributional characteristics of each metric: a Gaussian model for strength and eigenvector and a negative binomial model for betweenness. We used the functions "glm" from the package *stats* version 4.0.2 in R, and "glm.nb" from the package *MASS* version 7.3-60 (Venables and Ripley 2002) in R, respectively. The betweenness variable was transformed to only

contain integers higher than one. For all models, we ran a series of diagnostics to test their suitability (i.e., variance inflation, correlation of fitted and residual values and Cook's distance). The interaction between age and sex was included in each model. Although information on dominance rank was available, we did not use it since it is representative of only eight adult males.

We tested the effects of the infectious period, the R_0 and their interactions on the proportion of infected individuals and on the duration of transmission events using glm. We then used the function "glht" from the package *multcomp* version 1.4-14 (Hothorn, Bretz, and Westfall 2008) in R to perform multiple comparisons of means under general linear hypotheses as post hoc tests to test for significant effects within each of these models.

To test the effect of centrality measures on disease dynamics (the probability of an individual i getting infected during the outbreak, the outbreak size and duration; once individual i is the first to be infected), we performed correlation tests using the Kendall method for each one of the centrality metrics and for all combinations of R_0 and infectious period. We also used node-label permutations to test the null hypothesis that individual centralities did not influence disease dynamics throughout the distinct values of R_0 and infectious period. For each disease dynamic and each individual centrality metric, under each combination of R_0 and infectious period, we performed 10,000 node-label permutations of the network. For each replicate we computed the studied centrality metric for the permuted network and built a glm with the disease dynamic as the dependent variable and the centrality metric as the independent variable. The null distribution was created by extracting the centrality metric estimate of each glm and the computation of the p value consisted in calculating which proportion of estimates from permuted networks was higher (for the individual probability of being infected and the outbreak size) or lower (for the outbreak duration) than the estimate from the real network. Finally, we replicated the previous methods on the residual centralities obtained from the models testing the effects of individual traits on centrality. We thus tested the predictive power of centralities from which we removed the influence of individual traits, hence interpreting the difference in predictive power before and after removal as the extent to which the effect of centrality on disease outcomes may be purely attributed to individual traits. All analyses were performed in R 4.0.2 and the significance threshold was set at 0.05.

3 | Results

3.1 | Individual Trait and Centrality

The chimpanzee network of party associations was composed of 52 individuals and every individual shared a link with all other individuals in the network (Figure 1). A description of the global network characteristics (density, diameter, and modularity) is available in the Supporting Information. The individual strength centrality ranged from 11.745 to 30.918 (mean = 23.394 ± 5.033 , median = 23.205) and the eigenvector

centrality ranged from 0.346 to 1 (mean = 0.744 ± 0.171 , median = 0.735) showing heterogeneous association patterns with some individuals having up to three times more contacts than others. The betweenness ranged from 0 to 33 (mean = 1.923 ± 5.249 , median = 0) showing that a few individuals monopolized the role of bridges between other pairs of individuals.

In general, our statistical models showed that age and sex, by themselves, were not good predictors of individual centrality (Table 1, Supporting Information S1: Table S1). For all centrality metrics, we observed that the older the male, the higher its centrality (Table 1, Figure 2).

3.2 | Global Outcomes of the Model

An increasing R_0 tended to increase the outbreak duration when it stopped due to a lack of infected individuals (e.g., 3 compared to 1.5 with an infectious period of 3 days; Figure 3A,B), but it tended to decrease the outbreak duration when it stopped due to a lack of susceptible individuals (e.g., 3 compared to 1.5 with an infectious period of 20 days; Figure 3A,B). In other words, when the outbreak size increased, the outbreak duration increased accordingly, until a maximum where the R_0 and infectious period started to allow for complete transmission. From that point, the more likely the complete transmission, the shorter the outbreak (Figure 4). Whatever the duration of the infectious period, when $R_0 = 10$, the outbreak lasted for approximately 10 days (Figure 3B). Outbreak sizes and durations produced by the various R_0 and infectious periods were all significantly different from each other (Supporting Information S1: Tables S2–S4), but a few combinations of parameters showed nonsignificant interactions in models for the proportion of infected individuals only (Supporting Information S1: Table S5).

3.3 | Individual Outcomes of the Model

3.3.1 | Individual Probability of Getting Infected

The probability of an individual being infected was positively and significantly correlated with all three centrality measures, but less strongly correlated with betweenness than with strength and eigenvector centrality (Figure 5; Table 2). Increasing R_0 and infectious period increased the correlation between the probability of being infected and the individual centrality measures (Table 2). Regardless of the combination of parameters, the node permutation test was significant (p -random < 0.05; Table 2), meaning that the individual probabilities of being infected resulting from the simulations were the result of a transmission pattern that is different from what is expected under a random network. This was not the case for a high R_0 and a high infectious period, which allowed a quasi-constant complete transmission in the betweenness model, thus causing a plateau (e.g., $R_0 = 10$ and infectious period = 20 days; Table 2). The correlations with residual centralities followed the same pattern and were generally no more than 0.5 times weaker than the correlations with the corresponding full centralities, except for residual betweenness which showed a proportionally much lower predictive power than betweenness (Table 2).

