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THÈSE présentée par:

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Discipline/ Spécialité: Sciences du Vivant/Ecologie & Ethology

Social networks as a trade-off between optimal information transmission and reduced disease transmission

Les réseaux sociaux comme compromis entre une transmission d'information efficace et une réduction de la transmission de maladie

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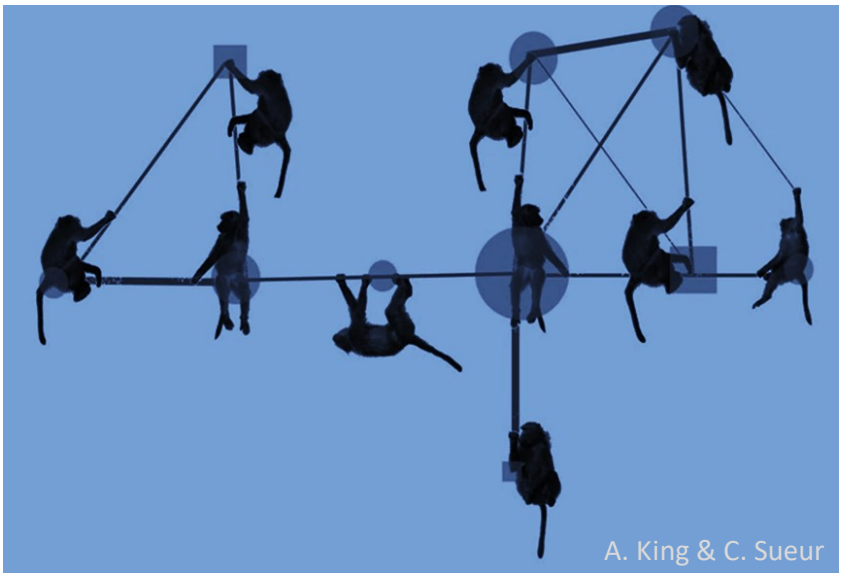

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To my parents,

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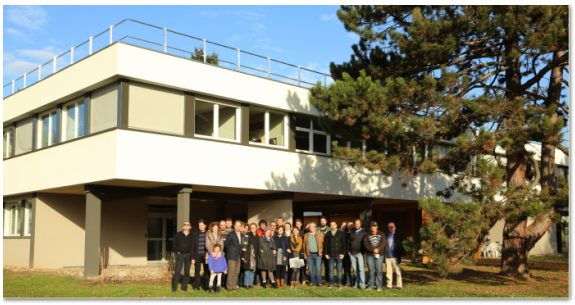

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- Duboscq J, **Romano V**, MacIntosh AJJ, Sueur C (2016) Social information transmission in animals: lessons from studies of diffusion. *Frontiers in Psychology*. 7: 1147. (doi:10.3389/fpsyg.2016.01147).

- Dusboscq J, **Romano V**, Sueur C, MacIntosh AJJ (2016) Scratch that itch: revisiting links between self-directed behavior and parasitological, social and environmental factors in a free-ranging primate. *Royal Society Open Science*. 3: 160571. (doi:0.1098/rsos.160571).

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- Duboscq J, **Romano V**, Sueur C, MacIntosh AJJ (2015) A social network perspective on macaque social styles. *American Journal of Primatology*. 42.

- MacIntosh AJJ, Sarabian C, Duboscq J, Thomas E, **Romano V**, Kaneko A, Okamoto M, Suzumura T (2015) Hidden constraits of chronic parasitism on health and fitness in Japanese macaques. *Primate Research Supplement*. The 31th Congress Primate Society of Japan. 103.

COMMUNICATIONS OF PHD RESULTS

- Duboscq J*, **Romano V***, Sueur C, MacIntosh AJJ (2013) Social networks as a trade-off between optimal decision-making, information transmission and reduced disease transmission. IX. Göttinger Freilandtage: the sociality-health-fitness link. Göttingen, Germany. *Poster Presentation*. *Co-presenters.

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SCIENTIFIC EDUCATION

- **Romano V** & Ruiz-Miranda C. (2014) Should I stay or should I go now? - report of dispersal in reintroduced and wild-born golden lion tamarins. *Tamarin Tales*. 12:9-10.

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General Introduction

Sociality, information and infectious disease

I. INTRODUCTION

"It is certain that either wise bearing or ignorant carriage is caught,
as men take diseases, one of another:
therefore, let men take heed of their company".
Shakespeare, Henry IV, part 2 (1600)

As observed by William Shakespeare in one of his memorable works, the rate of contact among individuals can lead to the transmission of knowledge among conspecifics, for better or worse, just as it can for infectious agents. It should therefore be upon each individual to decide with whom to interact. Social animals make daily decisions regarding their social lives, for example to reinforce or break relationships, to determine where to sit among conspecifics or which sexual partner to choose. This set of decisions affects the number and quality of an individual's social relationships, which in turn reflects the social structure into which those individuals are embedded (Hinde 1976). Sociality has evolved repeatedly throughout the animal kingdom (Maynard & Szathmary 1995), and undoubtedly brings many benefits for individuals, such as defense against predators, increased foraging efficiency, and increased offspring survival (Wilson 1975; Krause & Ruxton 2002), as well as certain costs such as within group competition where resources are limited in space and time (Wrangham 1980) and infectious disease transmission due to the frequent contact among conspecifics (White et al. 2017). In consequence, individuals face trade-offs while maximizing the

benefits and minimizing the costs of group-living. Social complexity emerges from these individual efforts or strategies to create solutions to deal with such inherent trade-offs in their social lives.

I.1. Animal societies and social structure

While there may be no unified definition of sociality in behavioral and evolutionary ecology (Kappeler et al. 2015), in general terms sociality refers to the degree to which individuals in animal populations tend to associate in groups. In vertebrates, individuals can be solitary but form temporary or facultative groups, e.g. for reproduction or nest sharing, or individuals may be involved in repeated social interactions with conspecifics, the core feature of stable groups[1].

Animals living in permanent (i.e. stable) groups often interact nonrandomly with their conspecifics, leading to complex patterns of social interaction manifest as intra-individual variation in the number of partners and the time allocated for relationships with each over time (Kurvers et al. 2014). Independent factors such as morphometric measures (Croft et al. 2005), individual personality (Pike et al. 2008), nutrient requirements (Sueur & Maire 2013), and hierarchical structure all influence the individual decisions that in turn affect interaction preferences observed. The emergent pattern of social interactions is called the social structure

(Hinde 1976) and it highlights individual differences in social behavior[2] (Whitehead 2008a). For example, the social structure of guppies (*Poecilia reticulata*) is influenced by body length and shoaling tendency, with individuals preferentially interacting with conspecifics of similar traits (Croft et al. 2005). Affiliative interactions in yellow-bellied marmots (*Marmota flaviventris*) is influenced by age and kinship, with younger animals being more active in grooming interactions, greeting, sitting in close proximity and play behavior (Wey & Blumstein 2010). Social patterns of shy sticklebacks (*Gasterosteus aculeatus*) are highly skewed in consequence of interactions concentrated to few individuals (Pike et al. 2008). How and why individuals interact is a long-standing question in biology, as it affects gene flow and spatial patterns (Wilson et al. 1975).

The conceptual framework presented in studies of animal societies and social structure is usually based on Hinde's (1976) classic paper, which introduced three levels of association: i) interactions, ii) relationships and iii) social structure (**Figure I.1**). At the fundamental basis of this framework is the *interaction*, denoting the nature of the mutual activities of each pair of individuals (content), as well as how they perform them (quality; Hinde 1976). An interaction, or an individual behavior, can comprise one or more types of activities, such as grooming, food transfer, fights or spatial proximity. The nature of interactions, whether they are affiliative or agonistic, defines the quality of interactions that subsequently affect the social relationships between a pair of individuals, i.e. a dyad. In the

second level of the framework, a *relationship* refers to the patterning of social interactions, the content and quality of interactions within each dyad (**Figure I.1**). For example, relationships might summarize how frequently individuals groom each other. At the last level, there is *social structure*, denoting the overall quality and patterning of social relationships in a group (Hinde 1976; Figure 1). Hinde's framework is multidirectional, indicating that social structure may feed back into social relationships as well, which in turn affects social interactions (**Figure I.1**). Revisiting the previous example of guppies, the social structure created as a consequence of social preferences for individual traits, i.e. shoaling tendency and body length, creates a feedback loop from which individuals sharing similar traits are in turn more likely to interact among themselves than with others of different traits; the social structure thereby directs social relationships and interactions (Croft et al. 2005).

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[1] GROUP: the definition of group might vary among observers and to the animals themselves. For most primatologists, "a group is usually a largely set of animals whose interactions are with each other" (Whitehead 2008a), whereas for researches studying cetaceans or chimpanzees, the same definition is considered a community (e.g. Cantor et al. 2015). In this thesis, I follow the definition used by primatologists, in which groups comprise individuals that actively maintain spatiotemporal proximity and keep the majority of their social interactions within the same set of individuals.

[2] SOCIAL BEHAVIOR: denotes behavior directed toward one or more individuals, usually of the same species, and in most cases mediated by communication or relationships (Kappeler et al. 2013). Examples are grooming, courtship, parental care, coalitions, fights, etc.

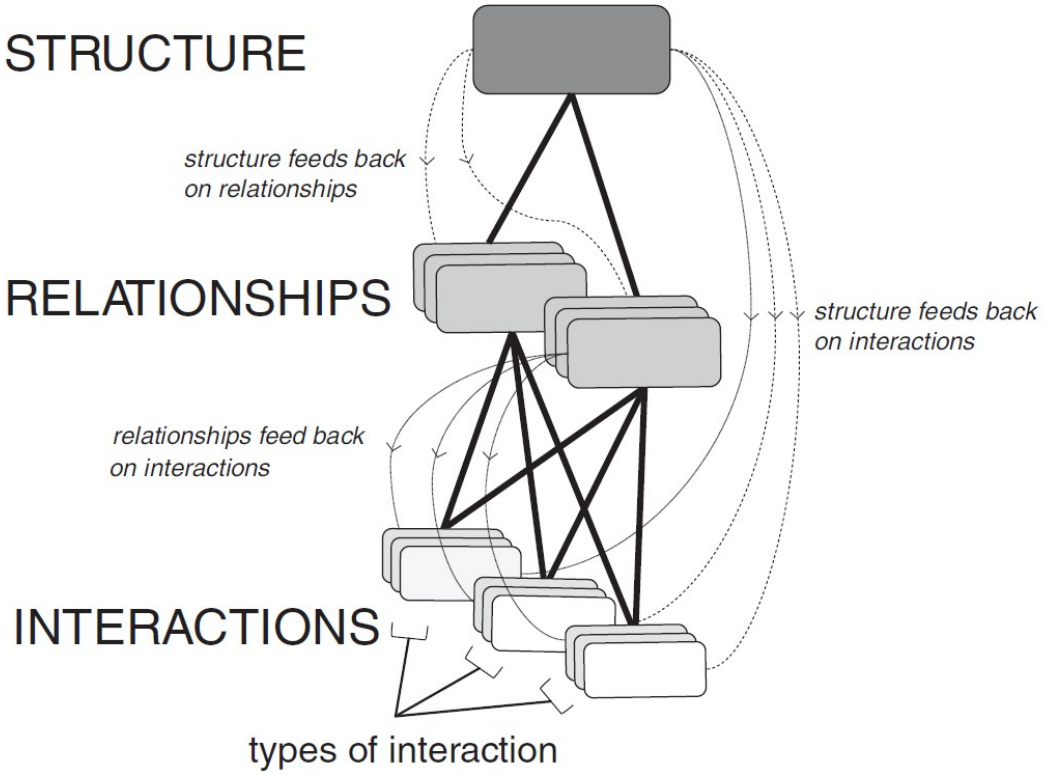

Figure I.1. Schematic representation of Hinde's (1976) conceptual framework for the study of animal societies. Interactions, relationships and social structure are represented as rectangles at three levels with multidirectional effect. Social structure feeds back to influence social relationships and interactions, and relationships similarly affect social interactions. Figure from Brent et al. 2011.

I.2. The analytical side of social structure

The study of social structure has a long history in Biology, with classical studies on social analyses conducted up to the 1960's being reviewed in Crook (1970), and then placed into a modern analytical framework as reviewed by Whitehead (2008a). This field has been frequently updated since then (e.g. Farine & Whitehead 2015; Scott 2017). For about 15 years there is a remarkable interest in investigating the social structure using a set of

sophisticated techniques called social network analysis (SNA; e.g. Lusseau 2003; Croft et al. 2008; Wey et al. 2008). SNA methodology originated from mathematical graph theory (see Scott 2017 for a review) and provides a refined estimation of the complex social structure into which individuals are embedded (Whitehead 2008a; see the section *Material and Methods* for a description of SNA). A social network comprises nodes, which represent individuals, connected by ties (also called links or edges), that represent the interactions between a pair of individuals. A synonym of social structure, social networks are pervasive in nature (**Box I.1**).

The conceptual framework of SNA is now consolidated in Ethology, Behavioral and Evolutionary Ecology, and it is broadly accepted as a means of investigating patterns of social interactions in animal groups (Croft et al. 2008; Whitehead 2008a; Krause et al. 2014; Sueur 2015), with applications in a variety of major taxa (e.g. fruit flies: Pasquaretta et al. 2016; sharks: Mourier et al. 2012; dolphins: Lusseau 2003; giraffes: Carter et al. 2013; birds: Oh & Badyaev 2010; meerkats: Blumstein et al. 2009; primates: Bret et al. 2013). The first generation of studies primarily introduced the concept of social networks analysis (Krause et al. 2007; Wey et al. 2008; Sih et al. 2009; Brent et al. 2011; Sueur et al. 2011a), but, more recently, finer analyses of social structure have allowed us to identify its effects on many ecological and evolutionary mechanisms (see Kurvers et al. 2014 for a review), such as animal dispersal (Blumstein et al. 2009), sexual selection (McDonald et al. 2013),

cooperation (Rand et al. 2011) and social transmission (Duboscq et al. 2016a, **Appendix B**; White et al. 2017).

Box I.1. Example of animal social networks.

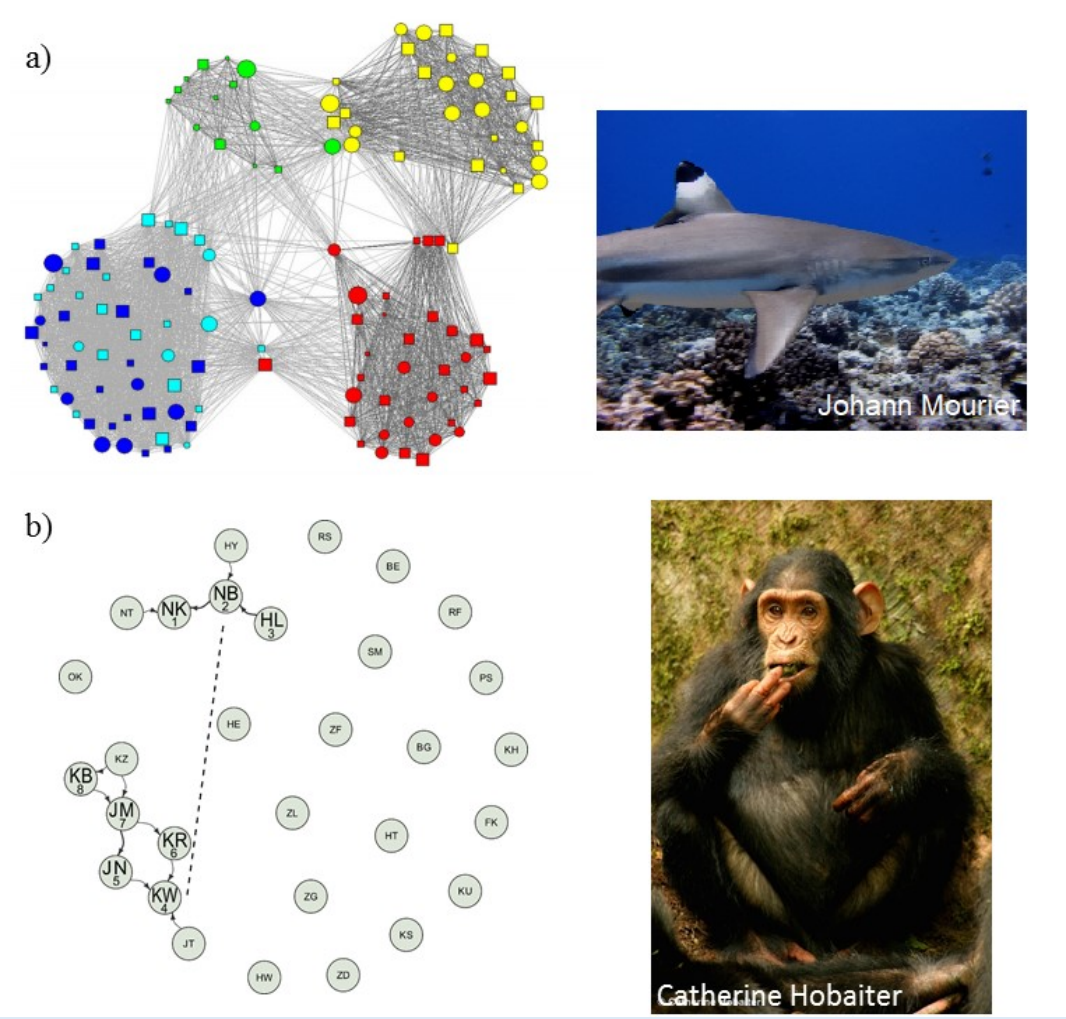

Figure BI.1. Social networks as evidence of social community in blacktip reef shark (a; Mourier et al. 2012) and the spreading of the moss-sponging behavior in the Sonso chimpanzee community (b; Hobaiter et al. 2014).

I.3. Social transmission

Social structure affects social transmission, and social transmission occurs through social interactions, which are mediated by relationships. We can envisage social transmission as involving any entity (e.g. knowledge, behavior, disease causing organisms) that can be transferred from one individual to another by direct contact or spatial proximity. Social transmission is an important component of animal society, with clear impacts on individual fitness. For example, the alarm calls of European robins (*Erithacus rubecula*) prevent infant predation (East 1981), and strong social bonds favor the spread of Tuberculosis among wild badgers (*Meles meles*) in Great Britain (Weber et al. 2013). The social interactions or relationships between individuals (e.g. body contact, proximity, grooming or fight, Croft et al. 2008) represent the possibilities for individuals to transmit such information and/or socially-transmitted pathogens.

I.3.1. Information transmission

In terms of animal societies, information is broadly understood as knowledge possessed by a potential resource-holder, which may benefit other individuals if transmitted (Stephens 1989, but see Dall et al. 2015 for uncertainty on information reliability). There is a range of possible information sources, including the environment – leading to "personal information" - or conspecifics – leading to "socially acquired information" if transmitted (Dall et al. 2015;

Figure I.2a). Personal information may include, for example, the observation of common landmarks, stars and the geomagnetic field to guide migrations (Grocott 2003). Socially-acquired information consists of the behavior, innovations or knowledge transferred from one individual to another (Dall et al. 2015; **Figure I.2b**). It can be produced advertently (a signal) or inadvertently (a social cue or public information). A signal is produced by an individual for the purpose of communication (Dall et al. 2015), with a classic example being predator alarm calls (Blumstein et al. 1997). As products of the individual emitting and mediating the signal, natural selection favors individuals whose signals provide fitness advantages, to both the signaler and the receiver in the case of honest communication (Krebs & Dawkins 1984). Inadvertent social cues, on the other hand, might also provide information about the presence or absence of a feature, such as the spatial location of a food patch (Galef & Giraldeau 2001). In this situation, however, the emitter has no control over the kind of information being transmitted, but natural selection might favor the abilities of other individuals to perceive such cues (Krebs & Dawkins 1984). Public information denotes graded information about a feature that can facilitate estimation of the quality of a feature in question, such as the aggressiveness of an opponent (**Figure I.2b**). Using social information is known to provide faster or better adaptations to environmental changes than using solely personal information. Information is thus an important currency of exchange among individuals in a society (Dall et al. 2015; Duboscq et al. 2016a).

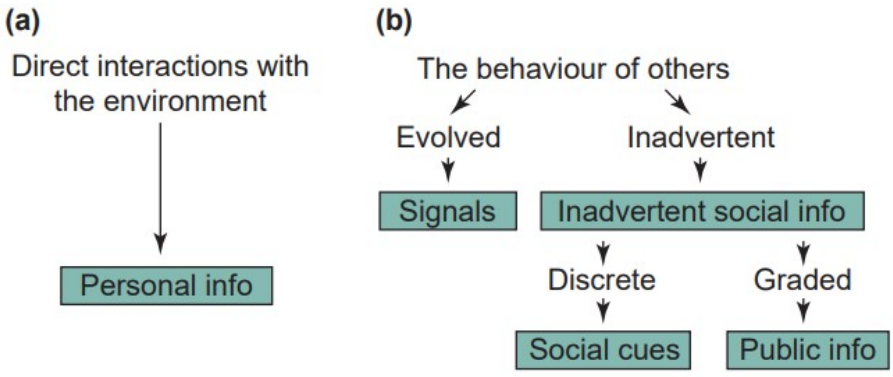

Figure I.2. Scheme representing the two types of information: personal information (a), directly acquired from the individual's interaction with the environment, or socially-acquired information (b), acquired from signals, social cues provided by conspecifics or public information. Figure from Dall et al. 2015.

In this thesis, I use the general definition of *socially-acquired information*, which can relate to any potential resource, such as the indication of a foraging patch, innovations, predator threats and/or partner selection, which provide evolutionary advantages (e.g. Danchin et al. 2004; Dall et al. 2015; Kendal et al. 2005; Aplin et al. 2012; Duboscq et al. 2016a). In behavioral and evolutionary ecology, information transmission is usually linked to social learning and cultural transmission (**Box I.2**). Cantor & Whitehead (2013), for instance, proposed an extension of Hinde's (1976) conceptual framework by including information transmission at the final level of the diagrammatic representation. In their review, they demonstrated that culture is shaped by social structure, and culture itself influences social structure, since individuals develop preferential patterns of interactions. For example, sympatric community of cetaceans sharing a vocal repertoire emerge from cultural

transmission, as individuals form bonds based on their behavioral similarity, which, as a consequence, creates a biased social learning of codas (i.e. units of communication; Cantor & Whitehead 2013; Cantor et al. 2015). If we express Hinde's (1976) scheme under the light of social networks, individuals (nodes), through their characteristics and behaviors, influence the pattern of social interactions and relationships (links) that affect the social structure (network topology). The network structure subsequently affects information transmission, through social learning and culture (Cantor & Whitehead 2013). This scheme also does not include causal directionality, meaning that the influence may circle back around to affect social relationships and individual behavior.

Box I.2. Social learning and cultural transmission.

Everyday, individuals acquire information provided by others to guide their own behavior (Morand-Ferron et al. 2010). There is an entire field dedicated to the mechanisms and patterns driving social learning in animals (Galef & Laland 2005; Hoppitt & Laland 2013). More specifically, if information is defined such that shared behavior is consistently different between groups in a population, it may be called culture (Whiten & van Schaik 2007). One example of information transmission comes from a wintering sub-population of great tits (*Parus major*) inhabiting the Wytham woods, England. Researchers aiming to investigate the

establishment of foraging techniques in the wild birds introduced a puzzle box with two opening options: slide right or left (**Figure BI.2a**). By examining the number of individuals within the flock that acquired the behavior, the team showed that from only two trained birds in each sub-population, the information spread quickly through the social network, reaching 75% of individuals in approximately 20 days (**Figure BI.2b**). Interestingly, the sub-populations were biased toward the foraging technique originally introduced (**Figure BI.2c**), demonstrating that informational conformity, in which individuals choose the most common variant when first learning a new behavior, is present in these wild birds (Aplin et al. 2015). This study contributed to the field of social transmission by showing that conformism is not restricted to humans.

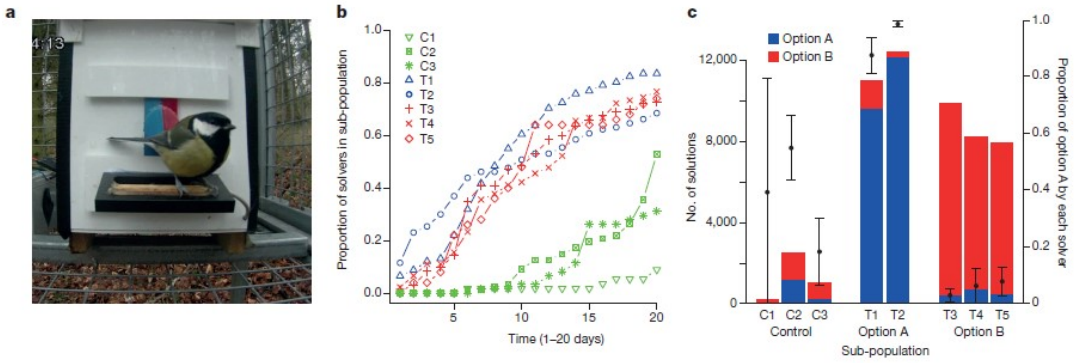

Figure BI.2. Transmission of foraging techniques in wild great tits. From an opening-box task (a), two demonstrators spread the behavior to the majority of their sub-population in about 20 days (b). Individuals kept the behavior learned using a specific technique, either by opening the box by sliding it to the right or to the left (c). Figure from Aplin et al. 2015.

I.3.2. Infectious agent transmission

Parasites[3] are pervasive in the lives of social animals, and although in some cases infection can appear benign, without visible symptoms or detectable impacts on individual fitness (Price 1980), infectious diseases can contribute significantly to the mortality and morbidity of numerous individuals (e.g. Rachowicz et al. 2006; Frick et al. 2010). For example, many microparasites, such as bacteria and viruses, are highly virulent and can cause significant population declines (e.g. Ebola in West Africa: Walsh et al. 2009; Anthrax in Central and West Africa: Hoffman et al. 2017). On the other hand, macroparasites such as helminths and arthropods are more likely to exhibit chronic effects on host survival and reproduction by decreasing the potential number and quality of offspring (e.g. May & Anderson 1978).

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[3] DEFINITION: The word parasite has different meanings and depends on the discipline in which it is used. I use the ecological definition of **parasite**, which refers to "any organism that lives on and draws nutrients or material resources, such as metabolism and behavior, from another living organism - the host" (Nunn & Altizer 2006). Parasites thus include the **disease-causing organisms** such as bacterial and viral pathogens, but also the many other species that live off other animals at some cost but are not associated with clinical disease or at least not present in the numbers required to cause overt disease in their hosts. **Pathogens** refers simply to any disease-causing organism, but this term is usually associated with microbial parasites in the medical and parasitological literature (viruses and bacteria; Nunn & Altizer 2006). In this thesis, I use the term parasite to denote any agent that can impact the health and/or fitness of its host, overtly or otherwise. However, because most of the work appearing in this thesis is theoretical, I also use the terms pathogen, infectious-agent, disease-causing organism and infectious disease interchangeably throughout the text.

The dynamics of infectious diseases spread via direct person-to-person transmission (such as influenza, smallpox, HIV/AIDS, etc.) are expected to be highly dependent on social structure (Pastor-Satorras & Vespignani 2001; Meyers et al. 2005; Salathé & Jones 2010; Griffin & Nunn 2012). The relationship between group-living and infectious disease thus seems to be generally straightforward: animals living in closer proximity and with higher contact rates should experience higher rates of pathogen transmission (e.g. primates, Nunn & Altizer 2006). However, individuals have also developed defenses to prevent or respond to pathogen invasions. These anti-parasite strategies include immunological defenses to combat infection (Wilson et al. 2003) as well as behavioral counterstrategies, such as hygiene, self-medication and social avoidance. In Japanese macaques (*Macaca fuscata*), for example, hygienic behavior in response to potential sources of contamination was linked to low geohelminth infection (Sarabian & MacIntosh 2015). Furthermore, infected horses and primates consume plants with medicinal properties to control parasite infection (Huffman 1997; Williams 2008). Finally, mandrills (*Mandrillus sphinx*) were shown to recognize parasitized individuals and avoid grooming their anogenital areas when shedding infective stages (Poirotte et al. 2017).

Changes in the rate of contact with conspecifics may therefore be one of the important mechanisms preventing pathogen transmission. Whether infected individuals actively avoid social interactions or become lethargic and therefore engage in fewer social interactions in

general as part of the sickness response (Hart 1988), or whether uninfected individuals actively avoid infected conspecifics (especially if they show signs of sickness), reduced social interactions might impact social structure in ways that can down-regulate social transmission and thereby constrain the infection to a few individuals (e.g. Lopes et al. 2016). For example, wild house mice (*Mus musculus domesticus*) challenged with lipopolysaccharide (LPS), which mimics bacterial infection, reduced their own rates of social contact by avoiding encounters with other group members (Lopes et al. 2016). In contrast, healthy bullfrogs (*Rana catesbeiana*) avoided individuals with a yeast infection (Kiesecker et al. 1999). The end result in both cases, despite the different mechanisms at play, is that uninfected or healthy individuals were less likely to interact with infected conspecifics, which should slow down the spread of infection through the population.

Finally, understanding the dynamics of infectious disease is important from various perspectives, from evolutionary to ecological and to health considerations, as well as to modern issues such as anthropozoonotic transmission and infectious disease in conservation (**Box I.3**). There is growing concern that accelerated environmental change may in turn accelerate the emergence of infectious diseases and potentially of disease outbreaks that can lead species toward local extinction (Deem et al. 2001; Leendertz et al. 2006), sometimes even putting our own human society at greater risk (Chivian & Bernstein 2008). Studies provide evidence on how network topology can not only be used to predict the chain of

transmission (who transmits pathogens to whom) but also to implement intervention strategies based on the identification and removal (e.g. quarantine or vaccination) of individuals more likely to spread disease-causing organisms (i.e. superspreaders; Silk et al. 2017; White et al. 2017). Network epidemiology, a framework combining components of epidemiology and network science, offers a sophisticated analytical platform to investigate the dynamic of infectious agent transmission between animal and/or human populations (Barabási 2016).

Box I.3. Transmission of disease, wildlife and human health: a holistic view

Epidemics, such as Ebola in 2014 (West Africa) and measles in 2015 (Canada), called the attention of agencies and governments to invest in intervention measurements (Fitzpatrick et al. 2017; Thomas et al. 2017). Outbreaks may occur stochastically, but future events are certain to happen (Ash 2017). Pathogen transmission thus poses substantial challenges for the conservation of wildlife, public health and ecosystem welfare.

Among current global concerns related to health are emerging infectious diseases (EID), the phenomenon being referred to as *pathogen emergence*, which are diseases that are increasing in incidence or expanding in geographic range into human populations for the first time (**Figure BI.3**; Jones et al. 2008). EID are mainly thought to be driven by socio-

economic, environmental and ecological factors (Jones et al. 2008) linked with anthropogenic modification of habitats and erosion of biodiversity (Keesing et al. 2010). Studies show that conservation of wildlife might be an efficient strategy for improving human health and well-being, since it maintains ecosystem health (Kilpatrick et al. 2017; Young et al. 2017). The modeling of infectious diseases in wildlife is then a valuable tool to study the mechanisms by which diseases spread, to predict the future course of an outbreak and to evaluate strategies to control an epidemic, providing the basis for understanding epidemiological processes with implications for species conservation, the overall well-being of animal societies, and the interrelationships between species and their ecosystems (e.g. One Health concept).

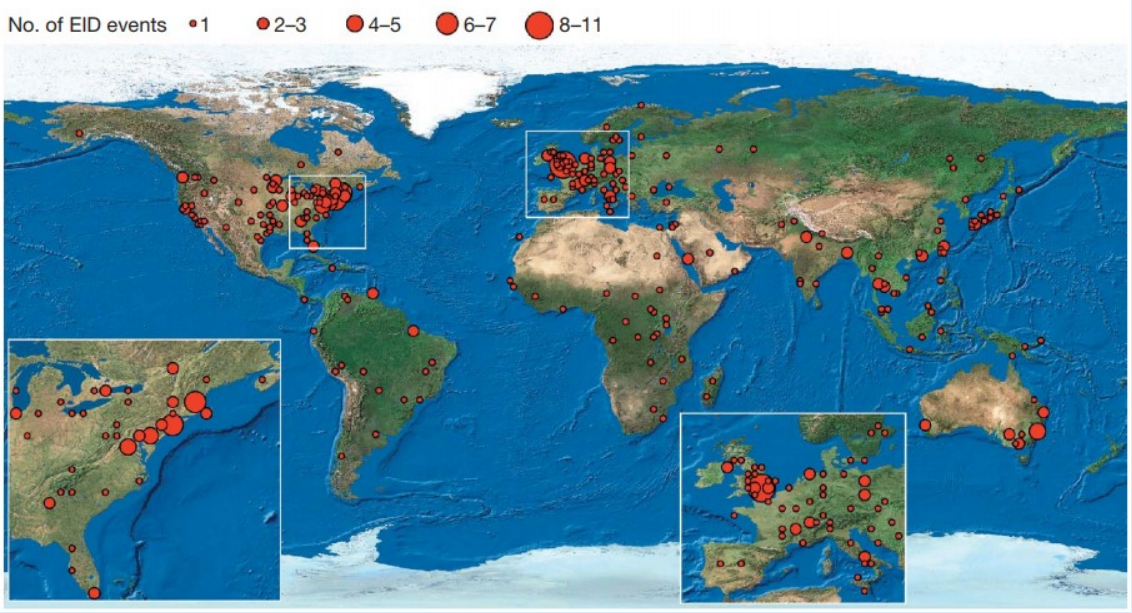


Figure BI.3. **Geographic origins of EID's from 1940 to 2004.** The large concentration of EID events per million square kilometers of land is concentrated in the northeastern United States, western Europe, Japan and southeastern Australia. Figure from Jones et al. 2008.

I.4. Increasing contagion risk and information flow via social networks

The spread of information and pathogens are both well-studied topics in network science (Barabási 2016). Multiple theoretical and experimental studies have indicated that the differentiation of social roles that individuals play, as well as the network structure – as estimated by global metrics such as density, which describes the proportion of relationships in relation to the potential total network connectivity, or modularity, which estimates the level of subgrouping present in the network – each influence both transmission process. These studies were mainly conducted using information and parasite flow networks independently, perhaps because of the challenges in investigating both within the same empirical framework or in accordance to the research questions of the scientist, but they provide evidence that the same properties of the network that favor transmission of information also favor the spread of pathogens. Further, a computer simulation study has shown that the spread of information and viruses happens as a function of similar network components, such as the position of individuals within the network (Weng et al. 2013).

I.4.1. Network position

An individual's network position is usually determined by the relative number and strength of its social relationships, hereafter called connections. Central individuals are those with larger

numbers of direct (e.g. number of social partners) or indirect (e.g. number of distinct subgroups an individual is related to) connections, while less central individuals are those with smaller numbers of connections. Thus, the position of an individual in its network alters the probability of acquiring or transmitting information and infectious diseases. Central individuals are expected to be key dispersers of information, controlling its quality and access (Vital & Martins 2009), but are also expected to spread infectious agents to a broader number of individuals and to be more vulnerable to pathogen exposure (Newman 2004). For example, central wild tits, estimated by their association patterns in artificial feeders, are more likely to be among the first-informed about new foraging patches (Aplin et al. 2012). At the same time, female Japanese macaques more central to their grooming networks exhibit higher parasite species richness and intensity of infection with nematode parasites (MacIntosh et al. 2012). Moreover, hunter-gatherer women in the Philippines that are more central in their proximity networks produced more living offspring but also suffered from greater disease burdens (i.e. gastro-intestinal disease, influenza and fever, respiratory tract infections and intestinal parasites; Page et al. 2017). Central individuals in terms of direct connections, at least considering the total number and strength of interactions, might indeed have more opportunities to observe and interact with others than less central individuals. However, the assumption that greater centrality is linked to greater social influence is not always straightforward, as this can also be affected by network subdivision, meaning the number of

subgroups or modules in the network, and synergies between direct and indirect connections for both information (e.g. Pasquaretta et al. 2016) and parasite transmission (e.g. VanderWaal et al. 2016).

I.4.2. Network structure

Emergent properties of the network at a global-level (**Table II.1**) are considered to distinctly affect transmission processes. For example, increases in network density (i.e. the total number of connections in proportion to the maximum possible number of connections in the network) implies faster social transmission: the more connected the network, the lower the number of connections necessary for information be transmitted from the spreader to the most peripheral individual in the group (e.g. Pasquaretta et al. 2014). The logic is simple, since social transmission is density-dependent, a higher number of individual connections will trigger faster transmission. However, the relationship between a network property and social transmission might not always be straightforward, since other properties may induce different effects in the processes. For example, group size is considered to have mixed effects on social transmission. While many studies provide evidence that infectious disease risk increases in larger groups (Ezenwa 2004; Whiteman & Parker 2004; Caillaud et al. 2013), others show the opposite relationship, with smaller groups having higher levels of parasite infection (Rubenstein & Hohmann 1989; Arnold & Lichtenstein 1993; Semple et al. 2002;

Bordes et al. 2007). Scientists claiming that sociality somehow should reduce the risk of acquiring directly-transmitted parasites led to the discovery that some network properties work as a buffer to reduce disease spread. For instance, high levels of clustering - the extent to which neighbors are connected to each other - decreases per capita infection risk in Lepidoptera (**Figure I.3**; Wilson et al. 2003).

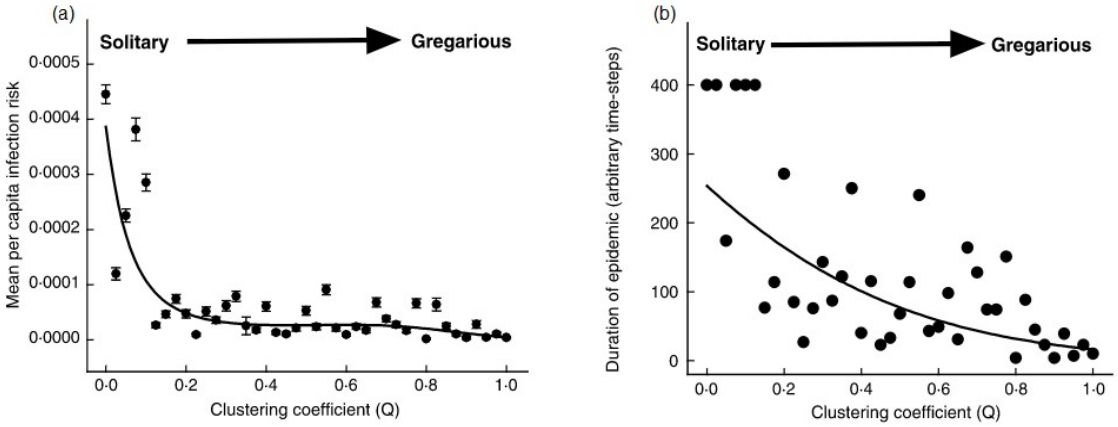


Figure I.3. Relationship between the clustering coefficient (Q) and (a) mean infection risk per individual during a simulated epidemic, and (b) duration of an epidemic. In (a), bars represent the standard error associated with the mean of infection risk per capita. Lines represent the best-fit exponential function. The higher the clustering coefficient, the lower the individual contagion risk (a) and the lower the duration of the epidemic (b). Figure from Wilson et al. 2013.

Other studies following this research line tested the "social bottleneck hypothesis", which predicts that the social network structure should modulate the association between group size and infectious disease risk: larger groups are expected to be more subdivided into

subgroups, and those subgroups should act as a social bottleneck by decreasing the potential for pathogen transmission (Nunn et al. 2015). Researchers examined the association between group size and social network metrics in 43 vertebrate and invertebrate species and showed that modularity, a measure of network subgrouping, acted as a buffer, reducing disease spread between subgroups (**Figure I.4**; Nunn et al. 2015). Prior to that, the same research group found evidences of a negative effect of modularity on non-human primate groups (Griffin & Nunn 2012). Modularity is now considered the main predictor of reduced disease spread according to the social bottleneck hypothesis (Nunn et al. 2015).

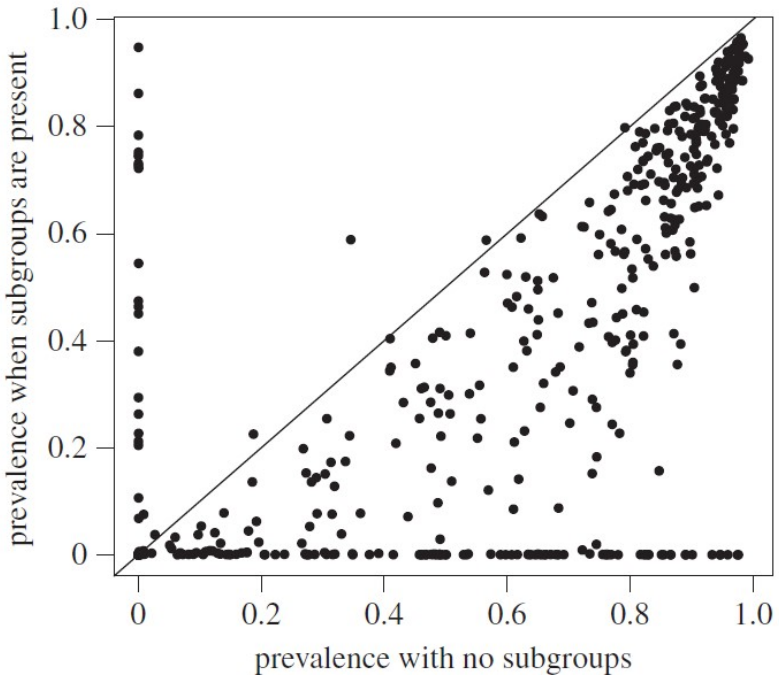


Figure I.4. Results of paired simulations showing the number of infected individuals (infection prevalence) in the presence (upper left triangle) and absence (lower right triangle) of subgroups in simulated networks. Maximum prevalence was substantially higher in the simulations without subgrouping. Figure from Nunn et al. 2015.

Network properties thus reflect a set of individual decisions and behaviors that can be dynamic, with links between individuals potentially changing in response to the behavior or status (informed, parasitized) of their social partners. For individuals showing flexibility in social behavior, it is expected that they manage their relationships according to the costs and benefits of each interaction. For example, humans were shown to cooperate, form new connections to cooperators, and break links with cheaters/defectors, leading to significant changes in network topology. This led to changes in social structure (Rand et al. 2011). Along similar lines, vervet monkeys were demonstrated to reinforce social bonds by increasing grooming bouts with individuals that provided more food to other conspecifics in an experimental foraging task, causing social preferences reflected in the network topology (Fruteau et al. 2009). Such evidence suggests that network properties at both the individual and global scale might fluctuate over time according to individual decisions. If the social structure is quantified, it may be the combination of properties rather than any specific property, such as being central in the group, that leads to optimization of social trade-offs, such as those that relate to information and pathogen transmission.

I.5. Framework and objective of study

I provide evidence that social structure can theoretically and empirically regulate information and disease transmission by mediating social contacts or spatial proximity (e.g. Aplin et al. 2012; Claidière et al. 2013; Kappeler et al. 2015). The social network properties optimizing the spreading of information may also increase the disease transmission rate, creating a trade-off between information transmission and infection risk. My research interests fit with the exploration of this trade-off by examining social network properties and investigating how they might interact to maximize information flow and minimize pathogen transmission. I work within the formal conceptual framework of studying the dynamic interactions between social structure and social transmission, with the aim of answering the following questions:

> *How do social networks shape information and infectious agent transmission in animal societies?*

In this thesis, I do not consider information solely in the light of social learning or cultural transmission (**Box I.2**), but with respect to any quality of a potential resource-holder that can be perceived by conspecifics as an indication of their likelihood to obtain beneficial information from them, including alarm calls against predator proximity, locations of new foraging patches, potential sexual partners, etc. The parasites referred to here include only

those that are or have the potential to be transmitted socially, either by direct contact or through proximity. I combined behavior sampling, social network analysis (SNA) and individual-based modelling (IBM), allowing for a resolute estimation of social structure (through SNA) and the ability to answer complex questions (through IBM) that would not be possible to address experimentally given the generation lengths of many social species and the number of behavioral scientists needed to score data at such volumes. In parallel, my research team and I also demonstrated that, while information transmission has been studied intensively in vertebrate networks (**Appendix B**, Duboscq et al. 2016a), studies of parasite transmission in networks has received less attention, especially in comparative perspective (for exceptions see Griffin & Nunn 2012; Gómez et al. 2013; Pasquaretta et al. 2014; Sah et al. 2017). During my PhD, I was also involved in experimental studies investigating parasite transmission in Japanese macaques (**Appendix C, D and E**, Duboscq et al. 2016b,c,2017), but I do not present them directly in this thesis. Instead, I focus on the observational and theoretical modelling approach I have used to understand the link between social structure and social transmission, with the ultimate aim of addressing the information/pathogen trade-off.

In this thesis, I chose primates as my main model of study since they are highly social animals, present complex social structure (Nunn & Altizer 2006), and there are vast amounts of social and behavioral information already available for this taxonomic group. It is also

worth noting that many of the studies of both information and parasite transmission on social networks have been conducted with primates (Voelkl & Nöe 2008, 2010; Walsh et al. 2009; MacIntosh et al. 2012; Claidière et al. 2013; Rushmore et al. 2013, 2014; Hobaiter et al. 2014; Pasquaretta et al. 2014; Coelho et al. 2015), making this a valuable group of organisms with which to work. Understanding the dynamic process involved in parasite transmission through primate networks might not only offer new perspectives to help us understand the variation observed in social structure across species, and its influence on disease outbreaks, but also may provide an important basis for discussion about conservation of species in light of infectious diseases and the implementation of intervention techniques (Silk et al. 2017). Furthermore, many of the hypotheses created to explain the evolution of group-living, the organizational structure of animal societies, and the links between social structure and social transmission were built through investigation of primate societies (e.g. Hinde 1976; Pusey & Packer 1987). The studies that form the components of this thesis have been conducted using association matrices from 40 wild groups of 21 primate species and published metrics of 68 networks of 21 primate species. See the section *Material and Methods* for a detailed list of species investigated.

I.6. Organizational layout

My thesis is organized into four chapters, increasing in complexity and moving from an evaluation of parasite transmission in empirical non-human primate groups to an investigation of social transmission in theoretical networks (**Figure I.5**). I first focus on understanding how individual attributes affect individual network position and how this affects transmission of theoretical pathogens in Japanese macaques – a well-studied species with data on ecology, social behavior and parasites (MacIntosh 2014) but not on epidemiology. Once the link between individual centrality and parasite transmission was established, my second chapter uses a comparative evaluation of primate societies (40 wild groups from 21 primate species) and disease outbreaks. In network epidemiology, studies have focused on the effects of network properties on the maximum reach of outbreaks. However, whether the influence of social structure on pathogen spread remains stable throughout the progression of the epidemic has not yet been considered. This dynamic approach is important since intervention strategies are being recommended in accordance with social structure (e.g. humans, Salathé & Jones 2010; chimpanzees, Rushmore et al. 2014). In the third chapter of my thesis, I extended my findings based on empirical networks by looking at the link between network properties and network efficiency (a proxy of social transmission) in theoretical networks with a larger variance in group size. In this chapter, I compared my predictions with published data on the network properties of 68 primate groups.

Finally, I finished the thesis predicting a scenario of optimal social relationships, to investigate which network properties emerge from conditions where individuals maximize the chances of getting information but minimize the risks of getting infected. I created an individual-based model with 20 different conditions and evaluated the emergent network properties. This study contributes substantially towards understanding how the trade-off between information and pathogen transmission may affect social networks.

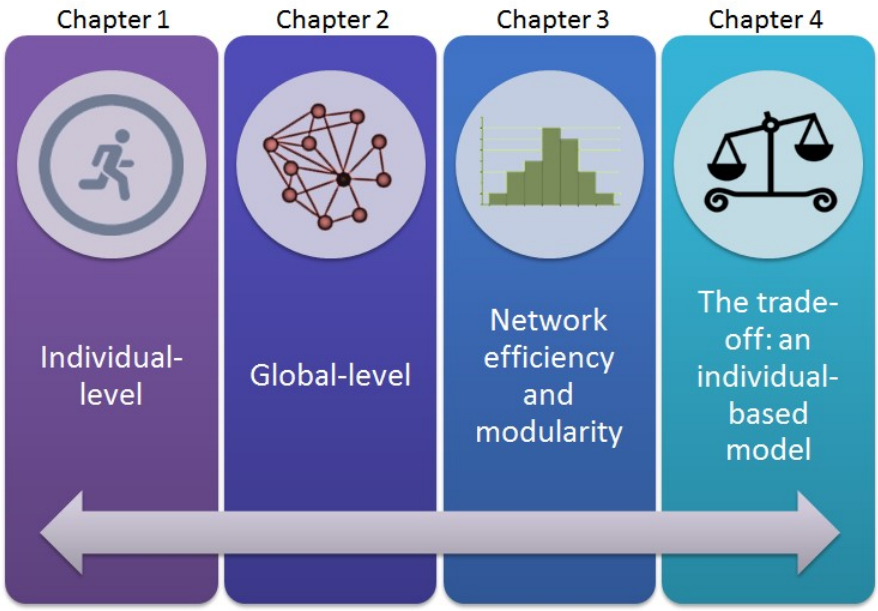


Figure I.5. Schematic representation of the structural framework developed in this thesis. Chapter 1 covers questions at the individual-level, chapter 2 at the network-level, chapter 3 presents the link between efficiency and modularity, and chapter 4 introduces the individual-based model created to investigate the trade-off between information and pathogen transmission.

Chapter 1 – Modelling infection transmission in primate networks to predict centrality-based risk

In the first study of my PhD, I addressed the question of how individual characteristics (dominance rank, family unit and age) predict individual centrality and how direct and indirect measures of individual centrality affect the chain of transmission in primate grooming and proximity networks. In network epidemiology, the importance of non-random connections in the spread of pathogen from one individual to other has been discussed at length, but fewer research has examined the vulnerability of central individuals to become infected in the chain of transmission. I combined social network analysis and an epidemiological approach to predict transmission of theoretical infectious agents in two wild groups of Japanese macaques (*Macaca fuscata*).

Chapter 2 – Pathogen spread and the dynamic social connectivity effect: an evaluation through epidemic time

In my second study, I moved from the individual metrics examined in the first chapter to an evaluation of global network properties with the aim of understanding the general rules driving outbreak size in primate networks. As infectious disease is considered one of the major threats to species survival, several studies have focused on the role of social structure

on the final size of the outbreak, but whether the influence of social structure on pathogen spread remains stable throughout the progression of the epidemic was not yet investigated. Here, I applied a comparative approach, using data from 40 wild groups of 21 non-human primate species, to investigate to what extent centralization, clustering coefficient, density, diameter and modularity interact with group size to enhance or constrain the spread of theoretical infectious agents at different stages of an epidemic.

Chapter 3 – Network efficiency peaks with intermediate levels of group substructure

In the third chapter of my thesis I focused on the relationship between transmission efficiency and modularity. Of the network properties under consideration, it has been argued that increased modularity is a major contributor to constraining transmission processes. However, research suggests that only beyond a threshold at high values of modularity do social networks result in decreased pathogen transmission. Within this framework, I created networks varying in network properties and compared my predictions with published data on the network metrics of 68 primate groups to investigate to what degree network efficiency is modularity-dependent, and whether a modularity threshold exists in the efficiency of social transmission processes.

Chapter 4 – Investigating the trade-off between information access and infection avoidance in animal societies: an individual-based model

In the last part of my PhD, I created an individual-based model, called the Optimal Relationships Model, to explore how individuals might deal with the inherent trade-off between information acquisition and pathogen avoidance in social networks. The model is programmed so that individuals favor interactions with others that exhibit higher information-sharing potential and avoid interactions with conspecifics that exhibit higher pathogen-spreading potential. I investigated the emergent network structure under conditions in which the distribution of values of information sharing potential and pathogen spreading potential varied widely across group members.

General Methods

Empirical and theoretical approaches

II. MATERIAL AND METHODS

II.1. Observational study: from field work to an extensive dataset

II.1.1. Fieldwork with Japanese macaques

Japanese macaques (*Macaca fuscata*), the northernmost extant nonhuman primate species, are endemic to Japan. It is a social primate species living in multi-female multi-male groups, where individuals form linear dominance hierarchies and differentiated affiliative social relationships. I studied Japanese macaques from Koshima island, located in Miyazaki prefecture, Japan (**Figure II.1**). Koshima is approximately 0.3km^2 in area and is mainly covered by secondary broadleaf evergreen forest (Iwamoto 1974). Koshima was at the time inhabited by approximately 100 individuals divided into two social groups, called Maki (ca. 15 individuals) and Main (ca. 60 individuals), along with an unknown number of solitary males. The main group of Koshima macaques has been periodically provisioned with wheat (currently ca. twice per week) and intensively studied, with group composition recorded for ca. 60 years (Iwamoto 1974). The Main group was habituated to the presence of humans and adults could be individually recognized by facial tattoos in combination with natural physical characteristics, such as scars or body shape. See **Figures II.2** and **II.3** for the Main group of Koshima.

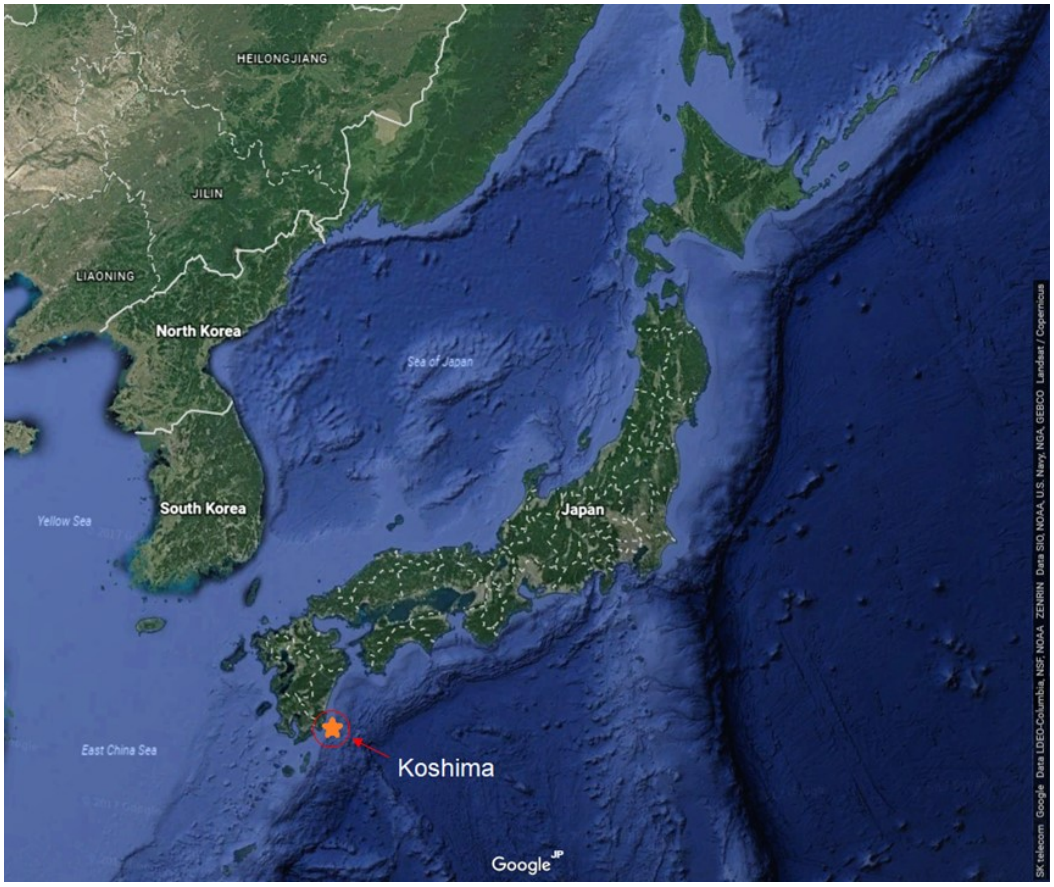


Figure II.1. Location of the field site in Japan. Koshima island is marked with an orange star. Picture extracted from Google Earth.

I collected behavioral data from 19-20 adult females (> 7 years old) of the Main group between January and August 2014. The reason I focused on female social networks in this study was that Japanese macaque societies are organized into female bonded groups in which females form the stable core (Yamagiwa & Hill 1998). Males usually emigrate from their natal groups at the sub-adult and adult age (Hamada et al. 1986). As a result, it is expected

that females dominate dynamics on social networks. Furthermore, it was difficult to adequately sample other members of the groups. For example, males are peripheral and their affiliation with the group is much less stable than that of females, while juveniles are often difficult to observe and identify reliably. Behavioral observations were balanced across females, and also by time of day (morning/afternoon).

Focal females were followed for 15 min with their main activities recorded at every minute. Amongst recorded activities, I distinguished between grooming given, grooming received, and simple body contact. Social, aggressive and other affiliative interactions as well as the identity of each social partner were collected continuously. During social and self-grooming bouts, I also counted the number of times per minute-scan the groomer conspicuously picked out something in the groomee's hair or her own and subsequently ate it. This conspicuous louse egg-picking behavior was later used to investigate the relationship between parasite burden and sociality in female Japanese macaques (**Appendix C**, Duboscq et al. 2016b), along with studies testing proximate hypotheses to explain variation in self-directed behavior, such as self-grooming and scratching (**Appendix D**, Duboscq et al. 2016c; **Appendix E**, Duboscq et al. 2017). Finally, fecal samples from adult females were opportunistically collected as part of another project I was involved with (MacIntosh et al. *in preparation*).

Figure II.2. The main beach of Koshima island with Japanese macaques foraging after a provisioning event. Photo credits: Valéria Romano.

Figure II.3. A dyad of female Japanese macaques during grooming interactions. Photo credits: Andrew J.J. MacIntosh.

II.1.2. Extending the study to another 20 primate species

Besides collecting data from Japanese macaques at Koshima, I used datasets (i.e. matrices of grooming and body contact) either shared by collaborators or from published sources (see **Table GS1, Appendix G** for the source of data). This extended my empirical dataset to a total of 40 wild groups from 21 primate species representing 4 families (Atelidae, Cebidae, Cercopithecidae, Hominidae; **Figure II.4**). Furthermore, I incorporate in my third chapter published metrics from previous social network analysis (Pasquaretta et al. 2014). This extended my analysis to 68 groups of 21 species (see detailed list at **Table HS1**, **Appendix H**).

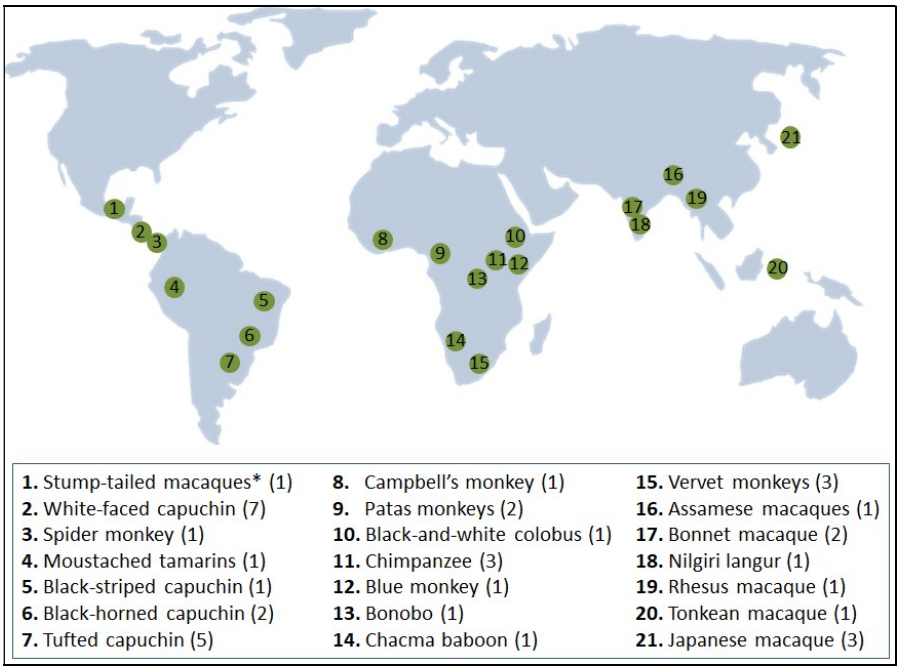


Figure II.4. Scheme representing the distribution of studied species. Data were kindly shared by collaborators or gathered from the literature (see **Table GS1, Appendix G** for the source of data). In parenthesis, the number of studied groups for each species. *The group of stump-tailed macaques was introduced to Catemaco (VeraCruz, Mexico) three years before the behavioral observation was conducted (Estrada et al. 1977).