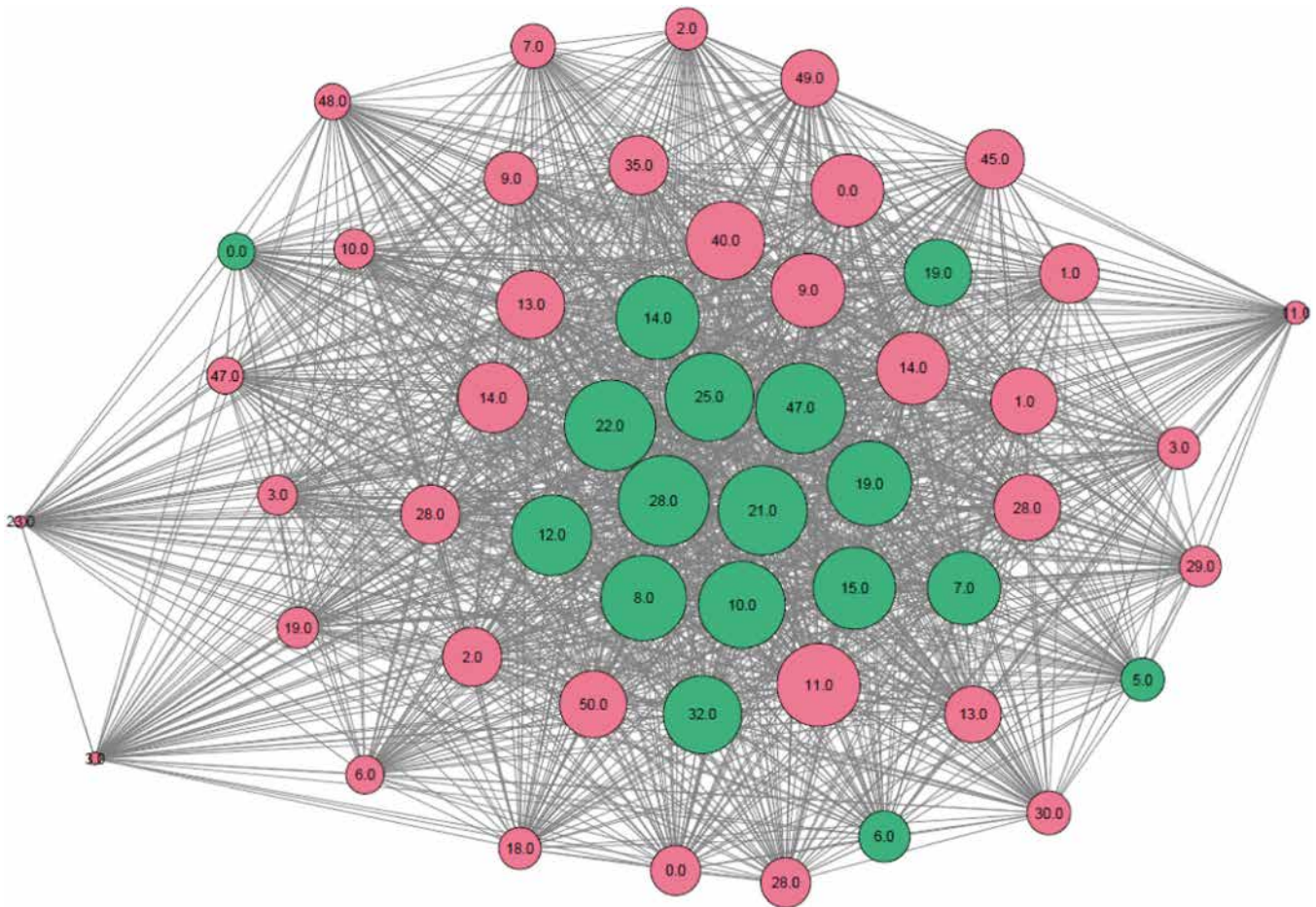


FIGURE 1 | Party association network of chimpanzees from M group—Mahale Mountains National Park, Tanzania. The node size represents the strength of the individual, the node color represents its sex (green = male; pink = female), and the label, its age in years. Individuals are positioned according to their strength centrality, following the layout Atlas Force 2 in Gephi 0.9.2 (Bastian et al. 2009).

TABLE 1 | Parameter estimates from generalized linear models explaining variation in individual centrality according to individual traits.

Factor	Estimate	Standard error	z Value	p Value
Model (gaussian): strength ~ sex × age				
Intercept	21.172	1.04	20.358	< 2e−16***
Sex (male)	2.463	2.043	1.206	0.234
Age	0.014	0.043	0.335	0.739
Sex (male): age	0.208	0.096	2.169	0.035*
Model (gaussian): eigenvector ~ sex × age				
Intercept	0.668	0.035	18.818	< 2e−16***
Sex (male)	0.083	0.07	1.195	0.238
Age	5.19E−04	0.001	0.358	0.722
Sex (male): age	0.007	0.003	2.166	0.035*
Model (negative binomiale): betweenness ~ sex × age				
Intercept	1.026	0.302	3.4	6.74E−04***
Sex (male)	0.391	0.55	0.711	0.477
Age	−0.014	0.013	−1.092	0.275
Sex (male): age	0.06	0.025	2.351	0.019*

Note: Significant codes are marked as follows: *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$.

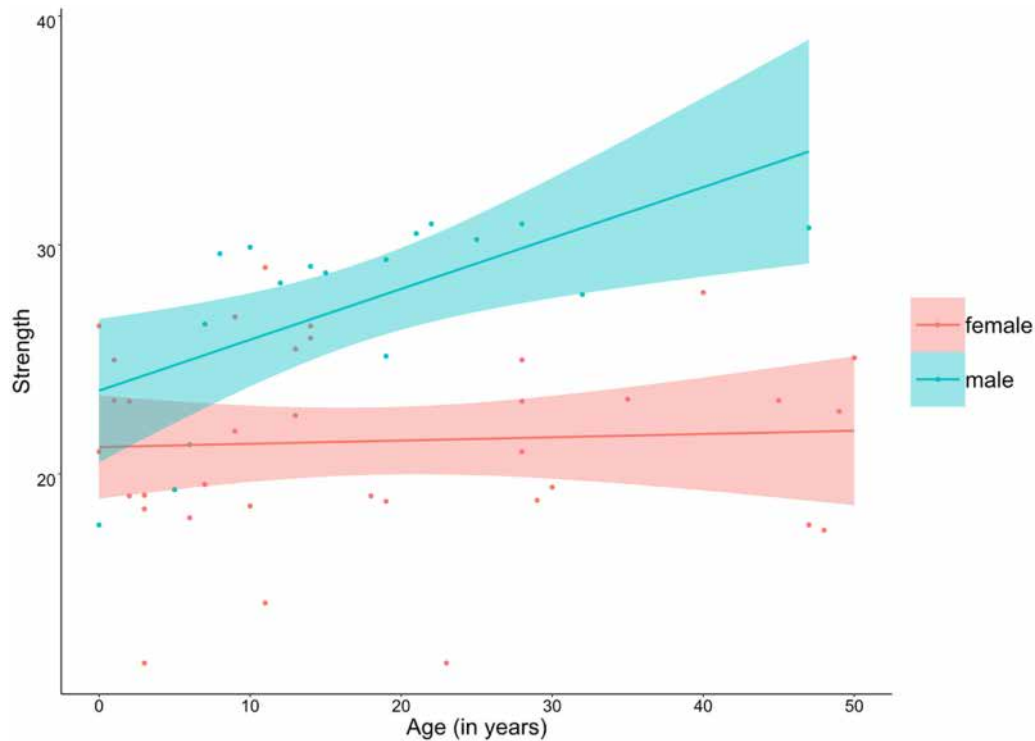


FIGURE 2 | Individuals' strength depends on both their age and sex. Observed patterns are the same with eigenvector and betweenness centralities.

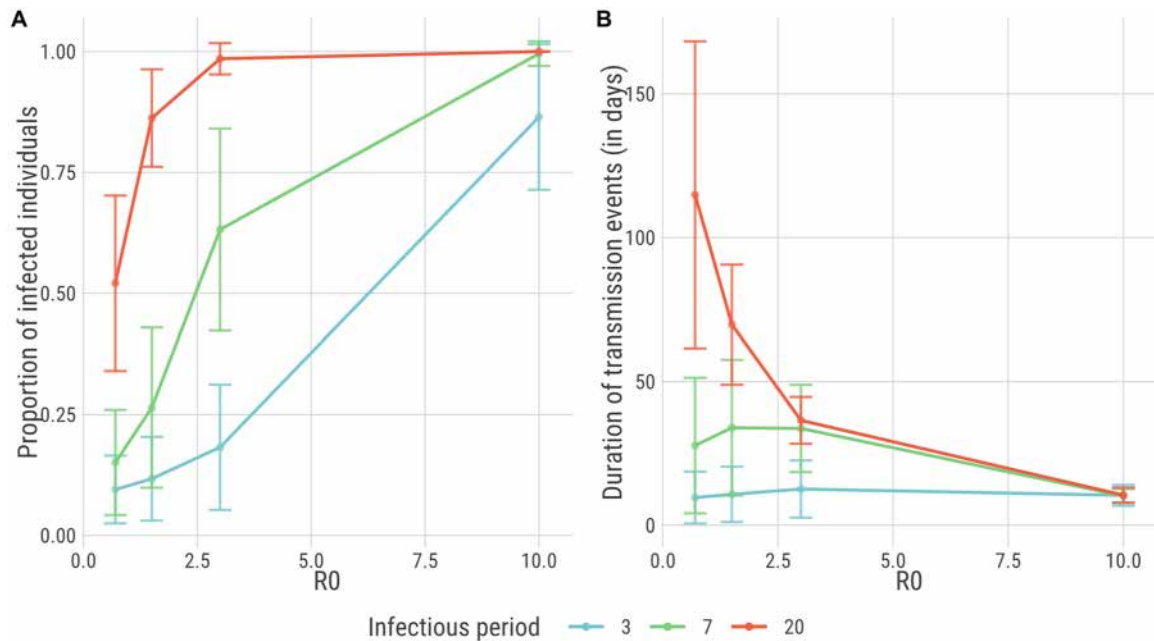


FIGURE 3 | Global outcomes of the model. Proportion of infected individuals (A) and duration of transmission events (B) depending on four values of R_0 (0.7, 1.5, 3, and 10) and three values of infectious period/recovery time (3, 7, and 20 days).