II.2. Social network analysis

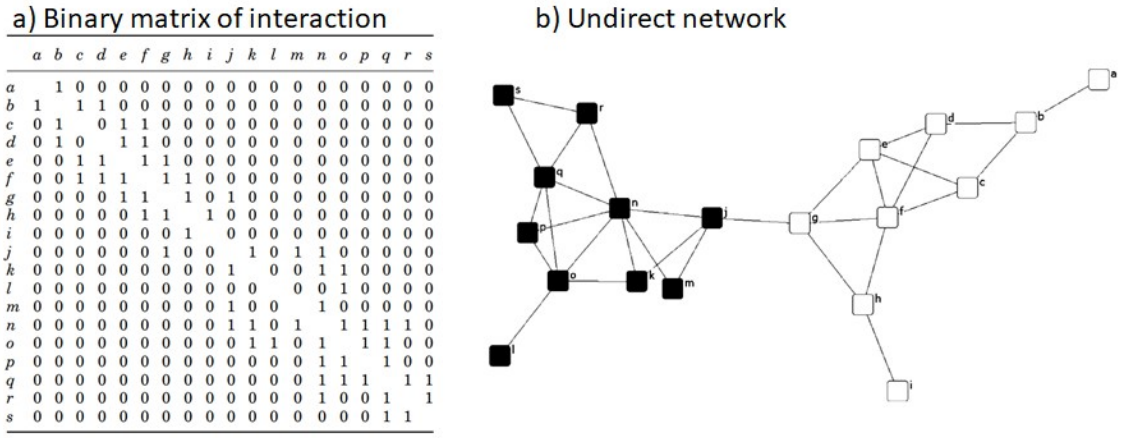
Basic Terms
Network: a set of nodes connected by links that reflect relationships.
Node: a component of the network with known relationship to others. In this thesis, it refers to individuals.
Links: the relationship (i.e. interaction or association) between a pair of individuals. They are also called edges or ties*.
Network topology: the arrangement of the nodes and the pattern of relationships between them. It is synonymous with network structure.
Centrality: a measure of a node's importance in a group based on its network position.

Social network analysis (SNA) is a toolbox providing a refined evaluation of social and ecological interactions in animal societies (Farine & Whitehead 2015). Network analysis envisages a social system as a *network*, with individuals, groups, communities or even different species being represented by nodes and their relationships represented by links (Whitehead 2008b). It provides both a visual and a mathematical analysis of social relationships. In this thesis, I will use the term "network" interchangeably with "group", "node" with "individual" and "link" with "relationship" or "edge".

II.2.1. Building social networks

An adjacency matrix is the classical representation of social association or interaction data (**Figure II.5a**). A matrix contains rows and columns denoting specific nodes (e.g. individuals, groups or communities, or different species). The data contained in the cells of the matrices reflect a representation of the relationships between the nodes. A network can be either directed or undirected, with the actor and target nodes being recorded or not,

respectively (**Figure II.5b**). Whether a network is directed or undirected depends on the type of links represented (interactions or associations). Undirected networks are usually typical of association data (e.g. proximity, body contact), in which the observer will score the time or occasions individual *i* and *j* spent together (with a spatio-temporal criterion; Sueur et al. 2011a). An interaction denotes direction, most usually, and it can refer to affiliative (e.g. individual A grooms individual B) or aggressive behaviors (e.g. individual B chases individual A), if individuals are placed as nodes in the networks. Networks can also be weighted and unweighted. When the duration and/or frequency of a given social relationship can be recorded, such as the time of grooming or the frequency individuals are in body contact, the resulting network is weighted. When building weighted networks, researchers usually normalize the links allowing for comparisons between nodes (Sueur et al. 2011a).

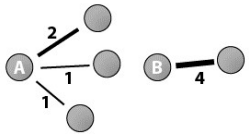
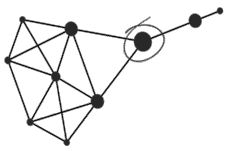
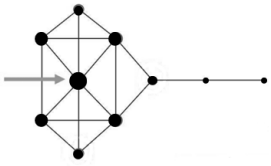


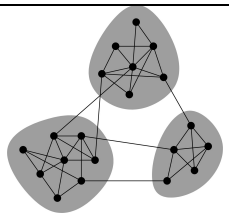
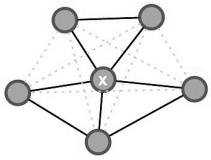
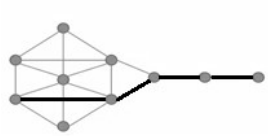
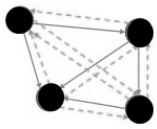
	a	b	c	d	e	f	g	h	i	j	k	l	m	n	o	p	q	r	s
a		1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
b	1		1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
c	0	1		0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
d	0	1	0		1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
e	0	0	1	1		1	1	0	0	0	0	0	0	0	0	0	0	0	0
f	0	0	1	1	1		1	1	0	0	0	0	0	0	0	0	0	0	0
g	0	0	0	0	1	1		1	0	1	0	0	0	0	0	0	0	0	0
h	0	0	0	0	0	1	1		1	0	0	0	0	0	0	0	0	0	0
i	0	0	0	0	0	0	0	1		0	0	0	0	0	0	0	0	0	0
j	0	0	0	0	0	0	1	0	0		1	0	1	1	0	0	0	0	0
k	0	0	0	0	0	0	0	0	0	1		0	0	1	1	0	0	0	0
l	0	0	0	0	0	0	0	0	0	0	0		0	0	1	0	0	0	0
m	0	0	0	0	0	0	0	0	0	1	0	0		1	0	0	0	0	0
n	0	0	0	0	0	0	0	0	0	1	1	0	1		1	1	1	1	0
o	0	0	0	0	0	0	0	0	0	0	1	1	0	1		1	1	0	0
p	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1		1	0	0
q	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1		1	1
r	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1		1
s	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	

Figure II.5. Representation of a binary matrix of interactions (a) and the graphical representation of a theoretical social network (b). Nodes represent males (black nodes) and females (white nodes) of a group. Links (lines) represent the interaction or association between two individuals. Figures from Sueur et al. 2011a.

II.2.2. Interpreting network metrics

Network metrics are statistical measures used to characterize properties of the network at individual (nodes) or global levels (the whole network). Another group of measures are edge metrics (e.g. edge betweenness), but they are rarely applied to animal social networks (Farine & Whitehead 2015) and thus are not covered in this thesis. In **Table II.1,** I provide definitions and the meaning of the more used network properties in the context of animal societies (Whitehead 2008a; Wey et al. 2008; Sueur et al. 2011a; Farine & Whitehead 2015). There is an extensive literature providing detailed explanations of many more network metrics (Newman 2010, Scott 2017). In the methods section of each chapter of my thesis, I re-visit the definition of individual and global properties, placing them according to each hypothesis under study. Network metrics can be calculated in network analysis programs, such as SocProg (Whitehead 2009) and Ucinet (Borgatti et al. 2002), or by using libraries in R (R Core Team 2016) such as igraph (Csárdi & Nepusz 2006), SNA (Butts 2008), tnet (Opsahl 2009) and ASNIPE (Farine 2013), which provide almost all network algorithms available. Because it is important to visualize the structure of networks to interpret the different roles of each metric, software packages such as Gephi (Cherven 2013) and Ucinet (Borgatti et al. 2002), or libraries in R such as igraph (Csárdi & Nepusz 2006) are also recommended.

Table II.1. Definition of network properties and their meaning in the context of animal societies.		
Network property	**Definition ***	**Biological meaning ***
Individual-level		
Degree 	The number of edges connected to the individual.	Represents the gregariousness of individuals, in terms of the total number of interaction partners or associates.
Strength 	The sum of the edge weights connected to each individual.	Represents the expected total interaction or association quantity per individual.
Betweenness 	The number of shortest paths that pass through the considered individual.	Indicates how important an animal is as a point of social connection and transfer. Animals with high betweenness are expected to bring stability to the network. Their removal may cause fragmentation of the network into smaller subgroups.
Eigenvector 	The weighted connectivity of an individual within its network, also considering the weighted connectivity of its neighbors.	Captures the potential of neighbors in determining centrality in the network. It is important for weighted networks and association data.
Global-level		
Eigenvector centralization 	Derived from individual eigenvector centrality, it estimates variation in connectedness across nodes in the network.	Denotes to what extent one or a few individuals monopolize(s) the social relationships in a network. It might range from 0 to 100, with values close to 100 denoting a network centralized around one individual (e.g. star network).
Modularity	The extent to which a network is subdivided into subgroups.	Denotes the set of individuals which interact or associate more frequently between each other than with individuals

		from other subgroups.
Overall clustering coefficient 	The mean of all nodes' clustering coefficients, which measures how densely one individual is connected within its neighborhood.	Describes how densely (or sparsely) the network is clustered around individuals.
Diameter 	The longest path edge of the network.	A basic measure of how well-connected is the network. Usually, individuals in a group with a smaller diameter are connected to each other through fewer intermediates.
Density 	The ratio between the number of observed edges and the number of possible edges in the network.	Represents the cohesion of all nodes in a network. A high dense network contains all possible relationships among individuals.

* Definition and biological meaning were based on Newman 2004, Hanneman & Riddle 2005, Newman 2006; Sueur et al. 2011a, Borgatti et al. 2013, Wey et al. 2013, Farine & Whitehead 2015.

II.3. Theoretical modelling

II.3.1. Individual-based model

In many disciplines, scientists face the challenges of understanding the inherent complexity of many ecological and sociological systems. Individual-based models (IBM), also called Agent-based models, is a class of computational models that provide a purposeful representation of real systems (Railsback & Grimm 2012). An IBM simulates the actions and

interactions of autonomous agents (which also includes collective entities such as groups and organizations), with the ultimate goal of assessing their effects on the system as a whole (Grimm et al. 2005; Railsback & Grimm 2012). An agent can be a social animal, a tree, a company, etc. Each agent is endowed with state variables, attributes or behaviors that modulate the pattern of interactions in a given environment. Behaviors can include reproduction, habitat selection, dispersal, etc. Modelling allow to change values of parameters and test per thousands of times the set of target values and conditions, which cannot be done in real situations. Advantages of individual-based models over traditional models include: i) IBM are bottom-up models providing an evaluation of how individual interactions affect the emergence of group or population structure; ii) they can incorporate multiple numbers of individual-level mechanisms, allowing a significant increase in the complexity of the artificial system (DeAngelis & Grimm 2014); and, iii) agent attributes and interactions are recorded through time, which allows for a refined evaluation of the simulated system. For roughly 40 years, individual-based models have been applied in ecology, with the first generation and some of the subsequent models being designed for the field of forest management (e.g. **Figure II.6**; DeAngelis & Grimm 2014).

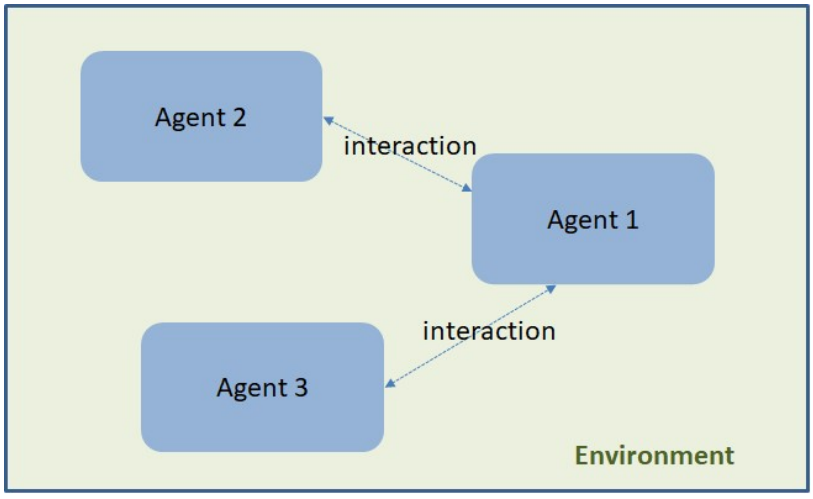


Figure II.6. Diagram representing the components of an individual-based model: agent, environment and links (i.e. interactions among agents). An agent represents a discrete entity with its own goals and behavior interacting and living in an environment.

Let me take as example the BEFORE model, designed to reconstruct the spatiotemporal dynamics of the natural beech (*Fagus silvatica*) forests of central Europe (Rademacher et al. 2004; Grimm et al. 2005, **Figure II.7**). Multiple patterns are expected to characterize the mosaic of successional stages in these forests, including patterns of vertical structure: the climax stage has closed canopy while decaying stages are characterized by canopy gaps. Yet, since in many places there are no longer beech forests, the structure and dynamics of these forests cannot be experimentally analyzed and compared with managed forests (Rademacher et al. 2004). In simple terms, the individual-based model then included a representation of agents (trees), with their state variables (e.g. size) in an environment representing the location of each tree (Rademacher et al. 2004). To increase the complexity of the model and to fit with the observed patterns, the model structure also included multiple characteristic patterns, such as the mosaic pattern determined by horizontal spatial scale and resolution, the vertical patterns determined by the need for height classes, and canopy gaps determined that large beeches must be described individually (Grimm et al. 2005). The BEFORE model predicted that natural beech forests show considerable fluctuations in forest structure, which indicates quasi-stationary dynamics (Rademacher et al. 2004).

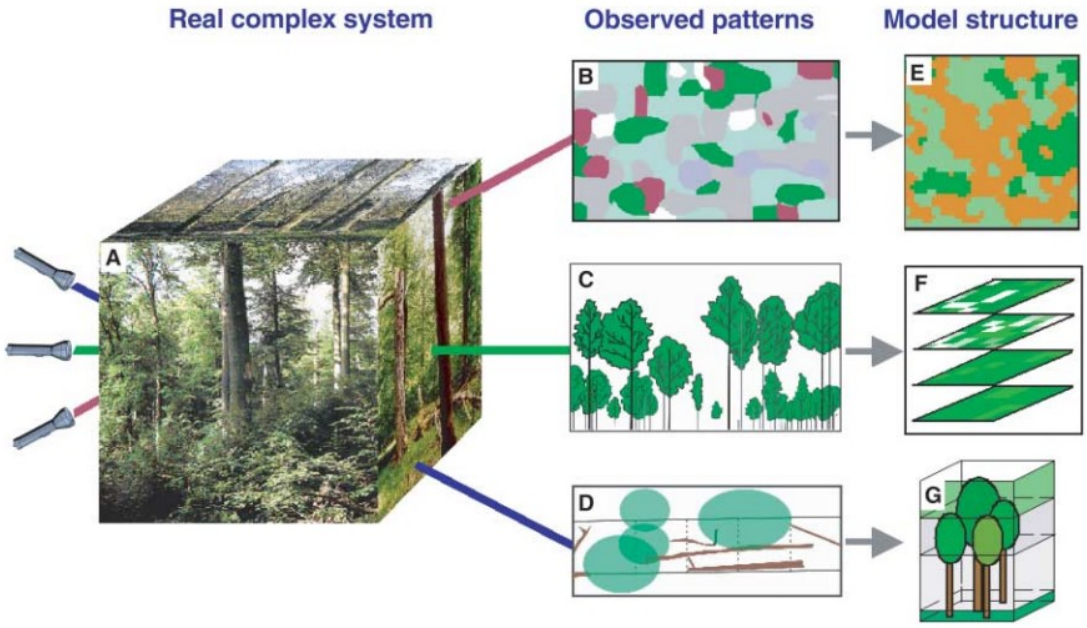


Figure II.7. Example of a model design. The BEFORE model is designed to simulate the spatiotemporal dynamics of beech forests of central Europe. From the real complex system (a), observations indicate multiple patterns of influence (a horizontal mosaic of developmental stages (b); the vertical patterns of tree size representing the developmental stages (c) and distributions of fallen large trees (d)) that drives the model structure (a grid-based horizontal structure (e), a grid-based vertical structure (f), and individual representation of large trees (g). Figure from Grimm et al. 2005.

II.3.2. Network epidemiology

Epidemiology consists of the study of the distribution and determinants of disease in a population (MacMahon & Pugh 1970). Classical epidemiological models have traditionally considered an equal probability of contact for each pair of individuals in a given population, which assumed that each individual was equally likely to acquire infectious agents from any other infected individual (Rothman et al. 2008). However, heterogeneity in spatial and social structure creates distinct routes of transmission (Bonnell et al. 2016; Silk et al. 2017; White et al. 2017). The network epidemiology field then emerged as a powerful tool to provide more realistic scenarios of disease spread (Pastor-Satorras & Vespignani 2001). In network epidemiology, an infectious agent spreads from individual to individual following the links

(i.e. social relationships) connecting them, such as those that represent the time spent in body contact, grooming, etc. If the transmission probability among individuals is high enough, an epidemic will occur (Craft & Caillaud 2011). The original studied in network epidemiology therefore takes advantage of the analytical set of tools created by classical epidemiological models and includes a new component related to the number of connections (i.e. degree) of each individual as an implicit variable (Pastor-Satorras & Vespignani 2001; Barabási 2016).

II.3.2.a. Susceptible-Infected (SI) Model

In a SI model, an individual can be in one of two states: susceptible (S) or infected (I; **Figure II.8a**). Susceptible denotes the number of individuals who are healthy at time (t) and Infected the number of individuals who already have been infected at time (t) (Barabási 2016). This model assumes that if a susceptible individual comes into a contact with an infected individual, it will become infected at rate β, the transmission rate, depending on the fraction of *degree-k* nodes that remains uninfected. After an individual is infected, it remains in this state, meaning that it does not recover or die, as would occur in an SIR model, R representing either removed or recovered individuals (Barabási 2016). At time (t) = 0, all individuals are susceptible and none are infected, but after few acquire a socially-transmissible pathogen, the proportion of infected individuals increases exponentially. As most of the infected individuals eventually meet fewer and fewer susceptible individuals, the fraction of infected individuals slows for large (t) (**Figure II.8b**). The rate is dependent on the values of social connectivity (k). The higher the degree of an individual, the more likely it will be infected (**Figure II.9**; Barabási 2016). A deeper explanation of the network epidemiological model used in this thesis is placed in the method sections of chapters 1 and 2.

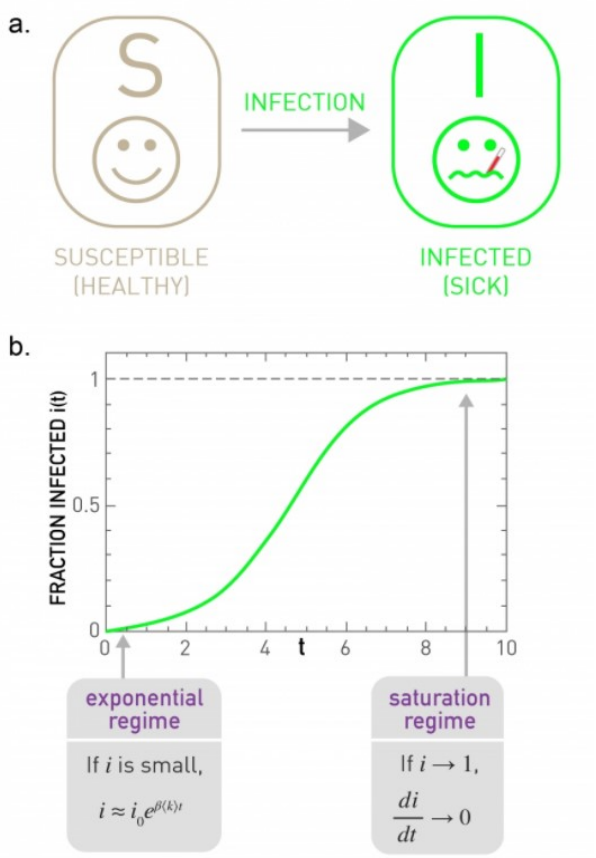


Figure II.8. Schematic representation of the SI epidemiological model. The model is organized into two compartments: susceptible (S) and infected (I) individuals. The arrow is undirected, indicating that once an individual becomes infected, it stays infected and thus does not recover or die. Figure from Barabási 2016.

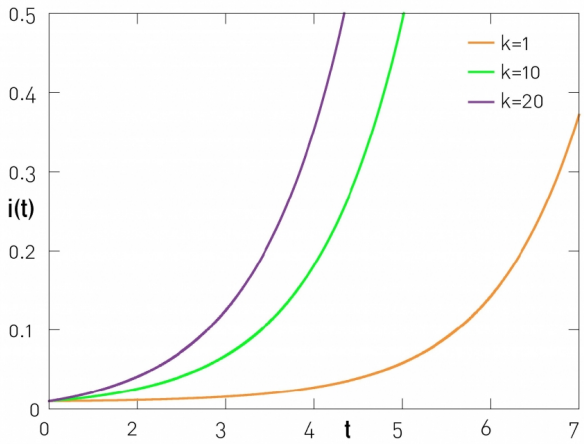


Figure II.9. Proportion of infected individuals (i(*t*)) in an SI model distributed according to values of degree from individuals of a hypothetical network. Degrees are equivalent to 1, 10 and 20 from a scale-free network with average degree (k) = 2. The higher the degree of the nodes, the faster the spread of pathogens among group members. At t = 3, less than 3% of the k = 1 nodes are infected. Contrary to k = 10, from which about 20% has already become infected. Figure from Barabási 2016.

Chapter 1

Individual centrality and pathogen transmission

III. CHAPTER 1:

Modeling infection transmission in primate networks to predict centrality-based risk

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III.1. Abstract

(English) Social structure can theoretically regulate disease risk by mediating exposure to pathogens via social proximity and contact. Investigating the role of central individuals within a network may help predict infectious agent transmission as well as implement disease control strategies, but little is known about such dynamics in real primate networks. We combined social network analysis and a modeling approach to better understand transmission of a theoretical infectious agent in wild Japanese macaques, highly social animals which form extended but highly differentiated social networks. We collected focal data from adult females living on the islands of Koshima and Yakushima, Japan. Individual identities as well as grooming networks were included in a Markov graph-based simulation. In this model, the

probability that an individual will transmit an infectious agent depends on the strength of its relationships with other group members. Similarly, its probability of being infected depends on its relationships with already infected group members. We correlated: (i) the percentage of subjects infected during a latency constrained epidemic; (ii) the mean latency to complete transmission; (iii) the probability that an individual is infected first among all group members; and (iv) each individual's mean rank in the chain of transmission with different individual network centralities (eigenvector, strength, betweenness). Our results support the hypothesis that more central individuals transmit infections in a shorter amount of time and to more subjects but also become infected more quickly than less central individuals. However, we also observed that the spread of infectious agents on the Yakushima network did not always differ from expectations of spread on random networks. Generalizations about the importance of observed social networks in pathogen flow should thus be made with caution, since individual characteristics in some real-world networks appear less relevant than they are in others in predicting disease spread.

Keywords: social relationship; wildlife epidemiology; agent-based model; social network analysis

(Français) La structure sociale peut en théorie le risque d'infection en modérant l'exposition au pathogène via la proximité sociale et le contact entre individus. L'étude du rôle des individus centraux au sein d'un réseau pourrait aider à prédire la transmission d'agents infectieux mais également la mise en œuvre de stratégies de contrôle des maladies. Cependant, nous avons actuellement très peu d'informations sur le rôle des individus centraux dans la dynamique de transmission des maladies au sein de groupes de primates. Nous avons donc ici combiné l'analyse de réseaux sociaux et de la modélisation afin de mieux comprendre la transmission d'agents infectieux théoriques au sein d'un groupe de macaque japonais en milieu naturel. Cette espèce est connue pour être très social, formant des

réseaux sociaux étendus mais très différents. Nous avons donc collecté des observations comportementales de macaques japonais femelles et adultes vivant sur les îles de Koshima et Yakushima, au Japon. L'identité des individus et les réseaux de toilettages ont été inclus dans une simulation de Markov. Dans ce modèle, la probabilité qu'un individu a de transmettre un agent infectieux dépend de la force des relations avec les autres membres du groupe. De façon similaire, la probabilité d'un individu à être infectée va dépendre de ces relations avec des individus du groupe déjà infectés. Nous avons donc corrélé : (i) le pourcentage d'individus infectés durant une épidémie dont la latence a été contrainte ; (ii) la latence moyenne de la transmission complète de l'agent infectieux ; (iii) la probabilité qu'un individu d'être infecté le premier au sein du groupe ; et (iv) le rang moyen de chaque individu dans la chaîne de transmission ayant différents propriétés de centralité de réseaux (eigenvector, strenght, betweenness). Nos résultats supportent ainsi l'hypothèse que les individus les plus centraux transmettent les infections plus rapidement et à plus d'individus, mais également que ces individus plus centraux se retrouvent infectés plus rapidement que les individus moins centraux. Cependant, nous avons également observé que la dissémination des agents infectieux au sein du réseau de Yakushima ne diffère pas toujours des prédictions au sujet de la dissémination au sein de réseaux aléatoires. La généralisation au sujet de l'importance des réseaux sociaux observés sur la dissémination des pathogènes doit ainsi être faite avec précaution, pour la bonne raison que les caractéristiques individuelles au sein de réseaux réelles apparaissent être moins pertinentes que dans des réseaux simulés.

Mots-clés: relation social; épidemiologie en milieu naturel; modèle multi-agents; analyse de réseaux sociaux

III.2. Introduction

In a social group, each individual is part of a network that varies in size, distribution and dynamics of relationships. Observed interactions between social animals are the outcome of trade-offs between the costs and benefits of sociality (Krause & Ruxton 2002) and one clear cost of being social is that many pathogens are transmitted via social interactions (Corner et al. 2003; Otterstatter & Thomson 2007; Drewe et al. 2011). Heterogeneity in host associations, for example, may influence the flow of disease-causing organisms through populations and mediate the risk of contagion across individuals (Newman 2002; Nunn & Altizer 2006). In consequence, there has been increased effort to investigate how association patterns and social positions of each individual in a network can affect disease transmission, via experimentation and/or modeling, in a vast range of species (humans: Bansal et al. 2007; Salathé & Jones 2010; non-human primates: Griffin & Nunn 2012, Carne et al. 2014; ungulates: VanderWaal et al. 2014a,b; cetaceans: Böhm et al. 2009; reptiles: Godfrey et al., 2009, Aiello et al. 2014). While the networking approach is appealing for its capacity to depict complex systems (Kurvers et al. 2014), modeling offers further utility for understanding and predicting the behavior of these systems (Newman 2003). Agent-based modeling, for example, represents individuals as unique entities in the environment; by simulating local interactions among agents and their environment, it offers a less simplified and thus more realistic representation of real systems (Amouroux et al. 2010, Railsback & Grimm 2012). For these reasons, combining network techniques with modeling has emerged as a powerful tool for examining dynamics of infectious diseases (Craft et al. 2010).

In this context, recent models exploring association patterns have shown that global network properties, such as modularity (Griffin & Nunn 2012; Nunn et al. 2015), as well as individual-level properties, such as node centrality (Salathé & Jones 2010; Rushmore et al.

2014; VanderWaal et al. 2014a), may regulate pathogen transmission. For instance, the way in which individuals are more or less central in a group's social network directly influences the way in which an infectious agent or information will be spread through a group and as a consequence, the relationship between centrality and probability of transmission (Griffin & Nunn 2012; Sueur et al. 2012). Central individuals may act as super-spreaders of disease-causing agents, and targeting them (e.g. during vaccination or culling efforts) can therefore be an efficient way to implement preventive measures against disease (Christley et al. 2005; Rushmore et al. 2014). However, similar studies on information transmission have shown contrasting results, with a demonstrated influence of social centralities and network structure in some groups but an absence thereof in others (Boogert et al. 2008; Kendal et al. 2010; Schnoell & Fichtel 2012). In this way, it seems crucial to understand how social network structure at both the global and individual levels might interact to predict transmission within a group.

Non-human primates are useful study subjects to investigate the influence of sociality on disease transmission. First, many species are obligate social animals. Second, their close phylogenetic relationship with humans means that many non-human primate diseases are also a concern for humans (Wolfe et al. 1998, Hahn et al. 2000). Likewise, human diseases are a concern for nonhuman primates, for example causing marked morbidity in apes through infection linked to tourism or research activities (Woodford et al. 2002, Köndgen et al. 2008). Furthermore, understanding transmission dynamics in primates is critical for development of conservation and management strategies, given that ca. 50% of primate taxa are now under threat of extinction (Mittermeier et al. 2009) and infectious disease is known to be a significant driver of population decline (Leendertz et al. 2006). Therefore, increasing fundamental understanding of how sociodemographic factors might interact with disease transmission among primates is now critical, particularly to predict how continued human

encroachment and habitat modification might impact primate health, fitness and population viabilities in the future (Chapman et al. 2005). Studies have thus begun to investigate the variable influence of specific individuals and the contexts in which they interact in the dynamics of disease spread using real world primate networks (Griffin & Nunn 2012, Carne et al. 2013, Rushmore et al. 2013, Carne et al. 2014, Rushmore et al. 2014).

In this study, we combine social network analysis of empirical data and agent-based modeling to investigate the theoretical relationship between network properties and the propagation of infectious agents. We focus on infectious agents transmissible through social contact networks in Japanese macaques, which provide a well-studied and thus tractable model system (MacIntosh 2014). Macaques are generally considered the most widely distributed and best studied group of non-human primates (Thierry et al. 2004, Thierry 2007), and in many parts of their range exist in extreme proximity to human settlements. There is also some empirical evidence that infection by nematode parasites in Japanese macaques specifically is related to network centrality and position within the dominance hierarchy (MacIntosh et al. 2012, MacIntosh 2014). However, many of the epidemiological processes involved, particularly those concerning other groups of socially-transmissible agents in these and other macaque species, remain poorly understood.

To test our hypothesis that the structure of social contact networks mediates the transmission of infectious agents, we first constructed networks of grooming, a very conspicuous behavior that provides a good approximation of social contact (Altizer et al. 2003) and which represents about 90% of body contact between female Japanese macaques (Duboscq et al. 2016). We then tested whether the spread of a theoretical infectious agent on these observed networks differed from its spread on random networks with the same number of individuals and degree distribution. Second, to further understand the role of individuals in transmission dynamics, we tested whether individual traits such as age, rank and family size

affected an individual's network position. Recent studies have suggested that such traits can be used as proxies of centrality and thus used to predict importance in disease dynamics (Rushmore et al. 2013, 2014). We then constructed an agent-based model to simulate the transmission of an infectious agent through the observed empirical contact networks. Based on the hypothesis that social network centrality and transmission dynamics are linked, we predicted first that central individuals in the contact network would transmit disease faster and to more individuals than less central individuals. To test this prediction, we modelled (i) the percentage of individuals infected before a latency threshold of transmission was reached, which should be higher when starting with more central individuals, and (ii) the latency between initial infection and the point at which the whole group became infected, which should be lower when starting with more central individuals. Second, we predicted that central individuals would also be at greater risk of being infected, which we estimated via (iii) the probability that an individual would be infected first among all group members, which should be higher for central individuals, and (iv) each individual's mean rank in the chain of transmission, which should be lower among more central individuals. This dual approach of social network analysis and simulation modeling allowed us to ascertain the importance of central individuals in disease transmission, not only as key agents of disease spread but also as those that are most vulnerable to being infected.

III.3. Methods

The research presented here complied with the Guidelines for the Care and Use of Nonhuman Primates established by the Primate Research Institute of Kyoto University, to the legal requirements of Japan and to the American Society of Primatologists (ASP) Principles for the Ethical Treatment of Non-Human Primates.

III.3.1. Study site and subjects

We studied two well-habituated groups of Japanese macaques, one provisioned but free-ranging on Koshima island (31°27'N, 131°22'E; *Macaca fuscata fuscata*) and the other wild (i.e. not provisioned) on Yakushima island (30°20'N, 130°30'E; *Macaca fuscata yakui*). Koshima is approximately 0.3km² in area and is mainly covered by secondary broad-leaved evergreen forest (Iwamoto 1974). The main group of Koshima macaques has been periodically provisioned with wheat (currently ca. twice per week) and intensively studied, with group composition recorded for ca. 60 years (Iwamoto 1974). During the study period (see below), the group included approximately 51 individuals, including 21-24 adult females (≥5 yo), 11-16 adult males (≥5 yo), 11-18 juveniles (1–4 yo), 1 infant (<1 yo) born in 2012.

The southernmost population of Japanese macaques living on Yakushima represents a distinct subspecies from those in the rest of Japan. Yakushima is a mountainous island of approximately 500km², much of which is protected as a UNESCO World Natural Heritage site and by the Kagoshima prefectural government. The study group ("Umi") inhabited the protected western coastal forest, which like Koshima is dominated by broad-leaved evergreen secondary forest, with an estimated home range size of roughly 0.8km² (Sueur et al. 2013). Umi group varied between 59 and 70 individuals during the study period, including 18 adult females, 11-15 adult males, and 20-31 juveniles, with 11 infants born in 2008 and 6 infants born in 2009 (MacIntosh et al. 2012). Ages of individuals were estimated based on body size and state of development of sexual organs and perianal regions.

III.3.2. Behavioral data collection and networks

We collected data on grooming interactions (both directions, received and given) of adult females over 8 months (between October 2012 – May 2013) in Koshima (N = 21) and 16 months (between October 2007 – August 2009) in Yakushima (N = 18). We focused on female social networks in this study. Japanese macaque societies are organized into female-bonded groups in which females form the stable core (Yamagiwa & Hill 1998). As a result, we expect females to dominate dynamics on social networks. It was also difficult to adequately sample other members of the groups, e.g. because male group affiliation is much less stable than that of females while juveniles are often difficult to observe and identify reliably. Both groups were habituated to the presence of human observers and adults could be identified using tattoos (Koshima only) and/or other individual traits. We used grooming networks because they are considered to be an excellent proxy of social contacts, and in addition can avoid issues arising from the gambit of the group (Franks et al. 2010). This concept underlies that all individuals seen grouping together, during an observation census, are associating with every other individual in that group. For example, if individual A is strongly associated with B and B is strongly associated with C, the gambit of the group assumes that A and C are strongly associated too. This can result in overestimation of real associations resulting in errors in estimating the disease transmission process. This overestimation is not observed when using body contact, especially grooming, between individuals. Previous studies have also shown no differences in transmission processes using either body contact or grooming interactions as the basis for network construction (Pasquaretta et al. 2014), but we focus on grooming to investigate infectious agents only transmissible trough social contact.

To collect data, we conducted 15-min instantaneous focal sampling at 1-minute intervals on Koshima, while all grooming activity performed during 60-min focal samples were recorded on Yakushima (Altmann 1974). To confirm compatibility in the data sets, which were collected using different sampling methods, we transformed the continuous-time grooming matrix constructed from Yakushima data into an instantaneous scan matrix after sampling the focal data at 1-min intervals. We observed that the matrices were 99.6% correlated (Mantel Z test: $p = 9.99^{e-13}$), showing that less than 0.5% of the data were lost when moving from one method to the other. We are thus confident that the Koshima and Yakushima networks are comparable. At both sites, we avoided re-sampling the same individual within a day wherever possible. When this was violated, individuals were not observed within 1h of a previous focal sample from the same individual. From the Koshima data set, we extracted minute-data points of grooming while for Yakushima we considered the total grooming time between two individuals. There was no difference in hourly observation time between individuals on either Koshima group (mean±SD: 12.96±0.50, $X^2 = 8.05$, $p = 0.99$) or Yakushima group (mean±SD: 45.61±0.81, $X^2 = 4.39$, $p = 0.99$), and the grooming frequencies were almost identical at both sites (Koshima: grooming given = 13.4%±6.3%; grooming received = 7.3%±3.6%; Yakushima: grooming given = 12.6%±3.6%; grooming received = 6.7%±1.5%; Yakushima data from MacIntosh et al. (2012)). Although there were differences in the total observation time between the Yakushima and Koshima groups, we believe the data set to be large enough in each group to decrease expected errors in social network measures (Whitehead 2008b). However, because of the differences in data collection and despite the high correspondence between methods using the Yakushima data, we remain cautious and make no direct comparisons between the two groups. Instead, we focus on the observed transmission events within each separate network based on grooming

behavior, a well-conserved and highly conspicuous behavior unlikely to differ substantially between groups and thus bias our results.

From the undirected and weighted grooming networks, we estimated the global measures of social networks, defined as follows:

- density: the ratio between the number of observed edges and the number of possible edges in the network (Sueur et al. 2011a);

- diameter: the longest path edge of the network;

- overall clustering coefficient: the mean of all nodes' clustering coefficients, which measures how densely one individual is connected to its neighbourhood (Hanneman & Riddle 2005);

- average degree: the average of sum of the number of edges of a vertex;

- network modularity: the extent of sub-grouping in a network (Newman 2004);

- transitivity: the circumstance where node *i* is connected to node *j,* node *j* is tied to node *k* and node *i* is also tied to node *k* (Hanneman & Riddle 2005).

We also calculated various weighted individual-level measures which are typically referred to as centrality coefficients to compare the roles of individuals in the transmission of infectious agents. These coefficients included:

- strength: the sum of each node's edge values. The individual with the most and strongest connections has the highest strength value (Sueur et al. 2011a). In our study, we have considered two different inputs. For Yakushima, strength indicates grooming time between individuals, while for Koshima strength indicates the number of times 2 individuals were observed to groom each other during sampling points.

- eigenvector: the weighted connectivity of an individual within its network, also considering the weighted connectivity of its neighbors. Individuals tied to central individuals (i.e., those with a high connectivity themselves) should have higher centrality than those connected to less central individuals (Borgatti et al. 2013);

- betweenness: the number of shortest paths that pass through the considered individual. The more connections that are made through one individual, the greater its value of betweenness becomes (Newman 2004, Hanneman & Riddle 2005).

Most of the global measures of social networks (density, diameter, overall clustering coefficient and transitivity) as well as the betweenness centrality coefficients were estimated using Ucinet 6.4 (Borgatti et al. 2002). Other network measures such as modularity, eigenvector and strength centrality were estimated via SocProg 2.4 (Whitehead 2009). Since we built our networks based on the weighted matrices, we estimated the weighted measure of each coefficient. The grooming networks were visualized using Gephi 0.8.2 beta (Cherven 2013).

III.3.3. Individual and social traits associated with network centrality

We categorized Japanese macaques by age, hierarchical rank and family size (the latter for Koshima only). Because Koshima group has been monitored for decades (Iwamoto 1974), exact ages are known for each individual. Such data were not available for Yakushima, so we instead distributed the sexually-mature females into three age classes (young adult ≥ 5 <10 yo, adult 10 – 14 yo, old adult >14) following MacIntosh et al. (2012). We also incorporated dominance ranks into our analysis, which were distributed within significantly linear

dominance hierarchies in both groups (Landau's linearity index corrected for unknown relationships: Koshima: h' = 0.68, $p < 0.001$; Yakushima: h' = 0.40, $p = 0.005$). Finally, family size was based on the definition of Rushmore et al. (2013), with a family unit including a mother and the total number of her living weaned offspring. An individual with no offspring, for instance, was counted as having a family size of 1 (Rushmore et al. 2013).

III.3.4. Random networks

To identify if the dynamics of disease transmission (i. percentage of infected individuals; ii. latency to complete transmission; iii. probability of acquiring an infectious agent; iv. latency of acquiring an infectious agent) in the Koshima and Yakushima networks differed from those in random networks, we compared the distributions of infectious agent transmission from the simulation on both random and observed networks. We created 100 random networks for each focal group, maintaining the original numbers of both individuals and observed bonds between them, via Ucinet 6.4 (Borgatti et al. 2002). Computer simulations were run 1000 times for each random network.

III.3.5. Disease transmission graph-based model

Individual identities as well as grooming interactions were included in an individual-based model using a Markov chain process developed by Sueur et al. (2009). This model is basically equivalent to the classical SI epidemiological model, in which individuals can only move from susceptible (S) to infected (I) classes with no possible recovery or death of an infected individual. Traditionally, such models considered the probability of contact for each pair of individuals to be equal, but we allowed for heterogeneity of relationships, which has

long been declared to improve the ability to understand and predict disease dynamics (Keeling & Eames 2005). To fit with the current model, grooming relationships were transformed following the description of Sueur & Deneubourg (2011). Observed frequencies were first transformed to relative frequencies (i.e. divided by the sum of observed frequencies) and then multiplied by N - 1, N being the number of group members, to obtain corrected frequencies implemented in the model. In the model, the probability that an individual will transmit an infectious agent to another depends on the strength of the relationships it has with each non-infected individual. In the same way, the probability that an individual will become infected depends on its relationships with already infected group members. Thus, the more a non-infected individual interacts with infected individuals, the greater is its probability of being infected. Likewise, the more an infected individual interacts with non-infected group members, the greater is its probability of infecting others.

In the model, each individual has the same probability of being the first infected, meaning that we focus only on the exposure to pathogens and assume a constant susceptibility to infection across individuals. This probability is named intrinsic probability λ. It is important to highlight that this lambda could be noted λ_i but as all individuals have the same intrinsic probability, we indicate λ. An individual i has to be infected to see its probability λ equal to 0, but if an individual k is infected and i is not yet infected, the probability λ is no longer 0. Thus, as soon as one individual is infected, the probability ψi for another individual i to be infected is:

$$\psi_i = \lambda + C \sum_{k=1}^{N} r(k,i) \qquad \text{With } C / \lambda = R_0 = 10,$$

where N denotes the group size, r(k,i) represents the social relationship that individual i has with individual k, and C is a mimetic coefficient, which means that the probability of being

infected is increased by a coefficient C following contact with conspecifics. R_0 is the basic reproduction number used to quantify the transmission potential of a disease; using $R_0 = 10$ allowed us to estimate properties of an outbreak with a highly contagious pathogen, such as was estimated for measles in humans and subsequently extrapolated for heuristic purposes to chimpanzees (Rushmore et al. 2014). While R_0 is known to differ dramatically across disease-causing organisms, using a high R_0 allows us more power to identify the influence of network structure in empirical data with small sample sizes. However, we also simulated the transmission of infectious agents with varying R_0 and found that their dynamics are consistent in both study groups, even though the total size of the outbreak varies (**Figure FS1, Appendix F**).

We implemented the model in Netlogo 3.1.5 (Railsback & Grimm 2012). At each run of the model, a number between zero and one was randomly attributed to each non-infected individual; when this number was lower than the theoretical infection probability ψi of each individual i, the individual i became infected; if this number was higher than the theoretical infection probability, the individual was not infected. The identity, infection rank (order of infection of individuals) and latency (number of runs) of infection for each individual were scored for each simulation. We induced two conditions, first considering the complete transmission latency and second restricting the latency of infection to three simulation runs. The first condition allowed us to investigate the period required to complete infection and the order of infection. One simulation corresponds to the infection of all group members. The total infection latency of a simulation is the number of runs required until all group members are infected. The second condition allowed us to investigate the number and identity of individuals infected during a set latency. We ran 10,000 simulations for each condition. The source code is available in **Appendix F**.

III.3.6. Data analysis

Generalized linear models were constructed to examine the impact of individual and social factors on centrality position. We tested for significant relationships between centrality coefficients (eigenvector, betweenness and strength) and the following predictor variables: age, dominance rank and family size (range: 1 – 5; Koshima only). Because eigenvector centrality is not truly independent of strength and betweenness coefficients, we estimated the effect of the predictor variables on each centrality measure separately. The distribution of all response variables (centrality measures) deviated from the Gaussian case, so all were square-root-transformed, which performed better than log-transformation and could accommodate the few zeroes in the data set, to meet the assumptions of the statistical models. For all models, we checked that the assumptions of normally distributed and homogeneous residuals were fulfilled by visually inspecting a qqplot and the residuals plotted against fitted values. We further ran a series of diagnostics to judge the validity of the models, including testing for variance inflation, correlation of fitted and residual values and Cooks' distance, all of which suggest the suitability of our models as no obvious violations of assumptions were detected (Field et al. 2012).

We compared the distribution of random and observed networks by Kolmogorov-Smirnov tests with Bonferroni correction (Abdi 2007). Regarding the dynamics of disease transmission, first we calculated the mean latency of complete transmission (to the whole group with no time constraint) and the percentage of infected individuals in a time-constrained simulation (3 runs). Second, we looked at the probability of each individual being infected given the number of times each individual was the first to be infected by the initial infected individual (i.e. the second to be infected among all group members). We also calculated the mean rank of infection in the transmission order (from 1 to $N - 1$ ranks, since

the initial infected individual is removed from the analysis). We then correlated centrality coefficients (eigenvector, betweenness and strength) and dynamics of transmission (complete transmission latency, percentage of infected individuals, probability of being infected and latency to being infected) using Spearman tests with Bonferroni corrections. A p value equal to or less than 0.05 was considered to be statistically significant, and all tests were two-tailed. Analyses were conducted in R statistical software v. 2.15.1 (R Core Team 2016).

III.4. Results

III.4.1. Network structure and centrality position

The two social grooming networks are illustrated in **Figure III.1** and the global measures are given in **Table III.1**. While the Koshima and Yakushima networks did not differ at a global level, differing results were found considering the interaction between individual/social traits and centrality position. Dominance rank, but not age, was a good predictor of eigenvector and strength centrality in Koshima, while we found weak evidence to suggest that age and dominance rank may influence eigenvector centrality in Yakushima (**Table III.2**; **Figures FS2 and FS3**, **Appendix F**). Family size had no effect on network position in Koshima.

Table III.1. Global measures of Koshima and Yakushima networks. Definitions of social network measures are presented in the methods section.

Group	Density	Diameter	Average degree	Overall clustering coefficient	Transitivity	Modularity
Koshima	0.41	3	8.19	1.38	0.52	0.38
Yakushima	0.44	3	7.50	1.37	0.50	0.63

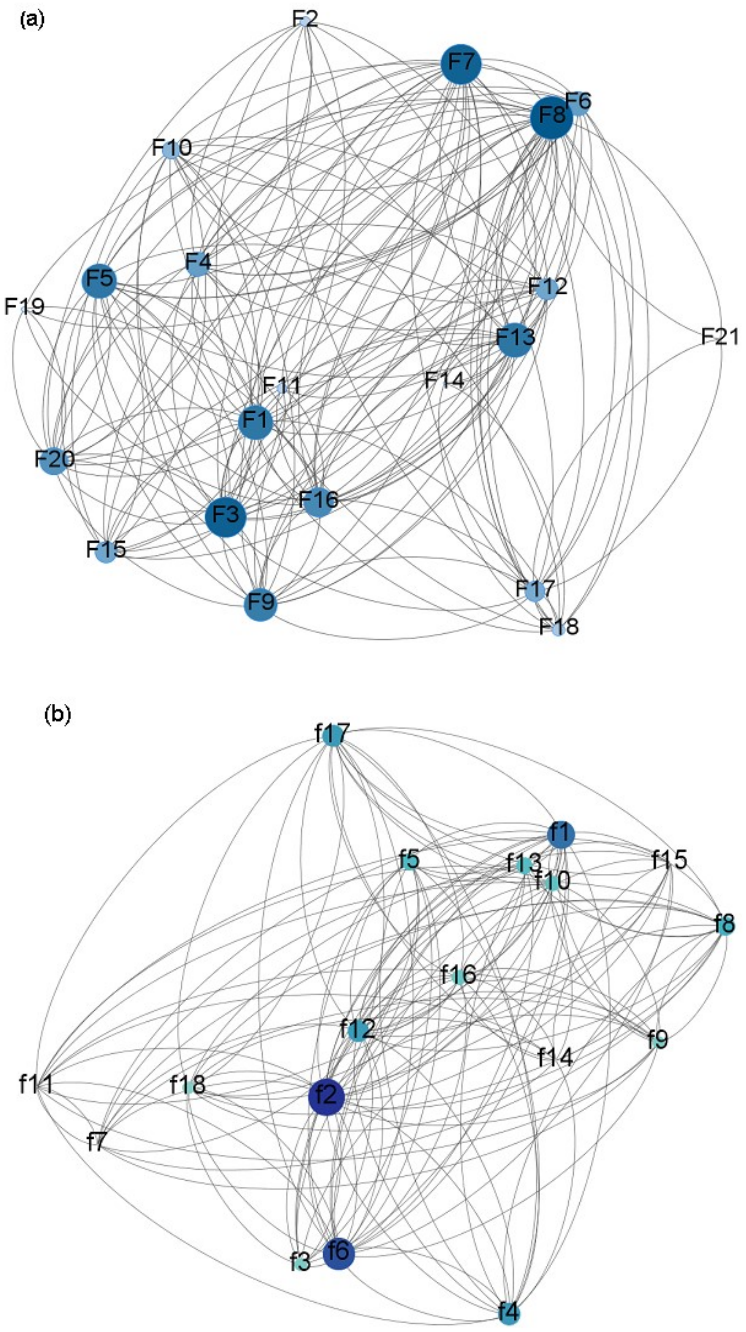

Figure III.1. Social networks of adult female Japanese macaques in Koshima group (a) and Yakushima group (b). Networks were built using Gephi 0.8.2 beta (Cherven 2013). A node (circle) represents a rank identifier, with its size and color directly related to the individual eigenvector centrality coefficient (the higher the centrality, the stronger is the color and the larger is the size of the node). Spacing of nodes was done using the option Force Atlas in Gephi. In this way, nodes are spaced according to their centralities but also according to whom they are connected.

Table III.2. Parameter estimates from generalized linear models explaining variation on network centrality among female Japanese macaques in Koshima and Yakushima groups.

Centrality Coefficients	Predictors	Estimate	Std. Error	t value	Pr(>|t|)
Koshima					
Eigenvector	(Intercept)	0.757	0.147	5.134	8.28e-05***
	Rank	-0.015	0.006	-2.405	0.028*
	Age	-0.033	0.018	-1.818	0.087 .
	Family Size	0.070	0.053	1.307	0.209
Strength	(Intercept)	1.587	0.294	5.389	4.9e-05***
	Rank	-0.032	0.012	-2.564	0.020*
	Age	-0.036	0.036	-1.003	0.330
	Family Size	0.059	0.107	0.555	0.586
Betweenness	(Intercept)	2.915	1.073	2.718	0.015*
	Rank	-0.041	0.045	-0.898	0.383
	Age	-0.053	0.132	-0.405	0.691
	Family Size	0.066	0.390	0.170	0.867
Yakushima					
Eigenvector	(Intercept)	0.278	0.035	7.821	4.73e-06***
	Rank	-0.005	0.003	-2.014	0.067 .
	Age *(adult)*	0.038	0.034	1.095	0.295
	Age *(old adult)*	0.082	0.039	2.079	0.059 .
Strength	(Intercept)	0.937	0.101	9.303	2.27e-07***
	Rank	-0.007	0.008	-0.963	0.352
	Age *(adult)*	0.156	0.096	1.626	0.126
	Age *(old adult)*	0.176	0.106	1.656	0.120
Betweenness	(Intercept)	2.785	0.687	4.054	0.001**
	Rank	-0.053	0.052	-1.020	0.325
	Age *(adult)*	0.870	0.654	1.330	0.205
	Age *(old adult)*	1.105	0.723	1.528	0.149

[a] All comparisons made against the intercept of the first level of each factor in Yakushima (age = young adult).
[b] Significant codes are marked as follow: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1

III.4.2. Observed versus random networks

We observed that two of the four distributions from the simulation on the Yakushima network did not differ significantly from those expected of a random network (Kolmogorov-Smirnov tests: probability of being infected: $D = 0.39$, $p = 0.2$; latency of being infected: $D = 0.5$, $p = 0.08$; **Figure FS4**, **Appendix F**). In contrast, the percentage of infected individuals ($D = 1$, $p < 0.001$) and latency to complete transmission ($D = 1$, $p < 0.001$) differed significantly between observed and random networks. Whereas in the Koshima networks, the probability of being infected ($D = 0.52$, $p \leq 0.05$), the percentage of infected individuals ($D = 0.81$, $p < 0.001$), latency to complete transmission ($D = 1$, $p < 0.001$) and latency of being infected ($D = 0.62$, $p \leq 0.05$) all significantly differed between observed and random networks (**Figure FS5**, **Appendix F**). Thus, in the majority of cases, infectious agents spread more readily in observed than in random networks, and the transmission properties of the Koshima network differed more strongly from those of a random network than did the transmission properties of the Yakushima network.

III.4.3. Transmitting an infectious agent

Individuals with higher centrality coefficients transmitted infectious agents to the entire group with a shorter latency in the Koshima group ($N = 21$) regardless of the centrality coefficient used (Spearman tests: eigenvector: $r = -0.70$, $p < 0.001$; betweenness; $r = -0.55$, $p \leq 0.05$; strength: $r = -0.75$, $p < 0.001$). This was also generally true in the Yakushima group ($N = 18$), though results depended more on the centrality index measured. The Yakushima group showed a strong relationship between transmission latency and eigenvector centrality ($r = -0.76$; $p < 0.01$) as well as betweenness centrality ($r = -0.56$; $p \leq 0.05$), but strength

coefficients were only marginally associated with transmission latency ($r = -0.51$; $p = 0.09$). Central individuals also transmitted infectious agents to a greater number of subjects when compared to less central individuals, but in both groups, betweenness centrality was not associated with the probability of infecting others (Koshima: eigenvector: $r = 0.95$, $p < 0.001$; betweenness; $r = 0.50$, $p = 0.07$; strength: $r = 0.99$, $p < 0.001$; **Figure III.2**; Yakushima: eigenvector: $r = 0.77$, $p < 0.001$; betweenness; $r = 0.45$, $p = 0.19$; strength: $r = 0.74$, $p < 0.01$; **Figure III.3**).

III.4.1. Acquiring an infectious agent

Central individuals were more likely to be infected than non-central individuals in the Koshima group (Spearman tests: eigenvector: $r = 0.68$, $p < 0.01$; betweenness; $r = 0.64$, $p < 0.01$; strength: $r = 0.82$, $p < 0.001$; **Figure III.2**), but only strength centrality was significantly correlated with probability of being infected in the Yakushima group (eigenvector: $r = 0.27$, $p = 0.94$; betweenness; $r = 0.08$, $p = 1.00$; strength: $r = 0.71$, $p < 0.01$; **Figure III.3**). Considering the mean rank of each individual in the chain of transmission, central individuals were more likely to be infected during the first transmission event than less central group mates, with the exception of those with high betweenness scores (Koshima: eigenvector: $r = -0.93$, $p < 0.001$; betweenness; $r = -0.51$, $p = 0.07$; strength: $r = -0.98$, $p < 0.001$; Yakushima: eigenvector: $r = -0.66$, $p \leq 0.05$; betweenness; $r = -0.35$, $p = 0.46$; strength: $r = -0.61$, $p \leq 0.05$).

Regarding analysis of data from Koshima, we identified one subject (f16) having a betweenness value 1.5 times higher than the third quartile, as well as two individuals (f12 and f13) in Yakushima with similarly high eigenvector coefficients. These individuals were therefore removed prior to the analysis. However, if included, correlations between centrality

and infection were similar to those presented above (Koshima: complete transmission latency: $r = -0.61$, $p < 0.01$; percentage of infected individuals: $r = 0.49$, $p = 0.07$; probability of being infected: $r = 0.68$, $p < 0.01$; latency to being infected: $r = -0.49$; $p = 0.07$; Yakushima: complete transmission latency: $r = -0.81$, $p < 0.001$; percentage of infected individuals: $r = 0.81$, $p < 0.001$; probability of being infected: $r = 0.27$, $p = 0.85$; latency to being infected: $r = -0.66$; $p \leq 0.05$).

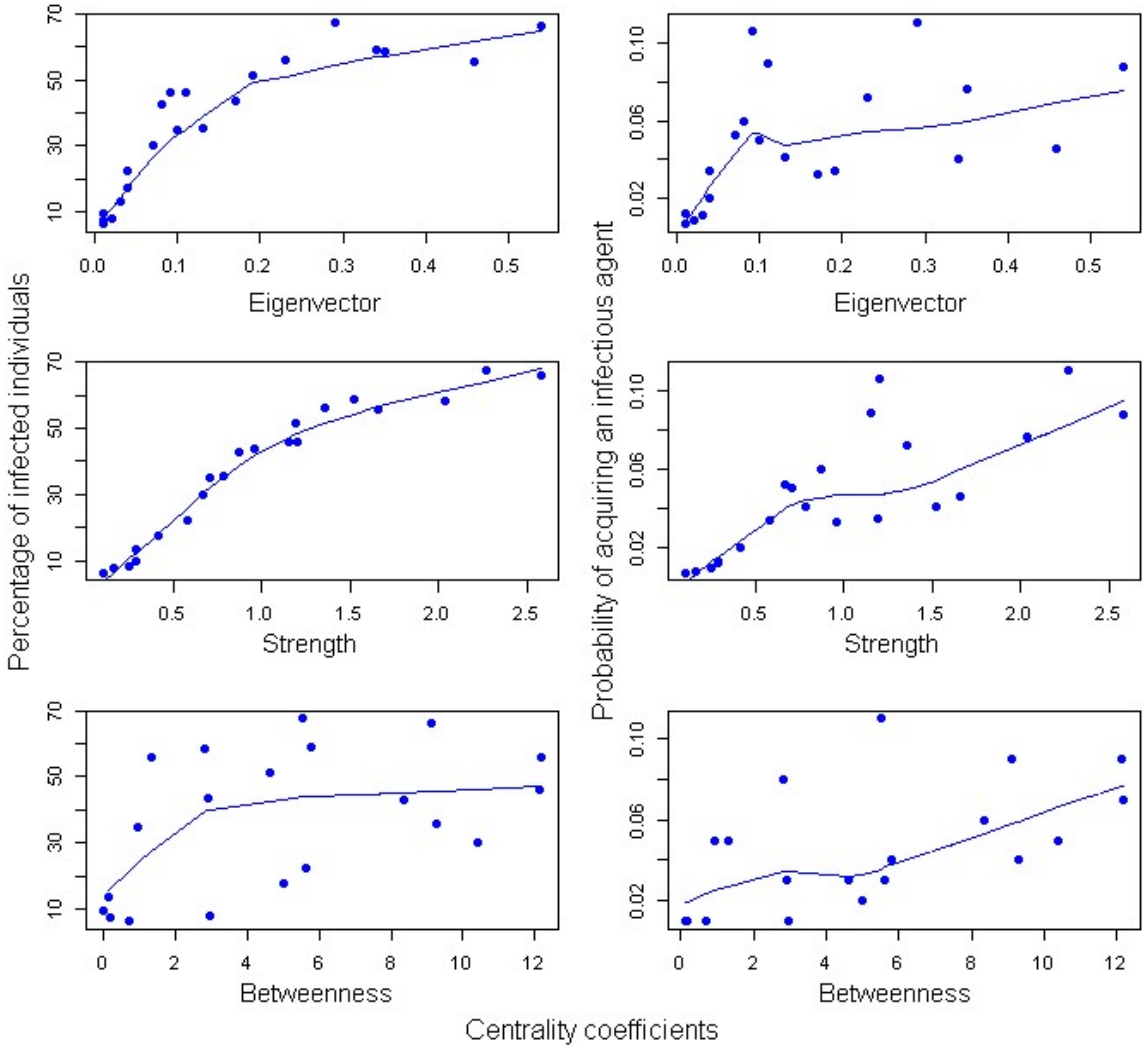

Figure III.2. Relationship between centrality coefficients and dynamics of disease transmission in Koshima group. Blue diamonds represent individual macaques and blue lines represent the mean of individuals infected and the probability of being infected. Outliers have been removed (see text for further information).