3.3.2 | Proportion of Infected Individuals According to the First Infected Individual's Centrality

The proportion of infected individuals (i.e., a proxy for outbreak size) depended on the centrality of the first individual infected. The higher the strength, betweenness, and eigenvector centrality of this individual, the larger the outbreak size

(Supporting Information S1: Figure S3). Correlations between outbreak size and betweenness were slightly weaker than with either strength or eigenvector, yet still significant (Supporting Information S1: Table S6). High values of R_0 tended to increase the correlation between the outbreak size and individual centrality, especially when the infectious period was short, up to a plateau (Supporting Information S1: Table S6). However,

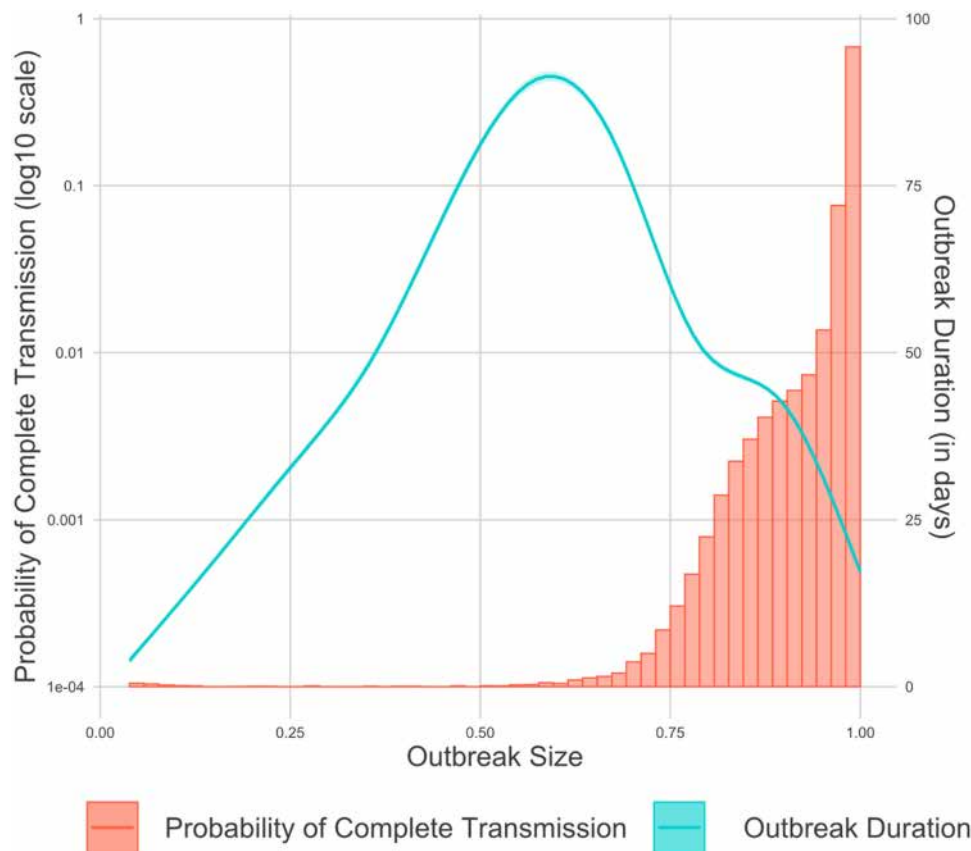


FIGURE 4 | Probability of complete transmission and outbreak duration depending on outbreak size over all simulations disregarding the R_0 and the infectious period.

increased infectious period reduced the correlation between the outbreak size and individual centrality, leading to a pattern in which the outbreak size was similar to what would be expected of transmission on a random network, according to the permutation tests (Supporting Information S1: Table S6). The correlations with residual centralities followed the same pattern and were generally no more than 0.5 times weaker than the correlations with the corresponding full centralities (Supporting Information S1: Table S6).

3.3.3 | Duration of Transmission Event According to First Infected Individual Centrality

In general, the duration of transmission events (i.e., a proxy for outbreak duration) was negatively correlated with all three centrality measures (Supporting Information S1: Table S7). Increasing R_0 tended to decrease the outbreak duration when complete transmission was achieved but increase it otherwise (Supporting Information S1: Figure S4). High R_0 also increased the negative correlations between outbreak duration and the centralities of the first infected individual (Supporting Information S1: Table S7). High infectious period increased the outbreak duration (Supporting Information S1: Figure S4) and strengthened correlations between the outbreak duration and the centralities of the first infected individual (Supporting Information S1: Table S7) by decreasing variance (Supporting Information S1: Figure S4). Low values of R_0 and infectious period were more likely to produce outbreak durations that

were not different from those expected if transmission was occurring on a random network (Supporting Information S1: Table S7). Finally, the combination of infectious period = 3 days and $R_0 = 3$ presented a surprising positive correlation between all tested centrality metrics and the duration of transmission events. The correlations with residual centralities followed the same pattern and were generally no more than 0.5 times weaker than the correlations with the corresponding full centralities (Supporting Information S1: Table S7).

4 | Discussion

This study investigated the impact of sociality on pathogen transmission in wild chimpanzees to check whether (i) individual traits can be used as proxies for individual centrality, (ii) how an individual's social centrality influences disease dynamics, and (iii) whether characteristics of infectious agents, such as their basic reproduction number (R_0) and infectious period, influence the predictive power of network centrality measures. We observed that an interaction between sex and age predicted centrality; as expected, males were more central than females, with older males but not females having increasingly strong relationships. Additionally, we demonstrated that direct measures of social interaction (strength centrality), as well as eigenvector centrality, had the highest predictive power in disease dynamics in this chimpanzee network (Table 2, Supporting Information S1: Tables S6 and S7). Key to our study aims, we found that individual traits (i.e., age and sex) were less

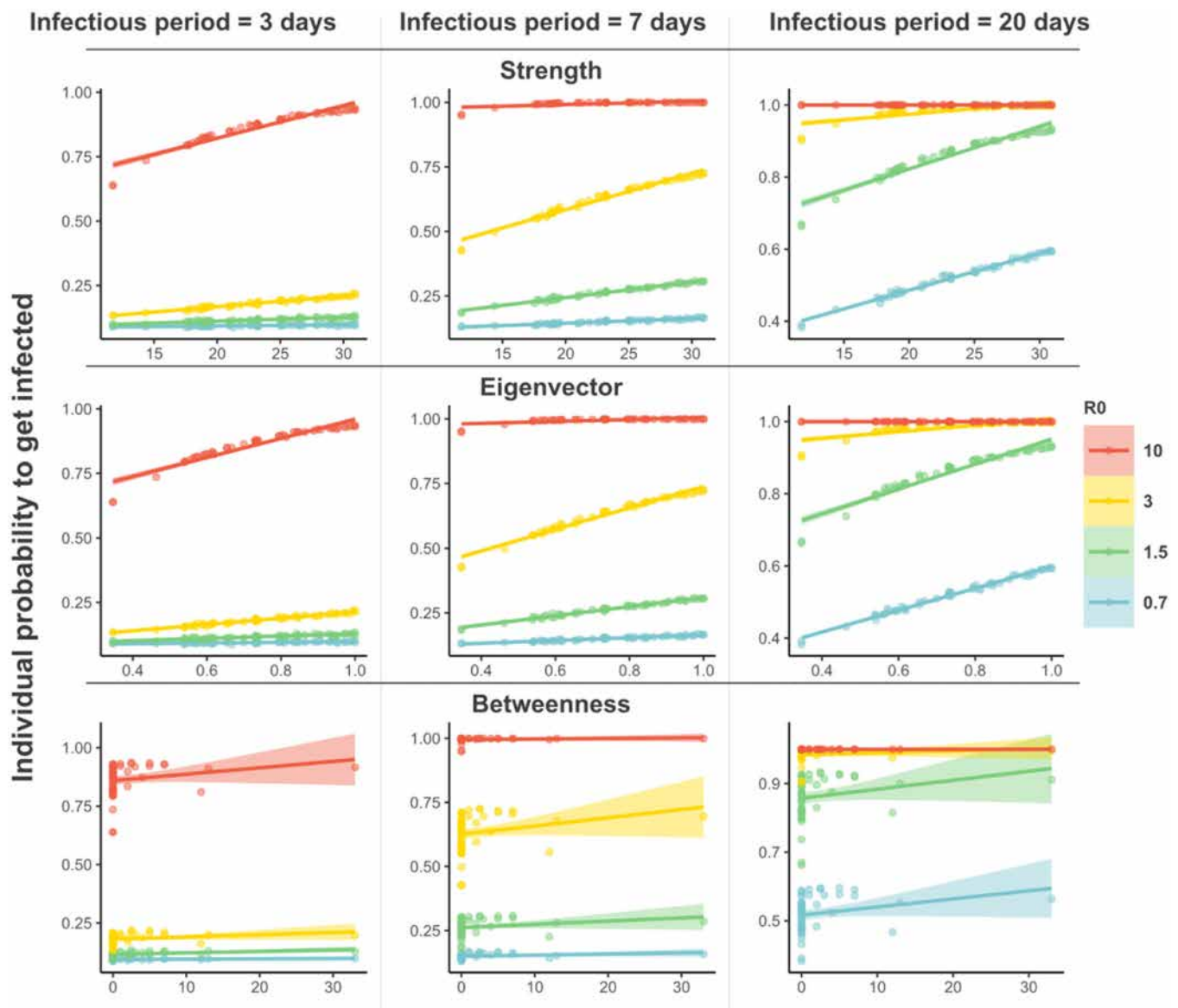


FIGURE 5 | Individual probability of being infected depending on individual's centrality (strength, betweenness, and eigenvector, from top to bottom), for four values of R_0 (0.7, 1.5, 3, and 10), and three values of infectious period (3, 7, and 20 days, from left to right).

efficient than individual centrality in predicting disease outcomes. Finally, our results suggest that the predictive power of the network is modulated by a given pathogen's R_0 and infectious period, with stronger predictive power for the most transmissible and longest-lasting infections.