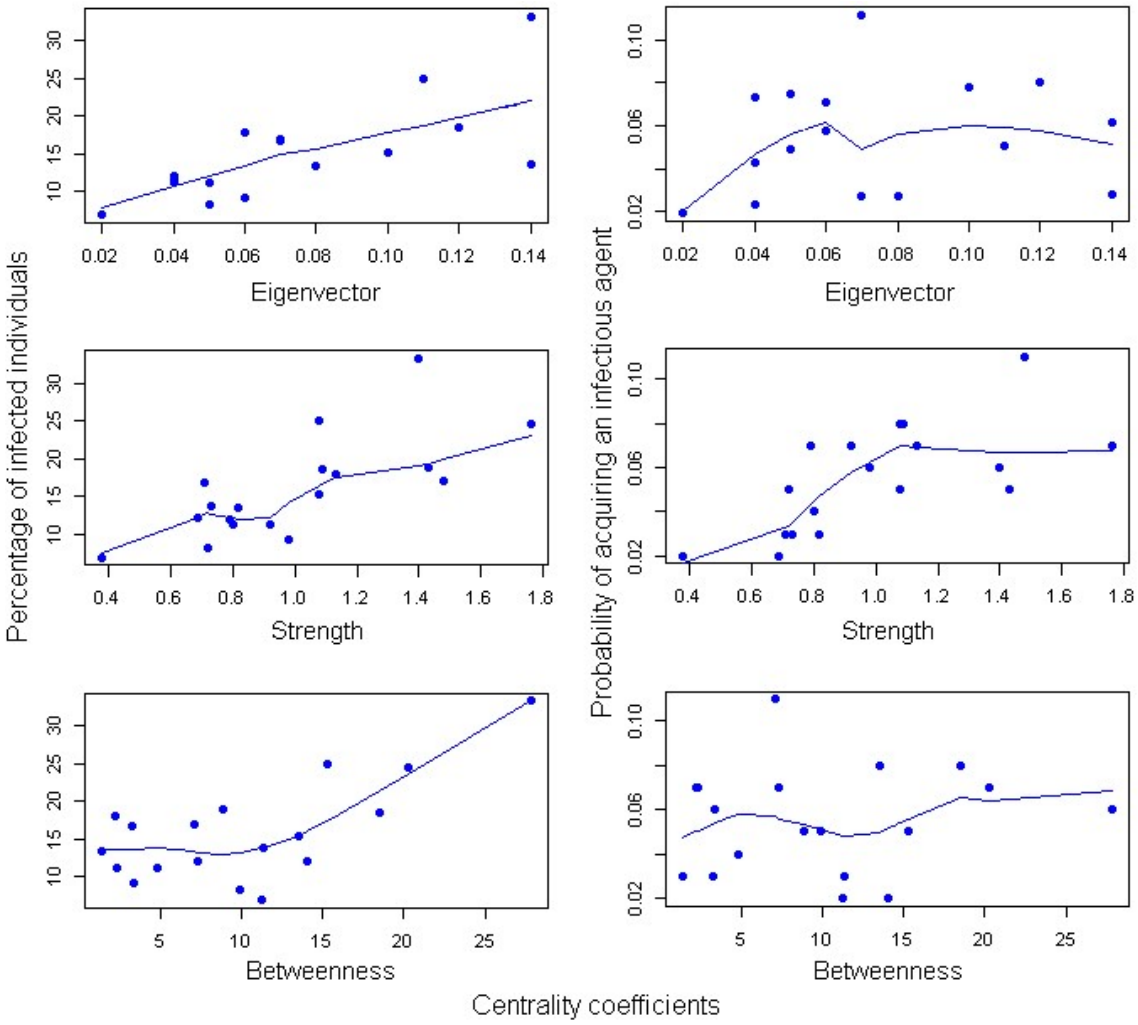

Figure III.3. Relationship between centrality coefficients and dynamics of disease transmission in Yakushima group. Blue diamonds represent individual macaques and blue lines represent the mean of individuals infected and the probability of being infected. Outliers have been removed (see text for further information).

III.5. Discussion

This study aimed to test the hypothesis that social network structure modulates disease transmission, and to better understand the influence of an individual's centrality on the transmission of infectious agents in real-world primate networks. In line with our predictions, individuals central to the grooming contact networks not only transmitted infectious agents with a shorter latency to other group members and to a higher percentage of individuals, but

were also more prone to infection themselves, being among the first to acquire infections and with a shorter latency than more peripheral individuals. Other simulation studies with transmissible agents whose parameters closely match those of the agents used here also show that direct contact interactions may facilitate the spread of infectious agents (Griffin & Nunn 2012, Rushmore et al. 2013). However, our study is among the first to model risks associated with pathogen transmission for central individuals, revealing their increased exposure (here investigated by the probability and latency of infection/transmission) to highly infectious agents. Nonetheless, even though we use the same fundamental data (observed grooming networks) in our simulations of transmitting or acquiring a theoretical infectious agent, results nonetheless show some differences in the importance of observed centrality distributions in disease spread; transmission was faster and affected more individuals in Koshima than in Yakushima, the latter group producing a network that behaved like a random network in two of our four simulation analyses. These divergences from our predictions suggest that caution should be exercised when making generalizations about transmission processes arising from the network structure. Ultimately, however, networks of proximity or contact interactions are generally accepted to underlie infectious disease dynamics in real groups of animals, including humans (Taylor et al. 2001; Altizer et al. 2003), to which our results further attest.

Our results also showed that both global (network-level, e.g. modularity) and individual (node-level, e.g. centrality) metrics must be considered when predicting disease transmission dynamics. Although the Koshima and Yakushima global measures did not differ from each other, we observed distinct distributions of centralities in each group - the variance in the Koshima network being higher than that in Yakushima - and a stronger influence of central individuals and a broader and faster transmission in the Koshima network. The clear relationship between centrality and infection found in the Koshima group may be explained

by the interaction between dominance rank and centrality index. In Koshima, dominance rank strongly correlated with eigenvector and strength centralities while age was marginally associated with eigenvector. In Yakushima, however, the relationship between individual/social traits and centrality measures were less consistent, with age and dominance rank only marginally associated with eigenvector centrality. Given that centrality measures showed slightly different relationships to both individual/social attributes and to transmission metrics, it is important to discuss the role of each metric in predicting pathogen transmission. Strength centrality refers to direct connections between individuals, while eigenvector centrality and betweenness coefficient refer to a combination of direct and indirect connections. The fact that in the Koshima group, transmission latency was strongly related to both eigenvector and betweenness centrality, but only marginally to strength suggests that indirect connections factor more strongly in the transmission chain/dynamics than do direct connections. By contrast, in the Yakushima group, the probability of being infected appears to be driven by direct rather than indirect connections. It thus seems that, while indirect connections are most predictive of latency to complete transmission (a group-level metric), direct connections are most predictive of an individual's probability of being infected (individual-level metric). Too few studies discuss the relative impacts of different levels of network connectedness (Christley et al. 2005); yet these details are essential to both advancing our understanding of the relationship between network structure and disease transmission dynamics, as well as to developing appropriate disease-intervention strategies.

In a handful of cases, targeting central individuals for disease control can reduce outbreak sizes (Salathé & Jones 2010) and should be more efficient than applying control efforts randomly (Böhm et al. 2009; Rushmore et al. 2014). Theoretical removal of individuals based on association networks of orangutans and chimpanzees, for instance, highlighted that the low level of association between orangutans may limit the spread of

disease through the population, in contrast to chimpanzees whose network structure may allow for faster spread of disease (Carne et al. 2014). In addition, an observational study with a wild giraffe population showed that transmission is more likely to occur between individuals that are more strongly connected within the network, indicating that an individual network position is a good predictor of transmission network position (VanderWaal et al. 2014a). We found that transmission in Japanese macaques is enhanced after central individuals become infected, which suggests the existence of super-spreaders in the population. However, the current results demonstrate this assumption through contrast; centrality-based vaccination, for instance, may be well suited to the Koshima group, but its efficacy would be more questionable in the Yakushima network given the somewhat reduced importance of centrality measures in our simulations. Ultimately, disease transmission is a stochastic as demonstrated by our multi-agent model, so observed results might diverge substantially and perhaps even unpredictably from models using deterministic methods, such as theoretically removing central individuals. Modeling can highlight differences observed at the level of the group, and these differences may have direct implications for disease spread and should therefore be taken into account in future endeavours designing disease management plans.

In this context, identifying relationships between individual traits and network centrality may be useful in assessing disease transmission dynamics, particularly because social roles of individuals can vary across groups. Rushmore et al. (2013, 2014) suggested that we might use individual traits that correlate with centrality in disease intervention strategies, based on their results from chimpanzee networks showing that high-ranking males and individuals with large family units were the most central individuals and thus best to target in vaccination efforts. A related outstanding question is whether or not high dominance rank might allow an individual to better tolerate certain infections (MacIntosh 2014), which

would make them even more capable of spreading infectious agents (Ezenwa & Jolles 2015). In our study, however, a middle-ranking old adult female and a low-ranking adult female exhibited the highest eigenvector centrality coefficients in the Yakushima group, while a middle-ranking adult female exhibited the highest betweenness coefficient in Koshima group, all by a very wide margin. The presence of such 'outliers' illustrates the need for caution here; even if rank and age are correlated with network position, we have to be careful about using dominance or age as a proxy for centrality in disease transmission. Analysis of the networks in this study highlights that using a trait-based vaccination strategy in cases with even one or two such outlying individuals might have less optimal results than one might hope in preventing further disease spread. Although almost all animal networks are structured according to sociodemographic variables (e.g. age, dominance, sex, etc.), some can also resemble random networks in their transmission dynamics, rendering them less relevant than others in modeling disease spread; i.e. our Yakushima networks which behaved as would random networks in some circumstances. In such cases, using dominance or centrality is no more useful in predicting who becomes infected or who transmits disease than making random predictions.

However, we are aware that our model represents a simplification of the real process. The models do not attempt to account for variation in individual susceptibility, which itself can relate to social (e.g. through kinship based shared immunological factors or even dominance hierarchy mediated variation in physiological stress, access to food and nutritional state, etc.) and other intrinsic characteristics (innate resistance factors, chronic stress, etc.). Other pertinent information relates to the variation we found between study groups, and how they might be related to different environmental effects (e.g. specific habitat characteristics, home range size), and of course to contrasting population management strategies (provisioned vs. non-provisioned), all of which can strongly affect the expression of social

behavior (Hill 1999). Provisioning food to non-human primates is expected to increase proximities between individuals and should increase aggression levels due to closer proximities. We tried to reduce the direct influence of provisioning on individual interactions by using only those data collected at least one hour after provisioning. Regardless, given a sample size of two networks, we hesitate to make any strong claims about what might have led to the networks observed, and instead focus only on the relative importance of central individuals and network structure in the social transmission of infectious agents in these divergent networks. Studying the influence of network properties in both groups allowed us to understand how different social network measures may affect the transmission dynamics irrespective of the factors that caused the networks to vary.

Another limitation of our study, which should be addressed in future work, is that we included only adult females in our social networks. This was mainly a practicality issue, as for example juveniles are extremely difficult to identify reliably and males, at least in the Koshima group, rarely if ever engage with the group outside of the mating season. Not including such individuals, however, leads to the construction of incomplete networks that may misrepresent true infection dynamics. For example, juveniles are often in contact with their mothers and each other, and may be the most susceptible individuals in the group to disease causing organisms (e.g. MacIntosh et al. 2010). Males are also known to harbour larger parasite infections than females virtually across vertebrates (Poulin 1996), and may therefore be key to the spread of infection on networks, even when not occupying central positions. Despite this limitation, however, female Japanese macaques do form the core of the group's social network, presenting the most stable social relationships organized into a rather rigid hierarchical arrangement. Female Japanese macaques are thus likely to be key players in the dynamics of disease transmission in this species.

In conclusion, our study suggests the importance of social network properties in disease transmission at both the global and individual levels, showing the role of central individuals (here in grooming networks) in spreading disease but also their vulnerability to becoming infected. Possible interactions between individual and global network measures might affect the outcome of disease dynamics. Furthermore, we show that the combined approach of network analysis and modeling provides a promising tool to predict epidemics in primates and other animals (Böhm et al. 2009; Salathé & Jones 2010; Craft & Caillaud 2011; Carne et al. 2014), but that caution should be exercised when generalizing since some networks or network properties may be less relevant than others in predicting disease dynamics. Indeed, it is well-known that detailed analyses of social structure are important in the broader scale of evolutionary and ecological process, further encouraging the use of network analysis across a vast range of topics (Kurvers et al. 2014). Here, understanding the role of networks in disease transmission has important implications for predicting disease spread from the perspectives of conservation and management, but also for understanding the evolution of social relationships in primates and other animals, and the trade-offs that may arise through group living.

Ethics: Specified in the methods section.

Data availability: Data available on request.

Competing interests: The authors declare no competing interests.

Authors' contributions: VR, CSueur and AM conceptualized the study. VR, JD, ET, CS and AJJM collected the behavioral data. VR performed the statistical analysis and wrote the manuscript. All authors significantly contributed to the review of the manuscript.

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Chapter 2

Global-network properties and epidemic stage

IV. CHAPTER 2:

Pathogen spread and the dynamics of social connectivity effect: an evaluation through epidemic time

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*Authors provided equal contribution.

IV.1. Abstract

(English) Because the intensity and strength of social interactions alter the risks of acquiring and transmitting infectious agents, there is an increasing interest in understanding how individuals are connected during outbreaks and how this might impact individual health and fitness. In a network of contacts, some properties of the social structure are suggested to favor pathogen spread while others, or even those same properties under different conditions, may constrain it, working as functional barriers of transmission. In consequence, studies have focused on the effects of network properties on the maximum reach of outbreaks. However, to our knowledge, whether the social structure influences pathogen spread dynamically throughout the progression of an epidemic has not yet been considered. We therefore investigated to what extent centralization, clustering coefficient, density, diameter and modularity facilitate or constrain the spread of theoretical infectious agents across five stages of a series of simulated epidemics in variable group sizes. We incorporated empirical social

contact data from 40 wild groups of 21 non-human primate species in a dynamic stochastic model, considering the strength of interactions between group members. We then examined the dynamics of pathogen flow by considering the mean percentage of individuals that were infected at each epidemic stage. Results show that the prevalence of infection was higher when groups were larger, but that centralization and modularity, and to a lesser extent clustering coefficient and diameter influenced epidemic size, though their influence depended on the stage of the outbreak and the virulence of the pathogen. Our results support the social bottleneck hypothesis, that social substructure reduces the costs of relationships. We also discuss how the temporal influence of network properties might direct the development of intervention strategies. These results help us understand which network properties might be targeted according to the epidemic stage in order to optimize intervention strategies during outbreaks.

Keywords: network epidemiology, agent-based modeling, comparative analysis, animal societies

(Français) L'origine de l'intérêt croissant porté à la compréhension des liens entre individus lors d'épidémies et comment cela peut impacter la santé et la fitness des individus se retrouve dans la découverte que l'intensité et la force des interactions sociales diminuent les risques d'infection et de transmission d'agents infectieux. Ainsi, il a été suggéré que certaines propriétés de la structure sociale d'un réseau favorisent la diffusion d'un pathogène quand d'autres (ou des propriétés similaires sous différentes conditions) peuvent la contraindre, œuvrant alors en tant que barrière de transmission. En conséquence, la majorité des études se sont concentrées sur les effets des propriétés du réseau au moment du stade maximal de l'épidémie. Cependant, à notre connaissance, aucune étude n'a pour le moment regardé comment la structure sociale influence la diffusion du pathogène de façon dynamique au cours de la progression de l'épidémie. Nous avons ainsi étudié ici comment la centralisation,

le coefficient de clustering, la densité, le diamètre et la modularité facilitent ou contraignent la diffusion d'agents infectieux théoriques dans des groupes de d'individus de taille variable au cours des cinq étapes d'une série d'épidémies simulées. Nous avons intégrés dans un modèle dynamique stochastique des données de contacts sociaux empiriques de 40 groupes de 21 espèces de primates non-humains en considérant la force des interactions entre les membres du groupe. Ensuite, nous avons examiné la dynamique de diffusion du pathogène en utilisant le pourcentage d'individus moyen infectés à chaque étape de l'épidémie. Nos résultats montrent que la prévalence de l'infection est plus importante quand les groupes sont plus grands, mais aussi que la centralisation et la modularité et en moindre proportion le coefficient de clustering et le diamètre influencent la taille de l'épidémie, bien que leurs influences dépendent de l'étape de l'épidémie et de la virulence du pathogène. Nos résultats supportent ainsi l'hypothèse du goulot social et que les sous-structures sociales réduisent les coûts des relations. Nous discutons également de comment l'influence temporelle des propriétés de réseau peut aider au développement de stratégies d'intervention. Ces résultats nous aident donc à comprendre quelles propriétés de réseau cibler en fonction de l'étape de l'épidémie et cela dans l'optique d'optimiser les stratégies d'intervention lors des épidémies.

Mots-clés: épidémiologie de réseau, modélisation multi-agent, analyse comparative, sociétés animales

IV.2. Introduction

How social animals, including humans, interact and develop relationships may deeply affect the dynamics and chain of infectious agent transmission (Kappeler et al. 2015). As the basis of most transmission processes, both global- (e.g. density and clustering) and individual-level (e.g. node centrality) network properties reflect social interactions that are influenced by

distinct factors such as the distribution of phenotypes across space (Farine et al. 2015b), loss of conspecifics (Firth et al. 2017), social status (Kanngiesser et al. 2011) and social systems (Sueur et al. 2011b). Recent studies focusing on the link between sociality, health and fitness have provided insight into the role of individual connections in pathogen transmission in animal societies (Nunn et al. 2015; Yang et al. 2016), highlighting that being social incurs higher exposure to pathogens, which may have implications for individual fitness (Weng et al. 2013). How individuals interact with each other and how the structure of a network affects transmission processes are crucial questions to better understand the costs of sociality, and thus how social structure might evolve.

In the last decade, theoretical models and field observations have broadly demonstrated that the strength of connections and the size of groups can alter the spread and persistence of infectious disease (Bansal et al. 2007; Godfrey et al. 2009; Salathé & Jones 2010; Aiello et al. 2014; Rushmore et al. 2014). Dense and well-connected networks might favor pathogen transmission (Keeling 1999), while highly subdivided networks might reduce the final outbreak size (Griffin & Nunn 2012). A study investigating parasite spread in two species of primates, for example, hypothesized that Ebola should spread faster in gorillas (*Gorilla gorilla*) and slower in chimpanzees (*Pan troglodytes*) given their smaller and more cohesive groups (Walsh et al. 2009). The relationship between group size and pathogen transmission, however, is not always clear-cut. While many studies, including large meta-analyses (Cote & Poulin 1995; Vitone et al. 2004), provide evidence that infectious disease risk increases in larger groups (Freeland 1979; Ezenwa 2004; Whiteman & Parker 2004; Caillaud et al. 2013), others show the opposite relationship, with smaller groups having higher levels of parasitism (Rubenstein & Hohmann 1989; Arnold & Lichtenstein 1993; Semple et al. 2002; Bordes et al. 2007; Snaith et al. 2008), or no relationship at all (Chapman et al. 2012). Further studies suggest that whether and how group size affects parasitism

depends on the type of parasite being considered (Cote and Poulin 1995; Vitone et al. 2004). This ambiguous relationship between group size, pathogen transmission and social organization has led to a further possibility, that network properties might interact with group size to predict the spread of infectious organisms in animal societies (Nunn et al. 2015).

The *social bottleneck hypothesis* predicts that social network structure modulates the association between group size and pathogen transmission: larger groups may be more subdivided structurally, and consequently such group substructure (i.e. modularity) may act as transmission bottleneck (Griffin & Nunn 2012; Nunn et al. 2015). Indeed, studies have provided support for the hypothesis that increased modularity can reduce the potential for pathogen transmission in large groups of mammals (Wilson et al. 2003; Salathé & Jones 2010; Griffin & Nunn 2012). Conversely, there is some contrasting evidence in which modularity was associated with increased transmission (Lentz et al. 2012; Nematzadeh et al. 2014), though the mechanism here is unclear. A recent study therefore suggested that modularity may only slow down transmission when the networks are extremely modular, via mechanisms such as network fragmentation and subgroup cohesion, which cause a structural delay in the spread of disease (Sah et al. 2017). These studies based their findings on the final size of the outbreak (i.e. total number of infected individuals in the group) and, consequently, generalize the effect of network properties on infection prevalence across a given period of time. Understanding whether the interaction between network properties and group size remains stable throughout the progression of an epidemic has, to our knowledge, not yet been investigated, despite its potential to highlight dynamic properties in the relationship between network structure and pathogen spread. Determining whether this relationship is stable or dynamic as an epidemic progress might have implications for the optimization and implementation of effective intervention strategies, in both human and wildlife populations.

In this study, we used estimations of network structure and group size from a broad dataset of Neotropical and Old-World primates in a stochastic model that simulates the spread of both moderately- and highly-contagious pathogens (e.g. Ebola and measles, respectively). Our goal was to examine the infection prevalence (number of individuals becoming infected) throughout the progression of the outbreak; here divided into five time-steps. We conducted this study with primates because many or most species are highly social and they represent the closest phylogenetic relatives of humans, which may help us to predict social transmission processes in our own societies. Furthermore, understanding variability in the spread of infectious agents has direct implications for disease control (Blanchard 2002; Barthélemy et al. 2004; Ryan & Walsh 2011), for the conservation of species (Leendertz et al. 2006), and for the optimization of public health services (Pisani et al. 2003).

We predicted that increased values of modularity, clustering coefficient and diameter, all of which are expected to increase in larger groups, should constrain transmission by down-regulating each individual's probability of encountering infected individuals, and therefore being exposed to infectious agents (**Table IV.1**). We predicted that network density, on the other hand, would exhibit the opposite effect by increasing the chance of disease spread: the denser the network, the higher the probability of transmission. Predictions about centralization, however, are not yet clear and may depend on per-contact transmission probabilities: highly-centralized networks might enhance transmission at high contact rates but decrease transmission at low contact rates (Griffin & Nunn 2012); a pattern that might be explained by the heterogeneous transmissibility of individuals in the population, i.e. whether or not 'super-spreaders' exist (Lloyd-Smith et al. 2005). It is important to highlight that in this study we only consider aspects of the emergent networks observed in real primate groups, irrespective of the mechanistic factors that modulate them or those that cause variation in susceptibility to infection.

Table IV.1. Predicted influence of network properties on the transmission of infectious agents. The third column (i.e.: Prediction) denotes whether a positive or negative relationship is expected between the network index and outbreak size.

Network index	Definition *	Prediction	Reference **
Centralization	The extent to which a single or few individuals monopolize relationships in the network. A highly-centralized network resembles a star, with an individual at the center of it.	- /+	1
Overall clustering coefficient	The likelihood that neighbors in a network are connected mainly to each other. It is a local measure of group substructure.	-	2
Density	The ratio of the number of observed edges and the number of possible edges in the network	+	3, 4
Diameter	The longest path (edge) in the network	-	5, 6
Modularity	The extent of sub-grouping in a network. The most supported prediction of the social-bottleneck hypothesis.	-	1, 7, 8

* Definitions are based on Croft et al. 2008.
** 1. Griffin & Nunn 2012; 2. Naug 2008; 3. Moller et al. 1993; 4. Otterstatter & Thomson 2007; 5. Centola 2010; 6. Eubank et al. 2004; 7. Nunn et al. 2015; 8. Pasquaretta et al. 2014.

IV.3. Material and Methods

IV.3.1. Empirical social networks

We compiled 40 social interaction matrices from 21 non-human primate species, including both Catarrhine (Old World) and Platyrrhine (New World) primates, living under natural conditions. Data were either contributed by coauthors or come from published sources. Because we were interested in infectious agents transmitted by social contact, we only used

data concerning body contact and grooming interactions to construct undirected, weighted networks. We did not differentiate social relationships based on the direction of the interactions, assuming instead that contact facilitates transmission regardless of the direction of the interaction. For eight species, observations of more than one group were included, so intraspecific variation could be assessed. Details on the sources of data and the group sizes are all available in the **Table GS1**, **Appendix G**.

IV.3.2. Social network measures

We estimated five metrics relevant to social network structure: centralization, clustering coefficient, modularity, density and diameter (definitions at **Table IV.1**). These measures are stated to be mathematically independent of, but empirically associated with, group size (Nunn et al. 2015), which we also consider in our analyses. The centralization index (CI), which increases as the network is centralized around fewer and fewer individuals, is an extension of eigenvector centrality (EGC): an individual-level measure considering the connectivity of an individual within its network as well as the connectivity of its neighbors. The CI was calculated as follows:

$$CI = \frac{100 * \sum_{i}^{N} (Cmax - EGC)}{Max \sum_{i}^{N} (Cmax - EGC)}$$

where $Cmax$ is the highest eigenvector centrality in the real network and $Max \sum_{i}^{N} (Cmax - EGC)$ considers what $Cmax$ would be under the largest possible centralization of the network (e.g. a star network in which CI≈100).

Clustering coefficient (CC) and modularity (Q) are both measures of connectivity among individuals, but clustering provides a more local level of estimation than does modularity. The CC takes into account the redundancy of connections among three individuals (i.e. whether an individual is connected to two other individuals, and whether those individuals are in turn connected to each other, forming a triplet; Croft et al. 2008; Nunn et al. 2015). CC ranges from 0 to 1, with values close to 1 indicating low levels of clustering in the network. In contrast, modularity evaluates how densely connected a group of individuals is and how sparse the connections between the subgroups are (Newman 2006). Among the several estimations of modularity that exist, we chose Newman's maximum modularity because it is expected to provide a more realistic estimation of network substructure (Newman 2006) and fits with previous studies testing the social bottleneck hypothesis (Nunn et al. 2015). Modularity ranges from 0 to 1, with values close to 1 indicating high levels of sub-grouping.

Network density (De), which is expected to increase in smaller groups (Lehmann & Dunbar 2009; Sueur et al. 2011a; Pasquaretta et al. 2014) takes the same range of values as modularity, with dense groups being characterized by values close to 1. Finally, network diameter (Di) is used here as a simple representation of the overall distance between individuals. Sparser networks usually have greater diameters. The centralization index and diameter were calculated using Ucinet 6 (Borgatti et al. 2002), while Newman's modularity was estimated using the function "cluster_leading_eigen" in the R package "igraph" (Csardi & Nepusz 2006). Network density and clustering coefficient were also estimated via the package "igraph", using the functions "graph.density" and "transitivity", respectively.

IV.3.3. Computer simulations

Our theoretical model on disease spread uses a Markov chain process, and is an implementation of an agent-based model previously used to study the transmission of pathogens in Japanese macaques (chapter 1, **Appendix A**) and collective movement in Tonkean macaques (Sueur et al. 2009). Developed in NetLogo version 3.1.5 (Wilensky 1999; Bryson et al. 2007), the model is basically equivalent to the classical Susceptible-Infected (SI) epidemiological model, in which individuals switch from a susceptible (S) to an infected (I) stage, but do not recover or die from the infection. Since we were interested in unidirectional transmission chains, we felt the SI model was appropriate, and its parsimony allowed us to focus on the role of networks in epidemic spread across stages of an outbreak. The SI model may also be appropriate when individuals are not likely to recover from infections caused by highly virulent pathogens (Rothman et al. 2008), as long as the outbreak continues to completion before individuals are removed from the population through death. We accept that this may only be true in a minority of cases, and that this is therefore a limitation of our study design. In the model, the spread of pathogens is network-dependent, meaning that the probability of an individual transmitting a pathogen to another depends on the strength of its relationships with each non-infected individual: the stronger the relationship, the greater the probability of transmission.

We included in the model data on group size, individual identities and their affiliative relationships. Grooming and body contact interactions were transformed to corrected frequencies according to previous studies so that the sum of relationship per individual equals $N - 1$ (Sueur & Deneubourg 2011, table 1; chapter 1, **Appendix A**). At the beginning of each simulation run, all individuals were given the same probability of becoming infected, meaning that we consider a constant susceptibility to infection across exposed individuals.

This probability was named intrinsic probability λ. As soon as an individual was infected, the probability Ψ for a conspecific i to be infected depended on:

$$\Psi i = \lambda + C\sum_{k=1}^{N} r(k,i)$$

where N represents the total number of individuals in the group, $r(k,i)$ denotes the social relationship individual k has with individual i, and C is the coefficient of network connectivity, the probability of being infected by other group members. Individuals with more links in the network are more likely to be in contact with an infected individual, thus they are more likely to be infected (Sueur et al. 2009). The basic reproductive number (R_0), defined as the mean number of secondary infections that arise from a randomly infected index case, depends on the network connectivity and intrinsic probability, with $R_0 = \frac{C}{\lambda}$. We calculated this ratio such that the resulting R_0 values matched those previously used for heuristic purposes in a study of chimpanzees (Rushmore et al., 2014), but also follow from epidemiological investigations of infectious agents circulating in human populations. Specifically, we used two values of R_0, one ($R_0 = 3$) representing a moderately-contagious pathogen such as Ebola (Legrand et al. 2007; Ndanguza et al. 2013), and the other ($R_0 = 12$) corresponding to a highly-contagious pathogen such as measles (Anderson & May 1991). It is important to note that, as highlighted in a previous study (Rushmore et al. 2014), estimations of R_0 are still not available for most wildlife pathogens, and more specifically, for those affecting non-human primates. The model is stochastic: at each time step, a number between 0 and 1 was randomly attributed to each individual; when this number was lower than the theoretical probability for an agent i to be infected, the agent gets infected; if this number was higher than the theoretical probability, the agent did not get infected.

From these models, we investigated two properties concerning the dynamics of disease transmission in primate networks: (1) the latency to complete, i.e. to the whole group, transmission; and, (2) the percentage of infected individuals. The first property allowed us to identify a range of epidemic stages varying in the number of groups completely infected. We plotted the distribution of the latency required to infect all studied groups and checked for the theoretical distribution that better fit with our data. From its observed exponential distribution (**Figure GS1, Appendix G**), we arbitrarily selected 5 stages, also following an exponential distribution, that covered the initial (few groups completely infected) to advanced (most groups completely infected) levels: stage 1 = 50, stage 2 = 100, stage 3 = 300, stage 4 = 1200 and stage 5 = 6000. These values (i.e. 50, 100, …) represent the number of time steps performed before the model is automatically stopped. It is important to note that these stages represent the progression of an epidemic divided by stage, or time period. The percentage of infected individuals calculated at each stage is a continuation of the previous stage, so if 20% of individuals are infected in stage 1 and 40% are infected at stage 2, 20% of the group was infected at each stage. In small groups, we expect an expedited progression in the epidemic, and consequently small groups are those that experience complete transmission earlier than the other, larger groups. We ran 10000 simulations for each property and took the average for each group of study.

IV.3.4. Statistical analysis

IV.3.4.a. Phylogenetic variance

We first conducted a comparative phylogenetic analysis to test for homogeneity of variance in network properties and group size across lineages (i.e. phylogenetic signal) using the

"phylosignal" function in the R package "picante" (Blomberg et al. 2003; Kembel et al. 2010). The K statistic is a measure of the phylogenetic signal. K values close to zero correspond to a random or convergent pattern of evolution and K values greater than 1 indicate a strong phylogenetic signal and conservatism in traits (Kembel et al. 2010). For species with more than one network, we avoided taking the average of network metrics and randomly chose one representative network following Nunn et al. (2015). We used an estimation of the branch scaling coefficient λ, which is a measure of phylogenetic signal (Freckleton et al. 2002) following the primate tree revised from Purvis 1995 and Rogers & Gibbs 2014. The estimated tree used for the comparative analysis is provided in the **Figure GS2**, **Appendix G**. The alpha level for all analyses was set at 0.05.

IV.3.4.b. Network properties, group size and pathogen spread

To examine how network properties and group size might affect disease dynamics, we applied Generalized Linear Mixed Effect Models (GLMMs) using the package "glmmADMB". We nested Genus within Family in the random effect structure to account for the hierarchical nature of our study (see Table S1 for more details on intraspecific variation). We applied the function "fitdist" from the package "fitdistrplus" from which we observed that the distribution of our response variable (percentage of infected individuals at the different epidemic stages) deviated from the gaussian case and better fit a gamma distribution. We ran a set of diagnostics to judge the validity of the models, including testing for correlation between fitted and residual values and using Cooks' distance to assess influential cases, all of which indicated that no major issues existed (Field et al. 2012). We also checked for multicollinearity between variables using the function "vif" in the R package

"car". Density and clustering coefficient were highly correlated (**Figure GS3, Appendix G**), with the latter presenting higher vif values (vif = 18.89) than the former (vif = 16.84). As density was not the major focus of our study – opposed to clustering coefficient and modularity which we used as measures of group substructure – density was removed from further analysis. Therefore, our GLMMs included centralization index, clustering coefficient, diameter, modularity and group size as fixed effects. As we expected to have interacting effects concerning group size and each of the network properties examined (Pasquaretta et al. 2014; Nunn et al. 2015), we included all possible two-way interactions involving group size in the model. Predictor variables were scaled and centered to facilitate comparison of effect sizes.

We built a series of five statistical models for each reproductive number ($R_0 = 3, 12$), focusing on the percentage of infected individuals at each epidemic time stage, increasing from stage one to stage five. We then used the "dredge" function of the R package "MuMIn" (Barton 2016) to streamline the process of candidate model selection according to Akaike's Information Criterion (Akaike 1985). We ranked all models by AIC corrected for small sample sizes (AICc) and normalized Akaike weights (AICw), and constructed a conservative candidate model set by removing all models that did not fall within 10 AICc units of the model with the lowest AICc (Burnham & Anderson 2002). Model parameter estimates were then averaged across the candidate set using the function "model.avg", and confidence intervals were computed with the function "confint", both of which are available in the "MuMIn" package (Gelman et al. 2013). We chose to use conditional averaging, i.e. to average parameter estimates only across models in which the variable of interest occurs. We present the model-averaged parameter estimates (β), adjusted standard errors (SE), confidence intervals (CI) and the relative importance of all predictor variables repeatedly occurring within the candidate set. Finally, to allow for the parsimonious interpretation of our

main predictor variables, we used the function "subset" available in the "MuMIn" package to select from the full candidate set those models with no interactions between the predictor variable of interest and group size. Models respecting this condition and with ΔAICc <10 were then averaged and confidence intervals were computed for each predictor. This allowed us to avoid the potential misinterpretation of main parameter effects caused by the presence of interactions involving that parameter in the models. All statistical analyses were performed in R version 3.0.1 (R Core Team 2016).

IV.4. Results

IV.4.1. Phylogenetic analysis

We first asked whether variation in our network measures could be explained by phylogenetic relationships. We did not observe a phylogenetic signal relevant to any of these network measures: clustering coefficient: K = 0.29, p = 0.12; centralization: K = 0.24, p = 0.23; diameter: K = 0.13, p = 0.82; modularity: K = 0.11, p = 0.95; group size: K = 0.20, p = 0.50.

IV.4.2. Network properties, group size and pathogen spread

We then tested whether network properties and group size influenced pathogen spread, and most importantly whether such influence remained constant throughout the progression of a simulated outbreak. Results show that the prevalence of infection was higher when groups were larger, but that centralization and modularity, and to a lesser extent clustering

coefficient and diameter influenced epidemic size. Their influence depended on the stage of the outbreak and the virulence of the pathogen (**Table IV.2**). The full set of candidate models, with their respective values of AICc, ΔAICc and AICw can be found in the **Lists GS1 and GS2**, **Appendix G**.

Group size was a good predictor of disease spread at all epidemic stages, being present in 90% of the candidate models and exhibiting the largest effect size (parameter estimate), with one exception at epidemic stage 4 (**Table IV.2**). The effects of interactions between network properties and group size were more variable, depending on the epidemic stage and the reproductive number of the pathogen (**Table IV.2**, **Figure IV.1**). During transmission of the moderately contagious pathogen, centralization negatively influenced transmission at earlier stages of the outbreak (*stage 1*: av.β±SE = -0.04±0.02, 95%CI = -0.07 to -0.01; *stage 2*: av.β±SE = -0.05±0.02, 95%CI = -0.09 to -0.02), whereas modularity constrained progression of the epidemic at more advanced stages (*stage 2:* av.β±SE = -0.04±0.02, 95%CI = -0.07 to -0.002; *stage 3:* av.β±SE = -0.09±0.03, 95%CI = -0.14 to -0.03; *stage 4:* av.β±SE = -0.06±0.03, 95%CI = -0.11 to -0.001). When simulating the spread of the highly transmissible pathogen, modularity constrained prevalence at the beginning of the outbreak (*stage 1:* av.β±SE = -0.08±0.02, 95%CI = -0.12 to -0.03; *stage 2:* av.β±SE = -0.10±0.03, 95%CI = -0.16 to -0.03; *stage 3:* av.β±SE = -0.06±0.03, 95%CI = -0.11 to -0.01) while at later stages, centralization positively impacted the outbreak size (*stage 3:* av.β±SE = 0.07±0.03, 95%CI = 0.001 to 0.13; *stage 4:* av.β±SE = 0.03±0.02, 95%CI = 0.0003 to 0.06). When looking at the effect of centralization and modularity without the interaction with group size, we observed their influence in the epidemic size at stage 2 for a moderately contagious pathogen (centralization: av.β±SE = -0.03±0.02, 95%CI = -0.06 to -0.002; modularity: av.β±SE = -0.06±0.02, 95%CI = -0.09 to -0.03) and the influence of modularity

at stage 1 for a highly contagious pathogen (av.β±SE = -0.06±0.02, 95%CI = -0.10 to -0.02; **Table IV.2**, **Figure IV.1**).

The interaction between network diameter and group size also predicted reduced prevalence: at stage 3 for the moderately-contagious pathogen (av.β±SE = -0.09±0.03, 95%CI = -0.15 to -0.02) and at stages 1 (av.β±SE = -0.06±0.03, 95%CI = -0.12 to -0.002) and 2 (av.β±SE = -0.10±0.04, 95%CI = -0.18 to -0.03) for the highly-contagious pathogen (**Table IV.2**, **Figure IV.1**). However, the relative importance of these factors was 0.33, 0.27 and 0.47, indicating that in most of the models, the interaction between diameter and group size did not appear to be an important predictor of outbreak size. Finally, clustering coefficient was also observed to negatively affect the outbreak size. Its influence was independent of group size, happening at stages 1 (av.β±SE = -0.05±0.02, 95%CI = -0.09 to -0.01) and 2 (av.β±SE = -0.04±0.02, 95%CI = -0.07 to -0.01) for highly and moderately contagious pathogen, respectively (**Table IV.2**).

Table IV.2. Moderately and highly contagious pathogen transmission in wild primate networks (N= 40). Conditional model-averaged parameter estimate (β) ± adjusted standard errors (SE) (95% unconditional confidence intervals - CI) and relative importance of variables in generalized linear mixed models of the percentage of infected individuals in 5 epidemic steps. Main effects are calculated based on models without interactions (see text). In bold are results for which CI do not include zero. Ccoef = clustering coefficient.

	Moderately contagious pathogen		**Highly contagious pathogen**	
Model parameter	β ± SE (95% CI)	Relative importance	β ± SE (95% CI)	Relative importance
Stage 1				
intercept	0.51±0.01 (0.48 to 0.54)	contained in all models	2.69±0.03 (2.63 to 2.75)	contained in all models
group size	**0.28±0.02 (0.24 to 0.32)**	**1.00**	**0.77±0.03 (0.71 to 0.82)**	**1.00**
centralization	0.01±0.01 (-0.02 to 0.04)	0.19	-0.03±0.02 (-0.07 to 0.01)	0.41
ccoef	-0.02±0.01 (-0.04 to 0.01)	0.31	**-0.05±0.02 (-0.09 to -0.01)**	**0.78**
diameter	-0.01±0.01 (-0.04 to 0.02)	0.19	-0.01±0.02 (-0.05 to 0.03)	0.19
modularity	-0.02±0.01 (-0.05 to 0.01)	0.39	**-0.06±0.02 (-0.10 to -0.02)**	**0.88**
centralization:group size	**-0.04±0.02 (-0.07 to -0.01)**	**0.61**	0.01±0.02 (-0.03 to 0.05)	0.07
ccoef:group size	-0.01±0.02 (-0.06 to 0.03)	0.10	0.01±0.04 (-0.06 to 0.08)	0.13
diameter:group size	0.01±0.02 (-0.02 to 0.05)	0.05	**-0.06±0.03 (-0.12 to -0.002)**	**0.27**
modularity:group size	-0.02±0.01 (-0.05 to 0.003)	0.29	**-0.08±0.02 (-0.12 to -0.03)**	**0.92**
Stage 2				
intercept	1.59±0.02 (1.56 to 1.62)	contained in all models	3.57±0.05 (3.47 to 3.68)	contained in all models
group size	**0.64±0.02 (0.60 to 0.67)**	**1.00**	**0.56±0.06 (0.43 to 0.68)**	**1.00**
centralization	**-0.03±0.02 (-0.06 to -0.002)**	**0.61**	-0.02±0.03 (-0.09 to 0.05)	0.20
ccoef	**-0.04±0.02 (-0.07 to -0.01)**	**0.82**	-0.03±0.04 (-0.11 to 0.06)	0.21
diameter	-0.06±0.02 (-0.02 to 0.02)	0.26	0.002±0.04 (-0.07 to 0.07)	0.16
modularity	**-0.06±0.02 (-0.09 to -0.03)**	**1.00**	-0.005±0.04 (-0.08 to 0.07)	0.16
centralization:group size	**-0.05±0.02 (-0.09 to -0.02)**	**0.88**	0.08±0.04 (-0.01 to 0.16)	0.12
ccoef:group size	0.04±0.03 (-0.02 to 0.09)	0.30	-0.03±0.09 (-0.21 to 0.15)	0.06
diameter:group size	-0.01±0.02 (-0.05 to 0.03)	0.05	**-0.10±0.04 (-0.18 to -0.03)**	**0.47**
modularity:group size	**-0.04±0.02 (-0.07 to -0.002)**	**0.72**	**-0.10±0.03 (-0.16 to -0.03)**	**0.42**
Stage 3				
intercept	3.25±0.05 (3.16 to 3.35)	contained in all models	4.28±0.03 (4.22 to 4.34)	contained in all models

group size	**0.65±0.06 (0.53 to 0.78)**	**1.00**	**0.19±0.03 (0.13 to 0.25)**	**1.00**
centralization	-0.02±0.03 (-0.08 to 0.03)	0.21	-0.05±0.03 (-0.11 to 0.004)	0.42
ccoef	-0.04±0.04 (-0.12 to 0.03)	0.29	0.02±0.03 (-0.04 to 0.08)	0.20
diameter	-0.01±0.03 (-0.06 to 0.05)	0.16	-0.004±0.03 (-0.07 to 0.06)	0.15
modularity	-0.03±0.03 (-0.09 to 0.02)	0.27	0.05±0.03 (-0.007 to 0.12)	0.59
centralization:group size	0.04±0.04 (-0.04 to 0.12)	0.06	**0.07±0.03 (0.001 to 0.13)**	**0.59**
ccoef:group size	-0.04±0.06 (-0.16 to 0.09)	0.07	-0.04±0.06 (-0.15 to 0.07)	0.06
diameter:group size	**-0.09±0.03 (-0.15 to -0.02)**	**0.33**	-0.06±0.03 (-0.12 to 0.01)	0.10
modularity:group size	**-0.09±0.03 (-0.14 to -0.03)**	**0.66**	**-0.06±0.03 (-0.11 to -0.01)**	**0.59**
Stage 4				
intercept	4.25±0.05 (4.16 to 4.34)	contained in all models	4.48±0.03 (4.42 to 4.54)	contained in all models
group size	**0.19±0.04 (0.12 to 0.26)**	**0.96**	0.04±0.02 (-0.001 to 0.07)	0.64
centralization	-0.06±0.03 (-0.12 to 0.01)	0.57	-0.02±0.01 (-0.05 to 0.005)	0.48
ccoef	0.03±0.04 (-0.04 to 0.11)	0.25	-0.01±0.02 (-0.05 to 0.02)	0.27
diameter	0.03±0.04 (-0.05 to 0.10)	0.21	-0.02±0.02 (-0.05 to 0.01)	0.31
modularity	0.04±0.04 (-0.04 to 0.11)	0.33	0.02±0.02 (-0.01 to 0.05)	0.33
centralization:group size	0.05±0.04 (-0.02 to 0.13)	0.30	**0.03±0.02 (0.0003 to 0.06)**	**0.35**
ccoef:group size	-0.01±0.05 (-0.12 to 0.09)	0.05	0.02±0.02 (-0.02 to 0.05)	0.07
diameter:group size	-0.05±0.04 (-0.12 to 0.02)	0.04	0.005±0.01 (-0.02 to 0.03)	0.05
modularity:group size	**-0.06±0.03 (-0.11 to -0.001)**	**0.31**	-0.01±0.02 (-0.04 to 0.02)	0.06
Stage 5				
intercept	4.47±0.02 (4.43 to 4.51)	contained in all models	4.52±0.01 (4.50 to 4.53)	contained in all models
group size	**0.05±0.02 (0.01 to 0.08)**	**0.80**	**0.04±0.01 (0.02 to 0.06)**	**1.00**
centralization	-0.01±0.02 (-0.05 to 0.03)	0.21	0.01±0.01 (-0.008 to 0.03)	0.29
ccoef	-0.01±0.02 (-0.05 to 0.04)	0.21	0.01±0.01 (-0.01 to 0.03)	0.34
diameter	**-0.04±0.02 (-0.08 to -0.001)**	**0.64**	0.01±0.01 (-0.01 to 0.03)	0.24
modularity	0.04±0.02 (-0.001 to 0.08)	0.61	0.005±0.01 (-0.01 to 0.02)	0.20
centralization:group size	0.02±0.02 (-0.02 to 0.06)	0.04	-0.001±0.01 (-0.02 to 0.02)	0.06
ccoef:group size	-0.01±0.02 (-0.06 to 0.03)	0.03	-0.01±0.01 (-0.03 to 0.01)	0.09
diameter:group size	-0.02±0.02 (-0.05 to 0.02)	0.14	-0.01±0.01 (-0.03 to 0.004)	0.14
modularity:group size	0.003±0.02 (-0.03 to 0.04)	0.09	-0.01±0.01 (-0.03 to 0.01)	0.09

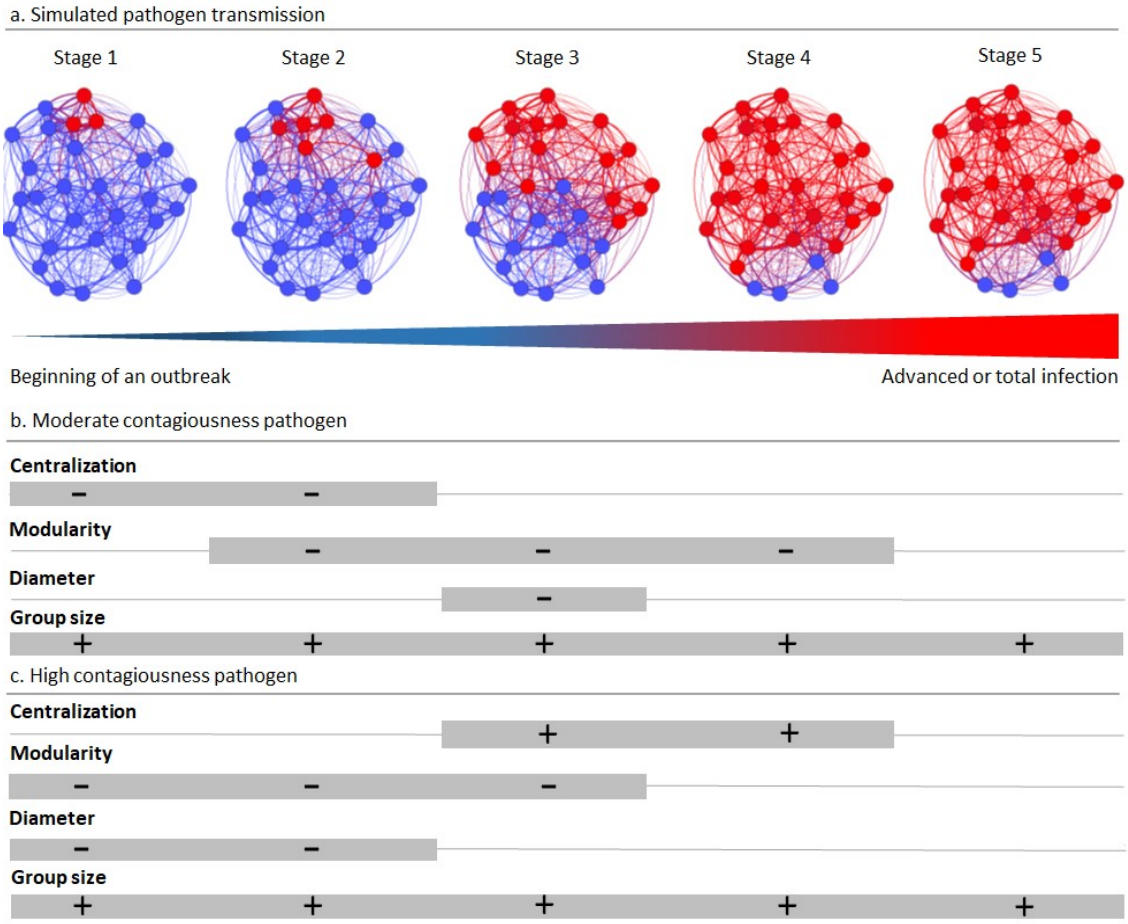

Figure IV.1. Social network properties interact with group size to predict prevalence through the advance of an outbreak. Depending on the stage of the outbreak (a), centralization and modularity interacted with group size to predict the percentage of infected individuals. Group size positively influences the spread of pathogens at all stages of an epidemic. However, during the transmission of a moderately-contagious pathogen, centralization at stages 1 and 2 negatively influence the spread of disease and in stages 2, 3 and 4, modularity does the same (b). If a highly-contagious pathogen is included in the system, modularity (stages 1, 2 and 3) decreases the epidemic size and then centralization (stages 3 and 4) positively influences the average number of infected individuals in each group (c). Diameter also reduces prevalence. Effects are present only at stages 1, 2 and 3 of the outbreak (b and c). In the networks (a), blue denotes non-infected individuals while red reflects infected individuals. The influence of network properties across outbreak stages are represented by the bars in parts b and c, with the sign reflecting the direction of the effect.

IV.5. Discussion

One of the intrinsic costs of sociality is the risk of acquiring socially-transmitted pathogens from conspecifics (Loehle 1995; Altizer et al. 2003; Kappeler et al. 2015). Although the rate

of pathogen transmission is expected to increase with group size (Freeland 1979; Ezenwa 2004; Whiteman & Parker 2004; Caillaud et al. 2013), some properties of the social network in which individuals are embedded can offset this transmission process. The so called *social bottleneck hypothesis* predicts that increased group substructure (i.e. modularity), formed in larger groups, could offset the spread of infection and thereby modulate the relationship between group size and infectious disease transmission (Nunn et al. 2015). Our agent-based SI model confirmed that increased modularity reduce pathogen spread (Wilson et al. 2003; Huang & Li 2007; Salathé & Jones 2010), and showed that network centralization (i.e. the tendency for a few individuals to dominate social interactions) also predicts pathogen transmission, though in contrasting ways depending on the reproductive number, i.e. transmissibility, of the pathogen. To a lesser extent, diameter interacted with group size and also decreased prevalence. Finally, the influence of these network properties was related to the stage of the outbreak and to the reproductive number of the pathogen in question, as described in the following paragraphs.

The interaction between Newman's modularity and group size, for example, influenced transmission of the highly-contagious pathogen at earlier stages (1, 2 and 3) than did the moderately-contagious pathogen (2, 3, 4). The contagiousness of a pathogen might increase the influence of the individual network properties compared to the global networks ones. This might cause variation in effects, not only of Newman's modularity but also of eigenvector centralization, diameter and clustering coefficient. It is known that increased transmissibility of a pathogen increases the per-contact transmission rate (β) - the probability that one individual will transmit an infectious agent to another (Rothman et al. 2008). When a moderately contagious pathogen is introduced into the system, the effects of individual properties may be less apparent. In consequence, properties of the network at the individual level might affect global network properties in distinct ways. For example, Griffin & Nunn

(2012) found that the effect of eigenvector centralization is dependent on β, the per-contact transmission rate, with centralization showing a negative effect on transmission when β is low and a positive effect when β is high. This might be a consequence of network heterogeneity, with peripheral individuals in highly centralized networks evading infection (Lloyd-Smith et al. 2005). Eigenvector centralization then acts as a facilitator of social transmission when highly-contagious pathogens are introduced into the system. The formula of Newman's modularity also is based on individual eigenvector centrality, but contrary to eigenvector centralization, we observed a continuous negative effect of modularity on epidemic size, as found by Griffin & Nunn (2012) and Nunn et al. (2015). This is again a consequence of the local structure, even if the pathogen is transmitted faster from one individual to another, once the pathogen falls into the subgroup, there will be a decrease in the rate of transmission outside of the subgroup. Recent studies suggest this is driven by two mechanisms of modular organization: network fragmentation and subgroup cohesion (Sah et al. 2017).

For the other properties, similar effects are expected to happen. For example, highly-contagious pathogens might transmit faster, but properties such as increased network diameter, i.e. the distance from the spreader to the most peripheral individual in the group, can reduce the number of individuals becoming infected. In relation to a network's clustering coefficient, i.e. how densely individuals are connected to their neighborhood, its relationship with pathogen spread will depend on the strength of the other individual connections. If central individuals are connected to another central individuals, pathogens may spreader faster, but if infected individuals have low clustering coefficients, this will reduce pathogen transmission.

Among the network properties we tested in this study, modularity has been considered the main predictor of pathogen transmission under the *social bottleneck*

hypothesis, and has furthermore been investigated through the lens of social evolution (Griffin & Nunn 2012; Nunn et al. 2015). As suggested by Nunn et al. (2015), increased group substructure could be a byproduct of natural selection to reduce the costs of connection between individuals, as it acts as counterstrategy to infectious disease risk in larger groups. As in any social system, what influences the social structure and consequently drives the emergence of network properties is the collection of individual decisions and behavior (Hinde 1976, Whitehead 2008a). Social relationships are dynamic and individuals can adjust their relationships according to their partners' behaviors (e.g. guppies: Croft et al. 2005; bullfrogs: Kiesecker et al. 1999; vervet monkeys: Fruteau et al. 2009; humans: Rand et al. 2011; mice: Lopes et al. 2016; mandrills: Poirotte et al. 2017). Modular structure then can emerge as a consequence of social preferences, social style (Sueur et al. 2012a) or avoidance of social interactions (Lopes et al. 2016). Indeed, theoretical studies investigating the evolutionary origins of modularity in other biological networks demonstrated that selection to reduce connection costs causes the emergence of modular networks (e.g. proteins and neural networks: Wagner et al. 2007; Clune et al. 2013). Our study offers evidence that modular networks may reduce the costs associated with infectious disease in mammalian societies as well, independent of the reproductive rate of the pathogens that were simulated here. However, it is important to bear in mind that not only one network property but the integration of many can affect pathogen transmission processes. Further studies manipulating the costs and benefits of social relationships are thus necessary to understand the mechanisms of social network evolution in terms of how individual behavior is selected and reflected in the social structure of a given group or species.

We reiterate that an integrative approach, combining epidemiological models, social network analysis and experimental studies to quantify variation in social structure in wildlife, is necessary to better understand the drivers of pathogen transmission and social evolution.

Here, we offer the first theoretical evidence, derived from real-world primate networks, showing temporal variation in the effect of network structure and group size on infectious disease transmission during simulated outbreak conditions. Whilst providing opportunities to better understand how social networks evolve through natural selection acting on individual behavior, these current results should also help us to better target which network properties are more important than others at each time step during an epidemic and contribute to targeted strategies to avoid or mitigate outbreaks in real-world situations.

Conflict of interests: The authors declare no competing interests.

Authors' contributions: VR, CS and AJJM conceptualized the study. VR wrote the manuscript and performed the statistical analysis. CS, CP, JD and AJJM provided support for the statistical analysis. All authors were involved in collecting behavioral data and/or managing field sites.

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Chapter 3

Network efficiency and modularity

V. CHAPTER 3:

Network efficiency peaks with intermediate levels of group substructure

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V.1. Abstract

(English) In myriad complex systems, including human societies, multiple lines of evidence indicate that group substructure, i.e. modularity, may delay transmission processes (e.g. socially-transmitted pathogens). There is increased interest in understanding variation in the effects of group substructure on transmission processes, as it may provide important insights into a given network's performance in addition to the ultimate consequences it may have for individual fitness in evolutionary terms. In this study, we investigated to what degree network efficiency, as a proxy for social transmission, is modularity-dependent. We created 2798 networks varying in group size and density, and tested whether network structure (density, modularity, centralization) and group size shape network efficiency. We also used published data from 68 primate social networks to test whether the predictions generated in our simulations were supported by empirical observations. Our results show a non-linear relationship between modularity and global efficiency, with the latter peaking at intermediate values of group substructure in both theoretical and empirical networks. This phenomenon may have relevance to observed variation in social structure and its link with network performance. Our results may thus provide a basis on which to discuss the evolution of complex systems, including animal societies.

Keywords: network efficiency, modularity, theoretical and biological networks, social transmission

(Français) Au sein d'une myriade de systèmes complexes, incluant les sociétés humaines, de multiples faisceaux de preuves indiquent que la sous-structure de groupe, comme la modularité, peut permettre de retarder le processus de transmission (dans le cas de pathogène transmis socialement). L'étude et la compréhension des variations des effets de la sous-structure de groupe sur les processus de transmissions pourrait apporter des éclairages importants sur la performance d'un réseau donné mais aussi sur les conséquences que cela pourraient avoir sur la fitness individuelle dans le sens évolutif du terme. Cela explique ainsi l'intérêt croissant des chercheurs pour cette thématique. Dans cette étude, nous avons étudié à quel degré l'efficacité de réseau, représentant la transmission sociale, est dépendant de la modularité. Nous avons créé 2798 réseaux de taille et densité différentes et testé comment la structure du réseau (densité, modularité, centralisation) et la taille du groupe affectent l'efficacité de réseau. Nous avons également utilisé des données publiées de 68 réseaux sociaux afin de tester si nos prédictions obtenues par simulation sont supportées par des observations empiriques. Nos résultats montrent une relation non-linéaire entre la modularité et l'efficacité globale, avec un pic tardif pour des valeurs de sous-structures de groupe intermédiaires dans les réseaux théoriques et empiriques. Ce phénomène pourrait être important dans l'étude des variations observées dans la structure sociale d'un groupe et son lien avec la performance du réseau. Nos résultats peuvent également servir de base à des futures discussions sur l'évolution des systèmes complexes, incluant les sociétés animales.

Mots-clés: Efficacité de réseau, modularité, réseaux théoriques et biologiques, transmission sociale

V.2. Introduction

Networks are ubiquitous in complex systems, ranging from human created communication networks to individuals' connections in companies, schools and local communities (Barabási 2016). Through network analysis, the frequency and strength of interactions are mathematically quantified to assess the level of connectivity, the importance of nodes in the network, and the impact of node removal in the chain of transmission. Besides the vast applicability in social science and computer engineering, social network analysis has in the few last decades been more frequently used in the biological sciences. In the field of animal behavior, for example, it is now known that the social structure is linked with individual fitness and that the network position can be used to infer the success of reproduction (Formica et al. 2012), the risk of contagion (Rushmore et al. 2013), and probability of survival (Silk et al. 2003). While previous studies have contributed significantly to the development of this field, much remains unknown (Kurvers et al. 2014), such as the mechanisms that shape social structure and the role of such structure in shaping transmission processes (Farine 2017).

In the animal kingdom, social transmission can occur in diverse ways (e.g. through body contact or inter-individual proximity favoring pathogen contagion or information spread, VanderWaal et al. 2014b, Aplin et al. 2015) and may directly affect individual survival. The use of social information, for example, is expected to have evolutionary advantages and may regard food location, increased predator protection and partner selection (Danchin et al. 2004; Dall et al. 2015; Kendal et al. 2005; Duboscq et al. 2016a). On the other hand, the spread of socially-transmitted pathogens may have profound impacts on animal populations (Plowright 1982; Heide-Jorgensen et al. 1988; Walsh et al. 2009) and is considered to be one of the main stochastic threats to wildlife (Deem et al. 2001). As such,

the heterogeneous distribution of social connections within a group or population create varying opportunities for social learning (Coussi-Corbel & Fragaszy 1995), decision-making (Strandburg-Peshkin et al. 2013) and contagion risk (MacIntosh et al. 2012; VanderWaal et al. 2014a,b; Duboscq et al. 2016b). The structure of a group, population or community thus has important consequences for the social transmission and fitness of individuals.

In terms of social network analysis, network efficiency quantifies how fast an element (e.g. computer viruses) can be spread through the network with the minimum number of connections. A more efficient network is known to have a higher number of informed/infected individuals, be more cohesive, and present hierarchical structure (Flack et al. 2015), which might be related to each individuals' role, as well as the social structure (Conradt et al. 2009; Sueur et al. 2012b; Pasquaretta et al. 2014; Flack et al. 2015). In a study comparing 80 groups of primates, for example, research demonstrated that more despotic groups had less efficient networks (Pasquaretta et al. 2014). Furthermore, leaders occupying central positions improved the efficiency of the decision-making process (Sueur et al. 2012b). Increased network efficiency is suggested to allow individuals to quickly adapt to changes in the environment, but it also should increase their connection costs (Sueur 2011). As a consequence of this fitness trade-off, there has been considerable discussion on whether and how network properties also down-regulate transmission processes by decreasing network efficiency.

Among the various emergent properties of networks, it has been argued that increased modularity - a global network index used to estimate the level of group substructure - is a major contributor to the capacity of biological networks to evolve, as seen in animal brains, protein networks, and bacterial metabolic networks (Wagner et al. 2007). Computational evolution experiments have also demonstrated that modular networks maximize network performance and minimize connection costs, and are therefore more likely to evolve and are

themselves more evolvable than less modular networks (Clune et al. 2013). Furthermore, theoretical and empirical studies of mammal social structure have highlighted that increased modular structure, formed naturally in larger groups, can act as a barrier to socially-transmitted pathogens (Griffin & Nunn 2012); the so-called 'social bottleneck hypothesis' (Nunn et al. 2015). Collectively, such evidence suggests that modularity can influence social transmission by decreasing the costs of relationships. More recently, however, Sah et al. (2017) suggest that disease risk is largely unaffected by modularity, and that only beyond a threshold at high values of modularity do social networks result in decreased pathogen transmission.