With regard to our first hypothesis, we tested whether individual traits might be a good proxy of individual centrality. This idea came from a longitudinal study of wild chimpanzees, which showed that females with large family sizes ranging in the core of their territory, as well as high-ranking males, are likely to be the most central in a party network (Rushmore et al. 2013). We did not assess female ranging behavior or family size, but consistent with the fact that male chimpanzees are more gregarious than females (Gilby and Wrangham 2008), we found that males were generally more central than females. We also found that their centrality increased with age, unlike females. In fact, when aging, male chimpanzees tend to develop numerous, strong, and mutual

rather than one-sided friendships (Rosati et al. 2020). Even though males generally seem more central than females in Figure 1, the effect of sex depends on the interaction with age. This is explained by the fact that the effect of sex is driven by older males being more central, while younger males are not more central than any female. Because age and sex are easy individual traits to identify in field studies, an open question is whether an intervention based on these attributes would be efficient. It is also important to investigate whether the predictive power of these traits differ between groups, since chimpanzees do exhibit intergroup variability in terms of social style and tolerance (Cronin et al. 2014; van Leeuwen et al. 2021). Thus, generalizations in using individual traits as predictors for individual centrality should be done carefully, and using centrality metrics directly to study their effects on disease dynamics should be preferred, when possible.

Our results show that individual centrality impacts disease dynamics, such as estimated through the probability of an

TABLE 2 | Parameter estimates from the correlation tests (Kendall's τ and p value) and node permutation (Perm p value) between individual probability of getting infected and centrality measures for all combinations of R_0 and infectious periods. A residual centrality is the part of a centrality metric that cannot be explained by age nor sex. The difference between the correlations of centrality and of the corresponding residual centrality can thus be interpreted as the predictive power of the individual traits on the probability of getting infected.

R_0	Infectious period = 3 days				Infectious period = 7 days				Infectious period = 20 days			
	Kendall's τ	p Value	Perm p value		Kendall's τ	p Value	Perm p value		Kendall's τ	p Value	Perm p value	
<i>Strength</i>												
0.7	0.510	1.12E-07***	0***		0.848	1.01E-18***	0***		0.930	2.99E-22***	0***	
1.5	0.756	3.32E-15***	0***		0.919	8.78E-22***	0***		0.960	1.50E-23***	0***	
3	0.910	2.35E-21***	0***		0.954	2.44E-23***	0***		0.927	4.36E-22***	0***	
10	0.948	5.29E-23***	0***		0.911	2.93E-20***	0***		0.276	0.017*	2.00E-04***	
<i>Residual strength</i>												
0.7	0.408	2.10E-05***	0***		0.565	3.56E-09***	0***		0.573	2.01E-09***	0***	
1.5	0.460	1.54E-06***	0***		0.561	4.33E-09***	0***		0.576	1.73E-09***	0***	
3	0.548	9.64E-09***	0***		0.588	7.51E-10***	0***		0.590	7.12E-10***	0***	
10	0.586	9.10E-10***	0***		0.633	1.25E-10***	0***		0.275	0.017*	2.00E-04***	
<i>Eigenvector</i>												
0.7	0.523	4.91E-08***	0***		0.828	5.14E-18***	0***		0.927	3.30E-22***	0***	
1.5	0.738	1.25E-14***	0***		0.901	4.40E-21***	0***		0.934	1.76E-22***	0***	
3	0.899	5.50E-21***	0***		0.958	1.22E-23***	0***		0.909	2.21E-21***	0***	
10	0.939	1.10E-22***	0***		0.900	6.72E-20***	0***		0.275	0.017*	6.00E-04***	
<i>Residual eigenvector</i>												
0.7	0.426	8.84E-06***	0***		0.559	5.22E-09***	0***		0.573	2.01E-09***	0***	
1.5	0.457	1.80E-06***	0***		0.549	9.21E-09***	0***		0.562	4.12E-09***	0***	
3	0.542	1.40E-08***	0***		0.588	7.51E-10***	0***		0.594	5.27E-10***	0***	
10	0.586	9.10E-10***	0***		0.632	1.39E-10***	0***		0.275	0.017*	2.00E-04***	
<i>Betweenness</i>												
0.7	0.199	0.067†	0.105		0.411	1.56E-04***	0.042*		0.374	5.52E-04***	0.030*	
1.5	0.463	1.99E-05***	2.00E-04***		0.370	6.39E-04***	0.049*		0.379	4.76E-04***	0.017*	
3	0.367	7.12E-04***	0.032*		0.366	7.38E-04***	0.023*		0.352	0.001**	0.030*	
10	0.361	8.81E-04***	0.022*		0.331	0.003**	0.036*		0.117	0.372	0.334	

(Continues)

TABLE 2 | (Continued)

R_0	Infectious period = 3 days				Infectious period = 7 days				Infectious period = 20 days			
	Kendall's τ	p Value	Perm p value		Kendall's τ	p Value	Perm p value		Kendall's τ	p Value	Perm p value	
<i>Residual betweenness</i>												
0.7	0.002	0.987	0.587		0.013	0.893	0.351		-0.037	0.699	0.530	
1.5	0.073	0.449	0.060 [†]		-0.060	0.533	0.630		-0.026	0.788	0.429	
3	-0.014	0.887	0.505		-0.042	0.664	0.498		-0.038	0.693	0.437	
10	-0.052	0.586	0.461		-0.041	0.679	0.402		0.025	0.830	0.496	

Note: Significant codes are marked as follows: *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$, [†] $p < 0.1$.

individual being infected, the outbreak size, and its duration. Although transmission of pathogens cannot always be explained by social networks (Morrison et al. 2021), direct connections between individuals, such as their strength or number of partners (degree) have generally been shown to be good predictors of disease dynamics and pathogen exposure, both in simulation (Christley et al. 2005; Rushmore et al. 2014; Romano et al. 2016; Rozins et al. 2018) and observational studies (Patrono et al. 2020; Sandel et al. 2020; Myall et al. 2022). Our simulations support this finding as strength predicted the probability of an individual being infected irrespective of the R_0 and infectious period of the pathogen (Table 2). Indirect connections such as betweenness, however, remained the least efficient predictors for all our measures of disease dynamics, even if it still proved a significant factor in many cases. This is partially divergent from a previous study showing that indirect metrics were good predictors of the latency to complete transmission in grooming networks of Japanese macaques (i.e., outbreak duration here; Romano et al. 2016). An explanation may be that different social styles across species, and the consequent variation in connectivity due to varying social tolerance rates (Sapolsky 2006; Sueur, Petit et al. 2011; Call, Amici, and Aureli 2012), may produce different network topologies. This, combined with the type of data (e.g., party network versus grooming network), may in turn produce different network structures, which may mediate the predictive power and relative importance of each centrality metric in disease dynamics.

In sum, although these studies found divergent results, it may shed light on the importance of network types in predicting pathogen transmission. In the case of our study, we used a party network that can be depicted as loose aggregations within acoustic range and moving in the same general direction (Itoh and Nishida 2007). This means that our chimpanzee network exhibits the maximum density and a very low modularity. In such a network, betweenness is expected to be of less importance than in a more modular network (Rozins et al. 2018), where individuals with high betweenness centrality may play a key role in the diffusion of pathogens between different regions of the network.

When evaluating the importance of individual traits to the relationship between centrality and disease dynamics we observed that these contributed to less than half of the observed effect. This contrasts with previous studies where individual traits could effectively predict disease outcomes (Rushmore et al. 2014), and targeting them could lead to efficient control of the epidemic even when not accurately representing centrality (Evans et al. 2023). Without having the same kind of data as these studies at our disposal (social: family size, and social rank, but also on ecological factors), it remains difficult to assess what drives this difference. We can note, however, that individual traits seem to have a proportionally higher contribution to the predictive power of betweenness on an individual's probability of getting infected (Table 2). Yet, betweenness remains our least influential predictor, as stated above. Generalizing thus seems difficult and using network data, when possible, should be preferred. Considering the relative ease with which data on age and sex can be collected—or indeed other sociodemographic characteristics—compared to network data, and its relative

efficacy at predicting disease outcomes, we encourage further research assessing proxies of centrality for epidemic control.

Regarding our third question, we investigated whether characteristics of infectious agents, such as their basic reproduction number (R_0) and infectious period, influence the predictive power of network centrality measures. As expected, longer infectious periods increased the probability of getting infected as well as the outbreak size and duration. With a stable R_0 , and by increasing the infectious period, Cross et al. (2005) found that more individuals were infected and that the outbreak lasted longer in structured populations. In accordance with previous simulation studies, increasing R_0 increased the probability of getting infected (Christley et al. 2005) as well as the outbreak size (Hamede et al. 2012; Griffin and Nunn 2012; Rushmore et al. 2014). Increasing R_0 also enhanced the predictive power of centrality metrics on the probability of getting infected (Christley et al. 2005). However, we showed that R_0 had a bimodal effect on outbreak duration, which seems related to infectious period (Figure 3B). Increasing R_0 with an infectious period too short to allow complete transmission increased the outbreak duration. This happened because of an increase in the proportion of infected individuals. When there was whole-group transmission, increasing R_0 reduced the time to achieve it and the predictive power of centrality on the outbreak size and probability of getting infected greatly decreased. Interestingly, in our study, a long infectious period tended to alleviate the importance of centrality to predict the outbreak size, and eventually produced results not different from what is expected under a random network, even when complete transmission was not consistently achieved. Nevertheless, it emphasizes its importance to predict the outbreak duration. In short, our work highlighted the interplay between R_0 , the infectious period and the individual centralities, all acting on the outbreak—which, to our knowledge, has not yet been considered on animal epidemiology and conservation.