Within this framework, we combined a theoretical and empirical approach to investigate to what degree network efficiency is modularity-dependent, and whether a modularity threshold exists in the efficiency of social transmission processes. Our assumptions are that density (Keeling & Rohani 2008) and group size (Patterson & Rusckstuhl 2013) shape network heterogeneity, here measured in terms of global network properties. Besides investigating how the degree of modularity might influence efficiency, and in order to take a broader view of network structure, we also investigated the effect of network centralization on network efficiency. Highly centralized networks are those in which one or few individuals are highly connected to other individuals in the group and dominate network interactions. In terms of social transmission, centralization is related to network efficiency (e.g. centralization index negatively affects efficiency, Pasquaretta et al. 2014) and decision-making processes (e.g. individuals lost their leadership by moving from decentralized to centralized networks; Sueur et al. 2012a).

In accordance with the social bottleneck hypothesis, we predicted that larger social groups would exhibit increased modularity and thus decreased network efficiency (i.e. social transmission, Nunn et al. 2015). Alternatively, incorporating recent evidence from Sah et al.

(2017), we might predict little significant change in network efficiency with increasing modularity until some threshold is reached, beyond which efficiency is expected to decline. As this is a current topic of debate, our focus on simulated truncated networks facilitates investigation into the direct influence of subgroups on network efficiency. While accounting for a threshold effect of modularity on transmission processes is certainly important in the field of epidemiology (Sah et al. 2017), we further suggest that if such a threshold exists, it should be broadly observable in theoretical and empirical social networks. We then tested our predictions with real association data collected from 68 primate groups to test the outputs of our theoretical models and explain the link between modularity and network efficiency. Finally, we also expect that if centralization is a good predictor of network efficiency, highly centralized networks will decrease transmission speed, as has been observed in an empirical study (Sueur et al. 2012a).

V.3. Material and Methods

V.3.1. Creating truncated networks

We constructed 2798 undirected binary networks in R v.3.3.1 (R Core Team 2016) with the function "sample_pa" from the "igraph" package v.1.0.1 (Csárdi & Nepusz 2006). This function allowed us to create truncated power-law-distributed networks according to the Barabási-Albert model (Barabási & Albert 1999) with degree exponent $\gamma=2$, an arbitrary number considering that most real systems present a degree exponent higher than 2 (Barabási 2016). Many systems are well-approximated by power-law-distributed network models (e.g. World-Wide Web, Albert et al., 1999; airport network of India, Bagler 2004; protein-protein interactions, Jeong et al. 2001; networks of market investments, Garlaschelli et al. 2004),

including in the animal kingdom (e.g. human sexual networks, Liljeros et al. 2001; bottlenose dolphins, Lusseau 2003; tonkean and rhesus macaques, Sueur et al. 2012b). We were interested in truncated power-law-distributed networks because they are structurally characterized by the numerous small-degree nodes coexisting with hubs, a few nodes that are highly-connected to other nodes in the network (Barabási 2016). In terms of social transmission, their presence may increase the speed of transfer, the so called super-spreader effect (Fujie & Odagaki 2005; Llyod-Smith et al. 2005), and were described during the outbreak of many infectious diseases (Stein 2011). We designed networks with sizes similar to those found in animal groups, and following a set of Fibonacci numbers (a pattern commonly observed in nature; 8, 13, 21, 34, 55, 89, 144) with different densities to explore the links between density, centralization, modularity and size. Network density was shifted between values of 0 and 1, meaning the denser the network the lower the truncated structure. We aimed to explore a broad combination of network properties, and 2798 networks were the possible number of networks we could create based on method used. R code is available in the **Appendix H**.

V.3.2. Estimating association indices

We estimated *eigenvector centralization* and *Newman's modularity*; two network properties that are expected to be mathematically, but not necessary empirically, independent of group size, and which provide a global view of network structure (Nunn et al. 2015). Both network metrics were estimated in R.

Eigenvector centralization captures variation in connectedness by comparing all centralities with that of the most central individual. It was calculated as:

$$C = \frac{\sum_{i}^{N} (Cmax - Ci)}{Max \sum_{i}^{N} (Cmax - Ci)}$$

where C is the centralization index, Ci is the centrality for individual i, C_{max} is the maximum value of Ci across all individuals and $Max \sum_{i}^{N} (Cmax - Ci)$ refers to what the sum would be under the largest possible centralization of the network (Pasquaretta et al. 2014). Highly centralized networks resemble a star, where all individuals are connected to one individual in the center of the group. In this condition, the centralization index would be approximately 1.

Modularity reflects the presence of large subgroups of nodes that are typically highly connected internally but only loosely connected to other subgroups in the networks (Girvan & Newman 2002). We estimated the degree of modularity in our theoretical networks using the function "cluster_leading_eigen" provided in the "igraph" package. We decided to use Newman modularity (Q) as it considers the eigenvalues of a matrix while avoiding estimation of modules using fixed values of group size. It is also expected to provide a more realistic estimation of network substructure (Newman 2006). When Newman's modularity is equal to 0, the density of interactions within subgroups is equivalent to the density of interactions between subgroups. Higher values of Newman's modularity indicate stronger substructuring of social networks, with values close to 1 denoting greater modular structure.

V.3.3. Estimating network efficiency

To represent social transmission, we used a measure of network efficiency called *global efficiency*, which considers the ratio between the number of group members N and the number of connections I multiplied by the network diameter D:

$$Global\ Efficiency = \frac{N}{I * D}$$

In other words, global efficiency denotes how quickly information is transmitted from the spreader to the most peripheral individual in the group. Global efficiency can range from 0 to 1, with more efficient networks having values closer to 1. This is a consequence of the nature of our networks, with most having diameters higher than 2; higher diameters decrease global efficiency. Highly dense networks are also expected to have reduced network efficiency, since in these cases *I* is considerably higher than *N*.

V.3.4. Empirical data

We obtained published data on global efficiency and modularity originally measured for 68 primate social groups of 21 species, including 4 groups of humans. Non-human data included groups from captivity and the wild (Pasquaretta et al. 2014; **Table HS1, Appendix H**). The authors did not found evidence that phylogeny affected the network measures observed (Pasquaretta et al. 2014). Networks were created based on socio-positive relationships (such as body contact, social grooming and/or proximities), and were in all cases weighted and symmetrized (Pasquaretta et al. 2014). We have used the same coefficients as those used in the reference study (Pasquaretta et al. 2014) to estimate modularity (i.e. Newman's Modularity) and global efficiency, but while they calculated Newman's modularity in SocProg 2.4 (Whitehead 2009), we used the package "igraph" in R.

V.3.5. Statistical analysis

To test whether network efficiency is influenced by network structure and group size, we constructed a generalized linear model (GLM) with a gamma error distribution and inverse link function (e.g. observed and gamma distribution functions of global efficiency: **Figure HS1**, **Appendix H**) using the "car" package v.2.1-3 in R. We ran a series of diagnostic tests to judge the validity of the models, including testing for variance inflation and Cooks' distance. Density was highly correlated with centralization and modularity (**Figure HS2**, **Appendix H**) and, as it was not a major focus of our study, was removed from the analysis. All diagnostic tests suggested the validity of our models (Field et al. 2012). Full models included centralization, modularity and group size as explanatory variables.

To assess whether there were multiple relationships between modularity and network efficiency, we used a piecewise regression performed with the package "segmented" v.0.5-1.4 (Muggeo 2008) in R. 'Breakpoints' are defined as the boundaries between the line segments that fit the regression, and are expected when the independent variable exhibits different relationships with the predictor in these regions. We performed the piecewise regression as a single analysis with all group sizes combined, and as separate analyses for the size categories small (8, 13), medium (21, 34, 55), and large (89, 144) to allow for better understanding of variation in the observed breakpoints. Exploratory analysis included the combination of 8, 13 and 21 individuals in small groups, but this did not change our general results. We then performed the same piecewise approach with the empirical data, which included only small- (N= 49; ranging from 5 to 14; mean= 9.4) and medium-sized (N= 19; ranging from 17 to 38; mean= 23.7) groups. Finally, to understand if the outputs from theoretical networks differed from the empirical networks, we compared (i) the distribution of modularity and global efficiency using Kolmogorov–Smirnov tests, (ii) the slopes and the

intercept values of the regression lines by applying an ANCOVA test and a linear regression, respectively, and finally (iii) we compared the median of global efficiency and modularity found in each set of networks using a Wilcoxon test. The alpha level was set at 0.05. Graphs were created using the package "ggplot2" v.2.2.1 (Wickham 2009) and the function "plot" from the base package in R.

V.4. Results

In our theoretical networks, global efficiency values ranged from 0 to 0.5, modularity ranged from 0 to 0.83 and centralization varied from 0 to 0.81. Modularity and group size were good predictors of global efficiency in theoretical networks, with larger group sizes having lower global efficiency scores (**Table V.1**). Results concerning modularity were less clear. When all theoretical networks were observed together, we found a positive association between modularity and global efficiency (**Table V.1**). However, modularity seemed to play two distinct roles in mediating transmission processes: increasing values of modularity at the lower end of its range tended to increase network efficiency, while increasing values at the higher end of its range decreased transmission rates (**Figure V.1**). A breakpoint at $Q = 0.58$ was observed in the regression line when considering all theoretical networks together. This result indicates the occurrence of two segments, one before the breakpoint with a slope of 0.02 and a secondary slope after the breakpoint at -0.44.

Table V.1. Parameter estimates from generalized linear models explaining variation in global efficiency. Data extracted from the theoretical networks. Bold values indicate p < 0.001.

Predictors	Estimate	Std. error	t value	Pr (>|t|)
Intercept	2.90^{e-02}	1.82^{e-04}	159.08	**<2e-16**
Centralization	5.67^{e-05}	1.29^{e-04}	0.44	0.66
Modularity	2.02^{e-02}	4.25^{e-04}	47.54	**<2e-16**
Group size	-8.82^{e-03}	1.24^{e-04}	-71.15	**<2e-16**

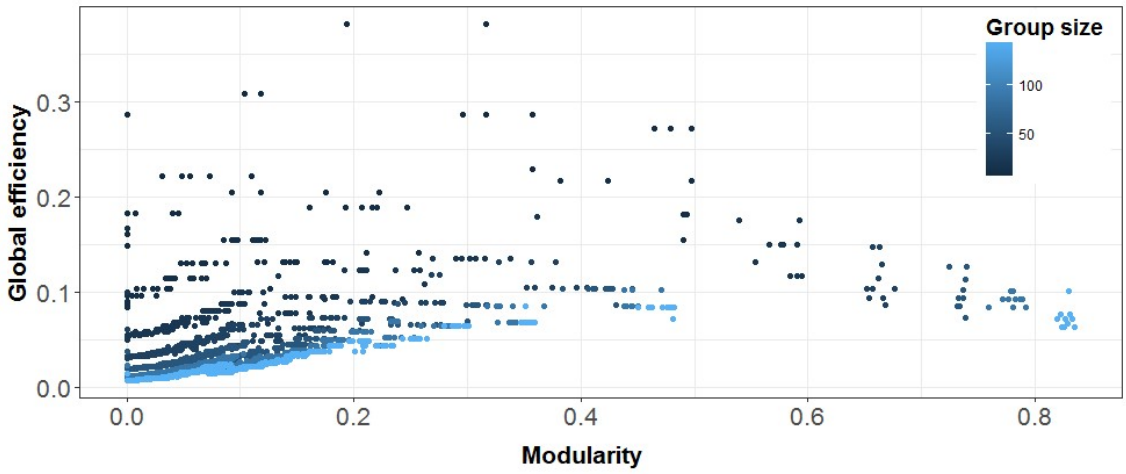

Figure V.1. Relationship between global efficiency and modularity from all groups of the theoretical networks. Data is presented in function of group size.

More specifically, we observed three distinct breakpoints at Q = 0.35, 0.53 and 0.69 when groups were classified as small, medium and large, respectively (**Figure V.2**). First slopes were equal to 0 at all group sizes, and the secondary slopes were -0.67, -0.52 and -0.51 for small, medium, and large groups, respectively. It is important to note that small groups were underrepresented by high values of modularity, but we could still identify a negative slope in the secondary segment. We then looked at the data from empirical networks and

observed that breakpoints also existed at 0.17 for small groups and 0.30 for medium groups. Slopes from the regression lines of empirical groups were as follows: 0.64 and -0.36 for small groups, and 0.55 and -0.02 for medium groups (**Figure V.3**). We observed that the distribution of modularity and global efficiency from small and medium theoretical networks were different from those of small (Modularity: D = 0.63, p = 1.09^{e-12}; Global efficiency: D = 0.24, p = 0.04; **Figure HS3, Appendix H**) and medium empirical groups (Modularity: D = 0.52, p = 0.0002; Global efficiency: D = 0.35, p = 0.03; **Figure HS4, Appendix H**). Overall, the median of global efficiency and modularity found in empirical networks were higher than those of theoretical networks, independent of group size category (small - global efficiency: W = 5442, p = 0.005; modularity: W = 5742.5, p = 0.0007; medium - global efficiency: W = 3829, p = 0.01; modularity: W = 5250, p = 6.5^{e-13}). However, the slope and intercept of regression lines were only distinct for small groups (p = 1.47^{e-08}), those being higher in empirical than in theoretical networks (**Figures HS3 and HS4, Appendix H**). In general, our results indicate that peaks at intermediate levels of social subgrouping may optimize social transmission, a predicted pattern in theoretical networks that was corroborated in empirical data, though at different absolute values.

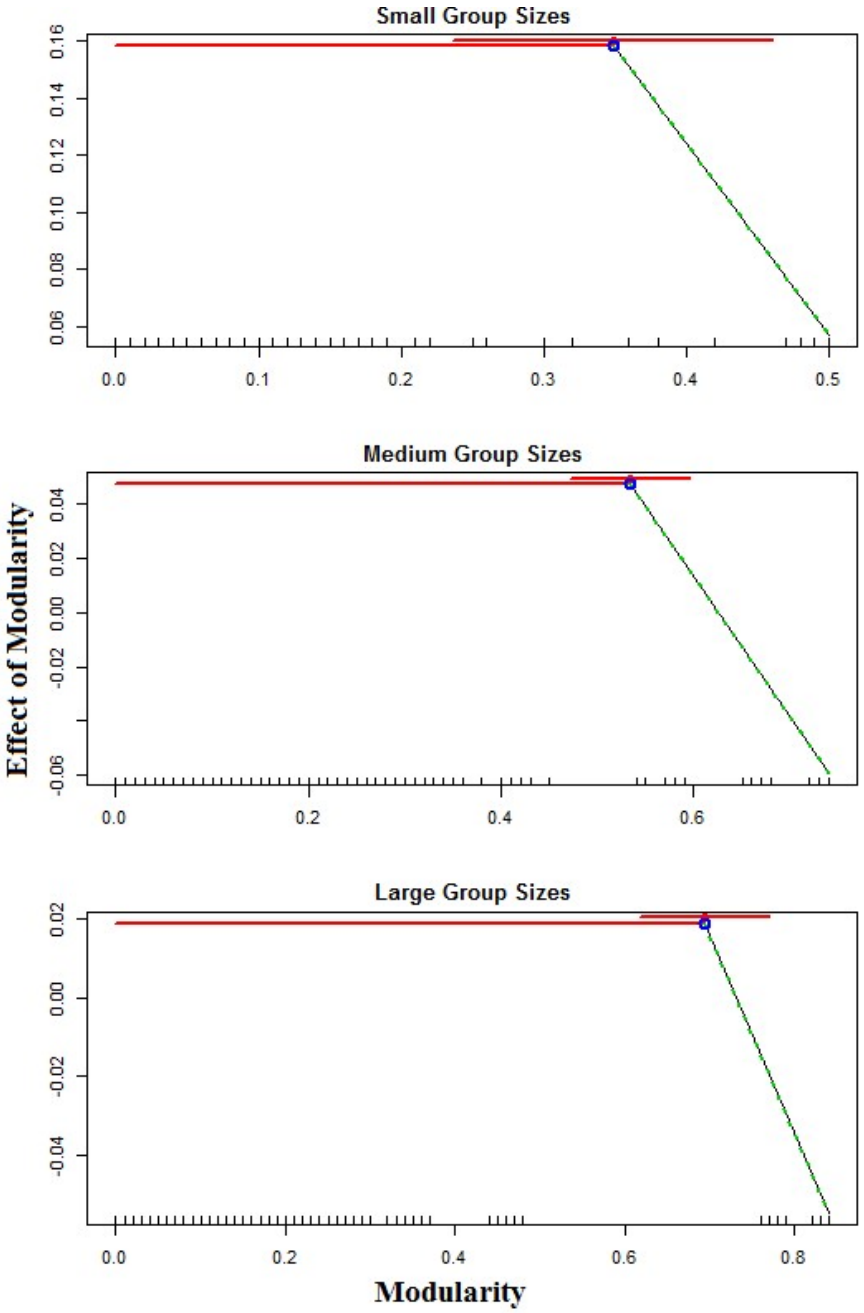

Figure V.2. Breakpoints in the regression lines observed in intermediate levels of modularity from small, medium and large theoretical networks. Red lines represent the first slope, dashed green lines represent the secondary slope and blue circles are the breakpoints in the regression lines (values in the main text). Red horizontal lines above the blue circles indicate variance around the breakpoint.

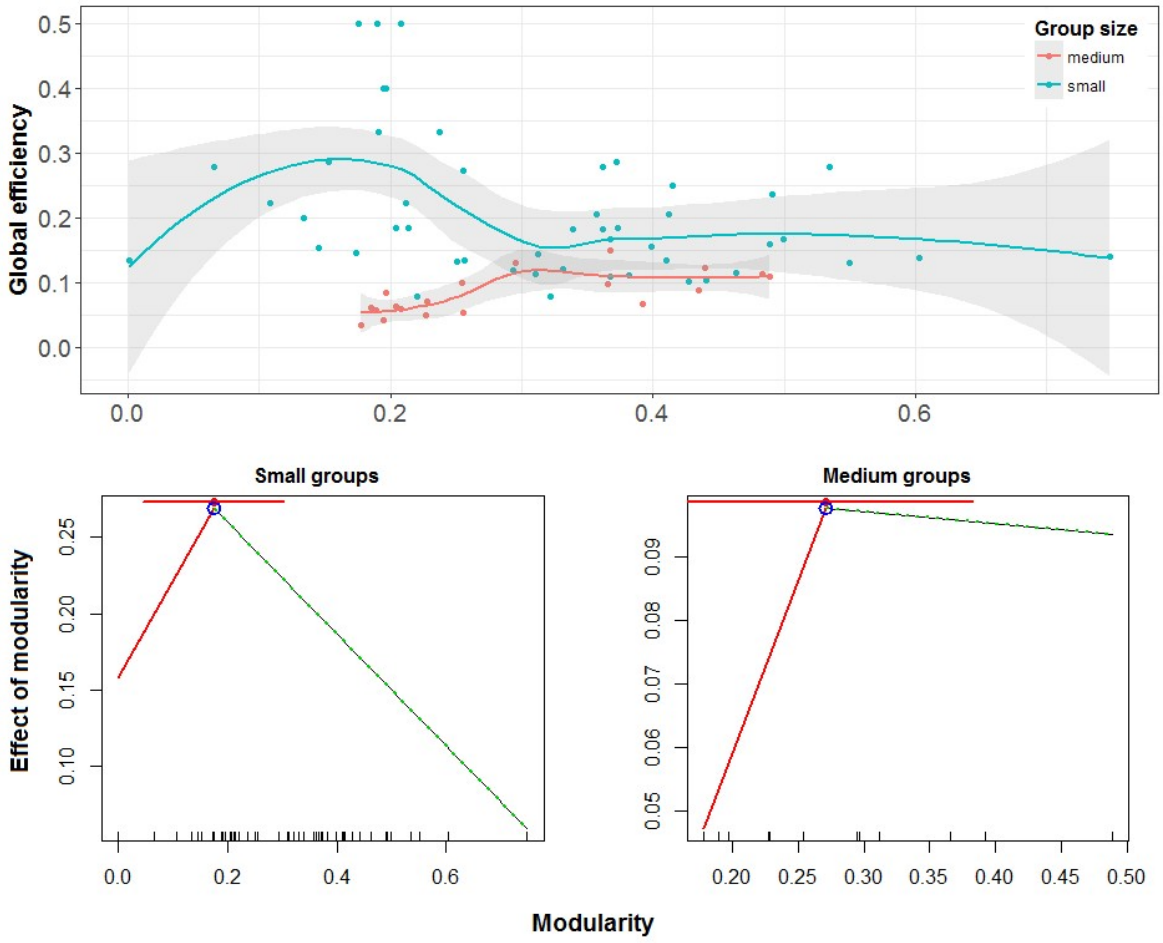

Figure V.3. Relationship between global efficiency and modularity for empirical networks (upper figure) and breakpoints in the regression lines observed in small and medium-sized groups (lower figures). Red lines represent the first slope, dashed green lines represent the second slope and blue circles are the breakpoints in the regression lines (values in the main text). Red horizontal line above the blue circle indicate the variance of data around the breakpoint.

V.5. Discussion

It is well-established that social connectivity modulates transmission processes in real networks (Kappeler et al. 2015; Duboscq et al. 2016a), but to what degree transmission efficiency is modularity-dependent has received less attention in the literature. We found evidence that modular structure in social groups can have nonlinear effects on transmission processes, with low values of modularity tending to positively influence social transmission

and high values tending to negatively influence transmission. This pattern was consistent across small, medium and large truncated networks and was corroborated by our empirical primate networks, including small- and medium-sized groups. With respect to the existing literature (Nunn et al. 2015; Griffin & Nunn 2012), our findings support the prediction that increased modularity constrains social transmission, but further suggest that intermediate levels of network substructure produce the highest efficiency.

Over the last decade, considerable evidence has arisen to suggest that modular organization might negatively influence social transmission processes in biological networks (Wagner et al. 2007; Clune et al. 2013; Sah et al. 2017). While some studies provide support for the "social bottleneck hypothesis", i.e. the larger the group size the higher the modularity and the lower the speed of transfer (Griffin & Nunn 2012; Nunn et al. 2015), few studies have found modular structure to increase diffusion due to higher connectivity within subgroups (Lentz et al. 2012; Nematzadeh et al. 2014). Recently, Sah et al. (2017) suggest that reduced outbreak size in highly modular social networks might be caused by two mechanisms related to modular organization: network fragmentation (i.e. the number of subgroups) and subgroup cohesion (i.e. preferential association within subgroups). In this study, however, reduced outbreak size was only apparent in social networks with $Q = 0.6$ for moderately spreading pathogens. The authors proposed that the negative effect might be related only to high values of modularity, and that an epidemic threshold might exist, below which there is a minimal risk of a large outbreak, and depend on pathogen transmissibility (Sah et al. 2017). While their focus was specific to epidemiology, we propose here that the peak in network efficiency, and thus transmission potential, at intermediate values of modularity might occur at a broader scale, meaning that it is not only related to outbreak size but also to other social transmission processes. While Sah et al. (2017) identified an epidemic threshold, our study suggests the existence of a peak, in which modularity at first tends to

favour transmission but at higher values tends to constrain social transmission. What mechanisms drive this phenomenon, however, remains an open question. For our theoretical networks, the pattern observed is a consequence of a correlation between network properties: high density implies low modularity, and vice versa. As the degree of modularity increases, network transmissibility decreases. However, for empirical networks, as modularity and efficiency are higher than for theoretical data, behavioral mechanisms might be at play.

In animal social networks, it is broadly accepted that sociality incurs both costs and benefits. Among them, the same properties of the social structure that favour the spread of benefits, such as behaviors that transmit information, might also favour the spread of costs, such as socially-transmitted pathogens that may cause disease, creating a trade-off between information and parasite transmission. If the topology of the network can be optimized in a way that balances the costs and benefits of interaction, the peak observed in global efficiency at moderate sub-structuring might be the first piece of evidence suggesting that variability in individual social behavior can lead to the emergence of global properties that might reflect this trade-off, the accommodation of both transmission processes. In nature, real networks are a consequence of individual decisions, and it has been empirically demonstrated that individuals can modulate their social connections to decrease the costs of sociality (e.g. immune-challenged mice reduced their own social connectivity: Lopes et al. 2016; wild mandrills avoided grooming at the peri-anal area of infected individuals: Poirotte et al. 2017) and increase their own benefits (e.g. wild vervet monkeys increased their grooming rates with individuals holding information, Fruteau et al. 2009). Social networks are thus expected to be dynamic, varying flexibly with the needs of the individuals involved according to the costs and benefits of their relationships and as a consequence of their interactions. Rather than proposing a single optimization point, we suggest a range of modularity across which we

might expect to find real-world networks if interactions were occurring to balance costs and benefits of relationships that depend on network structure.

The results of our compiled set of empirical networks demonstrate that real systems also exhibit a non-linear relationship between modularity and efficiency, although to a lesser degree than our theoretical networks. We observed that the slope and intercept of the regression line from small empirical groups were both higher than those from theoretical networks. Interestingly, the slope of the first regression line was positive for the empirical networks, unlike our theoretical networks, raising the possibility that observed networks reflect the dual role of modularity in favoring social transmission at small values and suppressing it at large values. The latter process has been already identified by theoretical researches (Nunn et al. 2015; Sah et al. 2017). While interesting, more data and further tests are required to resolve this possibility. An important avenue for future research will be to link variation in individual behavior, which affects modularity and efficiency of networks, to both positive and negative elements of social transmission. In animal social systems, behaviors stemming from nepotism (higher tolerance of kin) or despotism (manifest as aggressive behavior linked to dominance hierarchy) are known to increase modularity (e.g. macaques social style, Sueur et al. 2011b; Puga-Gonzales & Sueur 2017), while discriminating between conspecifics with different levels of infection or information (Fruteau et al. 2009; Poirotte et al. 2017) may further influence emergent network properties like modularity.

In conclusion, our approach revealed that network efficiency is modularity-dependent, with the highest values of social transmission occurring at intermediate levels of modularity. The description of such phenomena suggests that future work manipulating the costs and benefits of social relationships, and ultimately network efficiency itself, might shed light on how networks develop and adapt through the behaviors of individuals given the prevailing environmental constraints. Looking at the mechanisms that generate flexibility in social

structure might contribute to network science and even enhance our understanding of social evolution.

Ethics: Do not apply.

Data availability: Data available on request.

Competing interests: We have no competing interests.

Authors' contributions: VR, CS and AM conceptualized the study. MS created the theoretical social networks and conducted exploratory analyses; VR performed statistical analyses and wrote the manuscript; AM and CS significantly contributed to the manuscript development; MS and JP provided additional comments.

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Chapter 4

Modeling the evolution of social networks

VI. CHAPTER 4:

Investigating the trade-off between information access and infection avoidance in animal societies: an individual-based model

Valéria Romano, Ivan Puga-Gonzalez, Andrew JJ MacIntosh, Cédric Sueur

VI.1. Abstract

(English) Sociality incurs costs and benefits for animals. While relationships between individuals may favor the spread of valuable information, they also increase the risk of acquiring socially-transmitted pathogens, which ultimately may cause disease, leading to a fitness trade-off of sociality. One of the open questions in behavioral ecology targets how local pressures related to the costs (e.g. contagion risk) and benefits (e.g. information) of specific interactions influence variation in social structure. In a first step toward answering this question, we used a theoretical modeling approach to investigate the evolution of network structure while varying each individual's value in terms of information sharing and pathogen spreading. We tested 20 conditions across group sizes of 10, 30 and 70 individuals. In our model, individuals are initially unaware of the potential for each other individual to share information or spread pathogens, but perceive which individuals are informed and which are infected as they interact. Individuals were programmed to favor informed individuals and avoid infected individuals, and adjust their probabilities of interacting with each group member accordingly. Our results demonstrate that variation in the network properties, in terms of density, centralization and modularity, is a consequence of individual

decisions given the trade-offs between collecting information and avoiding infection. Highly centralized networks, for example, emerge when information was monopolized by few individuals since group members favor informed over uninformed individuals. Unlike previous theoretical studies, which have focused on information and pathogen transmission networks independently, our model accounts for the potential trade-off between information access and infection avoidance in animal societies, and therefore the conditions under which social structure is expected to adapt and evolve.

Keywords: group-living, social behavior, trade-off, network structure, individual-based modeling, network analysis

(Français) Vivre en groupe induit des coûts et des bénéfices pour les animaux. Bien que les relations entre les individus puissent favoriser la diffusion d'informations, ils augmentent également le risque d'acquisition et d'infection par des pathogènes transmis socialement, conduisant ainsi à l'équilibre entre coûts et bénéfices de la vie en groupe. Une des questions toujours en suspens en écologie comportementale s'intéresse à comment des pressions locales liées à des coûts (risque de contagion) et bénéfices (information) d'une interaction spécifique va influencer les variations de la structure sociale. Pour aider à répondre à cette question, nous avons utilisé une approche de modélisation théorique pour étudier l'évolution de la structure du réseau pendant en même temps que les valeurs individuelles en termes de partage de l'information et diffusion du pathogène varient. Nous avons testé 20 conditions différentes au sein de groupe de 10, 30 ou 70 individus. Dans notre modèle, les individus sont initialement non conscient du potentiel de chaque individu de partager des informations et de diffuser des pathogènes, mais perçoivent quels individus sont informés and lesquels sont infectés quand ils interagissent. Les individus ont été programmés afin de favoriser les individus informés et éviter les individus infectés, mais également d'ajuster en fonction les probabilités d'interactions avec chaque individu du groupe. Nos résultats montrent que la

variation des propriétés de réseau en termes de densité, centralité et modularité est la conséquence de décisions individuelles au regard de l'équilibre entre collecte d'information et évitement d'infection. Par exemple, des réseaux très centralisés vont émerger lorsque l'information est monopolisée par quelques individus et que les autres membres du groupe vont favoriser les individus informés par rapport aux non-informés. A rebours d'autres études théoriques qui se sont concentrées sur la transmission d'information et la transmission de pathogène au sein de réseaux de façon indépendantes, notre modèle prend en compte les potentiels compromis entre accès à l'information et évitement de l'infection dans les sociétés animales, et par conséquent les conditions sous laquelle une structure sociale est supposée de s'adapter et évoluer.

Mots-clés: vie en groupe, comportement social, compromise, structure de réseau, individual-based modeling, analyse de réseau

VI.2. Introduction

Understanding the link between individual behavior and the organization and functioning of a group or population has long been central to ecology and evolutionary biology (Hinde 1976; Wilson 1975; Krause et al. 2007). Animals living in groups often interact nonrandomly with conspecifics, leading to variable patterns of social structure (i.e. who interacts with whom), which ultimately feeds back into variation in individual fitness (Sih et al. 2009). Sociality offers many benefits to individuals (e.g. defense against predators, increased foraging efficiency, increased offspring survival: Wilson 1975; Krause & Ruxton 2002), but also comes with costs (e.g. within-group competition for limited resources: Wrangham 1980; increased exposure to infectious agents: White et al. 2017). Social complexity, which can be expressed as patterns of social interaction, then emerges from individual decisions and

strategies to deal with the inherent trade-offs of social-living. Among them, how individuals manage the potential fitness trade-off between information acquisition and infectious disease avoidance has received little direct attention in the biological literature.

Studies of transmission on social networks have suggested that social structure can regulate information transmission and mediate exposure to pathogens via social contact or social proximity (Duboscq et al. 2016a; Rushmore et al. 2017; White et al. 2017). In the majority of studies, the most interconnected individuals in the group (i.e. central individuals), are characterized by fast acquisition of information (Claidière et al. 2013) but also increased exposure to socially-transmitted pathogens (MacIntosh et al. 2012). For example, Aplin et al. (2012) demonstrated that the most central individuals in a wild population of three sympatric tit species (family Paridae) were the first to be informed about new foraging patches. On the other hand, VanderWaal et al. (2014b) demonstrated that the probability of sharing microbial (*Escherichia coli*) subtypes was related to the frequency of association among ungulates (such as Grant's gazelles and zebras). Finally, Page et al. (2017) showed that more central hunter-gatherer women in the Philippines that are more central in their proximity networks produced more living offspring but also suffered from greater disease burdens (i.e. gastro-intestinal disease, influenza and fever, respiratory tract infections and intestinal parasites). This growing body of evidence demonstrating that network structure shapes both information and pathogen transmission raises questions, such as to what extent might network properties interact to modulate transmission processes (Farine 2017; chapter 3). Since the same properties that increase information flow on networks are also expected to increase pathogen spread, it is important to investigate how individuals might balance these and other costs and benefits of social transmission.

Studies in animal biology have typically investigated the flow of information or infectious agents on networks independently, perhaps because of the challenges associated

with exploring both within the same empirical framework, or because of variation in study aims and researcher expertise. However, a computer simulation study has shown that the same properties of the network that influence information flow also influence pathogen transmission (Weng et al. 2013). Of the several network properties under investigation, modularity – a global index that estimates the degree to which a network is divided into subgroups – might emerge if individuals are naturally selected to avoid association costs. For example, avoidance of connection costs has been suggested to drive modularity in other biological networks such as protein and neural networks (Wagner et al. 2007; Clune et al. 2013). In animal societies, it has been suggested that increased modular structure, which is typically associated with larger animal groups, might decrease costly social transmission, such as that involving pathogens (Griffin & Nunn 2012). Although this is currently in debate (Sah et al. 2017), evidence does suggest that the higher is the modularity, the lower is the degree of social transmission observed. Furthermore, studies have shown that other network properties that may be more salient to the individual, such as the number of connections an individual has (individual degree) or the weighted value of their interactions (individual strength), can be used to estimate their degree of exposure to infectious agents in chains of social transmission (e.g. badgers: Weber et al. 2013; Japanese macaques: chapter 1, Romano et al. 2016; chimpanzees: Rushmore et al. 2013; giraffes: VanderWaal et al. 2016). Since these individual-level metrics of sociality influence global properties of networks such as modularity, and can further mediate the costs and benefits individuals receive through social interactions, network structure should reflect the accumulation of individual decisions in the context of social interactions.

Indeed, studies have shown that individual decisions about with whom to interact appear sensitive to the various costs and benefits of sociality. For example, vervet monkeys (*Chlorocebus aethiops*) reinforced social bonds with individuals that provided more food

during an experimental foraging task (Fruteau et al. 2009). Humans were shown to cooperate, form new links (i.e. social bonds) to cooperators, and break links with cheaters/defectors, leading to significant changes in network topology (i.e. social structure; Rand et al. 2011). More recently, studies have begun to provide empirical evidence that individuals can modulate their behavior to decrease connection costs as well. Mandrills (*Mandrillus sphinx*), for instance, may be able to recognize individuals infected with protozoan parasites and avoid grooming their anogenital areas when shedding infective stages (Poirotte et al. 2017). Similarly, healthy bullfrogs (*Rana catesbeiana*) avoided individuals with a yeast infection (Kiesecker et al. 1999). Such findings suggest that individuals may modulate their behavior to increase benefits and avoid costs of social interactions, which should lead to detectable changes in the global network structure.

In the present study, we aimed to explore how individuals might deal with the trade-off between information acquisition and pathogen avoidance in social networks. We developed an individual-based model in which individuals were programmed to favor interactions with others that exhibited higher values of information-sharing potential (hereafter, 'information') and avoid interactions with others that exhibited higher values of pathogen-spreading potential (hereafter, 'pathogen'). Our ultimate goal was to explore the structure of the social networks that emerged under a set of conditions (N = 20; **Figure VI.1**) in which the distribution of information and pathogen values varied systematically across individuals. At the start of each simulation, individuals were unaware of the values of information and pathogen of others, but they learned which individuals were likely to share information and which were likely to spread pathogens over time as they interacted. In the model, we omitted social (e.g. dominance rank: Puga-Gonzalez & Sueur 2017) and environmental (e.g. resource availability: Foster et al. 2012) factors that may also drive individual social preferences and thus network structure. We did so purposefully to create a

parsimonious system that would allow us to better understand the development of social networks when the only variables were individual attributes related to information sharing and pathogen spreading – a first step toward investigating this potential trade-off.

We made a number of predictions regarding the structure of the networks that we would expect to emerge under the set of conditions tested. First, under conditions in which the values of information were high and those of pathogens were low (conditions 5, 8, 9, 12, 13; **Figure VI.1**), we predicted the emergence of: (i) *centralized and low-density networks* when information was monopolized by a few individuals (one or two), because group members should favor informed over uninformed individuals; (ii) *centralized, modular and low-density networks* when values of information were distributed more evenly among group members, because information could be obtained from numerous sources and therefore individuals might form relationships stochastically with some informed individuals but not with others. Conversely, under conditions in which the values of pathogen were high and those of information were low (conditions 2, 3, 4, 14, 15; **Figure VI.1**), we predicted the emergence of: (iii) *non-modular, non-centralized, and high-density networks* when few individuals had pathogens and none has information or very low values of information, because individuals would avoid infected individuals but would not develop preferences for other group members in so doing. Yet, in conditions in which there are some highly informed individual (e.g. condition 14), we predict the emergence of *centralized* networks; (iv) *fully connected networks* when pathogen values were distributed evenly among group members, because individuals should avoid all group members equally as they frequently change interaction partners seeking information. Under intermediate conditions, in which the values of information were both higher and lower than those of pathogens (conditions 7, 10, 17, 18, 19, 20; **Figure VI.1**), we expected (v) *centralized, modular and low-density networks*, because individuals would develop social preferences only with those informed individuals.

Finally, under conditions in which the levels of information and pathogen were similar (conditions 1, 6, 11 and 16), we had (vi) *no specific expectations*. In terms of individual centrality, the most inter-connected individuals were predicted to be those endowed with the highest values of information, particularly when combined with the lowest values of pathogen.

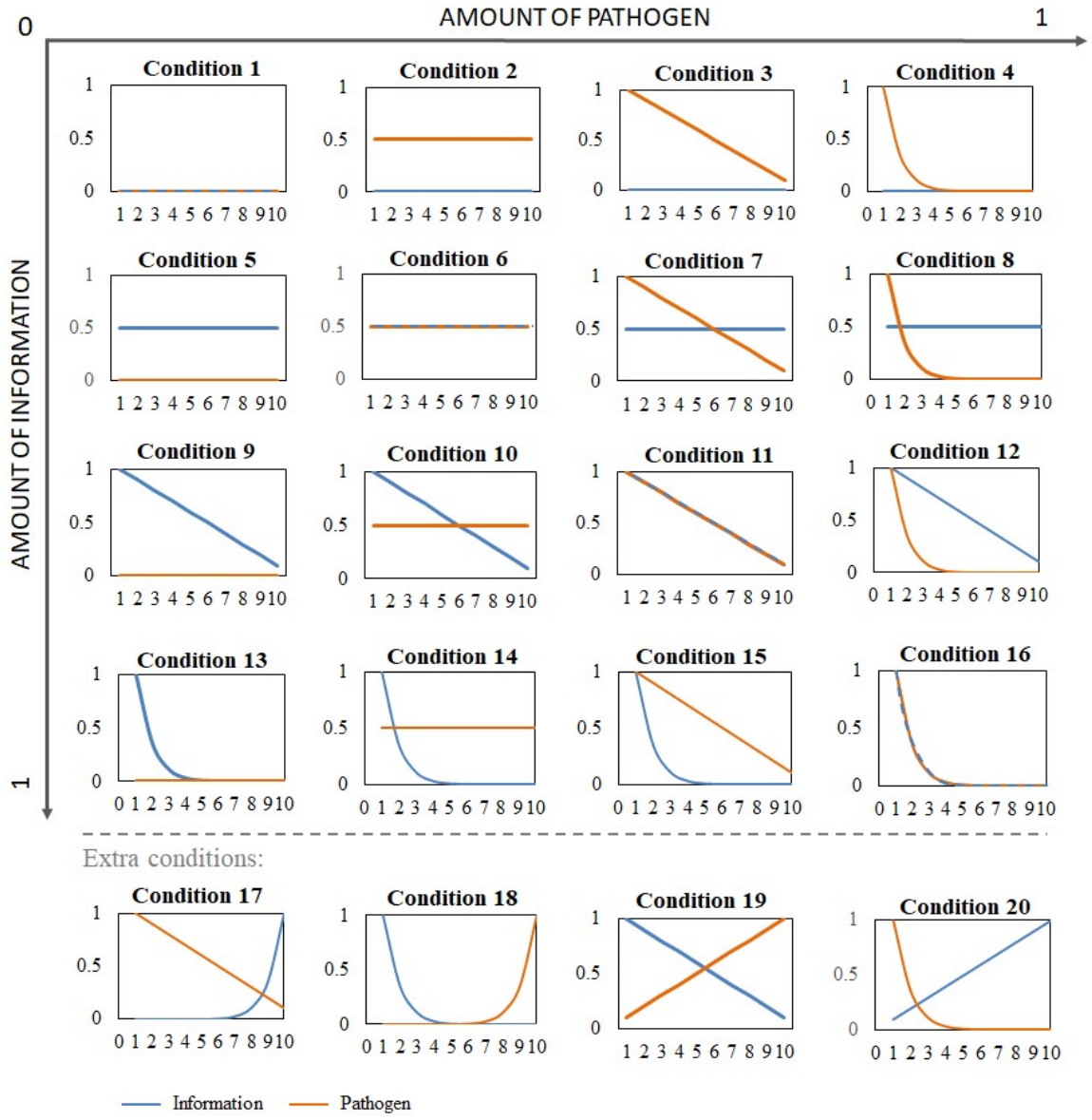

Figure VI. 1. Schematic representation of each theoretical condition. Values of information (blue line) and pathogen (orange line) vary from 0 to 1 (y axis) across individuals (x axis: group size of 10 represented here, each number representing the ID of an individual).

VI.3. Methods

VI.3.1. The Optimal Relationships Model

This section describes the model ("Process overview and scheduling") and the testing conditions ("Testing conditions"). A detailed description of the model according to the ODD Protocol (Grimm et al. 2006, 2010) and the source codes for the model are given in the **Appendix I.**

VI.3.1.a. Process overview and scheduling

The purpose of the model is to identify the type of social network structure that arises from relationships that maximize interactions with informed individuals and minimize interactions with infected individuals. Individuals are endowed with values of *My-information* and *My-pathogen*, which remain stable throughout the simulation, and will interact with other individuals according to their own values and the values of conspecifics. At the beginning of each simulation, individuals are not aware of the values of information and pathogen exhibited by the others. However, by interacting with other individuals, they perceive which individuals are informed and which are infected. Because individuals are actively seeking information for their own benefit, and trying to limit their interaction with infected individuals to avoid the associated costs, they adjust their interactions probabilistically to optimize their social relationships.

All individuals are activated at each time step, and at each time step each individual chooses to interact with a single partner. One or more individuals can interact with the same target individual at any given time step. All individuals are forced to interact at each time

step. The probability of interacting with a specific individual is given by the weight of the relationship: the stronger the weight, the higher the probability. At the initial time step, all individuals have the same probability of interaction (see **section 1**, equation 1). An individual will increase or decrease the weight of its relationship with its interaction partner according to whether it perceives a pathogen and/or information from its partner. The amount of increase or decrease in the weight of the relationship is controlled by the parameters *social-increase* and *social-decrease*. After each social interaction, the weights of relationships, and thus the future probability of interaction, are updated (see **section 2**). Note that the weight between individual *i* and all other group members is constrained to sum to 1.

Definitions of indices and coefficients can be found in **Table VI.1**. A schematic diagram illustrating the individual-based model can be found in **Figure VI.2**. The model was written in Netlogo v. 6.0 (Wilensky 1999).

Table VI.1. Glossary of parameters used in this study.

Parameter	Definition
N	Number of individuals in the group. Group size is set as 10, 30 or 70.
My-information	The probability of being perceived as informed. Values are chosen between 0 and 1.
My-pathogen	The probability of being perceived as infected. Values are chosen between 0 and 1.
Social-increase	The percentage increase in the weight between individuals *i* and *j*. The default value is 20%. See section 2.
Social-decrease	The percentage decrease in the weight between individuals *i* and *j*. The default value is 20%. See section 2.

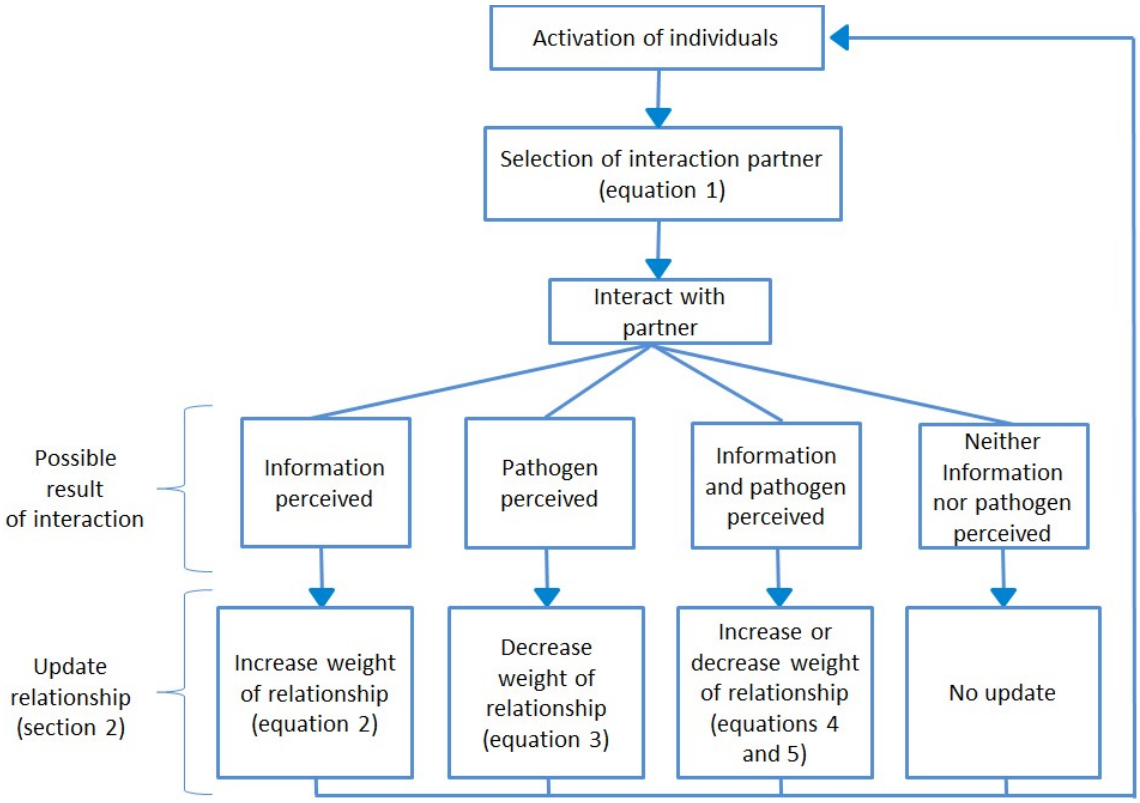

Figure VI.2. Flow diagram of each time step in the Optimal Relationships Model. Schematic interaction rules happening at each time step.

Section 1. Activation of individuals and interactions

At each time step, all individuals are activated. The probability of an individual *i* selecting an interaction partner *j* is given by the weight of their relationship: the higher the weight of their relationship, the higher the probability of interaction. The probability of interaction φ_{01} at the beginning of a simulation is thus given by the initial weight, which corresponds to:

Equation (1) $$\varphi_{01} = \frac{1}{N-1},$$

where *N* is the number of individuals in the group. The probability of successive interactions is equivalent to the updated weight (see section 2).

After an individual selects an interaction partner, there are 4 possible outcomes of the interaction: (1) it perceives its partner as informed; (2) it perceives its partner as infected; (3) it perceives its partner as informed and infected, but either more informed than infected or more infected than informed; or, (4) it perceives neither. Each output depends on the following probabilities:

1. Probability of perceiving an individual as informed (*Pinf*):

Pinf = *My-information* of InteractionPartner * (1 – *My-Pathogen* of InteractionPartner)

2. Probability of perceiving an individual as infected (*Ppat*):

Ppat = *My-pathogen* of InteractionPartner * (1 – *My-Information* of InteractionPartner)

3. Probability of perceiving an individual as informed and infected (*Pinfpat*):

Pinfpat = *My-information* of InteractionPartner * *My-Pathogen* of InteractionPartner

If the difference between *My-information* and *My-pathogen* is higher than 0, the agent perceives more about the informed status than about the infected condition of the interaction partner. If the difference is equal to or less than 0, the agent perceives more about the infected condition than about the informed status of the interaction partner.

4. Probability of not perceiving the status of the interaction partner (*Pnone*):

Pnone= (1 - *My-Information* of InteractionPartner) * (1 - *My_Pathogen* of InteractionPartner)

The model is stochastic: to determine the output of the interaction, first a random number between 0 and 1 is drawn. This random number is compared with Pinf, if the number is less than or equal to that probability, the individual perceives the interaction partner as informed only. Otherwise, it compares the random number with Pinf + Ppat. If the number is less than or equal to the sum of these probabilities, the individual only perceives the interaction partner as infected. If the number is greater than the sum of these probabilities, it compares the random number to Pinf + Ppath + PInfPath. If the random number is less than or equal to the sum of these probabilities, the individual perceives the interaction partner as infected and informed. If the random number is higher than the sum of these probabilities, the individual perceives nothing. At the end of this step, the relationship between *i* and *j* is updated, as described in the next section.

Section 2: Updating relationships

After every interaction, individual *i* will update the weight of its relationship with individual *j* according to whether or not it perceived the potential to acquire pathogens and/or information. To present the social network dynamics, the model includes a *social-relation transfer*, the percentage of transfer within social relationships. We follow Sueur & Maire (2014), who proposed that a rate of 20% is adequate to predict differences in social interactions preceding the fission or social instability of a group. We include stochasticity in this process and at each time step a random value is chosen from a normal distribution with mean equal to 20% of the social relationship and a standard deviation of 5%, hereafter represented by “20%±5%".

In our model, if pathogens are perceived, the weight of interaction between individual *i* and *j* will decrease by “20%±5%" (controlled by the *social-decrease* slider in the model

interface; **Figure VI.3**). The “20%±5%" subtracted from this relationship is distributed proportionally among the weights of the relationships the agent *i* has with all other group members, meaning that the stronger relationships will be reinforced to a greater degree than the weaker ones. Conversely, if information is perceived, the weight of interaction between individual *i* and *j* will increase by “20%±5%" (controlled by the *social-increase* slider in the model interface). The “20%±5%" added to this relationship is proportionally obtained from the weights of the relationships the agent *i* has with other group members (**Figure VI.3**). In this way, the weight of relationships is constrained to vary between 0 and 1, and the sum of the weights between individual *i* and all other group members must equal 1. The updating of relationships is given by the equations in **Table VI.2**.

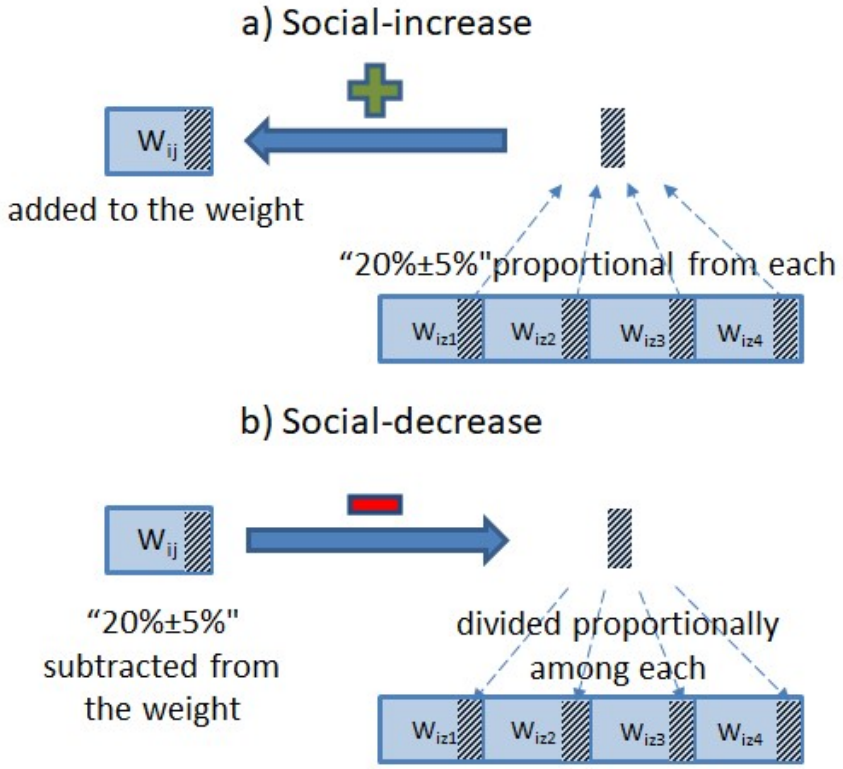

Figure VI.3. Updating the weight of relationships according to the mechanisms of social-relation transfer (i.e. social-increase or social-decrease). If the output of the interaction is positive (individual *i* perceives information from its interaction partner *j*), a total of "20%±5%" will be reduced from the weights of the relationships between individual *i* and all other group members; excluding the interaction partner *j* (a). If the output of the interaction is negative (individual *i* perceives pathogens from its interaction partner *j*), a total of "20%±5%" of the weight of the relationship between individual *i* and *j* will be removed and distributed proportionally among the weights of the relationships that *i* and *j* have with the remaining group members (b).

Table VI.2. Update of relationships.

	Weight of relationship (W)	
Outcome of the interaction	**Agent and interaction partner (W_{ij})**	**Agent and other group members ($W_{iz\ (z\neq j)}$)**
Individuals perceive the interaction partner as informed (resulting from Pinf)	Equation (2) $W_{ij(t+1)} = W_{ij(t)} + \Delta$	Equation (2.1) $W_{iz(t+1)} = W_{iz(t)} - (W_{iz(t)} * \text{social-increase})$
	where: $\Delta = \sum_{z\neq j}^{N} W_{iz(t)} * \text{social-increase}$	
Individuals perceive the interaction partner as infected (resulting from Ppat)	Equation (3) $W_{ij(t+1)} = W_{ij(t)} - \Delta$	Equation (3.1) $W_{iz(t+1)} = W_{iz(t)} + (\Delta * \frac{W_{iz(t)}}{\sum_{z\neq j}^{N} W_{iz(t)}})$
	where: $\Delta = W_{ij(t)} * \text{social-decrease}$	
Individuals perceive the interaction partner as more informed than infected (resulting from Pinfpat)	Equation (4) $W_{ij(t+1)} = W_{ij(t)} + \Delta$	Equation (4.1) $W_{iz(t+1)} = W_{iz(t)} - (W_{iz(t)} * \text{social-increase} * \text{dif})$
	where: $\Delta = \sum_{z\neq j}^{N} W_{iz(t)} * (\text{social-increase} * \text{dif})$ $\text{dif} = \text{abs}(\text{Pinf} - \text{Ppat})$	
Individuals perceive the interaction partner as more infected than informed (resulting from Pinfpat)	Equation (5) $W_{ij(t+1)} = W_{ij(t)} - \Delta$	Equation (5.1) $W_{iz(t+1)} = W_{iz(t)} + (\Delta * \frac{W_{iz(t)}}{\sum_{z\neq j}^{N} W_{iz(t)}})$
	where: $\Delta = W_{ij(t)} * \text{social-decrease} * \text{dif}$ $\text{dif} = \text{abs}(\text{Pinf} - \text{Ppat})$	
Individuals do not perceive the status of the interaction partner (resulting from Pnone)	No update	

VI.3.1.b. Testing conditions

We tested 20 different conditions representing different combinations of *My-information* and *My-pathogen* among group members (**Figure VI.1**). Values endowed to each individual were exclusive, meaning that the values of *My-information* and *My-pathogen* of one individual do not overlap. These attributes were either linearly- or power-law-distributed across individuals (Figure 1) and were chosen arbitrarily. Linear distributions were classified as being a constant function of individual identity (*My-information* and/or *My-pathogen* = 0 or 0.5), a positive function of individual identity ($r_s = 1$), or a negative function of individual identity ($r_s = -1$), assuming a constant ordering of individual identification numbers from 1 to N. Power-law distributions were set with a degree exponent of $\gamma = 10$, which allowed us to have approximately 25% of the individuals with values between 1 and 0.1. We tested each of the conditions for theoretical group sizes of 10, 30 and 70 (representative of animal group sizes, Vital & Martins 2009) to investigate whether the emergent network properties differed as a function of the number of individuals involved. We ran 20 simulations, with a total of 10000-time steps (= number of interactions) for each of the 20 conditions tested. The number of simulations run is conventional and is usually used to show that some conditions are indeed different from others. It provides a way of understanding whether the variation within conditions is lower than between conditions. We expected to have high variance within conditions when we had no predictions about the emergent networks, in contrast to some of the more extreme cases in which the predictions are clear and uncontroversial.

VI.3.2. Data collection

We recorded each individual's identity, previous weights, updated weights and type of interaction at each time step. A matrix of interactions was then created including the total

number of interactions in a simulation, between each pair of individuals in the group. Matrices were undirected, meaning that we assumed no differences between *ij* and *ji* interactions. We extracted the dataset in two ways: i) the matrix of interactions separated into 10-time periods, containing 1000 time-steps in each period and ii) the matrix of interactions at the end of the 10000 simulations (i.e. complete dataset). While the former allowed us to check the development of networks through time, the latter gave us the resulting network properties found in the system.

VI.3.3. Global index

To evaluate the overall influence of information (i.e. *My-information*) and pathogen (i.e. *My-pathogen*) on the resulting global properties of the networks, we created an *Information-* and a *Pathogen-Index* ($Index_{Pat}$). The *Information-index* ($Index_{Inf}$) is a function of:

Equation (6) $$Index_{Inf} = Imax - (\bar{x} (Imax_{(z)})) ,$$

where *Imax* is the maximum value of *My-information* in the group and $Imax_{(z)}$ regards the values of *My-information* from the remaining individuals in the group.

The same formula, but considering *My-pathogen* values, was calculated to estimate the $Index_{Pat}$. High values of $Index_{Inf}$ refer to few individuals monopolizing high values of information compared to others. High values of $Index_{Pat}$ denote few individuals monopolizing high values of pathogen compared to others.

VI.3.4. Social Network Analysis

Visualization: We constructed association networks in R v.3.3.2 (R Core Development 2016) with the "igraph" package v.1.0.1 (Csárdi & Nepusz 2006).

Network indexes: We chose to estimate network metrics that have previously been demonstrated to influence social transmission processes in animal networks (Nunn et al. 2015), and that capture different aspects of network structure, at both global and individual levels. All properties were estimated using the "igraph" package in R.

Global metrics:

Density: an index that estimates the ratio between the number of observed edges and the number of possible edges in the network (Sueur et al. 2011a). Values range from 0 to 1, with 1 reflecting a completely-connected network with maximal density. We estimated density using the function "graph.density".

Eigenvector centralization: an index that estimates variation in connectedness across nodes in the network. Higher eigenvector centralization values denote a centralized network, where one or a few individuals monopolizes most of the interactions in the network (Kasper & Voelkl 2009). We estimated eigenvector centralization as:

$$C = \frac{\sum_{i}^{N} (Cmax - Ci)}{Max \sum_{i}^{N} (Cmax - Ci)}$$

where C is the centralization index, Ci is the centrality for individual i, C_{max} is the maximum value of Ci across all individuals and $Max \sum_{i}^{N} (Cmax - Ci)$ refers to what the sum would be under the largest possible centralization of the network.

Newman modularity: an index that estimates the extent of sub-grouping in a network. We used a commonly eigenvector-based measure that is claimed to be independent of group size (Newman 2006). High levels of modularity denote greater subdivision of the social group into subgroups. Although modularity is commonly considered to be an important factor regulating disease flow (Griffin & Nunn 2012; Nunn et al. 2015), there is mixed evidence regarding its influence on social transmission (e.g. mammals: Nunn et al. 2015; Sah et al. 2017). We estimated Newman modularity using the function "cluster_leading_eigen".

Individual metrics:

Betweenness: the number of shortest paths that pass through the considered individual. The more connections that are made through one individual, the greater its value of betweenness becomes (Hanneman & Riddle 2005).

Eigenvector: the weighted connectivity of an individual within its network, also considering the weighted connectivity of its neighbors. Individuals tied to central individuals (i.e. those with a high connectivity themselves) should have higher centrality than those connected to less central individuals (Borgatti et al. 2013).

Strength: the sum of each node's edge values. The individual with the most and strongest connections has the highest strength value (Sueur et al. 2011a).

VI.3.5. Statistical analyses

In a first set of analyses, we created general linear models (glm) using the package "car" v.2.2.4 in R to test for an influence of *Information-index*, *Pathogen-index*, and group size on the global metrics of the emergent network properties (centralization, density and modularity). The distribution of all response variables deviated from the Gaussian case, so we

applied Box-Cox transformation using the "MASS" library v.7.3-45 in R (Venables & Ripley 2002) to fit the assumptions of the statistical models. Since our data for modularity and centralization included many zeros, we followed the method applied by Hyndman & Grunwald (2000) to deal with skewed data and calculated the log of our response variable summed with lambda 2 ($\log(y+\lambda_2)$), here equal to 2. This allowed us to correct the data using an approximation of values according to our own dataset. We assumed lambda values ranging from -2 to 2 with 0.1 intervals (Box & Cox 1964; Venables & Ripley 2002). We further ran a series of diagnostics to judge the validity of the models, including testing for variance inflation, correlation of fitted and residual values and Cooks' distance, all of which suggested the suitability of our models, as no obvious violations of assumptions were detected (Field et al. 2012).