In this study, we tested an array of theoretical values of R_0 and infectious periods that may not necessarily represent reality. However, our results can still provide valuable information, as recent outbreaks in humans and other great apes have been shown to have a R_0 (from 1.27 to 9.42) and an infectious period—the duration during which an infected individual is infectious—from 0.38 to 20 days, with an outlier at 40.84 days) similar to the ones tested in this study (Chowell et al. 2004, 2006; Legrand et al. 2007; Tuite et al. 2010; Ndanguza, Tchuente, and Haario 2013; Scully et al. 2018; Negrey et al. 2019; Byrne et al. 2020; Morrison et al. 2021). In most of these outbreaks, the R_0 was less than 3 and the infectious period less than 7 days. For such pathogens, our model indicates that centrality is a good predictor of the individual probability to get infected and, to a lesser extent, of the outbreak size. Regarding outbreak duration, our model anticipates a relationship with centrality that ranges from positive to negative depending on how often complete transmission occurs.

Taken together, our results illustrate that understanding the impact of sociality on pathogen transmission is important for developing conservation strategies for primates and other species. For the previously described pathogens, because centrality is a predictor of individual probability to get

infected, one may consider adopting a network-based strategy. For example, targeted health monitoring of males, in particular—as they are more central—could allow an earlier detection of outbreaks and a consequently faster intervention. Alternatively, wildlife vaccination is increasingly being discussed as a viable option to avoid disease outbreaks in the wild (Warfield et al. 2014; Capps and Lederman 2015; Leendertz et al. 2017). We are aware that wildlife vaccination is time-consuming and costly (Plumb et al. 2007; Barnett and Civitello 2020), and immunity may be short-lived, but targeting individuals as well as developing new vaccines with relevant, cost-effective administration strategies may be advantageous for wildlife protection when the disease could cause the species' extinction and as long as benefits outweigh the risks (Capps and Lederman 2016). The model presented here could help explore the effects of various pathogens and test various transmission mitigation scenarios in disease-threatened populations.

We recognize there are limitations to our study, including the simplification of a real process. For example, we did not consider that individuals may have varied immune responses that depend on their genetics, social rank, or previous exposure to the pathogen. These are difficult data to gather, even in humans (Il'Yasova and Kinev 2019). Nonetheless, such data would improve the accuracy of these epidemiological models and contribute to unraveling the complexity behind individual traits and disease dynamics. For instance, high-ranking male chimpanzees seem to have higher levels of immunosuppressing testosterone (Muller and Wrangham 2004), which could result in greater susceptibility to infection, and consequently, probability of infecting others. This, combined with their moderate to high centrality (Rushmore et al. 2013; Watts 2018; Sandel et al. 2020), could accentuate male impacts in the transmission chain. Furthermore, we built our network with only 2 months of observations. Even though every possible dyad exists in our network, it represents just a fraction of all the variability in associations that may exist in the group throughout a longer time scale (Rushmore et al. 2013). Additionally, our network was based on “loose party associations,” which may not be ideal given the characteristics of socially transmissible disease agents (different nuances of interactions, and transmission according to a given proximity and behavior) but this method does estimate the “probability of interaction” among party members, at least relative to nonparty members. Another aspect for improvement is the potential for pathogen transmission between parties, as estimated in other studies with Great Apes. For example, in the highly modular network of a gorilla population, respiratory diseases spread quickly within a group but not to the other groups because of the rarity of interactions between them (Morrison et al. 2021). In the case of our study, chimpanzees' fusion-fission social structure implies regular interactions between parties that are not accounted for by the party-level AI (like the nocturnal fusion periods), and which may have critical impacts on disease dynamics at the community scale. Finally, our model also assumes that networks are static throughout the outbreak, without considering behavioral changes, such as lethargy, that can decrease social interactions during epidemics (Rushmore, Bisanzio, and Gillespie 2017; Patrono et al. 2020). This could be implemented in future studies.

In conclusion, by studying the social behavior of wild chimpanzees, we showed that males and especially older males are more central than females in the group, as expected, and that individual centrality is a strong determinant of the probability of being infected during an outbreak (especially in terms of strength and eigenvector centrality). Our results demonstrate that individual centrality is a good predictor of disease dynamics regardless of the value of R_0 used in the model. In the context of targeted intervention, because centrality can be challenging to measure in the field, a previous study proposed to instead use proxies of centrality which they found were family size and social rank (Rushmore et al. 2013). In our study, age only predicted individual centrality among males. The ability of these individual traits (in our case, age and sex) to predict disease dynamics was therefore limited, highlighting the need for caution when exploring the potential for trait-based interventions. This study highlights the importance of using animal social networks in wildlife epidemiology, encompassing how different centrality metrics predict simulated disease dynamics. We hope this stimulates further discussion into the roles of individuals in pathogen transmission, and their consequences for wildlife conservation.

Author Contributions

C.S., A.J.J.M., and V.R. conceptualized and supervised the research. C.S., M.S., A.J.J.M., and V.R. acquired the financial support to conduct this research. M.S. conducted the field investigation by collecting nonhuman primate data. M.P., C.S., A.J.J.M., and V.R. developed the methodology and software. M.P. conducted the formal analysis, visualization, and wrote the original draft. All authors contributed to the review and editing of the drafts and gave final approval for publication.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The source code that supports the findings of this study is openly available in Zenodo: <https://zenodo.org/record/7377819> (Pierron, Sueur, and Romano 2022). The data that support the findings of this study are available on request from MS. The data are not publicly available due to privacy or ethical restrictions.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.