We then tested whether the values of *My-information* and *My-pathogen*, which characterize an individual's status, predicted individual centrality (betweenness, eigenvector and strength) by applying Spearman tests with Bonferroni correction (Abdi 2007). All analyses were performed in R statistical software v.3.3.2, with the alpha level set at 0.05.

VI.4. Results

The association networks constructed from the data collected in five-time periods are illustrated in **Figure VI.4**, showing the network reach stability after the second-time period. It allows us to check for the emergence of network properties in the complete dataset.

We first sought to understand the influence of group size, *Information-* and *Pathogen-index* on the emergence of global network properties (density, eigenvector centralization and Newman's modularity). Our results indicate that *Information-index*, which quantifies the

overall values of *My-information* for a given condition, is a good predictor of density and eigenvector centralization (**Table VI.3**), meaning that the high values of information endowed to few individuals created a tightly connected "core" made of a few individuals who monopolized a significant part of the social relationships (**Figure VI.5**). As such, *Information-index* reduced the potential of full connectivity in the network (i.e. density). Group size also predicted a reduction in network density but an increase in Newman's modularity: as the group size increased, the number of subgroups also increased. *Pathogen-index*, however, had no influence on any of the network properties estimated in this study (**Table VI.3**; **Figure VI.5**).

Our next analyses focused on the measures of individual centrality (betweenness, eigenvector and strength). They demonstrated that individuals with the highest values of *My-information*, and thus the largest potential benefits for others, generally were those most inter-connected in the group (strength: $r = 0.27$, $p = 1.32^{e-15}$; betweenness: $r = 0.64$, $p = 1.32^{e-15}$; **Figure VI.6**). Confusingly, the opposite relationship was observed for eigenvector centrality ($r = -0.15$, $p = 2.8^{e-11}$). Finally, individuals endowed with increased values of *My-pathogen* were those that were avoided during social interactions, expressed by the strong negative correlation with strength ($r = -0.19$, $p = 1.32^{e-15}$) and betweenness ($r = -0.10$, $p = 2.08^{e-05}$) centralities. We found no relationship between *My-pathogen* and eigenvector centrality ($r = 0.0007$, $p = 1$).

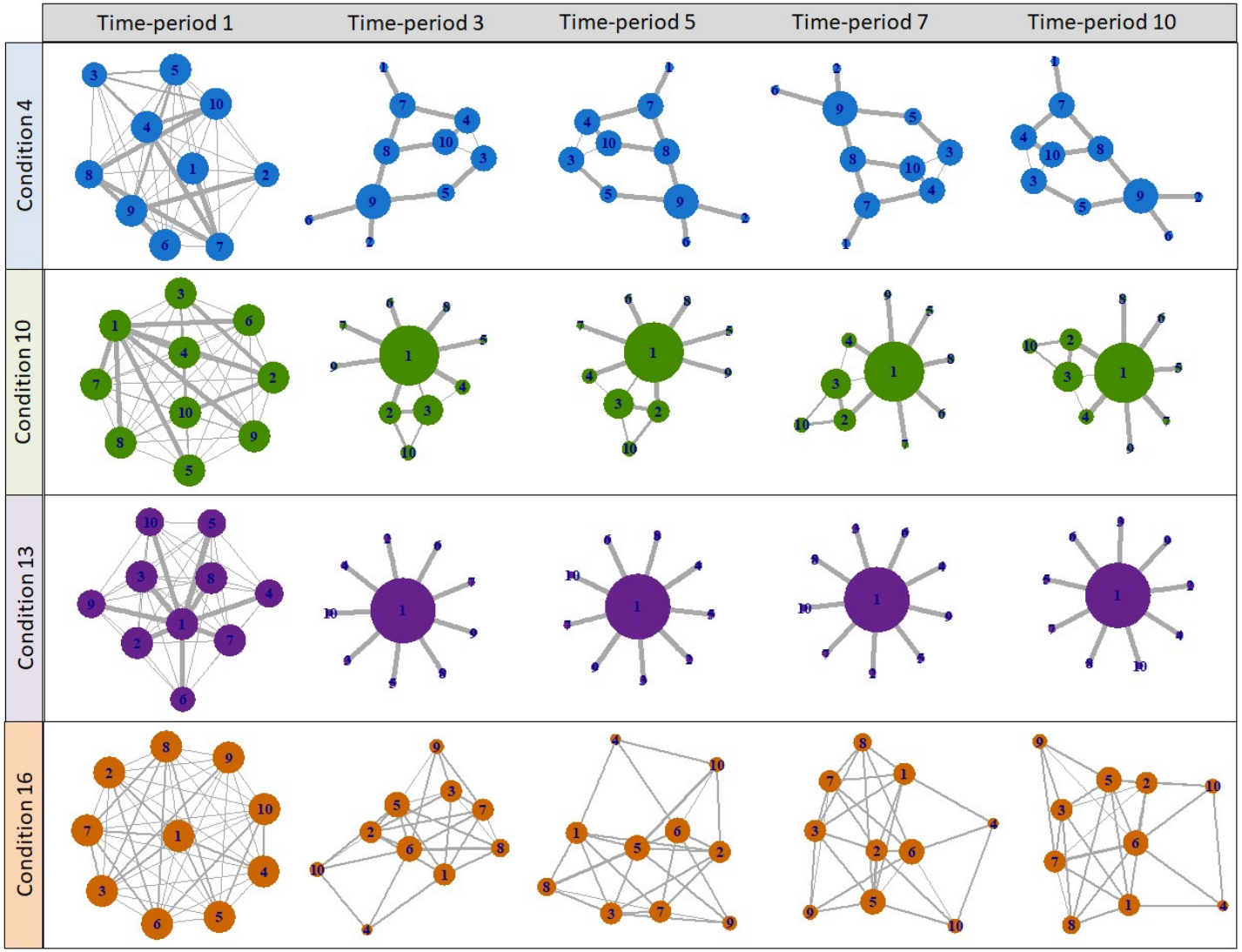

Figure VI.4. Association networks for time-periods of one simulation from conditions 4, 10, 13 and 16. Nodes (circles) represent individuals in the model (N = 10), with its size related to the degree centrality (i.e. number of edges connected to the individual; the higher the centrality, the larger is the size of the node). Edges are undirected and weighted, such that pairs with higher association indices had thicker edges. Each condition represents a prediction of the study. Networks were built using the package "igraph" (Csárdi & Nepusz 2006) available in R (R Core Team 2016).

Table VI.3. Parameter estimates from linear models explaining the global network structure.

Global metrics	Predictors	Estimate	Std. error	t value	Pr(>|t|) [a]
Density					
	(Intercept) [b]	-0.90	0.46	-1.97	0.05*
	Information-Index	-1.29	0.52	-2.46	0.02*
	Pathogen-Index	0.23	0.52	0.44	0.66
	Group size (N=30)	-1.14	0.49	-2.33	0.02*
	Group size (N=70)	-1.96	0.49	-3.99	1^{e-4}***
Modularity					
	(Intercept) [b]	0.43	0.01	52.76	$<2^{e-16}$ ***
	Information-Index	0.005	0.01	0.50	0.62
	Pathogen-Index	0.003	0.01	0.39	0.70
	Group size (N=30)	0.02	0.01	2.47	0.02*
	Group size (N=70)	0.03	0.01	3.65	0.001***
Centralization					
	(Intercept) [b]	334.98	56.26	5.95	1.9^{e-07}***
	Information-Index	201.89	64.32	3.13	0.003 **
	Pathogen-Index	44.85	64.27	0.70	0.49
	Group size (N=30)	-38.10	60.21	-0.63	0.53
	Group size (N=70)	-19.75	60.18	-0.33	0.74

[a] Significant codes are marked as follows: "***"p<0.001, "**"p<0.01, "*"p<0.05, "†"p<0.1.
[b] All comparisons made against the intercept of the first level of each factor of the Group size (N = 10).

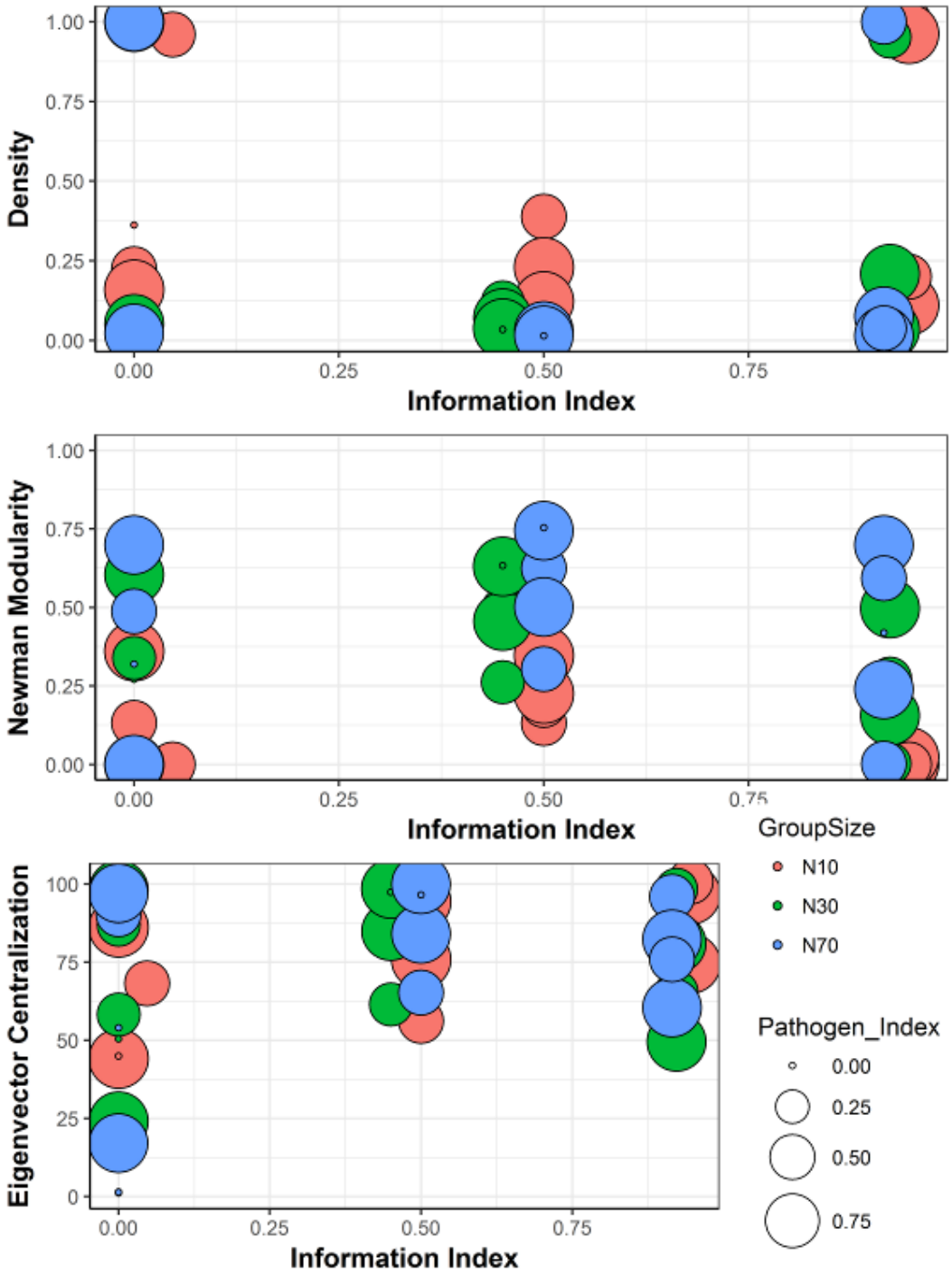

Figure VI.5. Global network metrics (density, Newman's modularity and eigenvector centralization) and their relationship with group size, *Information-* and *Pathogen-index*. The color of each cell represents the group size (N = 10, 30 or 70) and the size of circumference denotes *Pathogen-index*. For a definition of *Information- and Pathogen-index*, see main text.

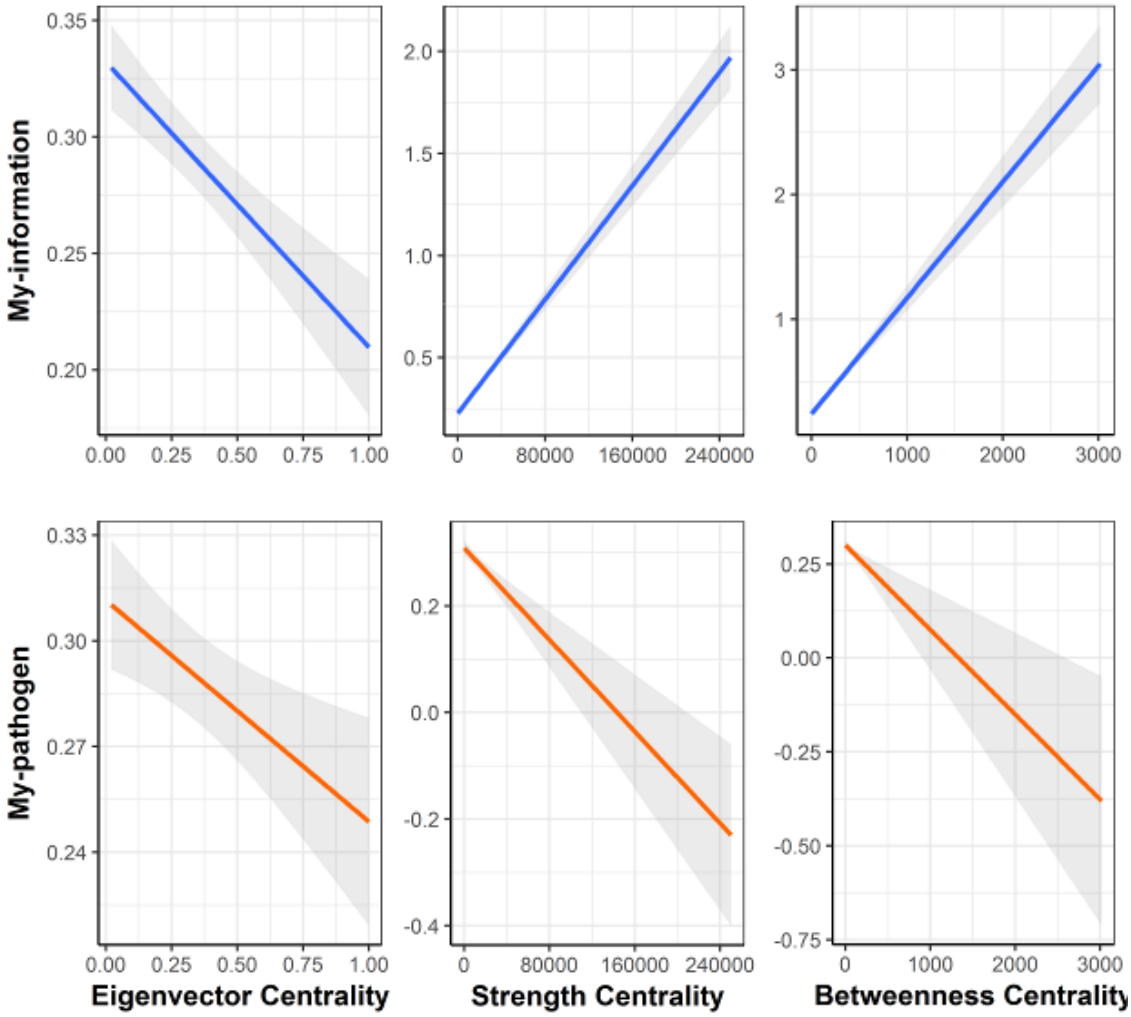

Figure VI.6. Relationship between individual centrality and the values of *My-information* and *My-pathogen*.

VI.5. Discussion

Our study test predictions about which network properties will emerge from a scenario in which individuals maximize the chances of getting information but minimize the risk of getting infected through social interactions. We demonstrated that few individuals monopolizing values of information (estimated by the *Information-index*) favor the formation of centralized networks and as such, a reduction in network density. Group size also influenced the network structure, showing a negative relationship with network density and a

positive influence on Newman's modularity: the larger is the group, the higher is the subdivision of the network into subgroups. The latter result is in accordance with several studies showing a positive relationship between group size and modularity (e.g. Griffin & Nunn 2012; Nunn et al. 2015; chapter 2). Based on the costs and benefits used in our model, optimal social relationships thus seem to be dependent primarily on the information each individual can obtain through social interaction.

From an evolutionary perspective, these results shed light on how an individual's decisions affect the development of social networks. Surely, in complex social systems, network structure is molded by numerous intrinsic and extrinsic factors (such as age, sex and morphometric characteristics: Croft et al. 2005), but our parsimonious model demonstrated how individuals might behave if constrained to deal solely with the potential trade-off between information and pathogen transmission. This is important since the acquisition of information is linked with chances to quickly adapt to a changing environment, which is expected to increase an individual's fitness (e.g. Dall et al. 2015). On the other hand, socially-transmitted pathogens are among the major causes of mortality (e.g. Walsh et al. 2009), and the removal of infected individuals or changes in their own behavior, or the behaviors of others in response to them, causes changes in the network structure (e.g. Carne et al. 2013; Lopes et al. 2016). Our results thus shed light on the mechanisms that cause variation in social structure and demonstrate how individual and global network metrics may reflect optimal social relationships, which is dependent upon the level of information with which each individual is endowed.

Individuals in real systems have developed many behavioral strategies to avoid infection (e.g. hygienic behavior: Sarabian & MacIntosh 2015). In this chapter, we focused specifically on social-avoidance, as it provides a direct response to the behavior or status of an infected conspecific (Loehle 1995). In group-living animals, social avoidance plays a role

in social cohesion among group members, which in turn affects pathogen transmission. Evidence that individuals can identify infections in conspecifics and modulate their own degree of interactions has grown in the past years (e.g. Kiesecker et al. 1999; Poirotte et al. 2017). Nonetheless, this remains an under-investigated area with respect to most mammalian species, including primates (e.g. Nunn & Altizer 2006).

We acknowledge that there are limitations in our study, including our ability to compare our results with real data. We suggest that future studies aiming to test our model consider social insects as models of study. Investigations have shown that wasps and ants have an efficient system to avoid socially-transmitted pathogens (e.g. corpse removal, Cremer et al. 2007; Stroeymeyt et al. 2014), and at the same time exhibit an extensive network of contacts through which they efficiently transmit information. Manipulating the contact network and the amount of information and pathogen each social insect is endowed with may provide a good opportunity to evaluate the predictions obtained from our Optimal Relationships Model.

Finally, it is well known that animals must deal with several trade-offs inherent to sociality, such as the increased foraging efficiency but higher intra-group competition, and the trade-off examined here involving information and pathogen sharing. Understanding how individuals respond to a particular trade-off should illuminate the mechanisms that favor social relationships and, more specifically, highlight the structural composition of a network if individuals are aiming to maximize the benefits (i.e. information) and minimize the costs (i.e. pathogen) of being social. Our study provides the first-step towards understanding variation emerging in network properties due to a potential optimization of the trade-off between information and pathogen transmission.

Ethics: Do not apply.

Conflict of interests: The authors declare no competing interests.

Authors' contributions: VR, AJJM and CS conceptualized the study. VR, IPG and CS created the individual-based model. VR and IPG performed the network analysis. VR wrote the manuscript and performed the statistical analysis. IPG, AJJM, CS significantly contributed to the manuscript development and review.

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General discussion

VII. DISCUSSION

"The most beautiful thing we can experience is the mysterious. It is the source of all true art and science. He to whom the emotion is a stranger, who can no longer pause to wonder and stand wrapped in awe, is as good as dead —his eyes are closed".

Albert Einstein, 1930

In my thesis, I investigated the interface between network topology and social transmission, incorporating data from group-living animals and integrating them with data from simulated networks. Specifically, I investigated the social structure of wild primate groups to understand the influence of central individuals in the chain of pathogen transmission (chapter 1), the effect of global network properties at different stages of an outbreak (chapter 2), the link between network efficiency and modular structure (chapter 3) and finally, I propose an optimal scenario for relationships where individuals minimize risk of contagion while maximizing potential information acquisition (chapter 4). Addressing these questions through a combination of classical methods, like sampling of social behavior (Altmann 1974), network analysis and theoretical modeling approaches (i.e. individual-based modeling; Whitehead 2008a; Railsback & Grimm 2012), allowed me to investigate more deeply the role of complex social structure on social transmission processes, and vice versa. My results on primate networks strengthen the evidence supporting social structure as a key feature mediating social transmission, while the individual-based model I developed provides insight into how individual characteristics related to their likelihood of transmitting information or pathogens might influence the social structure.

VII.1. On the interface between social structure and social transmission

Hinde's (1976) seminal paper introduced a conceptual framework for associations at the individual level and their influence on the emergence of social structure and variation therein. More recently, a scheme proposed by Cantor & Whitehead (2013) extended Hinde's conceptual framework by incorporating bidirectional effects between social structure and information flow: group structure influences the way information is transmitted, while the flow of information in turn can affect the social structure. For example, similarities in behavior, as expressed in the vocal repertoires of cetaceans or the opinions of humans, cause clustering of individuals. Within these groups, associates are also more likely to share information (Iñiguez et al. 2009; Rosvall & Sneppen 2009, Cantor et al. 2015). While the relationship between network structure and information flow has been empirically demonstrated in animal social networks (human: Iñiguez et al. 2009; birds: Farine et al. 2015a, Kulahci et al. 2016; non-human primates: Voelkl & Nöe 2010), multiple lines of evidence also suggest the influence of social structure on parasite transmission (human: Jones & Salathe 2010; lizards: Godfrey et al. 2009; ungulates: VanderWaal et al. 2014a,b; non-human primates: Nunn & Altizer 2006; Rushmore et al. 2017, honeybee: Naug 2008). Nevertheless, the dynamic feedback loop between social structure and social transmission has been discussed mainly in terms of information as the dominant network flow, specifically as it pertains to cultural transmission (Cantor & Whitehead 2013).

Here, I propose to extend this framework of social transmission and incorporate the costs of socializing in networks by including the spread of socially-transmitted pathogens (**Figure VII.1**). Instead of considering transmission as the final factor in the loop, or assume that information flow is the only relevant factor, I suggest simultaneous examination of

information and pathogen transmission as explicit and opposing entities. Each feed back into individual behavior and thereby influence social structure. Indeed, while access to crucial information is expected to drive individuals to cluster around knowledgeable individuals, affinity for enemy-free space, i.e. avoidance of potential social sources of infectious disease, should operate to reduce connectivity in a network. This presents a classical fitness for trade-off for individuals that aim to exploit social relationships for their own benefit on the one hand while avoiding potential costs on the other. While information transmission has been well placed into the classical framework proposed by Hinde (1976; **Box VII.1**), whether and how parasite and pathogen pressures interact with social structure in ecological and evolutionary terms, e.g. via their dependence on social contact or proximity for transmission, has received far less attention.

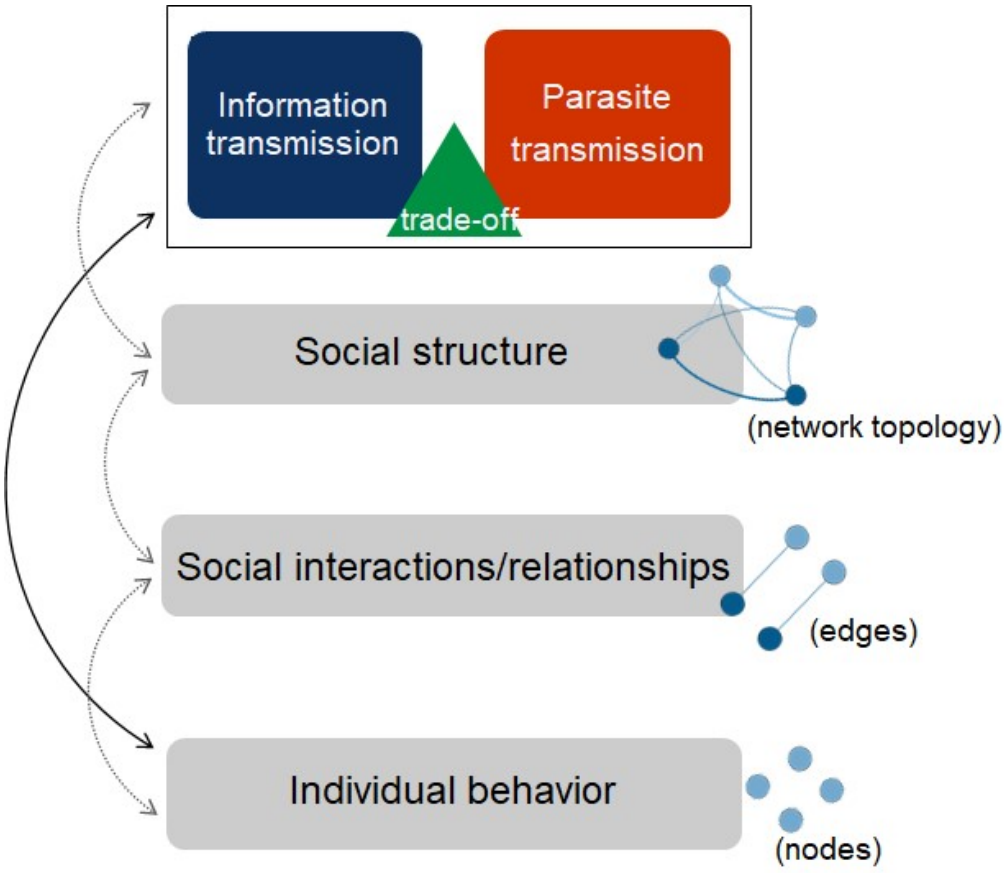

Figure VII.1. Schematic representation of the feedback loop between social interaction (or social relationship), social structure and social transmission. Individual behavior leads to different patterns of social interactions, that in turn influence and are influenced by social structure and social transmission. The boxes in grey represent the scheme proposed by Hinde (1976), and in blue the implementations proposed by Cantor & Whitehead (2013), which include the effect of information transmission in the loop (specifically cultural transmission) and the incorporation of the terminology of social network analysis (noted at right in the figure). The orange box and green triangle reflect the implementation I propose in this thesis: a simultaneous examination of information and pathogen flow as explicit and opposing entities with the network properties representing the trade-off between both transmission processes. Diagram adapted from Hinde (1976) and Cantor & Whitehead (2013).

Infectious agents that are transmissible via direct contact (such as influenza, Ebola, HIV/AIDS, etc.) depend on the host network for propagation (Pastor-Satorras & Vespignani 2001; Meyers et al. 2005; Guimarães et al. 2007; Salathé & Jones 2010; MacIntosh et al. 2012; Duboscq et al. 2016). Structuring of social relationships often creates heterogeneous

contact patterns that differentially affect the spread of parasites and pathogens (e.g. microbial transmission pathways in giraffes: VanderWaal et al. 2014a). At the same time, parasite and pathogen acquisition affects social structure directly through mortality rates, as well as indirectly depending on the degree to which individuals avoid social interactions involving infected individuals (i.e. avoidance behavior[4]; Krause & Godin 1994; Keisecker et al. 1999; Behringer et al. 2006; Poirotte et al. 2017). For example, Caribbean spiny lobsters (*Panulirus argus*) avoid individuals that are infected with PaV1, a lethal virus (Behringer et al. 2006), while wild mandrills show some avoidance toward grooming group members infected with and shedding gastrointestinal protozoan parasites (Poirotte et al. 2017). Group size is also considered an influencer of disease spread, and there is evidence that social animals change their activity budgets, including social contact, depending on group size (e.g. colobus monkeys, Gogarten et al. 2014). Although the relationship between group size and infection risk is inconsistent across species (Rifikin et al. 2012; Ezenwa et al. 2016), multiple examples of this effect suggest that individuals in larger groups may be more exposed to pathogens (Altizer et al. 2003). Therefore, pathogens that are or can be transmitted through social channels may negatively affect social cohesion, directly or indirectly, by reducing social connectivity, while social structure continues to set the conditions under which individuals are exposed to potentially deleterious infectious agents, creating a bidirectional feedback effect.

[4] AVOIDANCE: The term "avoidance" refers to actions taken by an animal to reduce the chances of becoming infected with pathogens or parasites (Curtis 2014). In this thesis, I use the term "social avoidance" to indicate avoidance of social contact or proximity with an infected individual.

In this thesis, I cover the first level of Hinde's framework – how individual attributes, such as age, dominance rank and family unit, affect social structure – in chapter 1. I demonstrate that group structure alters parasite transmission in animal-derived and theoretical networks (chapters 1, 2 and 3), covering the latter part of the social scheme proposed by Cantor & Whitehead (2013), with the addition of transmission costs not addressed by these authors. And finally, I explicitly demonstrate that the likelihood with which each individual is expected to transmit information and infectious agents drives social interactions, potentially in a way that optimizes the resultant social network (social structure) to deal with the costs and benefits of group-living (chapter 4). Behavioral ecologists have been aware of the complex relationships formed between individuals, but few have attempted to evaluate the resulting social system quantitatively, accounting for the interplay between these complex relationships (e.g. Sueur 2008, Cantor 2016, the current thesis). In addition to an extensive evaluation of both social structure and transmission dynamics underlying primate networks, I believe my work is the first to investigate the trade-off between information and parasite transmission in the social interaction-social structure scheme.

Box VII.1. How information transmission affects social structure: a brief summary

Information transmission has been demonstrated both theoretically and empirically to cause clustering of individuals (Rosvall & Sneppen 2009; Cantor & Whitehead 2013). In the context of cultural transmission, for instance, it is known that some individuals share a given behavior while others do not (Franz & Nunn 2009). Such phenomena cause variability in social connections to the effect that individuals sharing the same behavior form strong links with each other, forming subgroups that increase the overall subdivision (i.e. estimated throughout network modularity) in the group, as well as social complexity (Boccaletti et al. 2006; Daura-Jorge et al. 2012; Cantor & Whitehead 2013). Culture, then, directly affects social structure, as individuals preferentially interact with others displaying similar behavior patterns (Centola et al. 2007).

VII.2. Do social network properties reflect a trade-off between information and pathogen transmission?

One of the main goals of my thesis was to investigate whether the network topology (social structure) would reflect a trade-off between information and pathogen flow. Besides the evidence that social structure mediates transmission processes involving information and/or infectious agents (Kappeler et al. 2013; Duboscq et al. 2016a; White et al. 2017), it has long been argued that some properties of the network might favor or constrain transmission, or act in both ways (Cantor & Whitehead 2013; Nunn et al. 2015). Among the global network properties examined in the first and second chapters of my thesis, many (e.g. modularity, centralization, diameter) interact with group size to predict pathogen spread: increased values of modularity and diameter, which are both expected to increase in larger groups, caused

declines in pathogen spread, while high centralization had mixed effects depending on pathogen transmissibility. Clustering coefficient negatively affected pathogen transmission, but its effect appears independent of group size. These results together indicate that network properties can be used as a powerful estimation of transmission processes in animal societies, with each property potentially working as a buffer or facilitator of social transmission. If individuals optimize their relationships to deal with costs and benefits of social relationships, network properties may reflect the potential trade-off between information and pathogen transmission.

My third and fourth chapters provide evidence that the structure of the network might be optimized to favor information flow and decrease contagion risk. For example, a peak at intermediate levels of modularity predicted high levels of network efficiency in both theoretical and empirical networks (chapter 3). The predictions generated in the simulations from this chapter, where I identified a non-linear relationship between network efficiency and modularity, were supported by empirical observations from non-human primate networks. This suggests that we might expect to find real-world networks in which values of modularity might vary across some range if social interactions are occurring to balance costs and benefits of social relationships. In the last chapter of my thesis, I showed that the likelihood with which individuals are expected to share information or pathogens with social partners might lead to the optimization of social networks, with relationships being formed and broken according to the different outcomes of social interactions. Highly centralized and low-density networks reflected a social structure optimized to avoid infectious agents and maximize information attainment in an artificial environment (chapter 4). In summary, if the topology of the network can be optimized in a way that balances the costs and benefits of social relationships, it reflects the variability in individual social behavior towards balancing the benefits and costs of social contact, as has been empirically demonstrated (Poirotte et al.

2017; Fruteau et al. 2009). Social networks are demonstrably dynamic (Zimmermamm et al. 2004; Castellano et al. 2009), and interaction costs such as parasite and pathogen acquisition (Poirotte et al. 2017) as well as interaction benefits such as food availability (Fruteau et al. 2009) each cause variation in individual decisions about with whom to interact, leading to the emergent social structure observed.

In this thesis, I do not intend to suggest that there is one single network structure whereby individuals optimize the costs and benefits of social relationships. Instead, I propose that the degree to which individuals interact socially varies dynamically, plausibly to optimize information flow and minimize connection costs, neither of which will be static across time nor stable across environments. Despite my focus on static systems (represented by one network per species of study and time-invariant conditions in the individual-based model), social networks encode dynamic relationships. Network topology evolves, as changes in node states affect edges, and changes in edges affect node states (for a review see Gross & Blasius 2008 and Castellano et al. 2009

VII.3. Individual flexibility in social behavior, social transmission and the evolution of sociality

Individual flexibility in social behavior arises from individuals showing changes in their interactions with conspecifics over time or under different circumstances. Flexible interactions allow individuals to deal with conflicts, social and environmental changes, which ultimately may result in significant increases in individual fitness. *Social flexibility* is also a term that denotes changes in individual behavior, but it is expected to happen only if individual behavior affects social organization (i.e. composition of the group) that consequently may influence the other components of the social system: social structure and

mating system (i.e. who mates with whom; Schradin et al. 2012). For example, African striped mice (*Rhabdomys pumilio*) change their social strategies according to population density: high density implies greater competition (Schradin 2004), which leads to small home ranges (Schradin & Pillay 2006), while low density drives solitary breeding as the best reproductive tactic to avoid reproductive competition (Schoepf & Schradin 2012).

Although *individual flexibility in social behavior* and *social flexibility* are inter-linked terms, species that are flexible in their social behavior might not necessarily exhibit social flexibility, or vice-versa. For example, primates are considered highly flexible mammals (Schradin et al. 2013), resulting in many changes in the patterns of social interaction observed, but few species, such as marmosets and tamarins, show social flexibility that leads to changes in the social organization (Garber 1997). In animal societies, variation in social behavior can occur between individuals, due to genetic variation and/or developmental plasticity, but differences within an individual are considered a consequence of individual flexibility in social behavior and/or social flexibility (for a review see Schradin et al. 2013; **Figure VII.2**). In this thesis, I am interested in how changes in individual behavior, in a broader sense, affect social structure and consequently social transmission.

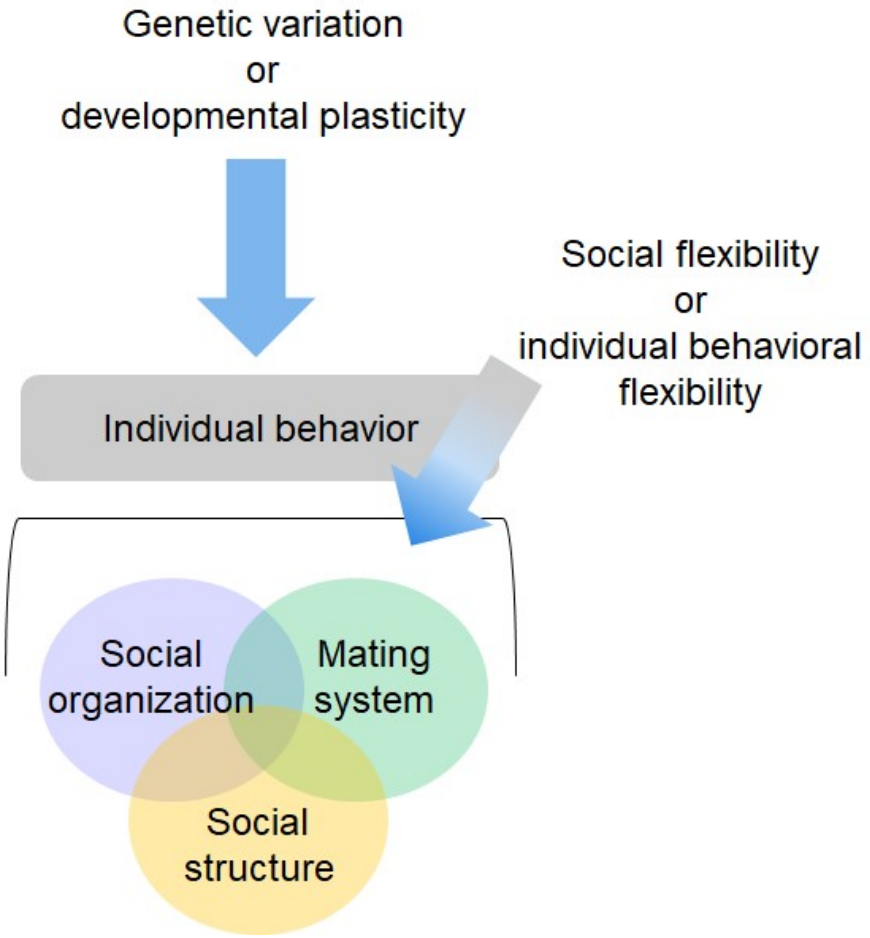

Figure VII.2. Schematic representation of the mechanisms influencing individual behavior and their influence on social system (social organization, mating system and social structure).

In the first and fourth chapters of this thesis, I investigated how individual attributes shape social structure. I showed that the emergent properties of a network are a consequence of individual behavior. In macaques, for example, where species are classified into a gradient of social tolerance (Thierry 2007), more egalitarian species (e.g. Tonkean macaques) have more connected networks than species with a despotic social style (e.g. Japanese macaques), which leads to increased levels of modularity in the latter (Sueur et al. 2011b, 2012; Pasquaretta et al. 2014). This is due to aggressiveness: strict hierarchies emerge when individuals are more aggressive and the risk of injury is higher (Thierry 2007). Individuals

differ in many ways in their social activities, and those differences affect the social structure. The flow of information and/or pathogens in animal networks is then a consequence of social structural patterns, as I demonstrate in chapters 1, 2 and 3.

From an evolutionary perspective, changes in social structure can affect individual fitness in many ways. For example, as social interactions are reduced with the isolation of sick individuals, social avoidance will limit the spread of pathogens through groups or populations. Surely, social animals exhibit multiple strategies and tactics to deal with pathogen transmission (e.g. the immune system), but the identification of sick individuals can be an evolutionary advantage allowing individuals to avoid contagion. Although the mechanisms from which animals identify sick individuals are still unclear, demonstrations of social avoidance in mammalian species are increasing (e.g. olfactory recognition of sick individuals, Poirotte et al. 2017). Yet, it is important to bear in mind that reduced social interaction of diseased hosts can also occur as a byproduct of lethargy caused by the pathogen, and the decrease of social interactions may represent the inability of sick individuals to maintain proximity to healthier group members (Huffman et al. 1997). In social insect colonies, however, in which social immunity is better understood, cooperation between group members constrains disease spread throughout the colony via several mechanisms, such as not cannibalizing infected corpses and guarding nest entrance to bar infected individuals from entering (Cremer et al. 2007; Stroeymeyt et al. 2014). Parasite transmission is a source of mortality and may also influence the social structure, as it causes the removal of individuals from the network.

Among the several trade-offs individuals must face in social groups, such as enhanced predator avoidance but increased intra-group competition and increased exposure to parasites but increased foraging efficiency, I focused on the trade-off between information and pathogen transmission caused by the social contact or proximity among individuals. From

this perspective, individuals that exhibit flexibility in their social behavior and that better adjust their behaviors to meet the challenges inherent in social relationships will be better able to increase their own fitness.

VII.4. Insights from non-human primate networks

My doctoral research used primates as the main model of study, building on the vast amounts of social and behavioral information already available for this taxonomic group. I showed how individual centrality can affect social transmission processes, and that generalizations about group structure must be made carefully when attempting to understand disease dynamics. In the past decade, network analysis has emerged as a powerful way to investigate social behavior in primates (Brent et al. 2011; Sueur et al. 2011a; for a recent review see Rushmore et al. 2017), as well as other taxa (e.g. e.g. ants: Pinter-Wolmann et al. 2011; beetles: Formica et al. 2012; shark: Mourier et al. 2012; dolphins: Lusseau 2003; giraffes: Carter et al. 2013; birds: Oh & Badyaev 2010; meerkats: Madden et al. 2009). Still, few studies have addressed research questions at the scale of multiple species and/or multiple groups. Among the exceptions to this, Nunn et al. (2015) highlighted important considerations regarding the interaction between group size and modularity in predicting infectious disease spread, which is largely a social process. Their framework, the "social bottleneck hypothesis", illustrates how network structure can decrease disease transmission in modular groups. Chapters 2 and 3 of this thesis were devoted to exploring this hypothesis, wherein I showed the variable importance of network properties in the advance of an outbreak and pathogen transmissibility (chapter 2), as well as that a peak in network efficiency is predicted at intermediate values of modularity (chapter 3). Addressing comparative studies is important not only to draw generalizations about our closest living

relatives, but also to understand, from an evolutionary perspective, the strategies species might have developed to deal with the trade-offs of sociality. Thereafter, I extended my questions to investigate the development of social networks when individuals strictly face the potential trade-off between information and pathogen transmission (chapter 4).

VII.5. SNA and IBM: combining a methodological approach in behavioral ecology

Quantifying social networks has proven an invaluable way to gain insight into the relationships between social structure and social transmission (Whitehead 2008a; Duboscq et al. 2016a; Rushmore et al. 2017). Besides the fact that social network analysis (SNA) provides a refined evaluation of the complexity in the social systems in which individuals are embedded, it also allows researchers to explore how fine-scale social structure can affect ecological and evolutionary processes (e.g. host-pathogen interactions and animal dispersal, Kurvers et al. 2014). Furthermore, SNA can be integrated easily with modeling to improve our understanding of how such complex systems evolve. For example, individual-based modeling (IBM) is a powerful tool to approximate real systems when the question under study is too complex or the model system evolves too slowly to be analyzed (Railsback & Grimm 2012). The advancement of quantitative techniques has thus allowed investigators to create purposeful representations of some real and complex systems, with clear applications in epidemiology and public health policy, but also wildlife ecology and conservation (Salathé & Jones 2010, Railsback & Grimm 2012, White et al. 2017). However, the combination of these methodologies remains under-utilized, and is especially uncommon in the field of behavioral ecology.

One particularity of my thesis is the application of a unique combination of approaches: observational field studies, network analysis and theoretical modeling. In this work, I explored disease outbreak scenarios by simulating the spread of theoretical pathogens with different degrees of transmissibility in empirical primate networks from multiple groups and species (chapters 1 and 2). I then evaluated the emergence of variation in network structure under variable degrees of relationship costs and benefits (chapter 3 and 4). Social network analysis not only provided information concerning the mechanisms of disease transmission within a group, but also showed how social structure varies depending on factors such as group composition and size. Moreover, my methodology and results could be applied more generally to any living social group with a broad array of ultimate applications, such as animal conservation and public health (e.g. Salathé & Jones 2010; Snijders et al. 2017). Drawing upon analytical tools applied to behavioral ecology was shown to improve our understanding of complex social systems. Incorporating observational and theoretical modeling work is a promising and perhaps even ideal next step in behavioral ecology, specifically to reveal patterns within complex social systems.

VII.6. Limitations of study

To afford a more comprehensive evaluation of the relationship between information and parasite transmission, there is room for improvement in at least two basic aspects not addressed in this thesis: i) data collection from individuals of all age classes and ii) experimental validation of the individual-based model I created, the Optimal Relationships Model. First, the more refined is the data that one can obtain to create the social networks (including age classes other than sub-adults and adults, which by and large formed the basis of the networks used in this thesis), the greater will be our ability to understand the dynamics

of transmission on those networks. Many studies have focused on the social networks of sub-adults and adults, often excluding immature group members (Lehmann et al. 2007; Bret et al. 2013; Duboscq et al. 2016b). The biggest reason for excluding other age categories is the difficulty of collecting reliable and resolute data from younger age classes, e.g. because of identification biases. However, recent studies have shown that juveniles can significantly contribute to the understanding of age and sex-specific social roles in the group. For example, the simulated removal of juveniles from a network of wild olive baboons (*Papio anubis*) significantly affected the resulting group structure, which leads to an incomplete representation of age- and sex-related social roles in animal societies (Fedurek & Lehman 2017).

Another goal of future studies might be the validation of the Optimal Relationships Model. Although it is logistically unfeasible to perform experiments over the large temporal scales relevant to social mammals, I believe a more simplified system, such as that of insects, might provide the adequate conditions to physically manipulate the costs and benefits of social relationships, and ultimately the network efficiency itself. The social system I modelled is a simplified representation of the complex organization observed in animal societies, and I am aware that multiple factors can simultaneously influence individual decisions (e.g. resource availability, age) - factors varying according to each species. By testing this model in a less complex social system, I believe I will be able to further understand to what degree the trade-off between information and parasite transmission affects individuals' decisions, and consequently, the evolution of social networks.

VII.7. Research contributions

The outcomes of my thesis may be of interest to researchers in many disciplines, including behavioral and evolutionary ecology, network science, and epidemiology. Besides the contributions this research brings in highlighting social transmission processes, I believe my findings are also of importance to a non-academic audience interested in animal societies and its evolution, epidemiology and conservation. The following paragraphs frame the specific contributions of my doctoral thesis research at three distinct levels.

The first application of my thesis regards the relationship between social structure and the evolution of society. One of the main questions in behavioral and evolutionary biology has been to understand how social animals deal with costly relationships. I present network properties that constrain or favor social transmission according to pathogen transmissibility, and I bring insights into the potential trade-off between information and pathogen transmission. By creating the Optimal Relationships Model, I could track back the formation of social structures to make inferences about the effects of individual decisions on the emergent properties of networks. My findings therefore add a fundamental piece towards the big puzzle surrounding how animal societies evolved, and more specifically, how variation in social behavior can drive changes in the emergent properties of social structure.

Second, my research adds important contributions to the picture of network epidemiology. My study design using data from 68 groups of 21 non-human primate species allowed me to draw strong comparisons and highlight variables that play similar roles in the different groups and species. My findings show that interactions between group size and network properties predict outbreak size, but their influence is dependent upon the stage of the outbreak and the transmissibility of the pathogen (e.g. modularity negatively affects prevalence either at the initial or advanced stages of an outbreak when a moderately- or

highly-contagious pathogen is introduced into the system, respectively). This result highlights that epidemiological models aiming to get a detailed picture of the dynamics of the system should take into consideration the different stages of an epidemic and the variation in effects of different network properties. Identifying optimal conditions or network topologies that constrain disease spread is one of the most important questions in network science, epidemiology and public health. The current study brings important theoretical insights through a comparative perspective to determine factors underlying infectious agent transmission in social organisms.

Finally, my findings have application in the design of pathogen control strategies for wildlife conservation and public health programs. I first provided support for the estimation of individual centrality as a key factor in the chain of social transmission, and showed that indirect connections (i.e. betweenness and eigenvector centralities) are the most predictive of group-level disease spread, whereas direct connections (i.e. strength centrality) are a major predictor of each individual's risk of acquiring an infection. Yet, I also demonstrated that slightly different relationships between individual and social attributes (such as age and dominance rank in Japanese macaques) in some networks appear less relevant than they are in others in affecting transmission metrics (e.g.: percentage of infected individuals and latency to whole group transmission). My findings thus suggest that generalizations about the importance of network position in pathogen flow is somehow problematic and should be made with caution.

VII.8. Future perspectives: where do we go from here?

VII.8.1. Understanding the mechanisms driving sociality

In my thesis, I have investigated the link between social structure and social transmission, but I did not focus on how the social environment (i.e. predation risk, feeding competition) drives social structure. It is well-known that parasites may either drive social structure (Freeland 1976) and/or be "passengers" of host sociality (MacIntosh et al. 2012), but how natural enemies (i.e. predators and parasites) or feeding competition modulate group structure is still an under-explored topic. In response, I propose to expand the fourth chapter of my thesis by increasing the complexity of the Optimal Relationships Model to include numerous competing external pressures such as the spatially-explicit availability of resources and predation, as well as infection risk, in addition to demographic factors such as birth, death, immigration and emigration, to further understand how social structure evolves and ultimately how it affects social transmission. This investigation will help to predict patterns of individual behavior under a complex of environmental pressures, and may shed light on our understanding of social evolution.

VII.8.2. Targeted vaccination in disease outbreaks: a wildlife conservation approach

The indication that trait-based vaccination (e.g. age, dominance rank, family size) could be used as a proxy for centrality to optimize pathogen control in wild chimpanzees (Rushmore et al. 2013, 2014) is providing future directions for research into population management and conservation strategies. This is important because disease interventions such as network-

based vaccinations in wildlife populations are being considered more and more given the threat of extinction facing many animals globally (Salathé & Jones 2010; Sih et al. 2017; White et al. 2017). However, analysis of the networks in my thesis highlights that using a trait-based vaccination strategy, which is thought to enhance the efficacy of the intervention because it should target key individuals ("super-spreaders"), might have less optimal results than one might hope in preventing further disease spread in cases with even just one or two outlying individuals. Indeed, Rushmore's (2013, 2014) conclusions are limited to only one habituated chimpanzee group, highlighting the need to validate those perspectives at a broader comparative scale. Thus, I propose to move from these preliminary observations of one or a few social groups to a much deeper understanding of the role of individual- and group-level traits in disease spread, adding critical knowledge to inform future conservation and disease management strategies (**Figure VII.3**).

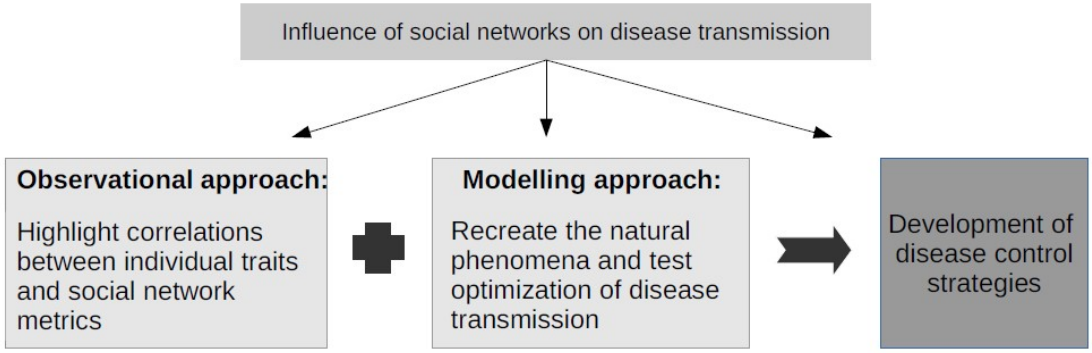

Figure VII.3. Simplified proposed plan of research.

VII.8.3. Are certain populations more vulnerable to disease outbreaks?

The field of network science has provided invaluable contribution on how social structure affects epidemic behavior (Keeling & Eames 2005; White et al. 2017; the current thesis). Anthropogenic activities, such as habitat fragmentation, have been documented to influence social structure, to have direct implications for group dynamics (e.g., decrease dispersal distance) and social systems. Habitat fragmentation is considered one of the major causes of reduced connectivity among groups/populations, and is hypothesized to significantly impact population health (Chapman et al. 2006; Bonnel et al. 2016), and consequently species survival (Chapman et al. 2013). Currently, 70% of the global remaining forest is within 1km of the forest's edge, due to anthropogenic causes of fragmentation (Haddad et al. 2015). A ramification of my work may be to assess whether primates are sensitive to environmental changes that are universal (e.g. higher temperature) or whether they are more sensitive to changes that are local (e.g. fragment isolation), which would complicate predictions of how primates in general will respond to landscape fragmentation. To answer this question, I propose an integrative approach combining i) experimental studies, ii) field studies, and iii) theoretical modeling to investigate the role of landscape change on the social structure of non-human primates and their vulnerability to stochastic processes such as disease outbreaks. To achieve this goal, an international network of collaborators has been created, combining experts from various field sites in the world, such as Brazil, China and Uganda (**Figure VII.4**).

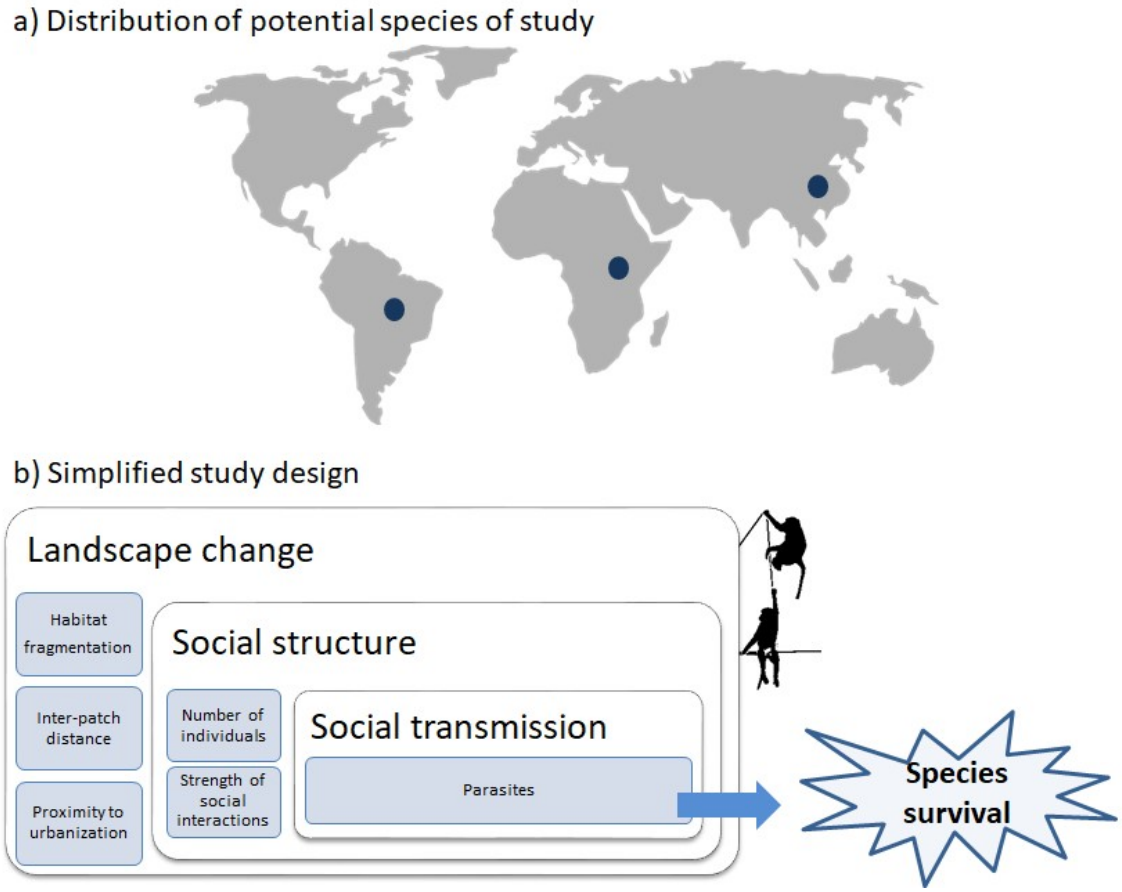

Figure VII.4. Schematic distribution of the studied species (a) and proposed study design to investigate the landscape's influence on primate networks (b).

VIII. Concluding remarks

(English) The research presented in this thesis investigated the relationship between social structure and social transmission with the ultimate aim of understanding how social animals deal with the potential trade-off between acquiring information and socially-transmitted pathogens. By encompassing the fields of behavioral ecology, network science and epidemiology, this work suggests that i) individual centrality in a social network determines the speed of pathogen transmission but also the probability of becoming infected, both of which are biased to central individuals; ii) some global network properties favor pathogen transmission (e.g. density), some constrain it (e.g. modularity and diameter), and still others

have mixed effects (i.e. centralization), depending on the stage of the epidemic and the pathogen's transmissibility; iii) network efficiency peaks with intermediate values of group substructure in theoretical and empirical networks; and, iv) variation in network properties is a consequence of individual decisions that reflect the trade-off between collecting information and avoiding infection.

Overall, my thesis reveals the importance of looking at mechanisms of social transmission with the potential to understand the complexity underlying individual relationships and, consequently, the great diversity in social structure observed across animal societies. I demonstrated that emergent network properties might reflect a trade-off between information and pathogen transmission if individuals optimize the costs and benefits of their relationships. Although this hypothesis arises from the results of a simulated system, empirical evidence does exist showing that individuals change their social relationships to increase benefits and decrease costs of social relationships. Social networks are dynamic, with individuals changing social relationships according to the behaviors and status of conspecifics. My thesis, besides contributing further to our understanding of pathogen transmission in empirical networks, represents the first step towards a more comprehensive framework for examining the potential trade-off between between the myriad connection costs and benefits inherent in animal societies.

(Français) Les recherches présentées dans cette thèse se sont intéressées aux relations entre la structure sociale et la transmission sociale dans le but ultime de comprendre comment les sociétés animales effectuent un compromise entre l'acquisition de l'information et la transmission sociale des pathogènes. Fruit d'un travail interdisciplinaire, à la croisée de l'écologie comportementale, de la science des réseaux et de l'épidémiologie, ce travail suggère que i) la centralité des individus au sein du réseau social determine la vitesse de transmission des pathogènes mais également la probabilité d'être infecté; ii) certaines

propriétés globales du réseau favorisent la transmission de pathogènes (comme la densité) alors que d'autres vont la constraindre (comme la modularité et le diamètre) et certaines vont avoir un effet different (comme la centralité) selon l'étape de l'épidémie et le capacité de transmission du pathogène; iii) un pic d'efficacité du réseau à des valeurs intermédiaires de sous-structure de groupe dans des réseaux théoriques et empiriques; et iv) la variation des propriétés du réseau est la consequence de décisions individuelles réflétant le compromis entre la collecte d'information et l'évitement de l'infection.

Globalement, ma thèse révèle l'importance d'étudier les mécanismes de transmission sociale et son potentiel dans la compréhension de la complexité sous-jacente aux relations individuelles et, par conséquent, de la compréhension de la diversité des structures sociales observée à travers les sociétés animales. J'ai ainsi démontré que les propriétés de réseau émergentes pourraient refléter un compromis entre transmission de l'information et transmission de pathogènes dans le cas où les individus optimisent les coûts et bénéfices de leurs relations. Bien que cette hypothèse est née des résultats d'un système simulé, des preuves empiriques montrant que les individus changent leurs relations sociales dans le but d'en augmenter les bénéfices et de diminuer leurs coûts. Les réseaux sociaux sont ainsi dynamiques, avec des individus changeant leurs relations sociales selon leurs comportements et le statut de leurs conspécifiques. Ma thèse, en plus de contribuer à la compréhension de la transmission des pathogènes dans des réseaux empiriques, représente la première étape dans le développement d'un cadre de recherche plus large afin d'examiner le potentiel compromis entre la myriade de coûts et bénéfices liés à la vie en groupe dans les sociétés animales.

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Appendices

X. APPENDICES

APPENDIX A:

Romano V, Duboscq J, Sarabian C, Thomas E, Sueur C, MacIntosh A (2016) Modeling infection transmission in primate networks to predict centrality-based risk. *American Journal of Primatology*. 28(7): 67–779.

APPENDIX B:

Duboscq J, Romano V, MacIntosh AJJ, Sueur C (2016) Social information transmission in animals: lessons from studies of diffusion. *Frontiers in Psychology.* 7: 1147. (doi:10.3389/fpsyg.2016.01147).

APPENDIX C:

Duboscq J, Romano V, Sueur C, MacIntosh AJJ (2016) Network centrality and seasonality interact to predict lice load in a social primate. *Scientific Reports.* 6: 22095. (doi:10.1038/srep22095).

APPENDIX D:

Duboscq J, Romano V, Sueur C, MacIntosh AJJ (2016) Scratch that itch: revisiting links between self-directed behavior and parasitological, social and environmental factors in a free-ranging primate. *Royal Society Open Science.* 3(11): 160571. (doi:10.1098/rsos.160571).

APPENDIX E:

Duboscq J, Romano V, Sueur C, MacIntosh AJJ (2017) One step at a time in investigating relationships between self-directed behaviors and parasitological, social and environmental variables. *Royal Society Open Science.* 4: 170461.

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RESEARCH ARTICLE

Modeling Infection Transmission in Primate Networks to Predict Centrality-Based Risk

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Social structure can theoretically regulate disease risk by mediating exposure to pathogens via social proximity and contact. Investigating the role of central individuals within a network may help predict infectious agent transmission as well as implement disease control strategies, but little is known about such dynamics in real primate networks. We combined social network analysis and a modeling approach to better understand transmission of a theoretical infectious agent in wild Japanese macaques, highly social animals which form extended but highly differentiated social networks. We collected focal data from adult females living on the islands of Koshima and Yakushima, Japan. Individual identities as well as grooming networks were included in a Markov graph-based simulation. In this model, the probability that an individual will transmit an infectious agent depends on the strength of its relationships with other group members. Similarly, its probability of being infected depends on its relationships with already infected group members. We correlated: (i) the percentage of subjects infected during a latency-constrained epidemic; (ii) the mean latency to complete transmission; (iii) the probability that an individual is infected first among all group members; and (iv) each individual's mean rank in the chain of transmission with different individual network centralities (eigenvector, strength, betweenness). Our results support the hypothesis that more central individuals transmit infections in a shorter amount of time and to more subjects but also become infected more quickly than less central individuals. However, we also observed that the spread of infectious agents on the Yakushima network did not always differ from expectations of spread on random networks. Generalizations about the importance of observed social networks in pathogen flow should thus be made with caution, since individual characteristics in some real world networks appear less relevant than they are in others in predicting disease spread.
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Key words: social relationship; wildlife epidemiology; agent-based model; social network analysis

INTRODUCTION

In a social group, each individual is part of a network that varies in size, distribution, and dynamics of relationships. Observed interactions between social animals are the outcome of trade-offs between the costs and benefits of sociality [Krause and Ruxton, 2002] and one clear cost of being social is that many pathogens are transmitted via social interactions [Corner et al., 2003; Drewe et al., 2011; Otterstatter and Thomson, 2007]. Heterogeneity in host associations, for example, may influence the flow of disease-causing organisms through populations and mediate the risk of contagion across individuals [Newman, 2002; Nunn and Altizer, 2006]. In consequence, there has been increased effort to investigate how association patterns and social positions of each individual in a network can affect

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disease transmission, via experimentation, and/or modeling, in a vast range of species (humans [Bansal et al., 2007; Salathé and Jones, 2010], non-human primates [Carne et al., 2014; Griffin and Nunn, 2012], ungulates [VanderWaal et al., 2014], reptiles [Aiello et al., 2014; Godfrey et al., 2009]). While the networking approach is appealing for its capacity to depict complex systems [Kurvers et al., 2014], modeling offers further utility for understanding and predicting the behavior of these systems [Newman, 2003]. Agent-based modeling, for example, represents individuals as unique entities in the environment; by simulating local interactions among agents and their environment, it offers a less simplified and thus more realistic representation of real systems [Amouroux et al., 2010; Railsback and Grimm, 2012]. For these reasons, combining network techniques with modeling has emerged as a powerful tool for examining dynamics of infectious diseases [Craft et al., 2010].

In this context, recent models exploring association patterns have shown that global network properties, such as modularity [Griffin and Nunn, 2012; Nunn et al., 2015], as well as individual-level properties, such as node centrality [Rushmore et al., 2014; Salathé and Jones, 2010; VanderWaal et al., 2014], may regulate pathogen transmission. For instance, the way in which individuals are more or less central in a group's social network directly influences the way in which an infectious agent or information will be spread through a group and as a consequence, the relationship between centrality and probability of transmission [Griffin and Nunn, 2012; Sueur et al., 2012]. Central individuals may act as super-spreaders of disease-causing agents, and targeting them (e.g., during vaccination or culling efforts) can therefore be an efficient way to implement preventive measures against disease [Christley et al., 2005; Rushmore et al., 2014]. However, similar studies on information transmission have shown contrasting results, with a demonstrated influence of social centralities and network structure in some groups but an absence thereof in others [Boogert et al., 2008; Kendal et al., 2010; Schnoell and Fichtel, 2012]. In this way, it seems crucial to understand how social network structure at both the global and individual levels might interact to predict transmission within a group.

Non-human primates are useful study subjects to investigate the influence of sociality on disease transmission. First, many species are obligate social animals. Second, their close phylogenetic relationship with humans means that many non-human primate diseases are also a concern for humans [Hahn et al., 2000; Wolfe et al., 1998]. Likewise, human diseases are a concern for nonhuman primates, for example, causing marked morbidity in apes through infection linked to tourism or research activities [Köndgen et al., 2008; Woodford et al., 2002]. Furthermore, understanding transmission dynamics in primates is critical for development of conservation and management strategies, given that ca. 50% of primate taxa are now under threat of extinction [Mittermeier et al., 2009] and infectious disease is known to be a significant driver of population decline [Leendertz et al., 2006]. Therefore, increasing fundamental understanding of how sociodemographic factors might interact with disease transmission among primates is now critical, particularly to predict how continued human encroachment and habitat modification might impact primate health, fitness, and population viabilities in the future [Chapman et al., 2005]. Studies have thus begun to investigate the variable influence of specific individuals and the contexts in which they interact in the dynamics of disease spread using real world primate networks [Carne et al., 2013, 2014; Griffin and Nunn, 2012; Rushmore et al., 2013, 2014].

In this study, we combine social network analysis of empirical data and agent-based modeling to investigate the theoretical relationship between network properties and the propagation of infectious agents. We focus on infectious agents transmissible through social contact networks in Japanese macaques, which provide a well studied and thus tractable model system [MacIntosh, 2014]. Macaques are generally considered the most widely distributed and best studied group of non-human primates [Thierry, 2007; Thierry et al., 2004], and in many parts of their range exist in extreme proximity to human settlements. There is also some empirical evidence that infection by nematode parasites in Japanese macaques specifically is related to network centrality and position within the dominance hierarchy [MacIntosh, 2014; MacIntosh et al., 2012]. However, many of the epidemiological processes involved, particularly those concerning other groups of socially transmissible agents in these and other macaque species, remain poorly understood.

To test our hypothesis that the structure of social contact networks mediates the transmission of infectious agents, we first constructed networks of grooming, a very conspicuous behavior that provides a good approximation of social contact [Altizer et al., 2003] and which represents about 90% of body contact between female Japanese macaques [Duboscq et al., 2016]. We then tested whether the spread of a theoretical infectious agent on these observed networks differed from its spread on random networks with the same number of individuals and degree distribution. Second, to further understand the role of individuals in transmission dynamics, we tested whether individual traits such as age, rank, and family size affected an individual's network position. Recent studies have suggested that such traits can be used as proxies of centrality and thus used to predict importance in disease dynamics [Rushmore et al., 2013, 2014]. We then constructed an agent-based model to simulate the transmission of an infectious agent through the observed empirical contact networks. Based on the hypothesis that social network centrality and transmission dynamics

are linked, we predicted first that central individuals in the contact network would transmit disease faster and to more individuals than less central individuals. To test this prediction, we modeled: (i) the percentage of individuals infected before a latency threshold of transmission was reached, which should be higher when starting with more central individuals, and (ii) the latency between initial infection and the point at which the whole group became infected, which should be lower when starting with more central individuals. Second, we predicted that central individuals would also be at greater risk of being infected, which we estimated via (i) the probability that an individual would be infected first among all group members, which should be higher for central individuals, and (ii) each individual's mean rank in the chain of transmission, which should be lower among more central individuals. This dual approach of social network analysis and simulation modeling allowed us to ascertain the importance of central individuals in disease transmission, not only as key agents of disease spread but also as those that are most vulnerable to being infected.

METHODS

The research presented here complied with the Guidelines for the Care and Use of Nonhuman Primates established by the Primate Research Institute of Kyoto University, to the legal requirements of Japan and to the American Society of Primatologists (ASP) Principles for the Ethical Treatment of Non Human Primates.

Study Site and Subjects

We studied two well-habituated groups of Japanese macaques, one provisioned but free-ranging on Koshima island (31°27′N, 131°22′E; *Macaca fuscata fuscata*) and the other wild (i.e., not provisioned) on Yakushima island (30°20′N, 130°30′E; *Macaca fuscata yakui*). Koshima is approximately 0.3 km^2 in area and is mainly covered by secondary broad-leaved evergreen forest [Iwamoto, 1974]. The main group of Koshima macaques has been periodically provisioned with wheat (currently ca. twice per week) and intensively studied, with group composition recorded for ca. 60 years [Iwamoto, 1974]. During the study period (see below), the group included approximately 51 individuals, including 21–24 adult females (≥5 yo), 11–16 adult males (≥5 yo), 11–18 juveniles (1–4 yo), 1 infant (<1 yo) born in 2012.

The southernmost population of Japanese macaques living on Yakushima represents a distinct subspecies from those in the rest of Japan. Yakushima is a mountainous island of approximately 500 km^2, much of which is protected as a UNESCO World Natural Heritage site and by the Kagoshima prefectural government. The study group ("Umi") inhabited the protected western coastal forest, which like Koshima is dominated by broad-leaved evergreen secondary forest, with an estimated home range size of roughly 0.8 km^2 [Sueur et al., 2013]. Umi group varied between 59 and 70 individuals during the study period, including 18 adult females, 11–15 adult males, and 20–31 juveniles, with 11 infants born in 2008 and 6 infants born in 2009 [MacIntosh et al., 2012]. Ages of individuals were estimated based on body size and state of development of sexual organs and perianal regions.

Behavioral Data Collection and Networks

We collected data on grooming interactions (both directions, received and given) of adult females over 8 months (between October 2012 and May 2013) in Koshima ($N = 21$) and 16 months (between October 2007 and August 2009) in Yakushima ($N = 18$). We focused on female social networks in this study. Japanese macaque societies are organized into female-bonded groups in which females form the stable core [Yamagiwa and Hill, 1998]. As a result, we expect females to dominate dynamics on social networks. It was also difficult to adequately sample other members of the groups, for example, because male group affiliation is much less stable than that of females while juveniles are often difficult to observe and identify reliably. Both groups were habituated to the presence of human observers and adults could be identified using tattoos (Koshima only) and/or other individual traits. We used grooming networks because they are considered to be an excellent proxy of social contacts, and in addition can avoid issues arising from the gambit of the group [Franks et al., 2010]. This concept underlies that all individuals seen grouping together, during an observation census, are associating with every other individual in that group. For example, if individual A is strongly associated with B and B is strongly associated with C, the gambit of the group assumes that A and C are strongly associated too. This can result in overestimation of real associations resulting in errors in estimating the disease transmission process. This overestimation is not observed when using body contact, especially grooming, between individuals. Previous studies have also shown no differences in transmission processes using either body contact or grooming interactions as the basis for network construction [Pasquaretta et al., 2014], but we focus on grooming to investigate infectious agents only transmissible trough social contact.

To collect data, we conducted 15 min instantaneous focal sampling at 1 min intervals on Koshima, while all grooming activity performed during 60 min focal samples were recorded on Yakushima [Altmann, 1974]. To confirm compatibility in the data sets, which were collected using different sampling methods, we transformed the continuous-time grooming matrix

constructed from Yakushima data into an instantaneous scan matrix after sampling the focal data at 1 min intervals. We observed that the matrices were 99.6% correlated (Mantel Z test: $P = 9.99^{e-13}$), showing that less than 0.5% of the data were lost when moving from one method to the other. We are thus confident that the Koshima and Yakushima networks are comparable. At both sites, we avoided re-sampling the same individual within a day wherever possible. When this was violated, individuals were not observed within 1 h of a previous focal sample from the same individual. From the Koshima data set, we extracted minute-data points of grooming while for Yakushima we considered the total grooming time between two individuals. There was no difference in hourly observation time between individuals on either Koshima group (mean ± SD: 12.96 ± 0.50, $\chi^2 = 8.05$, $P = 0.99$) or Yakushima group (mean ± SD: 45.61 ± 0.81, $\chi^2 = 4.39$, $P = 0.99$), and the grooming frequencies were almost identical at both sites (Koshima: grooming given = 13.4% ± 6.3%; grooming received = 7.3% ± 3.6%; Yakushima: grooming given = 12.6% ± 3.6%; grooming received = 6.7% ± 1.5%; Yakushima data from MacIntosh et al. [2012]). Although there were differences in the total observation time between the Yakushima and Koshima groups, we believe the data set to be large enough in each group to decrease expected errors in social network measures [Whitehead, 2008]. However, because of the differences in data collection and despite the high correspondence between methods using the Yakushima data, we remain cautious and make no direct comparisons between the two groups. Instead, we focus on the observed transmission events within each separate network based on grooming behavior, a well-conserved and highly conspicuous behavior unlikely to differ substantially between groups and thus bias our results.

From the undirected and weighted grooming networks, we estimated the global measures of social networks, defined as follows:

- Density: the ratio between the number of observed edges and the number of possible edges in the network [Sueur et al., 2011];
- Diameter: the longest path edge of the network;
- Overall clustering coefficient: the mean of all nodes' clustering coefficients, which measures how densely one individual is connected to its neighborhood [Hanneman and Riddle, 2005];
- Average degree: the average of sum of the number of edges of a vertex;
- Network modularity: the extent of sub-grouping in a network [Newman, 2004];
- Transitivity: the circumstance where node i is connected to node j, node j is tied to node k and node i is also tied to node k [Hanneman and Riddle, 2005].

We also calculated various weighted individual-level measures which are typically referred to as centrality coefficients to compare the roles of individuals in the transmission of infectious agents. These coefficients included:

- Strength: the sum of each node's edge values. The individual with the most and strongest connections has the highest strength value [Sueur et al., 2011]. In our study, we have considered two different inputs. For Yakushima, strength indicates grooming time between individuals, while for Koshima strength indicates the number of times two individuals were observed to groom each other during sampling points.
- Eigenvector: the weighted connectivity of an individual within its network, also considering the weighted connectivity of its neighbors. Individuals tied to central individuals (i.e., those with a high connectivity themselves) should have higher centrality than those connected to less central individuals [Borgatti et al., 2013].
- Betweenness: the number of shortest paths that pass through the considered individual. The more connections that are made through one individual, the greater its value of betweenness becomes [Hanneman and Riddle, 2005; Newman, 2004].

Most of the global measures of social networks (density, diameter, overall clustering coefficient, and transitivity) as well as the betweenness centrality coefficients were estimated using Ucinet 6.4 [Borgatti et al., 2002]. Other network measures such as modularity, eigenvector, and strength centrality were estimated via SocProg 2.4 [Whitehead, 2009]. Since we built our networks based on the weighted matrices, we estimated the weighted measure of each coefficient. The grooming networks were visualized using Gephi 0.8.2 beta [Cherven, 2013].

Individual and Social Traits Associated With Network Centrality

We categorized Japanese macaques by age, hierarchical rank and family size (the latter for Koshima only). Because Koshima group has been monitored for decades [Iwamoto, 1974], exact ages are known for each individual. Such data were not available for Yakushima, so we instead distributed the sexually mature females into three age classes (young adult $\geq 5 < 10$ yo, adult 10–14 yo, old adult >14) following MacIntosh et al. [2012]. We also incorporated dominance ranks into our analysis, which were distributed within significantly linear dominance hierarchies in both groups (Landau's linearity index corrected for unknown relationships: Koshima: $h' = 0.68$, $P < 0.001$; Yakushima: $h' = 0.40$, $P = 0.005$). Finally, family size was based on the definition of Rushmore et al. [2013], with a family

unit including a mother and the total number of her living weaned offspring. An individual with no offspring, for instance, was counted as having a family size of one [Rushmore et al., 2013].

Random Networks

To identify if the dynamics of disease transmission ((i) percentage of infected individuals; (ii) latency to complete transmission; (iii) probability of acquiring an infectious agent; and (iv) latency of acquiring an infectious agent) in the Koshima and Yakushima networks differed from those in random networks, we compared the distributions of infectious agent transmission from the simulation on both random and observed networks. We created 100 random networks for each focal group, maintaining the original numbers of both individuals and observed bonds between them, via Ucinet 6.4 [Borgatti et al., 2002]. Computer simulations were run 1000 times for each random network.

Disease Transmission Graph-Based Model

Individual identities as well as grooming interactions were included in an individual-based model using a Markov chain process developed by Sueur et al. [2009]. This model is basically equivalent to the classical SI epidemiological model, in which individuals can only move from susceptible (S) to infected (I) classes with no possible recovery or death of an infected individual. Traditionally, such models considered the probability of contact for each pair of individuals to be equal, but we allowed for heterogeneity of relationships, which has long been declared to improve the ability to understand and predict disease dynamics [Keeling and Eames, 2005]. To fit with the current model, grooming relationships were transformed following the description of Sueur and Deneubourg [2011]. Observed frequencies were first transformed to relative frequencies (i.e., divided by the sum of observed frequencies) and then multiplied by $N-1$, N being the number of group members, to obtain corrected frequencies implemented in the model. In the model, the probability that an individual will transmit an infectious agent to another depends on the strength of the relationships it has with each non-infected individual. In the same way, the probability that an individual will become infected depends on its relationships with already infected group members. Thus, the more a non-infected individual interacts with infected individuals, the greater is its probability of being infected. Likewise, the more an infected individual interacts with non-infected group members, the greater is its probability of infecting others.

In the model, each individual has the same probability of being the first infected, meaning that we focus only on the exposure to pathogens and assume a constant susceptibility to infection across individuals. This probability is named intrinsic probability λ. It is important to highlight that this lambda could be noted λ_i but as all individuals have the same intrinsic probability, we indicate λ. An individual i has to be infected to see its probability λ equal to 0, but if an individual k is infected and i is not yet infected, the probability λ is no longer 0. Thus, as soon as one individual is infected, the probability ψi for another individual i to be infected is:

$$\Psi_i = \lambda + C\sum_{k=1}^{N} r\ (k,i) \quad \text{With } C/\lambda = R_0 = 10,$$

where N denotes the group size, $r\ (k,i)$ represents the social relationship that individual i has with individual k, and C is a mimetic coefficient, which means that the probability of being infected is increased by a coefficient C following contact with conspecifics. R_0 is the basic reproduction number used to quantify the transmission potential of a disease; using $R_0 = 10$ allowed us to estimate properties of an outbreak with a highly contagious pathogen, such as was estimated for measles in humans and subsequently extrapolated for heuristic purposes to chimpanzees [Rushmore et al., 2014]. While R_0 is known to differ dramatically across disease-causing organisms, using a high R_0 allows us more power to identify the influence of network structure in empirical data with small sample sizes. However, we also simulated the transmission of infectious agents with varying R_0 and found that their dynamics are consistent in both study groups, even though the total size of the outbreak varies (Suppl. Fig. S1).

We implemented the model in Netlogo 3.1.5 [Railsback and Grimm, 2012]. At each run of the model, a number between 0 and 1 was randomly attributed to each non-infected individual; when this number was lower than the theoretical infection probability ψ_i of each individual i, the individual i became infected; if this number was higher than the theoretical infection probability, the individual was not infected. The identity, infection rank (order of infection of individuals) and latency (number of runs) of infection for each individual were scored for each simulation. We induced two conditions, first considering the complete transmission latency and second restricting the latency of infection to three simulation runs. The first condition allowed us to investigate the period required to complete infection and the order of infection. One simulation corresponds to the infection of all group members. The total infection latency of a simulation is the number of runs required until all group members are infected. The second condition allowed us to investigate the number and identity of individuals infected during a set latency. We ran 10,000 simulations for each condition. The source code is available in the supporting information.

Data Analysis

Generalized linear models were constructed to examine the impact of individual and social factors on centrality position. We tested for significant relationships between centrality coefficients (eigenvector, betweenness, and strength) and the following predictor variables: age, dominance rank, and family size (range: 1–5; Koshima only). Because eigenvector centrality is not truly independent of strength and betweenness coefficients, we estimated the effect of the predictor variables on each centrality measure separately. The distribution of all response variables (centrality measures) deviated from the Gaussian case, so all were square-root-transformed, which performed better than log-transformation and could accommodate the few zeroes in the data set, to meet the assumptions of the statistical models. For all models, we checked that the assumptions of normally distributed and homogeneous residuals were fulfilled by visually inspecting a qqplot and the residuals plotted against fitted values. We further ran a series of diagnostics to judge the validity of the models, including testing for variance inflation, correlation of fitted and residual values and Cooks' distance, all of which suggest the suitability of our models as no obvious violations of assumptions were detected [Field et al., 2012].

We compared the distribution of random and observed networks by Kolmogorov–Smirnov tests with Bonferroni correction [Abdi, 2007]. Regarding the dynamics of disease transmission, first we calculated the mean latency of complete transmission (to the whole group with no time constraint) and the percentage of infected individuals in a time-constrained simulation (three runs). Second, we looked at the probability of each individual being infected given the number of times each individual was the first to be infected by the initial infected individual (i.e., the second to be infected among all group members). We also calculated the mean rank of infection in the transmission order (from 1 to $N-1$ ranks, since the initial infected individual is removed from the analysis). We then correlated centrality coefficients (eigenvector, betweenness, and strength) and dynamics of transmission (complete transmission latency, percentage of infected individuals, probability of being infected, and latency to being infected) using Spearman tests with Bonferroni correction. A P-value equal to or less than 0.05 was considered to be statistically significant, and all tests were two-tailed. Analyses were conducted in R statistical software version 2.15.1.

RESULTS

Network Structure and Centrality Position

The two social grooming networks are illustrated in Figure 1 and the global measures are given in Table I. Of the global network measures presented, only modularity differed considerably between groups. We also observed some differences concerning the interaction between individual/social traits and centrality position. Dominance rank, but not age, was a good predictor of eigenvector and strength centrality in Koshima, while we found weak evidence to suggest that age and dominance rank may influence eigenvector centrality in Yakushima (Table II; Suppl. Figs. S2 and S3). Family size had no effect on network position in Koshima.

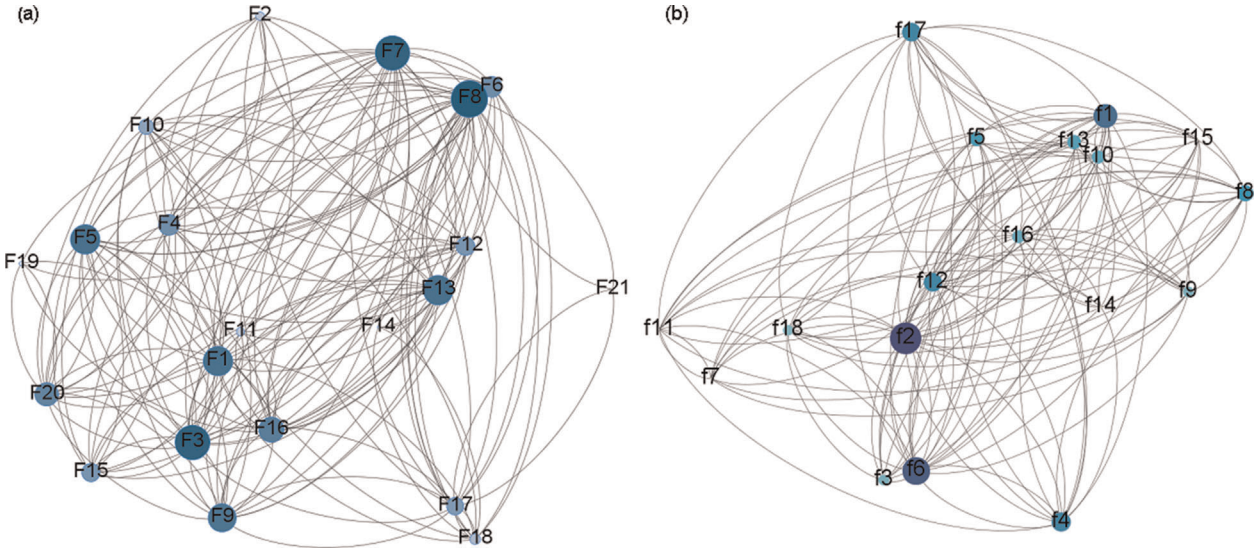

Fig. 1. Social networks of adult female Japanese macaques in Koshima group (**a**) and Yakushima group (**b**). Networks were built using Gephi 0.8.2 beta [Cherven, 2013]. A node (circle) represents a rank identifier, with its size and color directly related to the individual eigenvector centrality coefficient (the higher the centrality, the stronger is the color and the larger is the size of the node). Spacing of nodes was done using the option Force Atlas in Gephi. In this way, nodes are spaced according to their centralities but also according to whom they are connected.

TABLE I. Global Measures of Koshima and Yakushima Networks

Group	Density	Diameter	Average degree	Overall clustering coefficient	Transitivity	Modularity
Koshima	0.41	3	8.19	1.38	0.52	0.38
Yakushima	0.44	3	7.50	1.37	0.50	0.63

Definitions of social network measures are presented in the methods section.

Observed Versus Random Networks

We observed that two of the four distributions from the simulation on the Yakushima network did not differ significantly from those expected of a random network (Kolmogorov–Smirnov tests with Bonferroni correction: probability of being infected: $D = 0.39$, $P = 0.2$; latency of being infected: $D = 0.5$, $P = 0.08$; Suppl. Fig. S4). In contrast, the percentage of infected individuals ($D = 1$, $P < 0.001$) and latency to complete transmission ($D = 1$, $P < 0.001$) differed significantly between observed and random networks. Whereas in the Koshima networks, the probability of being infected ($D = 0.52$, $P \leq 0.05$), the percentage of infected individuals ($D = 0.81, P < 0.001$), latency to complete transmission ($D = 1, P < 0.001$) and latency of being infected ($D = 0.62$, $P \leq 0.05$) all significantly differed between observed and random networks (Suppl. Fig. S5). Thus in the majority of cases, infectious agents spread more readily in observed than in random networks, and the transmission properties of the Koshima network differed more strongly from those of a random network than did the transmission properties of the Yakushima network.

Transmitting an Infectious Agent

Individuals with higher centrality coefficients transmitted infectious agents to the entire group with a shorter latency in the Koshima group ($N = 21$) regardless of the centrality coefficient used (Spearman tests with Bonferroni correction, eigenvector: $r = -0.70$, $P < 0.001$; betweenness: $r = -0.55$, $P \leq 0.05$; strength: $r = -0.75$, $P < 0.001$). This was also generally true in the Yakushima group ($N = 18$), though results depended more on the centrality index measured. The Yakushima group showed a strong relationship

TABLE II. Parameter Estimates From Generalized Linear Models Explaining Variation in Network Centrality Among Female Japanese Macaques in Koshima and Yakushima Groups

Centrality coefficients	Predictors	Estimate	Std. error	t value	Pr(>|t|)[b]
Koshima					
Eigenvector	(Intercept)	0.757	0.147	5.134	8.28e-05***
	Rank	−0.015	0.006	−2.405	0.028*
	Age	−0.033	0.018	−1.818	0.087†
	Family size	0.070	0.053	1.307	0.209
Strength	(Intercept)	1.587	0.294	5.389	4.9e-05***
	Rank	−0.032	0.012	−2.564	0.020*
	Age	−0.036	0.036	−1.003	0.330
	Family size	0.059	0.107	0.555	0.586
Betweenness	(Intercept)	2.915	1.073	2.718	0.015*
	Rank	−0.041	0.045	−0.898	0.383
	Age	−0.053	0.132	−0.405	0.691
	Family size	0.066	0.390	0.170	0.867
Yakushima					
Eigenvector	(Intercept)[a]	0.278	0.035	7.821	4.73e-06***
	Rank	−0.005	0.003	−2.014	0.067†
	Age (adult)	0.038	0.034	1.095	0.295
	Age (old adult)	0.082	0.039	2.079	0.059†
Strength	(Intercept)	0.937	0.101	9.303	2.27e-07***
	Rank	−0.007	0.008	−0.963	0.352
	Age (adult)	0.156	0.096	1.626	0.126
	Age (old adult)	0.176	0.106	1.656	0.120
Betweenness	(Intercept)	2.785	0.687	4.054	0.001**
	Rank	−0.053	0.052	−1.020	0.325
	Age (adult)	0.870	0.654	1.330	0.205
	Age (old adult)	1.105	0.723	1.528	0.149

[a]All comparisons made against the intercept of the first level of each factor in Yakushima (age = young adult).
[b]Significant codes are marked as follows: "***" $P < 0.001$, "**" $P < 0.01$, "*" $P < 0.05$, "†" $P < 0.1$.

between transmission latency and eigenvector centrality ($r = -0.76$; $P < 0.01$) as well as betweenness centrality ($r = -0.56$; $P \leq 0.05$), but strength coefficients were only marginally associated with transmission latency ($r = -0.51$; $P = 0.09$). Central individuals also transmitted infectious agents to a greater number of subjects when compared to less central individuals, but in both groups, betweenness centrality was not associated with the probability of infecting others (Koshima, eigenvector: $r = 0.95$, $P < 0.001$; betweenness: $r = 0.50$, $P = 0.07$; strength: $r = 0.99$, $P < 0.001$; Fig. 2; Yakushima, eigenvector: $r = 0.77$, $P < 0.001$; betweenness: $r = 0.45$, $P = 0.19$; strength: $r = 0.74$, $P < 0.01$; Fig. 3).

Acquiring an Infectious Agent

Central individuals were more likely to be infected than non-central individuals in the Koshima group (Spearman tests with Bonferroni correction, eigenvector: $r = 0.68$, $P < 0.01$; betweenness: $r = 0.64$, $P < 0.01$; strength: $r = 0.82$, $P < 0.001$; Fig. 2), but only strength centrality was significantly correlated with probability of being infected in the Yakushima group (eigenvector: $r = 0.27$, $P = 0.94$; betweenness: $r = 0.08$, $P = 1.00$; strength: $r = 0.71$, $P < 0.01$; Fig. 3). Considering the mean rank of each individual in the chain of transmission, central individuals were more likely to be infected during the first transmission event than less central group mates, with the exception of those with high betweenness scores (Koshima: eigenvector: $r = -0.93$, $P < 0.001$; betweenness: $r = -0.51$, $P = 0.07$; strength: $r = -0.98$, $P < 0.001$; Yakushima: eigenvector: $r = -0.66$, $P \leq 0.05$; betweenness: $r = -0.35$, $P = 0.46$; strength: $r = -0.61$, $P \leq 0.05$).

Regarding analysis of data from Koshima, we identified one subject (f16) having a betweenness value 1.5 times higher than the third quartile, as well as two individuals (f12 and f13) in Yakushima with similarly high eigenvector coefficients. These individuals were therefore removed prior to the analysis. However, if included, correlations between centrality and infection were similar to those presented above (Koshima: complete transmission latency: $r = -0.61$, $P < 0.01$; percentage of infected individuals: $r = 0.49$, $P = 0.07$; probability of being infected: $r = 0.68$, $P < 0.01$; latency to being infected: $r = -0.49$; $P = 0.07$; Yakushima: complete transmission latency: $r = -0.81$, $P < 0.001$; percentage of infected individuals: $r = 0.81$, $P < 0.001$; probability of being infected: $r = 0.27$, $P = 0.85$; latency to being infected: $r = -0.66$; $P \leq 0.05$).

DISCUSSION

This study aimed to test the hypothesis that social network structure modulates disease transmission, and to better understand the influence of an individual's centrality on the transmission of infectious agents in real-world primate networks. In line with our predictions, individuals central to the grooming contact networks not only transmitted infectious agents with a shorter latency to other group members and to a higher percentage of individuals, but were also more prone to infection themselves, being among the first to acquire infections and with a shorter latency than more peripheral individuals. Other simulation studies with transmissible agents whose parameters closely match those of the agents used here also show that direct contact interactions may facilitate the spread of infectious agents [Griffin and Nunn, 2012; Rushmore et al., 2013]. However, our study is among the first to model risks associated with pathogen transmission for central individuals, revealing their increased exposure (here investigated by the probability and latency of infection/transmission) to highly infectious agents. Nonetheless, even though we use the same fundamental data (observed grooming networks) in our simulations of transmitting or acquiring a theoretical infectious agent, results show some differences in the importance of observed centrality distributions in disease spread; transmission was faster and affected more individuals in Koshima than in Yakushima, the latter group producing a network that behaved like a random network in two of our four simulation analyses. These divergences from our predictions suggest that caution should be exercised when making generalizations about transmission processes arising from the network structure. Ultimately, however, networks of proximity or contact interactions are generally accepted to underlie infectious disease dynamics in real groups of animals, including humans [Altizer et al., 2003; Taylor et al., 2001], to which our results further attest.

Our results also showed that both global (network-level, e.g., modularity) and individual (node-level, e.g., centrality) metrics must be considered when predicting disease transmission dynamics. Although most global network measures did not differ between groups, we did observe a large difference in modularity, with the Yakushima group exhibiting a more modular network than the Koshima group. This difference may in part explain the reduced capacity for transmission in the Yakushima network, as increased modularity is known to constrain disease transmission (the "social bottleneck" hypothesis; Nunn et al., 2015). At the same time, we observed distinct distributions of centralities in each group - the variance in the Koshima network being higher than that in Yakushima - and a stronger influence of central individuals and a broader and faster transmission in the Koshima network. The clear relationship between centrality and infection found in the Koshima group may be explained by the interaction between dominance rank and centrality index. In Koshima, dominance rank strongly correlated with eigenvector and

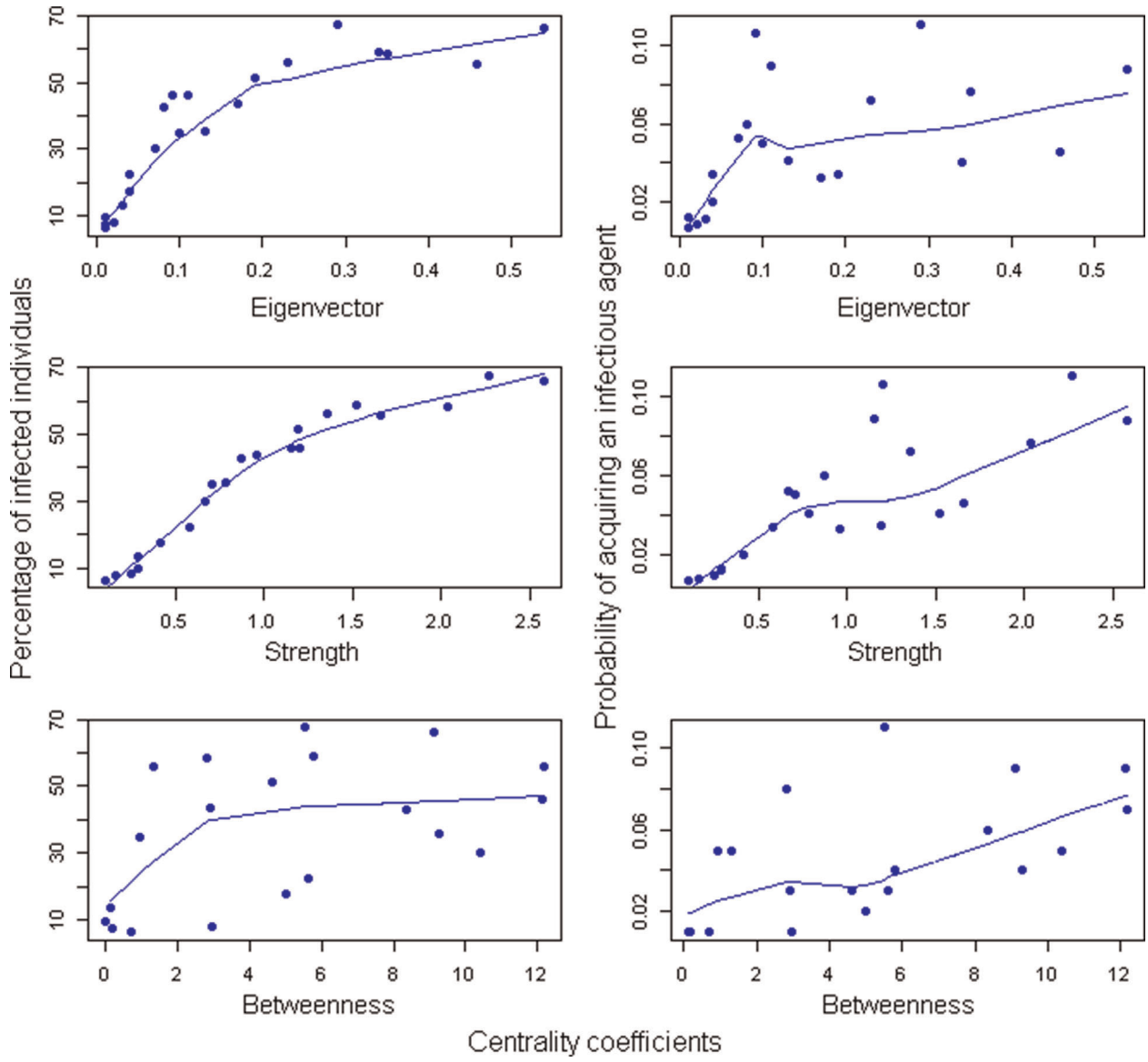

Fig. 2. Relationship between centrality coefficients and dynamics of disease transmission in Koshima group. Blue diamonds represent individual macaques and blue lines represent the mean of individuals infected and the probability of being infected. Outliers have been removed (see text for further information).

strength centralities while age was marginally associated with eigenvector. In Yakushima, however, the relationship between individual/social traits and centrality measures were less consistent, with age and dominance rank only marginally associated with eigenvector centrality. Given that centrality measures showed slightly different relationships to both individual/social attributes and to transmission metrics, it is important to discuss the role of each metric in predicting pathogen transmission. Strength centrality refers to direct connections between individuals, while eigenvector centrality and betweenness coefficient refer to a combination of direct and indirect connections. The fact that transmission latency was strongly related to both eigenvector and betweenness centrality in the Yakushima group, but only marginally to strength, suggests that indirect connections factor more strongly in the transmission chain/dynamics than do direct connections. By contrast, the probability of being infected appears to be driven by direct rather than indirect connections. It thus seems that, while indirect connections are most predictive of latency to complete transmission (a group-level metric), direct connections are most predictive of an individual's probability of being infected (individual-level metric). Too few studies discuss the relative impacts of different levels of network connectedness [Christley et al., 2005]; yet these details are essential to both advancing our understanding of the relationship

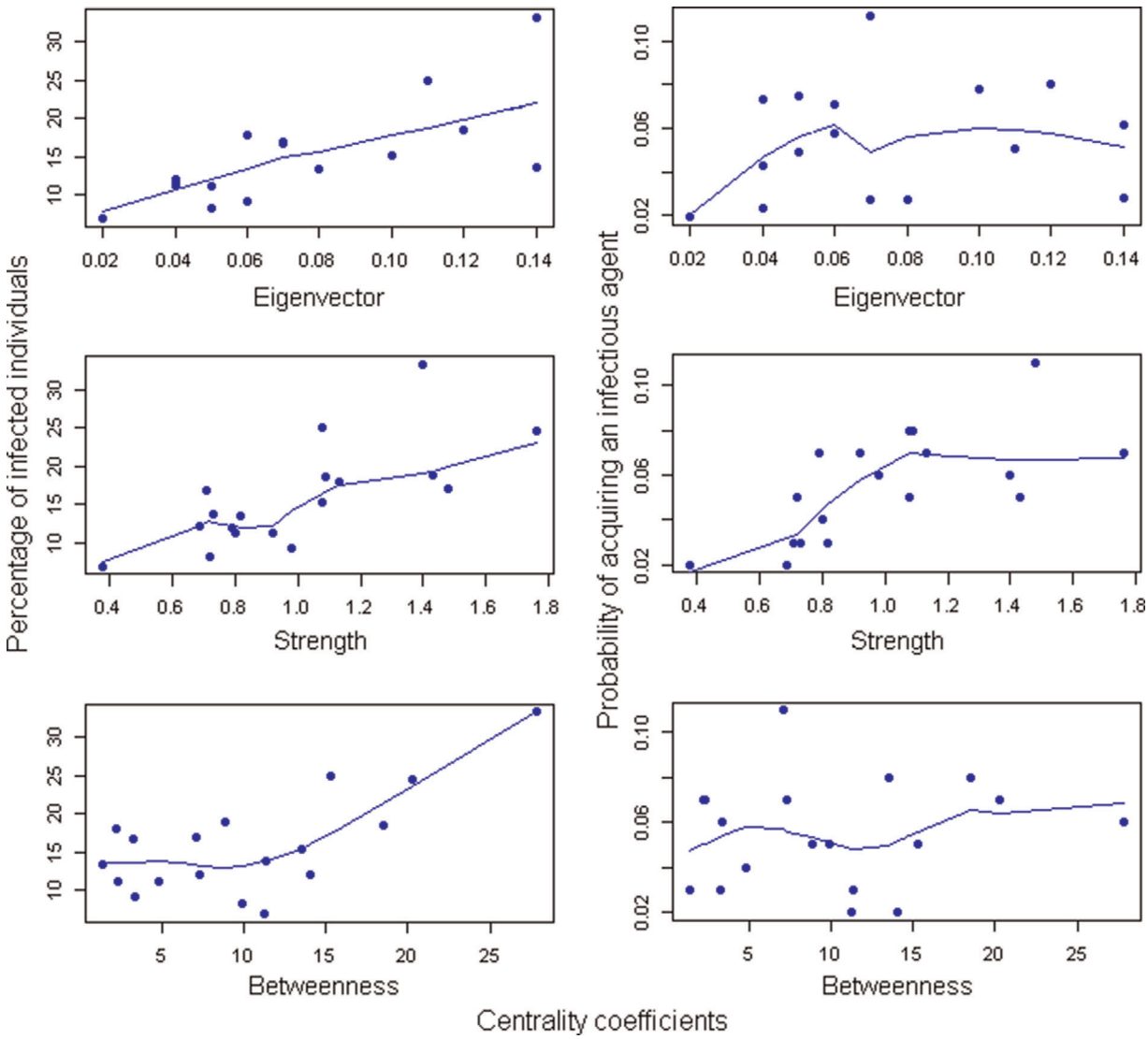

Fig. 3. Relationship between centrality coefficients and dynamics of disease transmission in Yakushima group. Blue diamonds represent individual macaques and blue lines represent the mean of individuals infected and the probability of being infected. Outliers have been removed (see text for further information).

between network structure and disease transmission dynamics, as well as to developing appropriate disease-intervention strategies.

In a handful of cases, targeting central individuals for disease control can reduce outbreak sizes [Salathé and Jones, 2010] and should be more efficient than applying control efforts randomly [Böhm et al., 2009; Rushmore et al., 2014]. Theoretical removal of individuals based on association networks of orangutans and chimpanzees, for instance, highlighted that the low level of association between orangutans may limit the spread of disease through the population, in contrast to chimpanzees whose network structure may allow for faster spread of disease [Carne et al., 2014]. In addition, an observational study with a wild giraffe population showed that transmission is more likely to occur between individuals that are more strongly connected within the network, indicating that an individual network position is a good predictor of transmission network position [VanderWaal et al., 2014]. We found that transmission in Japanese macaques is enhanced after central individuals become infected, which suggests the existence of super-spreaders in the population. However, the current results demonstrate this assumption through contrast; centrality-based vaccination, for instance, may be well suited to the Koshima group, but its efficacy would be more questionable in the Yakushima network given the somewhat reduced importance of centrality measures in our simulations. Ultimately, disease transmission is a stochastic process as demonstrated by our multi-agent model, so observed results might diverge

substantially and perhaps even unpredictably from models using deterministic methods, such as theoretically removing central individuals. Modeling can highlight differences observed at the level of the group, and these differences may have direct implications for disease spread and should therefore be taken into account in future endeavors designing disease management plans.

In this context, identifying relationships between individual traits and network centrality may be useful in assessing disease transmission dynamics, particularly because social roles of individuals can vary across groups. Rushmore et al. [2013, 2014] suggested that we might use individual traits that correlate with centrality in disease intervention strategies, based on their results from chimpanzee networks showing that high-ranking males and individuals with large family units were the most central individuals and thus best to target in vaccination efforts. A related outstanding question is whether or not high dominance rank might allow an individual to better tolerate certain infections [MacIntosh, 2014], which would make them even more capable of spreading infectious agents [Ezenwa and Jolles, 2015]. In our study, however, a middle-ranking old adult female and a low-ranking adult female exhibited the highest eigenvector centrality coefficients in the Yakushima group, while a middle-ranking adult female exhibited the highest betweenness coefficient in Koshima group, all by a very wide margin. The presence of such "outliers" illustrates the need for caution here; even if rank and age are correlated with network position, we have to be careful about using dominance or age as a proxy for centrality in disease transmission. Analysis of the networks in this study highlights that using a trait-based vaccination strategy in cases with even one or two such outlying individuals might have less optimal results than one might hope in preventing further disease spread. Although almost all animal networks are structured according to sociodemographic variables (e.g., age, dominance, sex, etc.), some can also resemble random networks in their transmission dynamics, rendering them less relevant than others in modeling disease spread; that is, our Yakushima networks which behaved as would random networks in some circumstances. In such cases, using dominance or centrality is no more useful in predicting who becomes infected or who transmits disease than making random predictions.

However, we are aware that our model represents a simplification of the real process. The models do not attempt to account for variation in individual susceptibility, which itself can relate to social (e.g., through kinship based shared immunological factors or even dominance hierarchy mediated variation in physiological stress, access to food and nutritional state, etc.) and other intrinsic characteristics (innate resistance factors, chronic stress, etc.). Other pertinent information relates to the variation we found between study groups, and how they might be related to different environmental effects (e.g., specific habitat characteristics, home range size), and of course to contrasting population management strategies (provisioned vs. non-provisioned), all of which can strongly affect the expression of social behavior [Hill, 1999]. Provisioning food to non-human primates is expected to increase proximities between individuals and should increase aggression levels due to closer proximities. We tried to reduce the direct influence of provisioning on individual interactions by using only those data collected at least 1 h after provisioning. Regardless, given a sample size of two networks, we hesitate to make any strong claims about what might have led to the networks observed, and instead focus only on the relative importance of central individuals and network structure in the social transmission of infectious agents in these divergent networks. Studying the influence of network properties in both groups allowed us to understand how different social network measures may affect the transmission dynamics irrespective of the factors that caused the networks to vary.

Another limitation of our study, which should be addressed in future work, is that we included only adult females in our social networks. This was mainly a practicality issue, as for example juveniles are extremely difficult to identify reliably and males, at least in the Koshima group, rarely if ever engage with the group outside of the mating season. Not including such individuals, however, leads to the construction of incomplete networks that may misrepresent true infection dynamics. For example, juveniles are often in contact with their mothers and each other, and may be the most susceptible individuals in the group to disease causing organisms (e.g., MacIntosh et al. [2010]). Males are also known to harbor larger parasite infections than females virtually across vertebrates [Poulin, 1996], and may therefore be key to the spread of infection on networks, even when not occupying central positions. Despite this limitation, however, female Japanese macaques do form the core of the group's social network, presenting the most stable social relationships organized into a rather rigid hierarchical arrangement. Female Japanese macaques are thus likely to be key players in the dynamics of disease transmission in this species.

In conclusion, our study suggests the importance of social network properties in disease transmission at both the global and individual levels, showing the role of central individuals (here in grooming networks) in spreading disease but also their vulnerability to becoming infected. Possible interactions between individual and global network measures might affect the outcome of disease dynamics. Furthermore, we show that the combined approach

of network analysis and modeling provides a promising tool to predict epidemics in primates and others animals [Böhm et al., 2009; Carne et al., 2014; Craft and Caillaud, 2011; Salathé and Jones, 2010], but that caution should be exercised when generalizing since some networks or network properties may be less relevant than others in predicting disease dynamics. Indeed, it is well-known that detailed analyses of social structure are important in the broader scale of evolutionary and ecological process, further encouraging the use of network analysis across a vast range of topics [Kurvers et al., 2014]. Here, understanding the role of networks in disease transmission has important implications for predicting disease spread from the perspectives of conservation and management, but also for understanding the evolution of social relationships in primates and other animals, and the trade-offs that may arise through group living.

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Social Information Transmission in Animals: Lessons from Studies of Diffusion

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The capacity to use information provided by others to guide behavior is a widespread phenomenon in animal societies. A standard paradigm to test if and/or how animals use and transfer social information is through social diffusion experiments, by which researchers observe how information spreads within a group, sometimes by seeding new behavior in the population. In this article, we review the context, methodology and products of such social diffusion experiments. Our major focus is the transmission of information from an individual (or group thereof) to another, and the factors that can enhance or, more interestingly, inhibit it. We therefore also discuss reasons why social transmission sometimes does not occur despite being expected to. We span a full range of mechanisms and processes, from the nature of social information itself and the cognitive abilities of various species, to the idea of social competency and the constraints imposed by the social networks in which animals are embedded. We ultimately aim at a broad reflection on practical and theoretical issues arising when studying how social information spreads within animal groups.

Keywords: information, sociality, experimental design, social cognition, social network, social competency

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INTRODUCTION TO SOCIAL DIFFUSION THEORY AND EXPERIMENTS

Many organisms, from plants to social animals, have the capacity to use information provided by others to guide their own behavior or decision (Morand-Ferron et al., 2010). Such information, the behavior of others or its product, constitutes social information. It can be advertently (a signal) or inadvertently (a cue) produced and may complement personal information acquired through trial and error and direct interactions with the environment (Bonnie and Earley, 2007). The use of social information is thought to allow individuals to adapt to their environment faster and/or better than through collecting personal information alone. Use of social information thus provides tremendous evolutionary advantages and is known to occur in many contexts, e.g., regarding food location, availability and palatability, predator threats, and finding and choosing mates (Danchin et al., 2004; Laland, 2004; Dall et al., 2005; Kendal et al., 2005; Bonnie and Earley, 2007; Taborsky and Oliveira, 2012). Even when the information or behavior appears non-adaptive, such as many of the behavioral traditions observed in non-human primates [e.g., hand-clasp grooming (McGrew and Tutin, 1978) or stone-handling (Leca et al., 2012)], such traditions may still be adaptive by preserving group cohesion or reinforcing group membership/identity through conformity for

example. In any case, the transmission of such traditions can be under the same social influences as that concerning more obviously adaptive social information. In this review, our main focus is on the transmission pathways of information between one individual (or group thereof) and another, regardless of its ultimate function/adaptive value. However, it must be kept in mind that low adaptive value may in itself partly explain a lack of diffusion of a given behavior, tradition or piece of information, and conversely that high adaptive value may facilitate and even enhance the diffusion process.

Within animal societies, an individual's ability to use social information and the properties governing its diffusion among group members or conspecifics have been studied under diverse frameworks, from evolutionary psychology (culture, social learning, and communication) and behavioral ecology (public information, eavesdropping) to neuroethology and economics of decision-making (information processing, social influences; Danchin et al., 2004; Dall et al., 2005; Kendal et al., 2005; Bonnie and Earley, 2007; Taborsky and Oliveira, 2012). The common threads binding all of these studies are twofold: (1) the source of information is the behavior of others and (2) the outcome of interest is the change in behavior associated with the acquisition and use of social information (Bonnie and Earley, 2007). Social information is thus a type of biological information, i.e., a property of some source that elicits a change in the state of the receiver in a (usually) functional manner. Differences between fields rest in the information content (who, what, and how) and packaging (signal vs. cue), as well as in the payoffs of using social information (Bonnie and Earley, 2007). For example, an animal's choice of a feeding site can be influenced by whether or not conspecifics are already feeding there (social influence or social learning), by the conspecifics' feeding behaviors that may be indicative of resource quality (public information), by how many other animals one can outcompete around the resource (eavesdropping), or by all of the above.

The acquisition and use of social information seems to be inherently adaptive, although some theoretical and empirical examples show that it could also be neutral (e.g., symbolic/arbitrary) and sometimes maladaptive (Rogers, 1988; Giraldeau et al., 2002). A maladaptive decision might also be defined as an inevitable by-products of an adaptive strategy that has evolved under strong selective pressures (Rieucau and Giraldeau, 2011; Pelé and Sueur, 2013). This probably relates to the existence of a trade-off between acquiring costly but accurate information through personal experience and using cheap but potentially less reliable information from others (Barnard and Sibly, 1981; Giraldeau et al., 2002; Laland, 2004; Kendal et al., 2005). Animals must thus adjust the weight they give to both sources of information depending on circumstance. Individuals may rely on social information when personal information is difficult to acquire or unreliable, and when they are uncertain about how to behave. They may instead rely on personally acquired information when the available social information conflicts with it or is incomplete, and/or when individuals are confident in the quality of their own information (Giraldeau et al., 2002; Laland, 2004; Kendal et al., 2005; Rieucau and Giraldeau, 2011). Most likely, decisions involve taking into account a combination of social and personal information and the diffusion of information is thus a function of the cost-benefit ratio of the different strategies available (Rieucau and Giraldeau, 2011). Yellow-bellied marmot (*Marmota flaviventris*) alarm calls, which are given to signal the presence of a predator, provide an opportunity to exemplify this because the caller's reliability in signaling danger is directly linked to the amount of time others allocate to personally assessing the threat: when the caller is judged unreliable, other marmots spend more time being vigilant (i.e., gathering personal information) before acting (or not) upon the threat (Blumstein et al., 2004). In species establishing recurrent and/or enduring social relationships between group members, reliability of social information also concerns these social relationships. For example, a middle-ranked female rhesus macaque (*Macaca mulatta*) will be more assertive toward an unfamiliar individual if she has seen a familiar subordinate individual defeating it in some competitive interaction (reliable social information), in contrast to conditions in which the interaction involved a familiar dominant or an unfamiliar individual (unreliable social information; cue reliability approach, Dewar, 2003).

Ways of testing functional and mechanistic hypotheses about social information and its use include: observing animals throughout their ontogeny, observing different populations of the same species with different behavioral traditions, or carrying out so-called social diffusion experiments in the lab or in the field. Social diffusion experiments investigate the transmission of social information from one individual (or group) to the next, seeding experimentally controlled innovations in behavior into groups of naïve individuals and tracking and documenting the spread (or otherwise) of the innovation (Whiten and Mesoudi, 2008; Whiten et al., 2016). A traditional experimental paradigm is to have two groups of subjects, an experimental group with a knowledgeable, proficient model that others can observe performing an action, and a control group without such an opportunity to observe. Alternatively, one of several new behaviors is seeded in one or few so-called informed individuals in a group of naïve individuals in order to artificially create behavioral variation amongst groups or populations. The aim is then to track the progressive acquisition of the new behavior in terms of pathways (from whom to whom the behavior is transmitted), speed, accuracy, and characteristics of individuals involved as compared to controls or variants (Whiten and Mesoudi, 2008; Whiten et al., 2016).

In this article, we first review such social diffusion studies and their goals, methods and outputs. We take a broad perspective on such studies, whether observational or experimental, with paired individuals or open groups, in a social learning or public information framework, but try to focus on salient research fitting our aims. We make no attempt to discuss what does or what does not constitute social learning (for comprehensive discussions of this see Galef and Laland, 2005; Hoppitt and Laland, 2008, 2013; Leadbeater, 2015, amongst others), nor to distinguish the mechanisms by which this particular use of social information occurs (see Laland, 2004; Hoppitt and Laland, 2013, amongst others), nor to debate whether the use of social information is adaptive (see Rogers, 1988; Giraldeau et al., 2002; Kendal et al.,

2005, amongst others). Hereafter, we instead focus exclusively on the possible pathways for information transmission within groups or aggregations of individuals, and the factors that may enhance or, more interestingly for us, inhibit information transmission. We pay special attention to studies in which the goals and outputs did not necessarily coincide because these studies tell us as much as do studies presenting "positive" results about how animals use, or do not use, social information. In the second part of this article, we return to essential concepts and expand our review on the nature of social information itself, the putative cognitive abilities of various species, the idea of social competency, and the influence of social networks on the use of social information in animal societies (**Table 1**). To paraphrase Bonnie and Earley (2007), our intention here is not to revolutionize the field, but rather to continue stimulating discussions about the abilities of animals to extract, use, and produce information from the social environment, and their influence on information diffusion.

SOCIAL DIFFUSION EXPERIMENTS: GOALS, METHODS, AND OUTPUTS

One of the earliest known accounts of social transmission of behavior is milk bottle opening among tits (*Parus major*, *Periparus ater*, and *Cyanistes caeruleus*) in England, where birds learned to pierce the lid of milk bottles left on doorsteps to drink the cream within (Fisher and Hinde, 1949; Aplin et al., 2013). Although this innovative behavior started in several places independently, once present in a population it would spread extensively, suggesting the influence of social processes (Fisher and Hinde, 1949; Lefebvre, 1995; Aplin et al., 2013). Another known example of social transmission among animals comes from Japanese macaques (*Macaca fuscata*) washing sweet potatoes in water, a behavior that spread gradually through the group (Kawai, 1965). In the years following the start of this seminal study, several other newly acquired behaviors (e.g., begging, stone-handling) emerged and spread through different groups of macaques in different regions of Japan following rules of acquisition dependent mainly on age, sex, and kinship (Kawai, 1965; Huffman et al., 2008). Since then, almost all published experimental or natural studies of social information transmission show that given the possibility to observe knowledgeable individuals performing a task, the majority of naïve, non-knowledgeable individuals subsequently use the same technique to accomplish the same task (Morand-Ferron et al., 2010). The non-random process of task acquisition is generally demonstrated if it occurs either above chance or above the proportion of naïve individuals performing the same task in a control group without knowledgeable demonstrators or in a group seeded with a different technique (Whiten and Mesoudi, 2008; Whiten et al., 2016). These results seem to be taxon-independent and pertain to insects, birds and mammals, demonstrating the overwhelming generality of social information use by animals (Laland, 2004; Chittka and Leadbeater, 2005; Galef and Laland, 2005; Whiten and Mesoudi, 2008; Rieucau and Giraldeau, 2011; Whiten et al., 2016). We can nevertheless distinguish these studies into three, non-exclusive categories: (1) those relating to the presence/absence of diffusion of the behavior; (2) those regarding individual characteristics and their influence on transmission; and (3) those interested in the pathways and characteristics of diffusion (e.g., persistence of transmission). Complementary to the ideas presented here, Whiten and Mesoudi (2008) and then Whiten et al. (2016) also provide extensive and updated reviews of diffusion studies in animals and humans.

Presence/Absence of Diffusion

A first step in studies of social diffusion is to show that information is actually transferred amongst animals in some way. The literature is vast and spans contexts such as foraging, breeding, anti-predation strategies, and social interactions. Examples range from bumblebees (*Bombus impatiens*) choosing the same-colored flowers as those chosen by conspecifics they previously observed (e.g., Leadbeater and Chittka, 2005; Worden and Papaj, 2005), to client fish (*Scolopsis bilineatus*) spending more time near cooperative cleaner fish (*Labroides dimidiatus*) than cleaner fish of unknown cooperative level after observing other clients' interactions with these cleaner fish (e.g., Bshary and Grutter, 2006), to flycatchers (*Ficedula albicollis*) using others' breeding outcomes (offspring quantity and/or quality) to select a breeding habitat (e.g., Doligez et al., 2002).

The interest here lies in where transmission apparently did not occur, because looking at how, why, and in what context animals do not use social information is just as telling as when they do. For instance, wild keas (*Nestor notabilis*), a mountain parrot, failed to solve a foraging task despite having the opportunity to observe proficient individuals solving the same task and to engage with the experimental setup immediately thereafter (Gajdon et al., 2004). When the experiment was repeated with captive keas, a majority of the birds solved the task after observing a proficient model (Huber et al., 2001; Gajdon et al., 2004). This indicates that the absence of social information transmission was independent of the task's level of difficulty. It could be that wild keas have the capacity to learn socially but some constraints prevent them to express it – maybe a question of opportunity or utility. This is similar to what is found in spotted hyenas (*Crocuta crocuta*), a social carnivore, where individuals in captivity seem more proficient at solving foraging tasks than those in the wild. This difference was attributed to personality rather than more trivial factors such as time-energy threshold, inasmuch as captive hyenas are more exploratory and less neophobic than their wild counterparts (Benson-Amram et al., 2013). In contrast, a novel foraging behavior (piercing a lid to access food) spread more quickly amongst groups of free ranging urban pigeons (*Columba livia*) than amongst captive groups. This was explained by the fact that urban pigeon groups are open to migrants which could enhance the degree of innovation and diffusion (Lefebvre, 1986).

Looking in more details at the hyena example, whether in captivity or in the wild, individuals presented with a box containing meat were more likely to approach and manipulate the box when they had seen others do it but were not more

TABLE 1 | Summary of points examined in this review.

Transmission process	Known influential factors	Directions for further studies
Initiation	– Producer characteristics (sex, age, dominance rank, and personality, motivation), – Environment (complexity, stability), – Type of innovation	– Competing solutions to the same problem – Suboptimal demonstrator characteristics – Seeding of information to individuals with different characteristics simultaneously
Pathway	– Producer/receiver characteristics – Producer/receiver relationships (kinship, dominance difference, "friendship") – Cognitive abilities (sensory output and processing) – Social network (openness, connectedness, tolerance) – Adaptive value – Information characteristics	As above, and: – Several information of varied types (e.g., social/asocial), qualities, relevance, or congruence presented at the same time – Social structure disturbance/manipulation (e.g., alone/in a social setting) – Same type of experiments to many different species/groups (including interspecies) – Different task complexity/difficulty concurrently
Establishment/ termination	– Cost/benefit ratio, – Conservatism level – Social network structure	– Comparison between initial transmission and long-term transmission patterns

Additional aspects:

– Technological equipment to track non-invasively: individuals' movements (GPS, accelerometer), physical states (heart rate monitor, blood glucose or glucocorticoid level monitor, infrared imaging), social proximities [radio-frequency identification (RFID) tags]
– Test apparatus version 2.0 with touch screens or panels, automated feeders, eye-trackers, face recognition
– Long-term population studies
– Heritability/evolution/environmental changes studies
– Taking inspiration in other diffusion domains such as epidemiology, informatics, or social media
– Building a database of protocols, pre-print, and published studies

likely to succeed in opening it (Benson-Amram et al., 2014). In this case, social information is used indirectly to enhance extraction of personal information but not directly to solve an environmental problem. This could be explained by the simplicity of the task (solvable by trial and error), or the characteristics of the demonstrator (not relevant or reliable). It could also be that social constraints, such as a rather competitive environment, affects the cost/benefit ratio of social information vs. personal information: hyenas are very good at solving goal-oriented cooperative tasks (Drea and Carter, 2009), which may be necessary to hunt large prey, but when they already have access to food, they may instead pay more attention to avoiding aggression than to new ways of obtaining the food *per se*. A lack of diffusion and establishment of a behavioral pattern can also occur when two alternatives are equally profitable. In meerkats (*Suricata suricatta*), individuals were at first more likely to feed on the same feeder as a demonstrator, but the more they explored the experimental apparatus, the more they realized they could easily get food at two "locations," making it less likely they would continue to use the demonstrator's feeder more frequently (Thornton and Malapert, 2009). In this example, although there was social transmission from one demonstrator to one observer, there was no establishment of behavioral tradition such that the behavior spread within the whole group according to individual's assortativity.

In other cases, the task presented seems too difficult, not appropriate or not ecologically relevant for the tested animals. For instance, laboratory-reared rhesus monkeys learned to fear snakes from watching videos of wild-reared conspecifics' reactions to snakes, but never learned to fear a flower on the same basis (Cook and Mineka, 1989). In a two-step foraging task, vervet monkeys (*Cercocebus aethiops*) had to remove a rope blocking a door before opening that door to retrieve food. Although the trained model was ultimately successful at the task, other individuals failed to master it although they were exposed to a successful model, suggesting that the link between one gesture and the next in a several-steps task was not evident (van de Waal and Bshary, 2011). Another example of a behavior, this time naturally occurring, that failed to spread is dental flossing in Japanese macaques (Leca et al., 2010). In their study, the authors reported several factors likely to constrain the diffusion of innovation such as belonging to a small grooming cluster relative to group size or having few close kin in the group, and the form, function and context of the behavior. The most interesting point that the authors made here is that the low adaptive value of dental flossing, a "comfort" innovation with such a "narrow window of applicability," may also account for its lack of diffusion (Leca et al., 2010).

Influence of Individual Characteristics on Diffusion

Because social groups are often mixed groups of individuals of different sexes, ages, and/or personalities, individuals' interest in, and experience and knowledge of, their environments vary. Thus, some individuals are potentially more likely to discover resources in the environment, to start innovating, or to correctly assess dangers than others, creating a differentiation in the availability and reliability of the social information produced within a group/aggregation of animals. On the other hand, some individuals are also more likely to learn from their conspecifics

because they are more social (in general terms), i.e., they are more often in proximity to others, they pay more attention to others, or they are more often engaged in social activities.

For instance, only 54% of naïve blue tits exposed to a proficient demonstrator solved a new foraging task (Aplin et al., 2013). Investigation of the variables that could explain this percentage showed that young females and subordinate males with higher innovative problem-solving capabilities were more likely to solve the task than others, whereas the characteristics of the demonstrator had no influence on the performance of naïve birds, i.e., there was no preferential attention to certain models (Aplin et al., 2013). On the other hand, studies on vervet monkeys demonstrated that social transmission is often influenced by kin relationships, i.e., vertical, from mother to offspring (van de Waal et al., 2014). When transmission is horizontal, from peer to peer, or oblique, from adults other than parents, vervet monkeys are more likely to copy the new foraging technique of an adult female compared to an adult/subadult male (van de Waal et al., 2010). Adult females of this species are philopatric and live their entire lives in the group in which they were born. This potentially makes them more reputable concerning food acquisition and processing because they have more experience and are the more familiar individuals in the group. They could also occupy more central positions in the social network of the group and may be more tolerant of individuals in proximity, all of which could potentially enhance social information transmission.

Similarly, it has been experimentally shown that, visually, monkeys do attend more to higher-ranking individuals than to lower-ranking individuals (e.g., McNelis and Boatright-Horowitz, 1998; Deaner et al., 2005), and to strong affiliates compared to average affiliates (Bonnie and de Waal, 2006; Micheletta et al., 2012). This pattern is interpreted as being more salient in terms of acquiring social information. As another case in point, the oldest living female in a group of African elephants (*Loxodonta africana*), the matriarch, often leads the group from one place to another and initiates group defense behavior (for example when encountering signs of unfamiliar individuals or of predators), potentially because she has enhanced local knowledge of the environment and group members defer the decision of travel/action to this informed individual (McComb et al., 2001, 2011; Mutinda et al., 2011). However, the best innovators, i.e., individuals more likely to start using a novel behavior, are not necessarily the best models for information transmission. For example, although male canaries (*Serinus canaria*) were better at solving a foraging task and thus could have been selected as demonstrators, their aggressive tendencies toward others prevented them from being good models (Cadieu et al., 2010). In this case, females constituted the best demonstrators because they tolerated individuals around them, so social transmission of an innovation mainly rested on females.

Diffusion Pathways

When social information is transmitted, determining the pathways taken by this information within a group of individuals as well as how fast and far it travels can give insights into the mechanisms of social information use. Indeed, animals living in groups or aggregations do not interact or associate randomly with one another, but have preferred associates or affiliates which are reflected in the heterogeneous structure of the social network of the group/aggregation. As such, the flow of social information is not random between individuals, but is in accordance with the structure of the social network of the population (Krause and Ruxton, 2002; Krause et al., 2007; Croft et al., 2008). Social transmission of information can thus fail not only because of some characteristics of demonstrators and/or naïve individuals, but also because the link between knowledgeable and naïve individuals may be suboptimal, e.g., the pair is not often together, not strongly affiliated or even avoids association, whatever the underlying causes. "Where" [i.e., with which individual(s)] to seed the social information diffusion within a network of individuals is thus as crucial as how connected the individuals are.

In brown capuchins (now *Sapajus apella*) for example, transmission during diffusion chain experiments was controlled in that pairs of demonstrators-observers were chosen amongst affiliates and the demonstrator was the higher-ranking of the two, which may have facilitated transmission (Dindo et al., 2008). In contrast, in a group of squirrel monkeys (*Saimiri sciureus*), where the chosen demonstrator of a new foraging technique was the alpha male, the open diffusion experiment demonstrated that more central individuals in the social network (those well connected and integrated in the group) were more successful at mastering the technique and quicker at using it than less central individuals (Claidière et al., 2013). Central individuals indeed may have more opportunity to observe the demonstrator and/or to manipulate the apparatus, especially if the demonstrator is itself central, which would enhance the use of social information. In a more natural setting, Brown (1986) showed that cliff swallows (*Hirundo pyrrhonota*) that were unsuccessful at bringing food back to the nest for nestlings were more likely to follow a successful individual on their next foraging trip than were successful foragers. Unsuccessful foragers were also more likely to follow their nest neighbors on subsequent trips, especially those within 1 to 5 nests away than further away in the colony. As there was intra-individual variation in foraging success, any bird could be a successful or unsuccessful forager and thus a follower or a leader to a foraging patch. This led Brown to coin the swallow colonies as "information centers" and is one of the earliest examples of diffusion analysis in a foraging context, albeit in a crude way (Brown, 1986).

A major step forward in the study of social diffusion is the development of network-based diffusion analysis (NBDA). NBDA is a tool now commonly used to demonstrate that the expression of a behavior by an individual is the result of it being associated with animals that themselves express this behavior with an increased probability compared to a model not including social effects (Franz and Nunn, 2009; Hoppitt et al., 2010). The model specifically illustrates directed social learning, in which information is transmitted at different rates depending on association patterns between individuals (Coussi-Korbel and Fragaszy, 1995). Such social effects explain variance in lobtail feeding in whales (Allen et al., 2013) or food patch discovery in tits (Aplin et al., 2012). The latter study not only demonstrated that tits use social information to locate new

food patches but also that the discovery success was linked to individual centrality in the flock association network: more central individuals were more likely to locate and use novel foraging patches than those with limited social connections. By looking at an animal or human group as a network of connected individuals, social network analysis has facilitated great progress in diffusion studies, and as a result, in the understanding of animal and human culture. Because culture is fundamentally based on the exchange of social information, social structure and culture are indeed linked (Cantor and Whitehead, 2013). In this perspective, diffusion studies, whether experimental or observational, coupled with social-network-based analysis brought substantial advances to our understanding of how animals use social information.

FURTHER PERSPECTIVES ON SOCIAL DIFFUSION STUDIES

Questions regarding the acquisition and use of social information are typically concerned with when to copy (e.g., when resources are easy or difficult to exploit/find, or when the environment is stable or unstable), who to copy (e.g., successful or reputable or familiar or genetically related individuals), what is copied (i.e., what kind of information is remembered and transmitted) and how individuals copy (i.e., the mechanisms or supports by which the information is reproduced; Laland, 2004; Bonnie and Earley, 2007; Whiten and Mesoudi, 2008; Whiten et al., 2016). The literature covering each of these aspects is vast and continues to expand almost exponentially (Galef, 2012; Whiten et al., 2016). The challenge that remains even today is to examine those questions in more integrative ways and to find the right experimental, empirical, and statistical paradigm to do so (Whiten et al., 2016).

Important aspects of diffusion that we feel deserve more attention include social information characteristics, what makes an animal a producer and/or a user of information, the cognitive capacities involved in acquiring, processing, and using social information, and finally the social competency of animals. We also think that future work could pay more attention to quantifying the rate at which information spreads, how far this information can spread in a network, and the factors that influence the flow of information. This means that an additional focus to factors favoring social transmission could be on those explaining an absence thereof. We now turn to these topics in a humble attempt to participate in advancing the field of social information use in animal societies.

Social Information Characteristics

The characteristics of social cues, i.e., information that is inadvertently produced through interaction with the environment, can greatly influence their transmission inasmuch as acquiring and using social information is directly related to the cost of acquiring and using asocial or personal information (Boyd and Richerson, 1988). These characteristics can be experimentally modified to assess which are important to the animals. For example, is the number of conspecifics performing a task sufficient, or are subtler cues necessary to decide to use social information? For instance, experiments of social transmission in fruit flies (*Drosophila megalonaster*) showed that within an aggregation, the number of informed individuals needed to be about twice the number of uninformed individuals in order to observe transmission of information from informed to uninformed individuals (Battesti et al., 2015). Experiments with fish and birds demonstrate that individuals without *a priori* information on environmental resources are more likely to follow a large group of conspecifics to a food location compared to a small group. But as soon as individuals can observe others actually feeding, they would rather follow few individuals feeding than many individuals not feeding (Kendal, 2004; Coolen et al., 2005; Rieucau and Giraldeau, 2011). This suggests that observing a direct link between a task and a reward is more salient than just observing a task. Similarly, individuals with *a priori* personal (or asocial) information are less influenced by their companions' behavior than those without. In an experiment with nutmeg mannikins (*Lonchura punctulata*), individuals without prior personal information consistently chose the feeder associated with previously acquired social information regardless of whether it was the mere numbers of companions present or the numbers of companions feeding. Individuals with prior personal information, however, did stick to their initial choice and switch feeders only if they observed companions actually feeding (Rieucau and Giraldeau, 2009). More subtly, homing pigeons were shown to adjust their flight routes, to which they generally show high fidelity, depending on those followed by conspecifics (Biro et al., 2006). When the pre-established routes of two pigeons did not differ greatly, a pair would converge on an average path, supporting the "many-wrong" hypothesis arising from a compromise between personal and social information. However, as soon as the routes diverged beyond a distance threshold, one individual became the leader, usually the pigeon most faithful to its own pre-established route, supporting the leadership hypothesis in which the most insistent, "confident," or less flexible individual imposes a social choice on the group. In other cases, both pigeons defaulted to their established routes and thus no use of social information was observed, again usually when the routes diverged beyond a distance threshold (Biro et al., 2006; Freeman et al., 2011).

Another characteristic of information that is likely to influence its transmission is complexity or difficulty. A one-step task may thus be acquired and spread faster between individuals than a task requiring four steps to be completed. For example, callitrichid monkeys used social information to solve a challenging foraging task involving pulling a door toward oneself and retrieving food inside a box, whereas they solved an easier foraging task involving pushing a door and reaching inside to retrieve food without using social information (Kendal et al., 2009). Similarly, vervet monkeys easily solved a simple foraging task such as pushing/pulling a door (van de Waal et al., 2013), but failed to solve a two-action foraging task, even when being provided with social information (van de Waal and Bshary, 2011). Information complexity or stability can also emerge from the environment. For example, the structure of the environment (open vs. closed, arboreal vs. terrestrial) can influence how communication

signals can be perceived (Maciej et al., 2011). Starlings (*Sturnus vulgaris*) in an unpredictable environment are better at foraging when in the presence of an informative demonstrator (who consistently indicated the same food location) than in the presence of an uninformative demonstrator, whereas individuals in a predictable environment performed equally well with or without an informative demonstrator (Rafacz and Templeton, 2003). The extent to which the complexity or stability of the environment affects the transmission speed, accuracy, and reach of social information is still not very clear, however. Ecological and social environments may very well interact to affect social information transmission inasmuch as an individual's perception and action are tightly linked to both (e.g., Barrett, 2011).

Some types of information are also more salient or relevant than others, which will influence their social transmission. For example, humans recall and repeat social information such as gossip involving third-parties with greater accuracy and in greater quantity than non-social information such as the geographical description of a city (e.g., Bartlett, 1932; Mesoudi et al., 2006). In animals, several studies hint that individuals would probably also pay more attention to information relating to social events as opposed to non-social events. For example, fish choose to take a long circuitous route with their mates rather than a shorter more direct route alone to access food. This preference persists over several generations even when founder demonstrators have disappeared from the population (Laland and Williams, 1998). Similarly, in a two-choice test paradigm where male rhesus macaques had to choose between receiving a fruit juice reward or receiving a fruit juice reward and seeing an image of a conspecific, they not only chose the latter option but sacrificed a bit of the amount of juice they could have received to do so (Deaner et al., 2005). This choice demonstrated that monkeys were ready to sacrifice a food reward to gather social information.

Another characteristic we briefly mentioned before concerns the adaptive value of a given piece of social information. If social information that is obviously adaptive, e.g., use of a tool to extract food among primates and corvids, versus that which is not-so-obviously adaptive, e.g., stone-handling among Japanese macaques, were to be seeded in the same group or aggregation, would the spread, speed and reach of diffusion of the former be more important than the latter? The relevance of the former compared to the latter would intuitively lead us to predict a positive relationship between adaptive value and these diffusion properties. However, if these not-so-obviously adaptive socially transmitted behaviors play a role in increasing group cohesion through conformity for example, the answer may not be so straightforward.

So, in general, although animals can display great interest in an experimental apparatus or a given situation, perform a task or a behavior to perfection, and readily observe and copy others, we still know too little about the nature of social information and its influence on transmission dynamics to predict when these behavioral aspects will coincide and result in diffusion. Is it about quality, quantity, complexity, congruence, relevance, or a mixture of all of these traits? Determining this requires long and patient trial-and-error tests, massive undertakings of experiments encompassing varied conditions, contexts, and characteristics, mathematical models and efforts in complex systems science and, importantly, although the information can sometimes be extracted from the study itself, a systematic report or test of the kind of information that is tested/used. Experiments combining tests of asocial and social information simultaneously are also important in determining characteristics of diffusion as it is likely that animals use a combination of both at every instant (Rieucau and Giraldeau, 2011).

Animals as Information Processors and Users

Each step of the transmission process requires individuals to "innovate" on a personal level, that is, they are not necessarily the first to express the behavior but this is the first time that they themselves express it. In this sense, understanding limits to innovation helps understanding constrains on social diffusion (Brosnan and Hopper, 2014). One of these limits is within the animals themselves, related either to individual characteristics – explored in this section – or to cognitive abilities – explored in the next section (for limits concerning the social environment, see "The social competency of animals or the social network effect").

Characteristics of the information producers, such as relative status, age, or sex, cannot only influence the performance of an individual in its environment but can also condition another animal's decision to observe such producers and to use the information gathered. Similarly, characteristics of the information receiver determine its processing and use of information and, as such, the speed, accuracy and extent of information transmission. Individual constraints on social diffusion (here, of innovations) stem from the propensity of individuals to be conservative, that is, individuals tend to persist with existing behaviors, or the existing uses of behaviors, rather than explore novel options (Brosnan and Hopper, 2014). As a case in point, bolder and less neophobic individuals are more likely to produce information and to innovate than shy and neophobic individuals because they tend to take more risks and explore their environments more (Wilson, 1998). Lower-ranking chimpanzees tend to be more innovative, probably because they are more constrained in their access to food and have to find an alternative solution more often than higher-ranking individuals (Reader and Laland, 2001). In great tits, variation in spontaneous problem-solving performance was unrelated to individual state (e.g., body condition) and not even associated with behavioral traits (e.g., neophobia), but most likely reflected inherent individual differences in the propensity to forage innovatively (Cole et al., 2011). In starlings, less neophobic and higher-ranking individuals were more likely to approach the experimental novel foraging tasks. Group mates of these first "contactors" approached the experimental apparatus more quickly as well if they themselves had a propensity to feed in a novel environment (Boogert et al., 2008).

Nevertheless, although some studies have determined which individuals tend to learn or innovate faster or better (see

references in previous paragraph), we are still at risk of making a lot of assumptions about who those individuals might be instead of testing who they actually are. When studying animals living in group, especially in natural conditions, researchers are indeed often constrained in the choice of knowledgeable demonstrator(s) vs. naïve observer(s), because high-ranking individuals monopolize the resources for example, or because bolder individuals are more explorative. It is also very difficult to disentangle which individual characteristics can have the most influence, as high-ranking individuals for instance can also be bolder than low-ranking individuals. Studies conducted with wild animals must keep these sociodemographic constraints in mind when being discussed or reported. Finding ecological validity in diffusion studies is a much needed challenge (Whiten et al., 2016).

Overall, what makes a producer and/or a user of information varies greatly according to ecological, social, and individual circumstances. What we need to be more aware of is that not all individuals will produce or use social information, in relative and absolute terms. Optimizing our knowledge and understanding of the speed, accuracy, and spread of social information transmission requires that the profiles of producers and users be more systematically reported. We also need studies that can select producers and users with suboptimal characteristics, for example a high-ranking individual with a lower-than-expected network centrality compared to a low-ranking individual with a higher-than-expected network centrality, or a lower-ranking individual with a higher-than-expected boldness profile compared to a high-ranking individual with a lower-than-expected boldness profile. For instance, in several groups of vervet monkeys tested in an experimentally induced coordination problem, dominant individuals naïve to a foraging task learnt to wait outside of an imaginary forbidden circle that the proficient but low-ranking individual approached and solved the task and allowed food access to the whole group (Fruteau et al., 2013). What is also needed is the assessment of the effects of individual characteristics on diffusion in naturally or spontaneously occurring innovations, observed from their birth to their establishment or disappearance, in a population where individuals are identifiable and their characteristics *a priori* known [e.g., dental flossing (Leca et al., 2010) and louse egg-removal techniques (Tanaka, 1998) in Japanese macaques, lobtail feeding in humpback whales (Weinrich et al., 1992; Allen et al., 2013), or moss-sponging in chimpanzees (Hobaiter et al., 2014)].

Cognitive Abilities

The social brain hypothesis states that increasing social complexity drives the evolution of large brains with more cognitive capacities, in the sense of information-processing, because of the challenges of managing complex social relationships (Whiten and Byrne, 1997; Dunbar, 1998; Pérez-Barbería et al., 2007). However, the use of social information is so widespread in the animal kingdom that one could contend that information-processing capabilities do not relate only to brain size (Barton, 2006; Morand-Ferron et al., 2010; Lihoreau et al., 2012). The fact that invertebrates such as wasps and bees are capable of memory and learning demonstrates how complex cognitive processes are possible even with a limited number of neurons (Lihoreau et al., 2012; Avarguès-Weber and Giurfa, 2013; Grüter and Leadbeater, 2014). Paper wasps (*Polistes fuscatus*) can recognize individuals and remember the identity of social partners, even after a succession of interactions with other individuals (Sheehan and Tibbetts, 2008). Honey bees (*Apis mellifera*) are well known for their symbolic "dance language," which they use to build consensus about relocating to a new home: the swarm integrates the different information given by different explorative scouts through their dancing and make a decision about a single location (Seeley, 2010). In the field of social learning, it has been argued that social learning does not depend on "advanced" cognitive adaptations, and that social and asocial learning alike depend on the same mechanisms (Heyes, 2012). This hypothesis is supported by the facts that social and asocial learning abilities covary across and within species (Bouchard et al., 2007; Reader et al., 2011), that social learning occurs also in solitary animals (Fiorito and Scotto, 1992; Wilkinson et al., 2010), and that social learning has the same key features in diverse species, including humans (Heyes, 1994, 2012). Heyes (1994, 2012) therefore argues that social and asocial learning depend on a common set of associative learning mechanisms and that social learning merely reflects the case in which the information is provided through a social channel (Heyes, 2012). This illustrates how the use of social information may in fact require relatively simple and computationally inexpensive forms of cognition (Lihoreau et al., 2012).

However, the use of social information also involves perceptual, attentional, and motivational processes specific to information coming from other individuals (Heyes, 2012). Acquiring and using social information requires animals to link other individuals' actions to environmental and/or social reactions or patterns. Feedback from the social domain also requires that individuals integrate and process stimuli not only related to the external (e.g., sex, size) but also to the internal (e.g., "emotional") states of other interacting agents, to the current social context, and to what this information means to the individual at that moment in time in order to respond with the appropriate behavior (Trimmer et al., 2008; Clutton-Brock, 2009; Taborsky and Oliveira, 2012). Throughout the evolutionary history of social species, these social-specific processes may have been selected for and may have further coevolved with the complexity of social life (Heyes, 2012; Leadbeater, 2015). For instance, Pinyon jays (*Gymnorhinus cyanocephalus*), a social corvid species, perform a social learning task better than an asocial learning task whereas Clark's nutcrackers (*Nucifraga columbiana*), a less social corvid, perform equally well in both tasks (Templeton et al., 1999). Based on these differences, social learning capabilities were interpreted as being adaptations to social life (Templeton et al., 1999; Heyes, 2012). This is essentially one of the tenants of the cultural intelligence hypothesis (Whiten and van Schaik, 2007; van Schaik and Burkart, 2011), which examines links between asocial and social learning and the development and maintenance of learned skills both horizontally and longitudinally in an effort to better

understand the emergence and maintenance of cultures and traditions.

From a neuroethological perspective, some parts of the brain are specifically dedicated to social stimuli, such as face recognition and processing, social approval (i.e., individuals tend to conform to social norms to "fit in"), selective social attention (e.g., individuals pay more attention to higher-ranking individuals), or recognizing and responding to socio-emotional signals such as fear and anger (Brothers, 1999; Insel and Fernald, 2004; Phelps and LeDoux, 2005; Barton, 2006; Adolphs, 2008; Rilling and Sanfey, 2011). Mirror neurons are specifically activated both when one performs an action such as reaching for food and when one observes someone else performing that same action (Gallese, 2007; Caggiano et al., 2009). In the broadest sense, emotions are "an evaluative response of the organism involving physiological arousal and expressive behavior," and "interfacing between sensory inputs and motor outputs in a way that allows flexibility in the response (to a stimulus)" (Aureli and Schino, 2004 for one definition amongst many). They function as adaptive responses to environmental demands, preparing individuals to cope with challenges (Aureli and Whiten, 2003; Aureli and Schino, 2004; Phelps and LeDoux, 2005; Naqvi et al., 2006; van den Bos et al., 2013). As shown in many (natural or induced) experiments of brain lesions/malfunctions in humans and animals (e.g., in the case of autism or brain damage due to an accident), individuals that are physiologically or neurologically stressed or impaired have difficulties making decisions in the social domain and may thus be poor users of social information, which would ultimately constrain social information diffusion without giving any indication about their cognitive abilities. For example, individuals with a damaged ventromedial prefrontal cortex have normal intellect and problem-solving abilities under test conditions in the lab, but make unfortunate decisions in real-life situations and do not learn from their mistakes. This is due to the fact that they have a generally "flat affect" and are thus unable to use emotions to aid in decision-making (Damasio, 1994; Naqvi et al., 2006).

From these perspectives, focusing social cognition research on sensory information, computational challenges, and neural networks, i.e., brain functioning, would be a rewarding way of looking at animal cognitive abilities in the social domain (Chittka and Niven, 2009; Barrett, 2011; Lihoreau et al., 2012). Designing experiments and observations where animals' motivational, emotional and perceptual capabilities concerning their social worlds are accounted for could give important insights into how social information is transferred within a group.

The Social Competency of Animals or the Social Network Effect

Ingenious mathematical models and experimental designs show that efficient transfer of information and decision-making can occur within animal groups in the absence of individual recognition, advanced cognitive abilities or complex mechanisms of transfer, and that individuals can respond spontaneously to others that possess information. All that is needed is variation in information holding among members of a population and simple mechanisms of coordination (e.g., Couzin et al., 2005).

However, these kinds of simple decision rules are more likely to be present in societies where individuals do not form differentiated relationships with each other. When group members have the opportunities to recognize each other and memorize past interactions that influence future ones, they do form differentiated relationships that can condition and influence their decision-making processes (Sueur, 2011; Lee and Harris, 2013; Pasquaretta et al., 2014). The heterogeneous distribution of social connections within a group also creates heterogeneous opportunities to observe and learn from certain individuals (as in directed social learning, Coussi-Korbel and Fragaszy, 1995). As such, the structure of the social network of a group can have important consequences for the social transmission of information (Coussi-Korbel and Fragaszy, 1995; Croft et al., 2008; Aplin et al., 2013; Cantor and Whitehead, 2013). For example, observer deer mice (*Peromyscus maniculatus*) have stronger reactions of preparatory analgesia and self-burying in reaction to biting flies when the observer is genetically related to or is more familiar with the demonstrator, although the demonstrator's behavior does not vary with social conditions (Kavaliers et al., 2005). High-ranking rhesus macaques solve a color-discrimination problem equally well when in a whole group or only amongst high-ranking individuals, whereas low-ranking individuals perform better when with other low-ranking individuals only than when with the whole group (Drea and Wallen, 1999). In a cooperation task, spotted hyenas adjust their behavior to the skills and capabilities of their partners (for example, when an adult is paired with a youngster) and their level of cooperation is modulated by the composition of their social group inasmuch as an individual's performance is better predicted by the presence of high-ranking individuals – which can be quite aggressive – than by the subject's prior experience in the task to solve (Drea and Carter, 2009). An entire field of research in animal communication is dedicated to these moderating effects of social context, so-called "audience effects," i.e., individuals adjust their decisions or behaviors depending on who is with or around them (Zuberbühler, 2008). Conformity, i.e., doing what the majority does, is a very influential mechanism by which culture emerges, evolves and persists (Laland, 2004; Morgan and Laland, 2012). Reaching a consensus decision, on where to go for example, is also a well-studied example of social modulation of behavior (Conradt and Roper, 2009).

Social network analysis (SNA) has proven a useful and powerful tool in understanding social influences on the patterns of acquisition and use of social information (Croft et al., 2008; Voelkl and Noë, 2010; Kurvers et al., 2014; Brent, 2015). A simulation study based on a substantial dataset of primate interaction matrices tested the hypothesis that the social structure of a group has a strong influence on patterns of social learning (Coussi-Korbel and Fragaszy, 1995) by comparing information flow within networks in empirical (structured) social groups and theoretical well-mixed groups in terms of propagation speed, path length of transmission and resilience against information loss (Voelkl and Noë, 2010). This study showed that information spreads faster in well-mixed groups compared to structured

groups. In structured social networks, information also spreads faster when the frequency of interactions was either disregarded (unweighted or topological networks) or distributed randomly amongst interacting individuals. Similarly, the number of transmission events (path length) from an innovator individual to a target individual was greater in structured groups compared to well-mixed groups and was related to reduced connectivity and variation in interaction frequencies. Furthermore, variance in average path length was related to variation in group size, the larger the group the longer the path length, but also to community modularity, a measure that quantifies the structuring of a group into subgroups (Voelkl and Noë, 2010). Actually, there is more and more evidence that the structure of a social group, rather than its absolute size, influences network flow (e.g., pathogens or diseases: Griffin and Nunn, 2012; Nunn et al., 2015; emotions, tastes, or health outcomes: Fowler and Christakis, 2008; Bakshy et al., 2012; Christakis and Fowler, 2014). At a more global level, this is illustrated by the differences found in cooperative performance, enhanced in socially tolerant bonobos compared to more aggressive chimpanzees (Hare et al., 2007), or in socially tolerant Tonkean macaques compared to non-tolerant rhesus macaques (Petit et al., 1992). Those differences have been attributed to the fact that social networks of tolerant species are more diverse and open because individuals tolerate each other's proximity better and this potentially offers a greater opportunity for information diffusion.

In humans, mathematical modeling has shown that social influences can lead to disproportionate diffusion of a trend or a fashion, an effect called the majority illusion (Lerman et al., 2015). In a network setting, behaviors can be contagious and spread to an entire population from a small subset of initial individuals. The speed and spread of this contagion has been shown to be heavily dependent on the network structure: a trend or a disease is transmitted faster if the initial adopters are very well connected and/or belong to very well connected clusters, e.g., superspreaders (Fujie and Odagaki, 2007; Garcia-Herranz et al., 2014). Because individuals take their social cues from their local neighbors, the characteristics and positions in the network of these initial adopters can greatly influence the contagion of a behavior, making it appear far more common locally than it is globally (Christakis and Fowler, 2014; Garcia-Herranz et al., 2014; Lerman et al., 2015). This has been termed the majority illusion and stems from the friendship paradox in which one's friends appear to have more friends than one has (it also concerns tweets and academic citations for instance). The mathematical model developed by Lerman et al. (2015) quantifies the strength of this phenomenon and shows that it is stronger in networks with active high-degree nodes (active knowledgeable individuals) and heterogeneous degree distribution (because active knowledgeable individuals are more attractive and others in the population, non-active non-knowledgeable, pay more attention to them). Similarly, in health programs dedicated to educate people about hygiene and safer practices, targeting friends of individuals – themselves chosen randomly – in the population can have greater effects on the spread of behavioral changes than targeting individuals with the most social ties (Kim et al., 2015). This effect is attributed to the specific structuring of human social networks, which show subgroups of interconnected individuals each with their own locally influential nodes (Newman and Park, 2003; Fowler and Christakis, 2008; Kim et al., 2015). It also suggests that the assumption of greater centrality linked to greater influence on social processes is not always straightforward as this relationship can be mediated by sub-structuring, individual role or position, and synergies between indirect and direct connections. In fruit flies, social network structure [for example, homogeneous (individuals behave similarly) vs. heterogeneous] also affects information use, specifically in oviposition site choice: uninformed flies would either follow or avoid choices of informed flies depending on the amount of variance in individual network centrality among informed group mates, the greater the variance the more uninformed individuals avoided the same site as informed individuals (Pasquaretta et al., 2016). Social network modeling can thus improve the underpinning social variance and the understanding of why some behaviors spread – or on the contrary do not spread.

A factor that is often overlooked is that, although social life is extremely beneficial, it can also be stressful because individuals not only have to satisfy their own needs but also must do so while coordinating with the needs of others (Krause and Ruxton, 2002). Whether test subjects are in their social group settings or tested singly can have tremendous effects on their stress level and cause concomitant effects on decision-making in the laboratory or under natural conditions (van den Bos et al., 2013). As such, on the one hand experimental studies done in isolation of the social context may have little predictive value in terms of social information use in general, although they allow for the dissection of mechanisms and functions quite difficult to achieve in natural settings. On the other hand, the social group context can be very inhibiting for some individuals and thus can impede social information diffusion, such as potential or actual conflicts with conspecifics, or the fact that performing a task in front of conspecifics can be overwhelming (van den Bos et al., 2013). Stress affects memory and learning (Schwabe et al., 2012) and biases decisions (Aureli and Schino, 2004; Naqvi et al., 2006; Starcke and Brand, 2012). For example, individual ravens (*Corvus corax*) approach a novel object faster but spend less time interacting with it when alone than when in pairs or groups, seemingly trading off vigilance against innovation depending on risk and opportunity assessment (Stöwe et al., 2006). Brown rats (*Rattus norvegicus*) experiencing stress significantly and progressively lose the ability to adjust their responses toward a larger reward when transitioning from equal to unequal reward quantities (Graham et al., 2009). The effect of stressors on decision-making may not be of great consequence in animal social diffusion studies apart from failed experiments, but in humans, having to make a decision under high stress is linked to variation and volatility which likely reflects uncontrollability and unpredictability and can lead people or groups to make irrational choices (Starcke and Brand, 2012).

A final aspect of the influence of sociality on social information use is the social competence of animals. Social competence refers to the ability of individuals to regulate the expression of their social behavior in order to optimize their social relationships

(Taborsky and Oliveira, 2012; Bshary and Oliveira, 2015). For instance, it allows individuals to avoid engaging in overly costly fights ("winner-loser" effect; Hsu et al., 2006; Taborsky and Oliveira, 2012) and to increase or decrease their degree of aggressiveness according to the familiarity of their opponents (familiar = "dear enemy" effect, stranger = "nasty neighbor" effect; Temeles, 1994; Taborsky and Oliveira, 2012). Social competence can also explain why individuals tend to cooperate more readily with social partners if they themselves have received help from others previously ("generalized reciprocity"; Pfeiffer et al., 2005; Taborsky and Oliveira, 2012). Although established from an evolutionary ecology point of view, with reference to phenotypic behavioral flexibility and plasticity, the vantage point of social competence provides an overview of the general ability and performance of individuals in a social environment (Taborsky and Oliveira, 2012). Recently, the social competence perspective has been paired with a game theoretic approach in animal cooperation with exactly this goal in mind. This more integrative framework also highlights the importance of studying the behavior and underlying decision rules/strategies of individuals across different social contexts, in the same way that behavioral syndromes encompass links and feedbacks of individual reaction norms across a variety of contexts (Bshary and Oliveira, 2015). Social diffusion studies would benefit enormously from taking such an integrative approach and accounting simultaneously for variation in the individual, social, and physical worlds.

SMART ANIMALS

Animals produce and receive, acquire and use social information from different individuals in different contexts and circumstances. The circumstances under which an animal uses social information rather than selects an option based on its own environmental sampling or the different rules animals adopt when making such decisions have been investigated in great details. Social diffusion experiments of all kinds are great tools to investigate the social insights of animals. Nevertheless, many important questions remain: how do animals distinguish informed and uninformed individuals? How do they judge the quality of a piece of information? What if several individuals are deemed knowledgeable but the information they provide conflict? What if the context in which social information is produced changes its value compared to another context? What if certain pieces of information are easier/less risky to get, but are also less accurate? To what extent the spread, reach and speed of transmission of a social information are affected by these parameters? Answering these questions, from our point of view, will require a more integrative approach, marrying different fields to reflect more realistically the probable holistic understanding animals have of their environments (Laland, 2004; Taborsky and Oliveira, 2012; Bshary and Oliveira, 2015).

On a practical side, with the accumulation of studies of diffusion, building a database of successful and failed experiments could better inform the scientific community. This could take the form of depositing protocols into an open-access database, such as the Dryad Digital Repository[1], with the advantage of having corresponding digital object identifiers (doi), or creating a dedicated website on which to aggregate studies, pre-prints, and protocols in the same fashion as the Global Mammal Parasite Database[2], with the advantage that it is searchable and collaborative. With the technology available today providing small cost-effective electronic devices [touch-screens, eye-trackers, automated feeders, accelerometers, radio-frequency identification (RFID) technology, GPS, etc.], broad-scale experiments and modeling could be possible as is now done regularly in cognitive science (Fagot and Bonté, 2010), ecology and social network studies (Rutz et al., 2012; Krause et al., 2013; Farine and Whitehead, 2015). One could setup providing automated food boxes with automatic food delivery devices and remote-controlled openings triggered by the approach of an animal equipped with RFID tags. Providing dozens of such boxes in a group setting would allow varying the quality, quantity, and reliability of the information available to group members both as producers and receivers. Tracking natural demographic changes or experimentally inducing changes by removing/adding individuals or manipulating the quality of a social bond could also give insights into the causes and consequences of social network structure on social information transmission.

This kind of diffusion experiments, with broad yet individualized parameters, could help tackle integrated questions related to variation and complexity of the environment, be it social or ecological. As has already been proposed for studies in cognitive science (see e.g., Barrett, 2011 and Wilson and Golonka, 2013 for an overview), social diffusion studies would also benefit from being more "embodied," i.e., investigating social information use within individual, social and environmental contexts. Furthermore, studies on social information transmission could get inspiration from other domains such as epidemiology, informatics security, or social media, especially in humans, where studies also account for and integrate social network processes in empirical and mathematical studies, thereby providing tremendously important insights into biological and social processes. Finally, most of the experimental examples are situated in foraging, mating and anti-predator contexts, but far less has been done in social contexts such as aggression or affiliation. We know that animals are socially aware in the sense that they recognize their group mates or conspecifics, that they can keep track of their relationships and that they can use social concepts such as dominance and triadic relations (Whiten and Byrne, 1997; Dunbar, 1998; Emery, 2004; Holekamp et al., 2007; Silk, 2007). We have evidence that animals can recognize facial expression in conspecifics (Micheletta et al., 2015), that emotional arousal can spread through a group (collective arousal or emotion contagion, e.g., De Marco et al., 2011) and that animals can also judge and use the social reputation of others in their decisions (Alexander, 1987; Bshary and Bronstein, 2010). How animals make use of these kinds of social information to guide their decisions in their social relationships is an open field of investigation where

[1]http://datadryad.org/pages/organization

[2]http://gmpd.nunn-lab.org/

social diffusion experiments can find their place. Better or further accounting for characteristics of information, of individuals, of cognitive and social competences is essential in making progress in the social information field and in the understanding of how animals make use – or not - of social information.

AUTHOR CONTRIBUTIONS

All authors made substantial contributions to the conception and design of the work; participated in the acquisition, analysis, or interpretation of data for the work; participated in drafting the work or revising it critically for important intellectual content; gave their final approval of the version to be published; and agree to be accountable for all aspects of the work.

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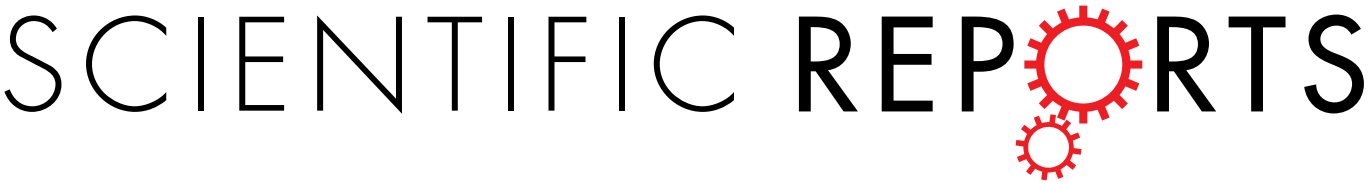

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Network centrality and seasonality interact to predict lice load in a social primate

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Lice are socially-transmitted ectoparasites. Transmission depends upon their host's degree of contact with conspecifics. While grooming facilitates ectoparasite transmission via body contact, it also constrains their spread through parasite removal. We investigated relations between parasite burden and sociality in female Japanese macaques following two opposing predictions: i) central females in contact/grooming networks harbour more lice, related to their numerous contacts; ii) central females harbour fewer lice, related to receiving more grooming. We estimated lice load non-invasively using the conspicuous louse egg-picking behaviour performed by macaques during grooming. We tested for covariation in several centrality measures and lice load, controlling for season, female reproductive state and dominance rank. Results show that the interaction between degree centrality (number of partners) and seasonality predicted lice load: females interacting with more partners had fewer lice than those interacting with fewer partners in winter and summer, whereas there was no relationship between lice load and centrality in spring and fall. This is counter to the prediction that increased contact leads to greater louse burden but fits the prediction that social grooming limits louse burden. Interactions between environmental seasonality and both parasite and host biology appeared to mediate the role of social processes in louse burden.

Many parasites, i.e. organisms that live and feed exclusively within or on other living organisms (hosts), are socially-transmitted, either directly through contact between individuals or indirectly through spatial overlap and resource sharing[1]. Risk of infection in social individuals can therefore depend partly upon the nature of their social interactions, making the risk of disease and pathogen transmission one of the major costs of sociality[2]. Highly social hosts are expected to encounter a more abundant and diverse parasite community than less social hosts, and thus to exert stronger influence on the transmission of parasites through their social networks[1]. Thus the heterogeneity and diversity of contacts between hosts must be considered when tracking parasite/disease transmission and attempting to understand infection risk[3,4].

From this perspective, social network analysis (SNA) provides a useful tool that captures such heterogeneity by taking into account direct and indirect connections (edges) between individuals (nodes), allowing for multilevel analyses from individuals to populations[3]. For example, the number of connections an individual has (network degree) and their combined 'weight' (network strength) can be used to assess an individual's risk of direct exposure to pathogens/parasites from social conspecifics, while other indices such as eigenvector centrality extend estimation of exposure risk to include the neighbourhood of an individual's neighbours[3,5]. The use of SNA in epidemiological studies has also highlighted that social transmission of pathogens can be dynamic because host sociality itself is intrinsically dynamic[3] and related to various factors which must also be taken into account such as environmental seasonality, age, sex, reproductive state, energetic needs and/or preferential attachment related to dominance rank, kinship, or friendship[2,6].

One common type of network relevant to social animals derives from social grooming (allo-body-care/allo-grooming/allo-preening, hereafter grooming). Grooming is often highlighted as a mechanism of establishing and maintaining group cohesion and social bonds[7,8], and at least in non-human primates, its frequency has been linked to kinship, dominance rank and access to resources[9,10]. In many social animals, grooming is also studied

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with respect to its direct and indirect health benefits: in addition to reducing physiological stress and releasing endorphins ("relaxing" hormones)[11–13], social grooming retains the original (hygienic) function of self-grooming which evolved to remove dirt and other debris, ectoparasites and/or dead skin. Evidence for this hygienic function is widespread in animal societies[14–16]. It is further supported by the preference of groomers to groom body parts likely infested with ectoparasites because they are not easily accessible to the individuals themselves (e.g. head, shoulders)[16–18], and by the finding that preventing grooming through physical incapacitation or social isolation leads to sharp increases in ectoparasite loads[19,20].

One common ectoparasite of mammals is the louse, which feeds on blood and requires a host during each stage of its life cycle. Adult lice typically lay one to several eggs (nitts) every few days, which are glued to the base of a hair. Like many organisms, lice respond to environmental conditions (e.g. temperature, humidity). In ungulates, for instance, adult louse populations peak in spring and drop in summer[20,21]. Lice are also susceptible to their host's physical condition (e.g. hormonal changes), which they perceive through blood meals (e.g.)[21–23]. Thus, louse reproduction can be triggered or hindered by that of their hosts[20]. Lice are further affected by changes in the pelage of their hosts–their habitat–such as those induced by moulting (sequential hair replacement) or shearing (e.g. in domestic sheep)[23]. All of these are relevant to the extent to which lice affect their hosts. Direct effects of infestation include activation of skin allergic reactions such as dermatitis or pruritis[20], which have pronounced effects on an animal's body condition, e.g. hair/feather quality[20], and ultimately its fitness[20]. Indirect effects include their potential to act as vectors or intermediate hosts of many pathogens (e.g., *Rickettsia prowazekii*, *Bartonella quintana*, and *B. recurrentis*[24]; *Rickettsia*, *Anaplasma*, and *Bartonella* spp. in various animals[20,25,26], including primates[27]).

Lice and their mammalian hosts are thus a good host-parasite system to study the likelihood or risk of disease/parasite transmission within host social networks[28]. Body contact, including grooming, provides an opportunity for these parasites to transfer from one host to the next. The host contact network structure is therefore paramount in patterns of louse transmission and even population viability, since lice are largely host specific, have a direct life cycle, and may not survive more than a few hours away from their host[20]. At the same time, much of the body contact observed in many social animals occurs in the context of social grooming, which may simultaneously constrain the spread of such organisms through parasite removal. An individual's lice load can thus be amplified or constrained by its social contact network, illustrating one of the trade-offs between costs and benefits of being social. Yet, studies investigating the links between network centrality and louse infestation and/or louse-mediated disease are still too few, and these trade-offs thus generally remain poorly understood.

In this study, we investigated the risk of louse infestation within Japanese macaque social networks. The Japanese macaque (*Macaca fuscata fuscata*) is a social primate species living in multi-female multi-male groups, where individuals form linear dominance hierarchies and differentiated affiliative social relationships[29]. Japanese macaques harbour two species of louse (*Pedicinis obtusus* and *P. eurygaster*). Based on quantification of egg and nymphal/adult stages of lice detected on culled macaques, it was estimated that an average-sized macaque could harbour up to 550 louse eggs[17]. This was approximated to represent 230 nymph/adult lice[17], with usually good correlation between these life stages (at least in domestic sheep)[21]. Thus, the number of louse eggs seems a good estimation of the host louse population[17]. Furthermore, video data analysis showed that 98.9% of what groomers conspicuously pick out and consume from the hair/skin of groomees consists of louse eggs[30]. Body parts estimated to have many louse eggs and associated with higher frequencies of conspicuous louse egg-picking gestures are groomed longer than other body parts[17,31]. This louse egg-picking behaviour can thus provide an ideal estimator of lice load among individuals observed under naturalistic conditions. We therefore used the number of louse egg-picking gestures during grooming, controlling for total number of observation records of grooming received, as a proxy for lice load (see Methods).

Specifically, we first assessed the extent of variation in centrality measures and lice load according to ecological and social contexts because host-parasite interactions can be mediated by such contexts[32]. Japanese macaques live in an environment with four distinct seasons and are strict seasonal breeders, with mating seasons occurring between fall and winter and birth seasons occurring between spring and summer (with strong regional variation)[29]. As such, host energetic demands and physiology as well as social contact and proximity networks also change seasonally and seasonal changes in host reproductive activity can induce variation in host immune defence[32] and social tendencies[33–35]. Individuals furthermore moult seasonally, hair being longest/densest in winter and shortest/sparsest in summer[36], which may strongly affect louse reproduction and survival but also louse detection during grooming. Host susceptibility to infection can also be related to an individual's general physical condition[37]. Higher-ranking females in the dominance hierarchy are generally fitter than lower-ranking ones[38–41], and they may thus better resist infection[35,42,43]. Such individual, social and environmental variables, along with the synergies among them, often correlate with variation in parasitism which in turn may influence transmission dynamics and infection risks[32]. As such we predicted that environmental seasonality, host reproductive status and dominance rank would affect host centrality, lice load and their interactions in a way that females, particularly low-ranking ones, may be less social and more prone to lice infestation during reproductive seasons (i.e. winter and summer), periods where lice population should also be either at its maximum or at its minimum due to local conditions (i.e. physiological and hair status of the host).

Then, we tested the relationship between centrality and lice load, accounting for the potential confounding factors presented above (also see Methods). Because body contact provides a transmission opportunity for lice, and thus an infection risk for hosts, yet grooming may constrain louse density through parasite removal, we made two opposing predictions: compared to less central females, 1/ more central females will be most infested with lice due to their higher diversity of interacting partners or their higher frequency of body contact in the network; or 2/ more central females will be least infested with lice because they have their louse eggs removed through grooming by a higher diversity of partners and/or more frequently. Evidence for a positive relationship between centrality and lice load would indicate that despite increasing exposure risk to potentially deleterious parasites, being

central still presents advantages. Conversely, a negative relationship would speak in favour of social grooming as an efficient antiparasite strategy that can be exploited by females through their grooming networks. To test prediction 1, we investigated the relationship between estimated lice load and centrality measures based on undirected weighted networks of body contacts, including grooming. To test prediction 2, we looked at whether lice load was related to in-centrality measures derived from directed weighted networks of grooming received. Testing different centrality measures allowed us to investigate whether it is the actual number (or diversity) of partners and/or the actual amount of social contact a female has that influences lice load. First, grooming skills and thus louse egg-picking efficiency significantly vary across individuals[44]. As such, being groomed for long duration by an individual poorly skilled at removing louse eggs can have less influence on lice load than being groomed for short durations by several individuals with varying degree of efficiency at removing louse eggs. Furthermore, female Japanese macaque society is based on strict hierarchical social rules determining who can interact with whom[45]. These social constraints can not only affect the number of partners as much as the frequency of social interactions of an individual, but also the areas of the body individuals have access to: lower-ranking females indeed tend to avoid eye contact when grooming higher-ranking ones, and thus avoid the head and frontal body parts (in bonobos)[46]. Given that lice are unevenly distributed on the body[17], this may also constrain louse egg removal and having diverse grooming partners may thus be as advantageous as being groomed for long durations.

Results

Grooming represented 27% (median, range = 14–39%, N = 20) of all activity scans of female Japanese macaques of Koshima during this study. Body contact without grooming represented only 9% (median, range = 1–20%, N = 20) of all scans with body contact. Females were in contact with other adult females in 48% of all scans with body contact (median, range = 4–88%, N = 20), with males in 6% (median, range = 0–61%, N = 20 females and 9 males) and sub-adults, juveniles and infants in 41% (median, range = 1–96%, N = 20 females and 23–31 non-adults). Total louse egg-picking events averaged 129 events per female over the whole study period (median, range: 36–320, N = 20), which represented less than one event per grooming minute-scan (median = 0.77, range = 0.34–2.23, N = 20).

Variation in centrality and lice load according to seasonal and individual factors. The modularity Q of social networks, representing the extent to which a network is partitioned in smaller units, shifted between seasons especially before and after summer where it was highest (Fig. 1). Randomisation tests showed that only the centrality measure degree in the contact network was significantly affected by seasonal and/or individual factors when compared to null models that randomised the network data. Degree in the contact network showed significant variation across seasons (Supplementary Table S2; Figs 1 and 2): it was significantly lower in summer and fall compared to winter and spring (Supplementary Table S2; Fig. 2), meaning that females had significantly more female social partners during the latter than the former seasons.

Lice load also varied across seasons, being higher in summer and fall compared to spring and winter (Supplementary Table S2; Fig. 2). Lice load changed marginally according to the females' reproductive state, being slightly higher in reproductively active females than others (Supplementary Table S2).

Testing prediction 1: increased parasitism with increased centrality in contact networks. Overall, only one of the centrality measures from contact networks was related to lice load and only through an interaction with season (Supplementary Tables S3 & S4, Figs 3–5): females with higher degree had a lower lice load than those with lower degree, but only in winter and summer, whereas there was no relationship between degree and lice load in spring and fall. Strength did not predict lice load (Supplementary Tables S3 & S4, Figs 3–5). Thus, in winter (mating season) and summer (birth season), females in contact with more female social partners had lower parasite burden compared to females in contact with fewer partners. In spring and fall, however, females showed similar parasite burden regardless of their centrality. The observed effect of degree centrality was significantly more pronounced than the same effect from a set of models that randomised the network data ($p\ \beta_{obs} < \beta_{rand} = 0.043$, Supplementary Table S4). Prediction 1 was thus not fulfilled.

Testing prediction 2: decreased parasitism with increased centrality in grooming received network. Models with grooming received centrality measures showed a tendency towards lice load being related to in-degree through an interaction with season and reproductive state separately (Supplementary Table S3 & S4, Figs 3–5): females with higher degree in the grooming received network tended to have lower lice load than those with lower degree in winter and summer whereas there appeared to be no relationship between degree and lice load in spring and fall (Supplementary Table S3 & S4, Figs 3–5). This negative effect of centrality on lice load was marginally greater in reproductively active compared to inactive females (Supplementary Table S3 & S4, Figs 3–5). This means that in winter and summer (the mating and birth seasons respectively, i.e. when some females were reproductively active), females receiving grooming from more female social partners had lower parasite burden compared to females receiving grooming from fewer partners. In spring and fall, however, females showed similar parasite burden regardless of their centrality. However, the observed effect of in-degree centrality was not quite more pronounced than the same effect from a set of models that randomised the network data ($p\ \beta_{obs} < \beta_{rand}\ p = 0.087$, Supplementary Table S4). Prediction 2 was thus partially fulfilled.

Discussion

In animal groups, increased centrality in social networks is often linked to increased parasite load and disease risk[3]. In female Japanese macaques of Koshima, centrality in terms of number of connections in contact and grooming received networks was negatively associated with lice load, as measured by louse egg-picking gestures performed during grooming. However, the relationship between degree and lice load was mediated by season in that females with fewer contact or grooming partners presented higher lice burden only during winter (mating

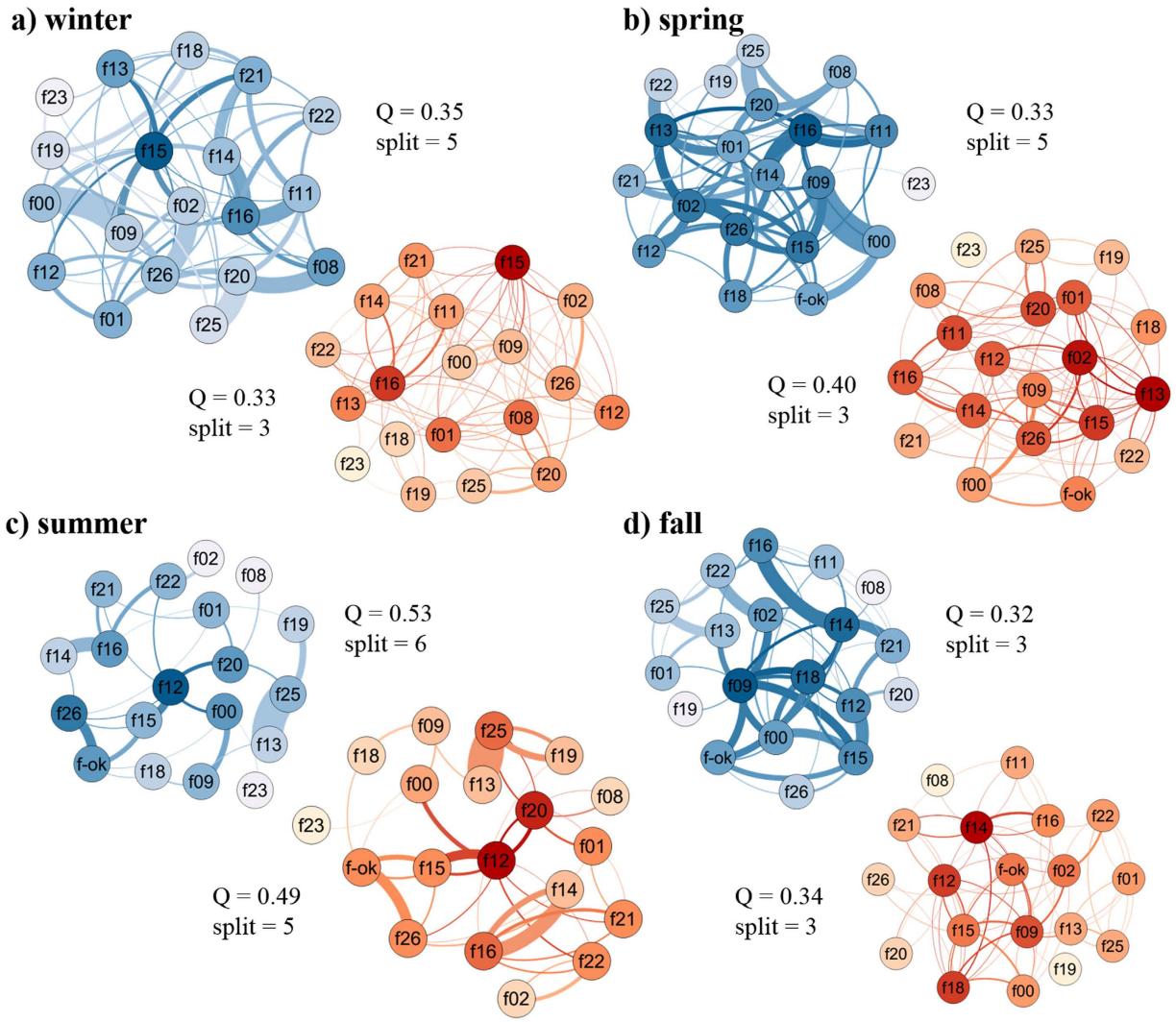

Figure 1. Seasonal variation in contact and grooming networks. Sociograms of weighted contact (in blue) and grooming received (in orange) networks across the four seasons. Node colour shades represent variation in number of connections (degree), the darker the higher, and edge thickness represents the strength of the connection between two nodes, the thicker the stronger. A bidirectional relationship between two nodes is indicated by two edges clockwise. On the side of each network is given the modularity Q as well as the number of communities (or splits) found according to eigenvector centrality (Newman 2006).

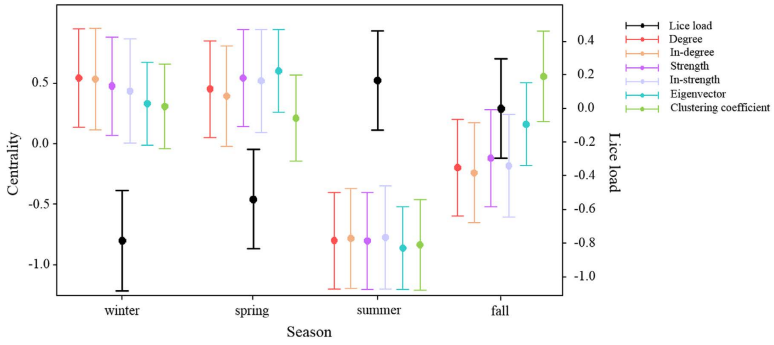

Figure 2. Variation in centrality indices and lice load across seasons. Coefficient plots of GLMMs testing the influence of seasonal and individual factors on centrality measures and lice load (here without the influence of centrality). Circle: coefficient value, bold line: one standard error. Levels of categorical predictors between parentheses indicate those not included in the intercept (the reference level is winter for season, not reproductively active for reproductive state).

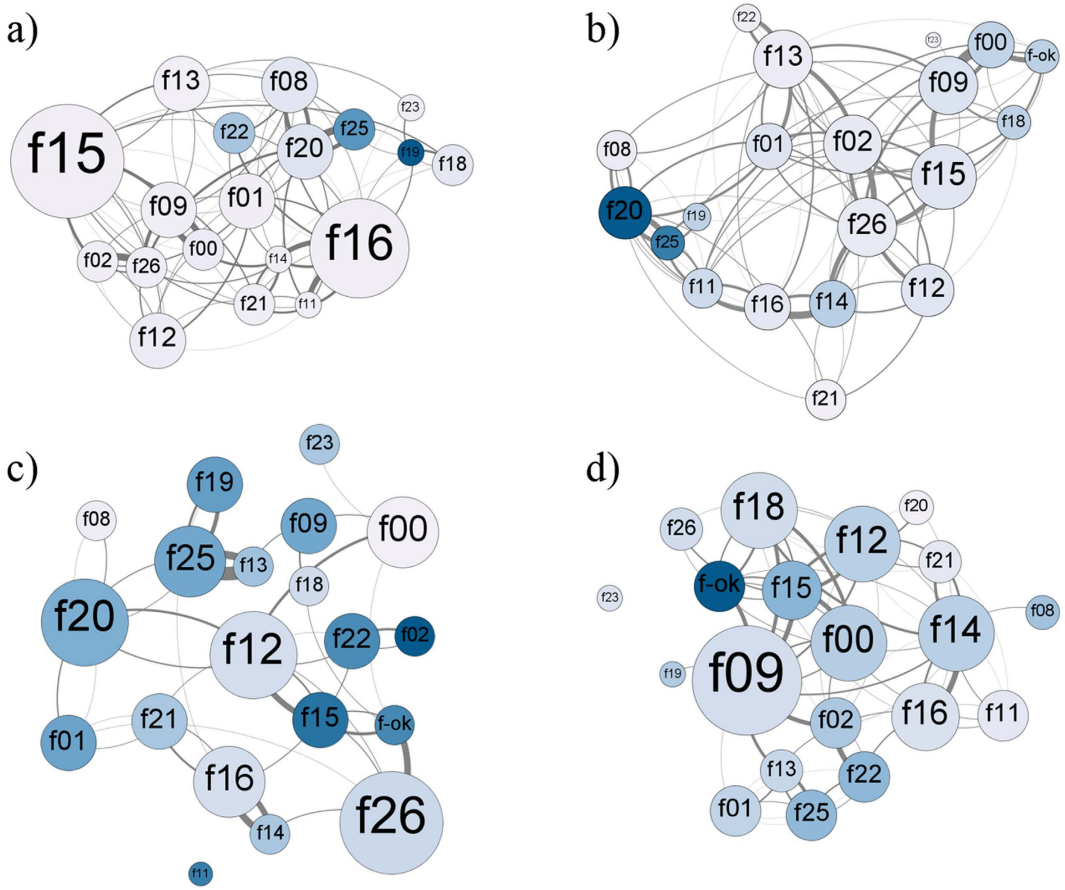

Figure 3. Interaction between centrality in grooming received network and seasonality in predicting lice load. Sociograms of weighted grooming received networks of adult female Japanese macaques of Koshima, divided according to season: (**a**) winter (N = 19, f-ok not followed), (**b**) spring (N = 20), (**c**) summer (N = 19, f11 no contact with others), and (**d**) fall (N = 19, f23 no contact with others). Variation in node colour represents variation in lice load per grooming unit, the darker the node, the higher the lice load. Variation in node size represents variation in degree, the bigger the node, the higher the degree. Variation in edge size represents the strength of interactions, the thicker the edge the more frequent grooming received between two nodes. Edge colour matches the target node, i.e. the node receiving grooming. A bidirectional relationship between two nodes is indicated by two edges clockwise.

season) and summer (birth season). This was concurrent with a tendency towards the negative effect of degree centrality in grooming received network on lice load to be greater in reproductively active compared to inactive females. These findings did not provide support to the prediction of increased parasitism with increased contact centrality, but were in accordance with other studies showing that grooming received can be a predictor of lower ectoparasite burden[15,19,20,47].

It must be stated up front that our measure of lice load is indirect, observational and dependent upon observing individuals grooming. Capturing monkeys and marking/collecting lice provides a direct estimate of lice loads and allows testing whether lice actually carry pathogens, but this approach is inconvenient and often impossible to implement in wild populations. That said, Japanese macaques are very conspicuous when picking items from the hair or on the skin of their grooming partners, and 98.9% of what is picked and consumed has been demonstrated to be louse eggs[30]. Furthermore, body parts estimated to have many louse eggs are groomed longer with more frequent louse egg-picking gestures[17,30]. Thus, despite the indirect nature of this measure, we believe it to be a fair estimate of lice load, particularly when coupled with the demonstration that the amount of eggs is approximately double that of adult lice[17]. Such conspicuous hygiene-related behaviours thus provide useful information with which to investigate risks of infestation with ectoparasites and/or disease spread in wild animals, at least in those species in which such behaviours are readily observed.

The study of this louse egg-picking behaviour led researchers to discover that macaques groom for demonstrably longer durations if they find many louse eggs to pick and eat (so called "grooming-related feeding"), irrespective of the relationship between groomer and groomee[31]. Studies on birds have also shown that allopreening of self-inaccessible body parts occurred regardless of dominance relationships, which was not true for self-accessible body parts[15]. These studies suggest that some grooming bouts or parts thereof may be less dependent upon social preferences than once thought, and instead linked to other ecological functions (e.g. hygiene, feeding) of social

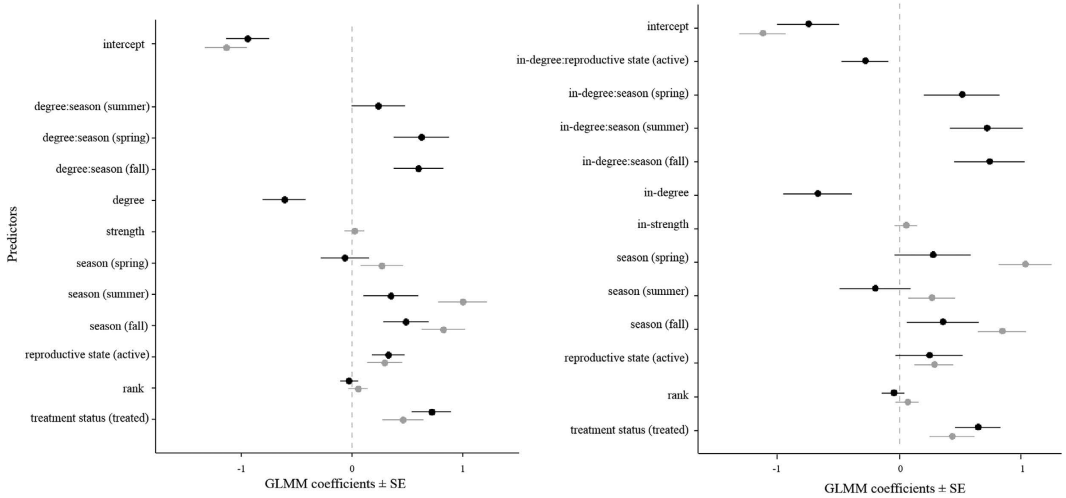

Figure 4. Coefficient plots of GLMMs. Testing prediction 1: left panel; Testing prediction 2: right panel. Circle: coefficient value, bold line: one standard error. Two variables separated by a column denotes an interaction. Levels of categorical predictors between parentheses indicate those not included in the intercept (the reference level is winter for season, not reproductively active for reproductive state, and non-treated for treatment). Black = degree (left) or in-degree (right). Grey = strength (left) or in-strength (right).

grooming[15,31]. This is remarkable because social interactions between group members are often influenced by similarity within dyads; kin, individuals of adjacent dominance ranks, or age-mates tend to interact more frequently with each other than with others. Such assortativity may ultimately reflect on the centrality of individuals within their network, and arises independently of the distribution of lice in the group. Yet, our study, like that of earlier works[17,30,31], hints at the possibility that grooming-related feeding may now prove influential in determining even partially the grooming network structure, at least in macaque societies, a link that should be investigated further.

One ecological factor that clearly affects numerous behavioural outcomes is seasonality. We considered seasonality of the environment as well as host and parasite biology, as it is generally highly relevant to infectious disease dynamics[32]. Indeed, the primary result of this study hinges on an interaction between seasonality, network centrality and lice load: the relationship between parasitism and sociality can only be interpreted in light of seasonal variation. Winter and summer represent the mating and birth seasons in Koshima, respectively. Both seasons show changes in female contacts and/or proximity behaviour[33–35] in two opposite fashions: an increase as solitary/floater males temporarily join social groups to gain mating opportunities during the mating season[48], and new individuals are born in the birth season; or conversely, a decrease as during the mating season, consortship pairs seek to be alone and during the birth season, females focus a great part of their attention on their newborns, somewhat decreasing their involvement in other social interactions[34]. These two seasons, winter (mating) and summer (birth), also have long-lasting effects on the physiology of females (e.g. immunosuppression, energy costs). It is therefore not surprising to observe that the greatest contrast observed in the relationship between lice load and centrality (from negative to almost absent) exist between the two most physiologically (reproductively) demanding seasons and the other less demanding seasons. Similarly, louse transfers and thus infection risk increased during the mating season in both chipmunks (*Tamias striatus)* and mouse lemurs *(Microcebus rufus)*, presumably because of the concomitant increase in both contact between individuals and host reproductive activity[28,49]. Because lice feed on blood, it has been hypothesised that their own reproduction is influenced by their hosts' sexual hormones[21,23], in that some stages of the host reproductive activity can trigger the parasite own reproductive activity. This is the case for another blood-feeding ectoparasite, the rabbit flea (*Spilopsyllus cuniculi)*, in which the reproductive cycle is triggered by pregnancy and parturition of the host doe. The fleas then migrate *en masse* to the nest to breed on the immunologically-naïve young[50,51], illustrating the tight links between host and parasite biology and the effect of environmental seasonality on the synergy between host and parasite (see also next paragraph).

These synergies between host and parasite biology also explain the results of this study. Lice load was highest in summer during the periparturient period. Females thus seemed more vulnerable to louse infestation at this time. This was particularly the case for reproductively active females, especially those that were less central in the social networks. This pattern could be due to the combination of several factors, also linked to the effect of environmental seasonality on host and parasite biology. First, the surge of sex hormones in the host around birth could have triggered louse reproductive activity and proliferation[20–23]. Second, the immunosuppressive effects of these sex hormones during challenging times such as birth and lactation could have decreased the host's defence against infestation, and made them more susceptible to it[52]. Third, the presence of immune-naïve hosts (newborns) could have constituted a newly available and easy breeding ground for lice, which could have then transferred to other individuals, especially vertically to the mother and then on to the mothers' social partners. Fourth, moulting induces changes in the habitat of lice, and in Japanese macaques, begins at the onset of spring

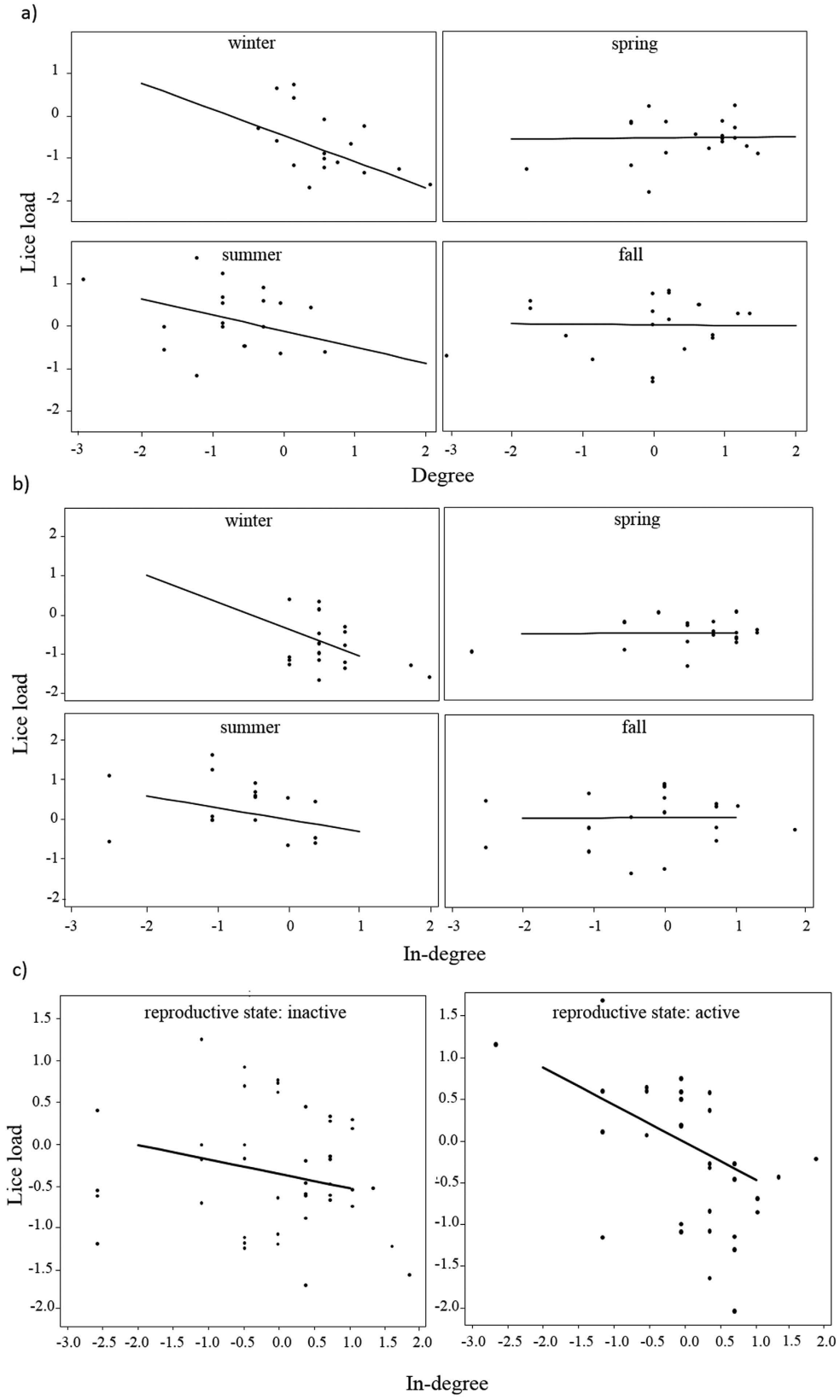

Figure 5. Effects of the interaction between number of connections in the network and seasonality or reproductive state on lice load. panel (**a**) shows degree (contact network) and season, panel (**b**) shows in-degree (grooming received network) and season, panel (**c**) shows in-degree (grooming received network) and reproductive state. The four seasons (winter, spring, summer, and fall) each have their own square, the two reproductive state (active, not active) as well. The line represents predictions from the model and dots the raw data transformed (i.e. log of the response (lice load) and square-root of the predictor (centrality)) and scaled.

and ends during summer. Hairs are therefore shortest in summer and continue to grow through autumn and winter when they are longest. These changes, in addition to natural variations in the louse microhabitat (like skin temperature or solar radiation higher in summer[21]), likely impact louse prevalence and intensity (*sensu* the effect of shearing on louse density in domestic sheep[21]). We also cannot rule out the possibility that moulting impacts the efficiency with which monkeys can find and extract louse eggs: beginning at the hair base, louse eggs would be pushed upward during regrowth and the shorter hair may increase their visibility. Increases in lice load in summer through fall may thus be linked to the synergistic effects of changing louse habitat, the interaction between lice and host reproduction, and changes in behavioural tendencies of hosts. Nevertheless, examining factors related to seasonal variation in lice loads, including pelage characteristics, is insufficient in itself to explain the observed negative relationship between centrality and lice load during reproductive seasons.

At Koshima, mean centrality indices generally seemed to vary in the opposite direction of mean lice load across seasons; the season in which mean centrality was lowest corresponded to the season in which mean lice load was highest. However, network modularity did follow somewhat closer to the pattern of mean lice load in that the season in which mean lice load was highest was also the season in which modularity was greatest. Greater modularity in a network means that the network decomposes into more modules than in a network with lower modularity. Greater modularity is hypothesized to negatively impact transmission of infectious agents throughout social groups by breaking down the chain of transmission[53]. In our study, periods of increased lice load seemed concomitant with periods of higher social network modularity, reflecting either a decrease in females' direct contacts and grooming exchanges or more focused exchanges within smaller cliques of individuals compared to periods with lower louse infestation. It seems likely, then, that decreases in grooming network centrality allow lice to multiply at both the individual and group levels, especially during periods of host immunological and physiological vulnerability (mating and birth seasons). Conversely, avoidance of social contacts is a well-known mechanism to limit the spread of socially-transmitted pathogens[53,54]. This requires that individuals can determine each other's infection status, which seems likely in many animals[55,56] but absent in others[57]. Of course, a third possibility is that changes in network structure and changes in lice load are concomitant but independent. While lice load may be environmentally determined, female hosts naturally increase social contacts to access mating opportunities and decrease them to care for their infants and minimise risks (of injury, disease), which may be stronger drivers of network changes compared to parasitism.

But if the network reflects pathways for parasite transmission, centrality indices such as degree and strength measuring risk of exposure are expected to be positively related to parasite infection patterns[3]. In our study, the negative relationship between degree and lice load during the winter and summer breeding seasons relative to spring and fall could suggest that individuals other than females have an impact on the lice burden or the likelihood of louse infestation during these periods: via vertical transmission between females and their immature offspring[21] or horizontal transmission from solitary/floater males[58]. Barring this third-party influence, individual variation in grooming/contact given and received could also influence lice load, inasmuch as the presence of a "super-groomer" for example could play the role of a super-spreader–a highly contagious individual or simply one with many social connections[59]. Alternatively, such a super-groomer could play the role of "super-delouser", as there seemed to be variation in louse egg-picking efficiency across individuals[44], or if there is variation in grooming site preferences across individuals[17]. Ultimately, amongst the centrality measures we tested, the only predictor of lice load was the number of direct connections females had with others in the social network. This implies that infection risk was more related to who is connected to whom in the network rather than how individuals are connected. Thus, as was also shown in meerkats (*Suricata suricatta*)[60], frequent social contact does not necessarily increase the risk of infection. From a parasite/pathogen perspective, one contact may be all it takes to change environment and continue reproducing on a new host. From a host perspective, having multiple social partners can either increase exposure to parasites through increased likelihood of interacting with a super-spreader, or increase parasite removal through increased likelihood of interacting with a super-delouser. These contradicting possibilities might also explain why observed relationships between centrality and lice load are not straightforward: grooming simultaneously offers lice a transmission opportunity and hosts a parasite removal opportunity. Our results are weighted toward the latter because less connected females generally exhibited higher louse burden, supporting the hypothesis that grooming is an effective antiparasite strategy to be exploited in a social context and providing further evidence for the benefits of being social despite the costs related to disease transmission and infection risk.

In this evolutionary arms race, parasites effectively use their hosts' behaviour to increase their own fitness[1], but hosts have evolved diverse social and ecological strategies of parasite avoidance and removal[61]. Due to the combined difficulties of accurately depicting animal contact networks from observation and of directly monitoring elusive parasite populations, experimental studies and/or alternative measures/estimations of both networks and parasites would help disentangling the synergistic effects of the environment and the interaction between host and parasite biology on transmission and infection risks from socially-transmitted pathogens[62,63]. For example, experimental reduction of parasite loads influences the frequency or patterns of host social interactions[64,65]. Conversely, manipulating host contact rates can also induce changes in infection risk[66].

Conclusion

In conclusion, our study shows that variation in contact and grooming network centrality in terms of number of connections explains variation in lice load in female Japanese macaques in that less central females have higher lice burden, providing further evidence of grooming as an efficacious anti-parasite strategy. However, our study also demonstrates that this relationship is dependent upon ecological and biological conditions, such as less central females have higher lice burden only during winter (cold–long, dense hair–mating season) and summer (hot–short, sparse hair–birth season). Our study is the first to estimate lice load from direct observation of wild animals and to link this estimate to the centrality status of individuals in their contact networks. This study also

control factor	type	control for (rationale)	interactions between control factors	contributions of interaction to model fit (LR tests**)
season	categorical: winter, spring, summer, fall	host: moulting, network changes, physiological changes[32–36,72,74] parasite: habitat change, fitness, population viability[21,23,32]	season* reproductive state* centrality	ns
reproductive state	categorical: active, not active	host: network changes, physiological changes[32–35] parasite: fitness, population viability[20,21]	season* reproductive state	ns
rank	continuous	host: physical condition, access to grooming partners[9,37,38,43] parasite: fitness, population viability[21,23,43]	repro* centrality	trend in model with in-degree as predictor
treatment*	categorical: treated, not treated	parasite: fitness[81]	season* centrality	significant in model with degree; trend in model with in-degree as predictors

Table 1. Summary of control factors included in GLMMs: type, rationale, interactions between them and whether or not interactions contributed significantly to model fit (ns = not significant). *Factor only included in models with lice load as response variable. **See Supplementary Table S2.

highlights the importance of seasonal variation both in parasite intensity and in host behaviour in explaining variance in infection risk and exposure from socially-transmitted external parasites.

Methods

Study site and subjects. We studied Japanese macaques on Koshima islet, Miyazaki prefecture, Japan (31°27′N, 131°22′E). The region has a warm-temperate climate with monthly mean temperatures ranging between 9.3 °C in January and 27.2 °C in August, and monthly mean precipitation between 35 mm in January (humidity 60%) and 730 mm in June (humidity 82%; data for 2014 from the Japan Meteorological Agency). Koshima is approximately 0.3 km^2 and covered by evergreen broadleaved forest[67]. Provisioning and behavioural observations of Koshima macaques started in 1952, and demographic, ecological, behavioural, and life-history data have been collected since then[68]. The main group of Koshima macaques is currently provisioned with ca. 3 kg of wheat ca. twice weekly. Koshima is now inhabited by approximately 100 individuals divided into two social groups, Maki (ca. 15 individuals) and Main Arctic monkeys (ca. 60 individuals), along with an unknown number of solitary males. Monkeys are individually recognisable by facial tattoos and natural physical characteristics (scars, body shape or hair colour).

We observed the 19–20 adult females (>7 years old) of the Main group. We focused on females because, in macaque societies, they form the core of the group and dominate dynamics of social networks, males are often few and not very social, and juveniles are difficult to recognise, often have their own subgroup, and usually engage in different age-typical activities than the adults[69–71]. Ten females were in oestrous during the mating season (winter); they were thus considered reproductively active during this period. Seven females gave birth between the end of June and the beginning of July (summer), and they were considered reproductively active in spring (as pregnant), summer and fall (as lactating). All other females were regarded as non-reproductive for this year. Additionally, as part of an on-going research project since 2012, half of the adult female cohort is orally administered an anthelminthic treatment several times a year (MacIntosh, unpublished data). During the observation period, treatment was administered to 11 females at the end of March, the middle of July and the middle of November (see Supplementary Table S1 and the Statistics part for more details).

Research adheres to the ASAB/ABS guidelines for the use of animals in research and was approved by the ethic committee of Kyoto University Primate Research Institute.

Data collection. Data were collected from January to November 2014 (total 142 days, 350 h of observation, 17h30 ± 1h15 per female, see Supplementary Table S1). Focal observations were balanced across females and time of day (morning/afternoon). To avoid the influence of artificial conditions on our data set, data other than dominance interactions were not collected during and up to an hour following provisioning. Focal females were followed for 15 min during which their main activities were recorded every minute, while their social, aggressive and other affiliative interactions as well as the identity of each social partner were recorded continuously. Amongst recorded activities, we distinguished between grooming given, grooming received, and simple body contact. We also collected data on dominance interactions (i.e. winner and loser of agonistic interactions) during focal observations and *ad libitum*.

During social and self-grooming bouts, we counted the number of times per minute-scan the groomer conspicuously picked out something in the groomee's hair or her own and subsequently ate it. The gesture is conspicuous in the sense that the groomer will focus on a narrow patch of hair, pinch the base of the hair with the thumb and index fingers or her teeth, pull the selected object (a louse egg in 98.9% of the cases) along the length of the hair, and eat it[30] (see Supplementary Video S1). If the focal female was the groomer and she picked louse eggs from the groomee, the louse egg counts were associated to the female groomee. If the focal female was the groomee, the louse egg counts were associated to her directly.

Data analysis. We divided our dataset in four parts to account for seasonal variations biologically relevant to the studied host-parasite system. The winter dataset included observations between January and March and corresponded to the macaques' mating season[72], as well as to the period in which macaque hair is at its longest and

densest[36]. The spring dataset included observations between April and June and corresponded to a non-breeding season and the period in which macaques started moulting. The summer dataset included observations between July and September and corresponded to the macaques' birth season as well as the end of moulting, when the hair is at its shortest. Finally, the fall dataset included observations between October and November, and corresponded to a non-breeding season and a period in which hair resumes growth to its full length and density. For each season, we computed each female's total number of scans of grooming received (including self-grooming), louse egg-picking gestures, centrality measures, dominance rank, reproductive state, and treatment status.

To compute lice load, we used minute-scans of grooming received, the focal female being either the groomer or the groomee, and minute-scans of self-grooming per female as well as minute-counts of louse egg-picking gestures across all grooming received bouts per female. Lice load was then calculated per female as follows:

$$\text{lice load} = \frac{\sum_1^n minute\ counts\ of\ louse\ eggpicking\ gestures}{\sum_1^n minute\ scans\ of\ grooming\ received + \sum_1^n minute\ scans\ of\ selfgrooming} \quad (1)$$

where n is the total number of grooming or self-grooming bouts.

We used SNA to investigate the risk of exposure to louse infestation of females within their social network of female group members. First, we calculated the modularity Q of the contact and grooming received networks (i.e., the number of clusters in the group) based on eigenvector centrality, which provides a descriptive statistic of detection and characterization of community structure in networks[73], the higher it is the more clustered the network is. Here we use it to describe the global connectedness of the female group and track its changes across seasons because inter-individual distances seem to vary across seasons in Japanese macaques: females showed lower cohesiveness in summer[74]. We then computed individual indices of network centrality based on two behavioural datasets different from those used to estimate lice load; these data sets only included adult female-female interactions. The first dataset included dyadic total numbers of scans of general body contact, including grooming, between females A and B, which was used to build *undirected weighted networks of body contacts* (Fig. 1). The second dataset included dyadic total numbers of scans of grooming received from female A to female B and from female B to female A, which was used to build *directed weighted networks of grooming received* (Fig. 1). Although the two networks are not entirely independent, their actual differences in terms of number and strength of connections, and thus in terms of risk of exposure, are meaningful to test our predictions. From these networks, we then computed centrality indices reflecting only direct exposure to lice through the network as lice are exclusively transmitted through direct contact between hosts. Node *degree* reflected the number of direct connections an individual has in the network, and node *strength*, the sum of the weights of an individual's direct connections (for a review, see)[3].

To assign dominance rank, we calculated normalised David's scores (normDS–package EloRating[75]), an individual score of relative power based on the successes (winning vs. losing) of an individual in agonistic interactions while accounting for the other group members' successes[76]. We based our calculations on matrices of decided aggressive interactions, in which we could define a winner and a loser, and of displacements or supplantations recorded *ad libitum*. As there was no change in female hierarchical order across seasons, but individual normDS varied in magnitude, we finally assigned females an ordinal rank with 1 being the highest and 20 the lowest.

Statistics. Centrality indices were calculated with the appropriate functions provided in the package igraph[73,77]. Network measures are not independent because they derive from a network where all individuals are linked to some extent and this non-independence violates many assumptions from most statistical tests. When testing the effect of factors on network measures or the effect of network measures on other variables this non-independence needs to be taken into account. A robust and modern standard way to do that is to compare statistical models based on the original observed data to a distribution of null models based on randomised data[78,79]. In this study, we randomised networks using the function rewire.edges of the package igraph, which rewires the end points (or nodes) of edges (i.e. edge rearrangement[78]) according to a probability of establishing connections that we set to vary randomly between 0 and 1 at each randomisation run. We used edge rearrangement to get a null model that randomly rearranges the observed interactions among pairs of nodes because we were confident in the observed edges and we constrained rearrangements by keeping the degree distribution of the original network to have a biologically meaningful null model[78]. Although the probability of a connection can be based on the number of individuals as potential partners, allowing the probability of establishing a connection to vary reflects natural processes of social partner choice. This approach thus provides a more conservative randomisation procedure. After each randomisation, network measures were recalculated and re-integrated in the statistical models (exactly the same models with all control factors but with the network measure derived from the randomisation). After 2000 randomisations, the statistical parameters of interest (e.g. model estimates or p values, see Supplementary Tables S2 & S4) were compared between models based on observed data and "null" models based on randomised data. If a substantial proportion (95%) of statistical parameters derived from models based on observed data were lower/higher than parameters derived from models based on randomised data, then we could conclude that the observed effects of or on sociality were different from those expected to arise by chance[78,79]. The randomisation procedure is exactly the same for all analyses.

To analyse relationships between the main variable of interest and the main predictor(s) while accounting for potential confounding factors, we built General Linear Mixed Models (GLMM) with Gaussian distributions and identity link functions using the function lmer from the lme4 package[80]. All models contained three main control factors: *dominance rank*, *reproductive state* and *season* (Table 1). In models with lice load as the response variable, an additional confounding factor, *treatment status* (binary: was/was not treated, Table 1), was included as the experimental anthelminthic treatment (Ivermectin) administered every 4 months to half the adult female cohort has been shown previously to affect louse numbers[81]. However, we observed only a small and transient decrease

in estimated louse infestation approximately one week after treatment (unpublished data), and did not expect this result to have a significant effect on overall or seasonal lice load. Regardless, treatment was included in our models to control for these potentially confounding effects.

Assessment of the variation in centrality and lice load according to seasonal and individual factors. To assess the variability present in host sociality and lice load that may affect the relationship between the two, we built GLMMs with each centrality measure as the response variable, season, reproductive state and rank as predictors, and individual identity as random effects. Variation in lice load was assessed from the null model that was built to test predictions 1 and 2 and that included season, reproductive state, treatment status and individual identity as well (see below).

Relationship between measures of centrality and lice load: testing predictions 1 and 2. To address our main question concerning the relationship between measures of centrality and lice load, we built GLMMs with *lice load* as the response variable, *centrality index* as the main predictor, *season*, *reproductive state*, *rank,* and *treatment status* as confounding factors and *individual identity* as a random effect.

We also included a three-way interaction between centrality, season, and reproductive state, and its associated two-way interactions (centrality by season, centrality by reproductive state, and reproductive state by season). The effect of centrality on lice load could indeed depend on seasonal variations in both lice load and centrality, and lice load could also be differentially influenced by some stages of host reproduction (e.g., cycling versus lactating), and these stages depend on season. Thus, it may be that the effect of the interaction between centrality and season on lice load varies according to female reproductive state, such as females more central in the network have more lice in winter and summer but only if they are reproductively active (Table 1). We used likelihood ratio tests (LRT) to compare models with and without interactions and removed them if they did not improve model fit (at p LRT > 0.100). If the p-value of the LRT was between 0.100 and 0.050, we considered the interaction to marginally improve model fit and kept it in the model (Supplementary Table S3). Supplementary Table S3 shows that amongst all interactions tested, only those including centrality and either season or reproductive state remained in the models. Note that if an interaction was significant, predictors can be interpreted only within this interaction. We transformed (log or square-root) whenever necessary and then standardized (z-transformed) all numeric predictors for more accurate model fitting and ease of interpretation/comparison of model estimates. We checked several model assumptions (normality and homogeneity of residuals, variance inflation factors[82]) and no obvious violations or influential cases were detected. Because all centrality measures were correlated to each other to some extent (r range = 0.17–0.92, p range = 0.15–0.01), and because our dataset is comparatively small and thus could not accommodate their inclusion at once, we ran one model for each centrality measure. We tested final fitted models against a null model, comprising only control factors not involved in an interaction and the random effect, with a LRT. Control factors included in null models were not considered further. Whenever this test showed that adding predictors induced a significant improvement in model fit, we proceeded in interpreting the significance of the predictors. All statistics were done in R version 3.1.2[83]. The full results are given in the Supplementary Information (Supplementary Tables S2–4).

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Author Contributions

J.D. designed the study, collected and analysed the data, and drafted the manuscript; V.R. collected data and helped draft the manuscript; C.S. and A.M. coordinated the study, provided financial and administrative support, participated in data analysis, and helped draft the manuscript. All authors gave final approval for publication.

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Scratch that itch: revisiting links between self-directed behaviour and parasitological, social and environmental factors in a free-ranging primate

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Different hypotheses explain variation in the occurrence of self-directed behaviour such as scratching and self-grooming: a parasite hypothesis linked with ectoparasite load, an environmental hypothesis linked with seasonal conditions and a social hypothesis linked with social factors. These hypotheses are not mutually exclusive but are often considered separately. Here, we revisited these hypotheses together in female Japanese macaques (*Macaca fuscata fuscata*) of Kōjima islet, Japan. We input occurrences of scratching and self-grooming during focal observations in models combining parasitological (lice load), social (dominance rank, social grooming, aggression received and proximity), and environmental (rainfall, temperature and season) variables. Using an information-theory approach, we simultaneously compared the explanatory value of models against each other using variation in Akaike's information criterion and Akaike's weights. We found that evidence for models with lice load, with or without environmental–social parameters, was stronger than that for other models. In these models, scratching was positively associated with lice load and social grooming whereas self-grooming was negatively associated with lice load and positively associated with social grooming, dominance rank and number of female neighbours. This study indicates that the study animals scratch primarily because

of an immune/stimulus itch, possibly triggered by ectoparasite bites/movements. It also confirms that self-grooming could act as a displacement activity in the case of social uncertainty. We advocate that biological hypotheses be more broadly considered even when investigating social processes, as one does not exclude the other.

1. Introduction

Self-grooming, scratching, rubbing or wallowing are forms of body care behaviours in which many animals engage frequently. In the literature, the frequency of these self-directed behaviours (SDB) has been linked to various factors, such as ectoparasite loads [1–5], environmental conditions [6–9] and social situations [10–16].

Ectoparasites are common parasites of many animals. Even on a small scale, infestation by ectoparasites such as lice, ticks and fleas can cause dermatitis, pruritis (itching), skin sensitization and other allergic reactions. Bites, stings, movements, released chemicals or body parts (e.g. urticating hair) of ectoparasites usually trigger an 'immune' or 'stimulus' itch [1,17,18]. Although ectoparasites can be susceptible to the immunological system of the host [19], anti-ectoparasite strategies nonetheless tend to involve non-immunological defences such as body care [2–5]. Experimentally preventing animals from grooming themselves or from being groomed generally leads to sharp increases in ectoparasite infestation [2–5], whereas decreasing ectoparasite loads (e.g. by administering anti-parasite drugs) drives reductions in social and self-grooming and scratching [20]. The prophylaxis or parasitic hypothesis thus predicts that the frequency of self-directed behaviour is directly linked to ectoparasite loads [2–5].

A major alternative hypothesis, at least in human and non-human primates, is the anxiety or social hypothesis, which instead links the frequency of SDB to indicators of emotional states and postulates that SDB function to mediate anxiety. SDB in long-tailed macaques are increased by administration of anxiogenic drugs and decreased by that of anxiolytic drugs [21]. Rates often increase in situations of social uncertainty linked to social (particularly aggressive) interactions, uncontrollable/unpredictable proximity of group members, or relative dominance rank [10–16,22,23]. High scratching frequency has also been linked to high degrees of restlessness [24,25], a symptom of generalized anxiety disorder in humans (e.g. [26]).

Increased frequency of body care has also been related to high ambient temperatures and humidity or rainfall [6–9]. Underlying mechanisms behind this environmental hypothesis are often linked to ectoparasite load because the life cycle of many ectoparasites is also influenced by environmental seasonality and their abundance thus fluctuates seasonally [8,27,28]. As the mammalian pelage constitutes the habitat of their ectoparasites, variation in its quality should greatly influence ectoparasite fitness and population dynamics [1], thereby creating the potential for pelage-associated variation in SDB frequency due to habitat-associated effects on ectoparasite loads. At the same time, however, variation in hair length and density also most probably influences the amount of time that animals devote to pelage care for thermoregulation [29], making it difficult to determine whether SDB frequency relates to ectoparasites or some other unrelated ecophysiological factor. Other factors such as sweating or pilo-erection could also play a role but have rarely been investigated [9,29].

To our knowledge, hypotheses relating to whether frequencies of SDB are better explained by one or a combination of the parasite, social and environmental hypotheses have previously not been considered together. To deepen our understanding of the underlying mechanisms and supposed functions of SDB, we simultaneously tested hypotheses explaining rates of such behaviours in female Japanese macaques (*Macaca fuscata fuscata)* of Kōjima islet, Japan. In particular, the possibility that variation in ectoparasite loads with seasonal factors may be an important predictor of SDB in primates has been largely dismissed, and in general, the role of ectoparasite loads has received little consideration in the primate SDB literature. Ectoparasites known to infest Japanese macaques include two species of lice (*Pedicinis obtusus* and *P. eurygaster)* [30] and one species of tick (*Haemaphysalis longicornis*) [31]. Lice and louse eggs are commonly observed on Japanese macaques during physical examination [30,31]. A previous study has shown that 98.9% of what individual macaques pick out of the hair while grooming themselves or others, using a very conspicuous sequence of behaviour, is louse eggs [30]. Ticks on the other hand are rarely found on Japanese macaques [30,31], and the gestural sequence of removing them when found differs from that of picking lice [30]. Fleas comprise another group of common ectoparasites, but have not been reported to infest Japanese macaques [30,31].

Thus, most of what is known about ectoparasites of Japanese macaques involves lice. Body parts estimated to have many louse eggs are generally inaccessible and cannot be self-groomed, and are thus socially groomed longer than other body parts [31,32]. The number of louse eggs present on a macaque was also estimated to correspond to approximately double that of the nymph and adult louse population [31], which are the stages that feed obligately on blood. Finally, lice loads estimated from louse egg-picking gestures during grooming were recently shown to vary seasonally in Japanese macaques, although the socio-ecological factors underlying such variation remain to be determined conclusively [33]. Japanese macaques live in a seasonal environment with substantial variation in temperature and rainfall throughout the year [34] and they moult seasonally, with hair being shorter and sparser in summer and longer and denser in winter [35], which could contribute to this observed variation.

In this study, we used an information-theory framework to examine simultaneously and objectively seven mutually non-exclusive hypotheses (formulated as statistical models) related to the occurrence of SDB. Depending on the level of support for each hypothesis among a candidate set, we interpreted the effect of the examined factors on SDB. Specifically, we tested a *parasite hypothesis* that SDB are best explained by lice load alone. Because louse egg removal by self-grooming should be prophylactic, i.e. it removes future blood-feeding, potentially infectious stages from the population, the occurrence of self-grooming and lice load should be negatively associated. Alternatively, as large numbers of louse eggs should be related to large numbers of blood-feeding nymphs/adults, i.e. those triggering the immune/stimulus itch, a positive relationship between lice load and rates of scratching might indicate that monkeys scratch because of their itch. We then tested a *social hypothesis* that SDB are best explained by social variables alone. This hypothesis is generally related to predicted levels of anxiety and included the following variables: aggression received, social grooming, proximity of higher-ranking individuals, number of neighbours, dominance rank and reproductive status. According to this hypothesis, high-ranking females should be less anxious about social outcomes and interactions because they receive less aggression and have more social options than low-ranking females. If SDB rates are indicators of anxiety, then they should be positively associated with dominance rank, rates of aggression received and proximity to higher-ranking individuals or even neighbours in general. SDB rates should also be higher when females are reproductively active, i.e. when cycling, pregnant or lactating, because they experience changes in their energetic needs, physiology, social interactions, especially increased aggression and coercion from males, and social networks [36–39], changes which can be sources of anxiety and thus be related to changes in SDB rates (e.g. [40]). We also tested an *environmental hypothesis* that SDB are best explained by environmental variables alone, such as seasonality, temperature and rainfall. Ambient temperature and humidity have differential effects on the pelage of animals and on their activities. For instance, according to the environmental hypothesis, we might predict that in summer (short hair), less pelage care is required than in winter (long hair) to achieve the same thermoregulation efficiency. However, hot and humid weather during summer may induce sweating, which may in fact increase the need for pelage care compared with winter. It was thus difficult to predict the sign of the relationship between the occurrence of SDB and environmental factors, so we left predictions open. Furthermore, Japanese macaques are strict seasonal breeders [34], so physiological and behavioural changes are tightly linked to season. In addition, lice loads themselves were shown to vary seasonally in Kōjima macaques [33], so it would be difficult in any case to separate the influence of these factors on SDB.

Because these hypotheses are not mutually exclusive, we also examined the explanatory power of models that included combinations of these main hypotheses. The *parasite–social hypothesis* predicted that a combination of parasitological and social factors best explains scratching and self-grooming. The *parasite–environmental hypothesis* predicted that a combination of parasitological and environmental factors best explains scratching and self-grooming. The *environment–social hypothesis* predicted that a combination of environment and social factors best explains scratching and self-grooming. Finally, the *integrated hypothesis* predicted that SDB are best explained by a combination of parasitological, social and environmental factors. After testing these hypotheses via model comparison, we present the results of the model or set of models that best explained the occurrence of SDB in our observed data.

2. Material and methods

2.1. Study site, study subjects and data collection

We studied Japanese macaques on Kōjima, a 0.3 km^2 islet in southern Japan (31°27′ N, 131°22′ E) [41]. Provisioning and behavioural observations of Kōjima macaques started in 1952, and demographic,

ecological, behavioural and life-history data are available since then [42]. The study group is currently provisioned with approximately 3 kg of wheat approximately twice weekly.

Data were collected on the 19–20 adult females (more than 7 years old; one female reached adulthood at the beginning of the study and was followed from April onwards) of the main group (approx. 60 individuals in total, including 9 adult males and 23–31 non-adult individuals) from January to November 2014. We focused on females because in Kōjima they form the stable core of the group and dominate dynamics of social networks, whereas males migrate between groups, are often few, peripheral and not very social, and juveniles are difficult to recognize and observe, and usually engage in different age-typical activities than adults (note that intraspecific variation exists in Japanese macaques' social structure [34,42,43]). Observations comprised 1265 15-min focal observations or a mean of 66 (±5 s.d.) per female. Females were observed following a randomized list updated day after day and focal observations were balanced across females and time of day (morning/afternoon). The main activities of females were recorded every minute, while their neighbours in proximity (including within 1, 5 and 10 m) were recorded every 2 min. Females were recorded as reproductively active in the mating (winter) and birth (summer) seasons, according to the occurrence of proceptive behaviours (e.g. approaching and presenting the hindquarters to males), male interest, and copulations, and to the birth and subsequent nursing of an infant respectively, and in the inter-season (spring and autumn) retrospectively if they had given birth. Data on agonistic interactions, i.e. those including bites, chases, hits, threats and displacements/supplantations, were collected during focal observations and ad libitum, and a winner and a loser was determined based on the receiver of the aggressive behaviour fleeing or submitting to her aggressor in order to establish a dominance hierarchy (see below). The number of scratching events and louse egg-picking gestures were counted in the interval between minute-scans. The occurrence of self-grooming was recorded as an activity state (i.e. on the minute-scan), but bouts falling within the interval between minute-scans were also counted in an extra column.

Scratching was operationally defined as moving the fingertips quickly and repeatedly across the same skin area [10,44]. New events started with changes in body area or breaks of more than 5 s (e.g. [25]). A self-grooming bout was defined as an individual grooming herself continuously until she stopped for more than 5 s. Counts of self-grooming bouts falling directly on as well as between minute-scans were pooled for analyses. To estimate lice load, we counted the number of times the groomer conspicuously picked something from the groomee's hair, or her own, for each minute-scan during social and self-grooming bouts. This louse egg-picking behaviour is defined as the groomer focusing on a narrow patch of hair, pinching the base of the hair with the thumb and index fingers or her teeth, pulling the selected object along the length of the hair, and eating the extracted item [30]. This louse egg-picking behaviour is a good estimate of lice load as it has been shown that in 98.9% of the cases, a louse egg is actually picked [30]. Louse egg counts during social grooming were assigned to the female from which eggs were removed, regardless of whether or not she was the focal female (e.g. [33]).

2.2. Data analyses

We built our dataset based on our focal observations, which we used as the unit of analysis. For each observation, we computed the variables listed in table 1. Because high numbers of zeros in count variables can lead to modelling issues (e.g. zero-inflation), we transformed several count variables into binary variables, i.e. presence/absence data, coding the occurrence of each behaviour or pattern during the focal observation as 1 and its non-occurrence as 0 (table 1).

2.2.1. Parasitological variables

Models including these variables, alone or in combination with others (table 1), tested the parasitic hypothesis that the occurrence of scratching and self-grooming is related to lice load.

The frequency of louse egg-picking gestures by unit of grooming received served as a proxy for louse infestation [33]. Females collected an average of 0.77 louse eggs per grooming minute-scan (median, range = 0.3–2.23, $N = 20$, 1885 louse egg-picking gestures in 5647 grooming minute-scans and 397 in 975 self-grooming minute-scans) [33]. Lice load was calculated as monthly average values of louse egg-picking counts divided by number of grooming minute-scans [33]. A month was the shortest timeframe under which lice load was accurately determined (i.e. the average per individual did not change after between 7 and 11 days of observation, and 11 days of observation sometimes constituted a whole month of data collection due to inconsistent access to the island).

Table 1. Summary of variables taken into account and their calculations (also see the text). *Per observation* indicates under which form the variable was entered in the models.

scratching	count of scratching events during minute-scans per observation: whether (1) or not (0) scratching occurred
self-grooming	sum of counts of self-grooming bouts between minute-scans and of self-grooming bouts falling on a minute-scan and written as an activity per observation: whether (1) or not (0) self-grooming occurred
lice load	count of louse egg-picking gestures during grooming divided by the number of grooming minute-scans per observation: monthly average
social grooming	minute-scan record of whether the focal individual grooms or is groomed by another individual per observation: whether (1) or not (0) social grooming occurred
aggression received	the focal individual receives a threat, a chase, a hit or a bite from another individual during either its focal observation or ad libitum per observation: whether (1) or not (0) the focal individual received aggression, separately during focal and ad libitum
provisioning day	the main group is regularly provisioned with 3 kg of wheat grains. Wheat is thrown on the sand of the main beach of the island over a limited area, which creates an increased potential for aggression to occur compared with when provisioning does not occur per observation: whether (1) or not (0) the group was provisioned on that day
dominance rank	dominance rank as determined by the calculation of David's scores (see the text) per observation: David's score of the focal individual (number between 1 and $N - 1$)
proportion of higher-ranking females within 10 m proximity	number of proximity scans with females that are higher ranking than the focal female as a proportion of all proximity scans with females as neighbours per observation: proportions between 0 and 1
number of female neighbours within 5 m proximity	number of different females within 5 m proximity for each proximity scan per observation: sum of those numbers (number between 0 and maximum 152 (19 potential female neighbours times 8 proximity scans))
reproductive status	reproduction is seasonal in Japanese macaques but females do not cycle every year and although they did cycle during the mating season, they may not become pregnant and give birth. Their reproductive status can thus vary per observation: whether (1) or not (0) the focal female was reproductively active, i.e. either cycling, pregnant or lactating
rainfall	total amount of rain in millimetres per day over the entire study period per observation: average amount of rain in millimetres over 3 days including 2 days before and the day of observation
temperature	average temperature in °C per day over the entire study period per observation: average temperature of the day of observation
season	climatic season during which the observations were carried out per observation: winter, spring, summer, autumn

2.2.2. Social variables

Models including these variables, alone or in combination with others (table 1), tested the social hypothesis that scratching and self-grooming are related to social factors and representative of social uncertainty or anxiety.

Social grooming reduces anxiety because it is linked with the release of rewarding opioid neuropeptide beta-endorphins [45,46] and has been connected with a reduction in heart rate [14,47] and SDB [10,16,22]. We thus included the occurrence of social grooming in the models as it is possible that

it influences the likelihood of occurrence of SDB compared to observations where social grooming did not occur.

Dominance rank is associated with social uncertainty because low-ranking individuals are more likely to receive aggression (in this study, correlation between rank and aggression received: $r_{\text{Pearson}} = -0.51$, $t = -2.54$, d.f.= 18, $p = 0.020$, $N = 20$). In a socially strict system such as that of Japanese macaques, low-ranking individuals are more constrained in their behavioural options than high-ranking individuals are [48]. Dominance rank was assigned through the calculation of normalized David's scores (normDS), an individual score of relative power based on the successes (winning versus losing) of an individual in agonistic interactions while accounting for the other group members' successes [49]. Calculations were based on matrices of decided agonistic interactions. The highest-ranking female receives the highest score.

To calculate individual rates of aggression received (number of events divided by observation time), we only considered focal data. This variable was then transformed into a binary variable, with the focal female either receiving or not receiving aggression. From the ad libitum data of each observation day, we additionally coded whether or not the focal female received or did not receive aggression during that day of observation, notably in order to account for the increased likelihood of aggression occurrence on provisioning days. The occurrence of provisioning on each observation day was also therefore included as a control factor. At the study site, provisioning involves providing the group with a small amount of wheat over a short duration in a limited area, which dramatically increases the frequency of aggression for the majority of the group and may thus have an influence on behaviours sensitive to social conditions.

The presence of high-ranking individuals has been shown to be a factor in social uncertainty inasmuch as their proximity can increase the rates of SDB [7,50,51]. We calculated the number of proximity scans up to 10 m in which higher-ranking females were present as a proportion of all scans in an observation, thereby giving per observation a number between 0 and 1. We also counted the number of different female neighbours within 5 m proximity for all proximity scans in an observation. We chose two different proximity thresholds, a radius of 5 m proximity representative of social integration and a radius of 10 m representative of social uncertainty potential. This was based on the facts that first, Japanese macaques living under natural conditions seem to tolerate each other without aggression above a proximity threshold of 1 m [52]. Second, given the high proportion (20%) of negative social interactions resulting from entering the proximity of another individual [48], it is fair to assume that the approach of a higher-ranking individual as far as 10 m can already potentially create uncertainty as to how this animal will behave.

We finally included the reproductive status of the females as either active, i.e. cycling, pregnant or lactating, or inactive. Indeed, reproductive activity is seasonal (with winter and summer being the mating and birth season respectively, with variation throughout Japan [34]) and induces drastic changes in the females' behaviour and physiology [36–38,53], which may influence rates of SDB, either directly or through interactions between reproductive state and social interactions, seasonal factors and/or lice load.

2.2.3. Environmental variables

Models including these variables, alone or in combination with others (table 1), tested the environmental hypothesis that scratching and self-grooming are related to climatic factors.

Daily rainfall and daily average temperatures were extracted *a posteriori* from the historical records of a meteorological service online provider (http://www.accuweather.com/en/jp/aburatsu/219041/weather-forecast/219041) based on data from the weather station nearest to the field site and on the same side of the coast (Aburatsu, 25 km). Because access to the island for observation was limited to days with relatively good weather (i.e. little rain or strong winds), thereby introducing a bias towards having no rain, we used the mean rainfall over three days including the two days preceding the observation and the day of observation itself. We also included the categorical variable season (winter, spring, summer, autumn) as Japanese macaques are highly seasonal animals at many levels (reproduction, moulting, sociality, etc.) [34].

The dataset is provided in electronic supplementary material, table S1.

2.3. Statistical analyses

Analyses were carried out in R v. 3.1.2 [54]. We ran generalized linear mixed models with a binomial error structure and logit link function with the function glmer from the lme4 package [55]. Models are presented in table 2. Focal animal identity, date and time of day (morning/afternoon) were included as

Table 2. List of models included in the comparison.

	independent variables											
testing the hypothesis(ses)	lice load	social grooming	aggression received (focal)	aggression received (ad libitum)	feeding day	David's score	prop. higher-rank nn10	number females nn5	reproductive status	rainfall	temperature	season
integrated	x	x	x	x	x	x	x	x	x	x	x	x
parasitic	x											
social		x	x	x	x	x	x	x	x			
environmental										x	x	x
parasitic–social	x	x	x	x	x	x	x	x	x			
parasitic–environmental	x									x	x	x
environmental–social		x	x	x	x	x	x	x	x	x	x	x

random factors to control for pseudo-replication and the effect of time of day on social interaction and the frequency/occurrence of SDB [7,9,50,56]. Model assumptions (homogeneity of residuals, variance inflation factors below or around 1, and stability of estimates [57]) were tested and found to be fulfilled and no influential cases were detected.

To compare all alternative hypotheses simultaneously and objectively, we used an information-theory approach based on Akaike's information criterion (IT-AIC) which provides an objective ranking of models from a candidate set and an estimation of their relative explanatory values [58]. The principle of this approach relies on assessing the likelihood and uncertainty of one or several models in a candidate set to represent the 'reality' or 'truth'. This can be judged by the AIC value as well as the difference in AICs between the model with the smallest AIC and the others (AICs in increasing order) and the likelihood and evidence ratio of each model compared with the one with the smallest AIC value [58]. In this way, we obtain a formal strength of evidence for each candidate model linked to a specific hypothesis. With the package AICcmodavg (function aictab) [59], we extracted the AIC of each model and ranked them accordingly. Convention sets a difference in AIC of more than two as indicative of a model having stronger explanatory value than another; we nevertheless considered models with AIC differences of up to four points as parsimonious candidate models to be conservative [58]. The function aictab also computes each model's Akaike's weight, or relative likelihood, which indicates to what extent one model is more likely than another in the candidate set to provide a reasonable explanation of the variance in the data. Akaike's weights were then used to compute evidence ratios (equal to the weight of the model with the lowest AIC divided by the weight of the model to compare it against), which determine the extent to which one model had stronger explanatory value over another, if any. We then used the modavg function of the same package to extract weighted parameter estimates, unconditional standard errors and 95% CIs of all predictor variables repeatedly occurring within the set of candidate models. Parameter estimates can be averaged across all models in the candidate set (full averaging), even those in which the variable of interest does not appear (in which case parameters are set to zero) or only across models in which the variable of interest appears (conditional averaging) [58]. We chose the latter strategy because we had strong *a priori* reasons to include specific variables in specific models. We also chose to show average parameter estimates instead of only those parameters estimated from the model with lowest AIC because, although the different models offer different interpretations of the data, all interpretations from models within the candidate set are plausible.

3. Results

Female Japanese macaques of Kōjima scratched on average 6.9 times per hour of observation (median, range = 3.7–11.0, $N = 20$) and groomed themselves 4.5 times per hour of observation (median, range = 2.0–5.7, $N = 20$).

Among the candidate models with the occurrence of scratching as the response variable, the parasite model with monthly lice load had the lowest AIC value and a weight of 0.63, followed by models including parasitological and social or environmental variables as well as environmental variables only (cumulative Akaike's weight of 0.92; ΔAIC up to 3.97; table 3 and figure 1). Within the model candidate set, the parasite model had 5.7–7.0 times more empirical support than the three closest competing models, i.e. those with the next lowest AICs (table 3 and figure 1). In models including lice load as a predictor, females were more likely to scratch if they had higher monthly lice loads (averaged $\beta = 0.26 \pm 0.15$ unconditional s.e., unconditional 95% CI = 0.02–0.55; table 4). Among social factors, there was a small tendency for the number of neighbours within 5 m proximity to increase the occurrence of scratching (averaged $\beta = 0.02 \pm 0.01$ unconditional s.e., unconditional 95% CI = −0.01–0.04; table 4). The occurrence of scratching was also positively associated with the occurrence of social grooming (averaged $\beta = 0.29 \pm 0.14$ unconditional s.e., unconditional 95% CI = 0.01–0.56; table 4). Other social variables and environmental factors explained little to none of the variance in the data (table 4).

Among the candidate models with the occurrence of self-grooming as the response variable, the parasite–social model with parasitological and social variables had the lowest AIC value and an Akaike's weight of 0.82. Within the candidate set, the parasite–social model had 10.3 times more empirical support than the second model in the list (table 3 and figure 1). Within this model, the occurrence of self-grooming was negatively associated with monthly lice load (av. $\beta = -0.27 \pm 0.14$ unc. s.e., unc. 95% CI = −0.53–0.00; table 4). Females were more likely to groom themselves if social grooming occurred (av. $\beta = 1.00 \pm 0.14$ unc. s.e., unc. 95% CI = 0.74–1.27; table 4), if they had a higher number of female neighbours within 5 m

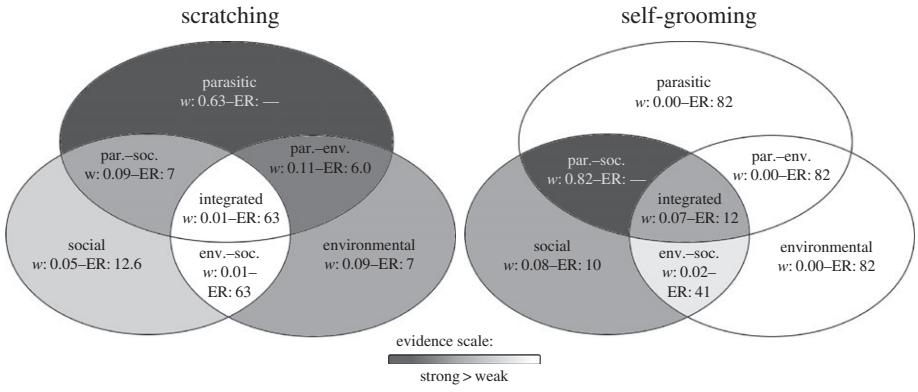

Figure 1. Schematic presentation of results. With the name of the model are given Akaike's weight (*w*) and evidence ratio (ER) (null '—' for the 'best' model) of each model. Colour darkness indicates level of support, from dark grey (model with the highest *w*) to white (model with lowest *w*) with intermediate *w* and ER in shades of grey in descending order of importance.

Table 3. Model characteristics. *K*, number of variables included; AIC, Akaike's information criterion; ΔAIC, difference in AIC between the model with the lowest AIC and the target model; weight, model probabilities (*sensu* Burnham & Anderson [58]); cum. weight, cumulative weight; ER, evidence ratio: weight of the model with the lowest AIC divided by weight of the target model. Models in italics are those within ΔAIC < 4 of the model with the lowest AIC (see the text and Burnham & Anderson [58]). Abbreviations: par, parasitic; soc, social; env, environmental.

models	*K*	AIC	ΔAIC	weight	cum. weight	log-likelihood	ER
scratching							
parasitic	*5*	*1499.94*	*0*	*0.63*	*0.63*	*−744.97*	*—*
par–env	*10*	*1503.44*	*3.51*	*0.11*	*0.74*	*−741.72*	*5.7*
par–soc	*13*	*1503.88*	*3.95*	*0.09*	*0.83*	*−738.94*	*7*
environmental	*9*	*1503.91*	*3.97*	*0.09*	*0.92*	*−742.95*	*7*
social	12	1504.84	4.90	0.05	0.97	−740.42	12.6
env–soc	17	1507.74	7.80	0.01	0.99	−736.87	63
integrated	18	1507.79	7.86	0.01	1.00	−735.90	63
self-grooming							
par– soc	*13*	*1492.37*	*0*	*0.82*	*0.82*	*−733.19*	*—*
social	12	1497.08	4.71	0.08	0.90	−736.54	10.3
integrated	18	1497.18	4.81	0.07	0.98	−730.59	11.7
env– soc	17	1499.50	7.13	0.02	1.00	−732.75	41
parasitic	5	1565.40	73.03	0.00	1.00	−777.70	>82
par– env	10	1570.78	78.41	0.00	1.00	−775.39	>82
environmental	9	1570.94	78.57	0.00	1.00	−776.47	>82

proximity (av. $\beta = 0.05 \pm 0.01$ unc. s.e., unc. 95% CI = 0.02–0.07; table 4) and if they were low ranking (av. $\beta = -0.03 \pm 0.01$ unc. s.e., unc. 95% CI = −0.05–0.00; table 4).

4. Discussion

SDB such as scratching and self-grooming can be explained by a number of factors related to parasites, sociality and the environment. Often enough, studies focus on a single hypothesis only. Taking an integrative approach and examining all hypotheses simultaneously and objectively, this study shows that in female Japanese macaques at Kōjima, scratching and self-grooming occurrences are better explained by models including lice load and social factors than other combinations of variables.

Table 4. Multi-model inference results: model averaged parameter estimates (β) ± unconditional standard errors (s.e.) (95% unconditional confidence intervals CI). In italics are variables for which CI does not include zero. Variable parameters are averaged only over models in which the variable appears, except for the intercept's, averaged across all models (see the text).

	scratching	self-grooming
	$\beta \pm$ s.e. (95% CI)	$\beta \pm$ s.e. (95% CI)
intercept	0.51 ± 0.31 (−0.10–1.12)	−0.53 ± 0.49 (−1.48–0.43)
monthly lice load	*0.26 ± 0.15 (0.02–0.55)*	*−0.27 ± 0.14 (−0.53–0.00)*
social grooming	*0.29 ± 0.14 (0.01–0.56)*	*1.00 ± 0.14 (0.74–1.27)*
aggression received (focal)	0.14 ± 0.20 (−0.25–0.53)	0.32 ± 0.19 (−0.05–0.70)
aggression received (ad libitum)	0.12 ± 0.15 (−0.19–0.42)	0.13 ± 0.15 (−0.16–0.43)
feeding day	0.18 ± 0.17 (−0.16–0.52)	0.19 ± 0.16 (−0.11–0.50)
David's score	−0.01 ± 0.02 (−0.05–0.02)	*−0.03 ± 0.01 (−0.05–0.00)*
prop. high-rank nn10	0.14 ± 0.16 (−0.18–0.46)	−0.20 ± 0.16 (−0.52–0.11)
nb females nn5	0.02 ± 0.01 (−0.01–0.04)	*0.05 ± 0.01 (0.02–0.07)*
reproductive status	−0.09 ± 0.18 (−0.45–0.27)	−0.01 ± 0.14 (0.28–0.27)
rainfall (3 days)	0.00 ± 0.01 (−0.02–0.01)	0.00 ± 0.01 (−0.01–0.02)
temperature	0.03 ± 0.02 (−0.02–0.07)	0.03 ± 0.02 (−0.02–0.07)
season winter	−0.37 ± 0.34 (−1.03–0.30)	0.58 ± 0.32 (−0.05–1.21)
season spring	0.00 ± 0.21 (−0.40–0.41)	0.28 ± 0.19 (−0.09–0.66)
season summer	−0.15 ± 0.32 (−0.78–0.47)	−0.23 ± 0.29 (−0.80–0.34)

Within models including lice load, the occurrence of scratching was positively associated with monthly lice loads. Chemicals in saliva, stings, body secretions or urticating hairs of ectoparasites all have the potential to induce an immune itch which triggers scratching, effectively relieving the itch [1,17]. Additionally, although scratching may not remove the egg from its position on the hair or feather, it may damage it and halt its development [5], adding a prophylactic benefit similar to that of self-grooming with the extra advantage that with scratching, an animal can reach areas inaccessible to self-grooming [5]. This link is commonly established in many animals such as ungulates and birds [3–5] but is neglected in primates because, among other reasons, they are social animals and scratching was linked early on to social events and anxiety due to social events.

Inversely, the occurrence of self-grooming was negatively linked to monthly lice loads. By grooming themselves, females thus may be able to prevent infestation by removing future blood-sucking ectoparasites [4]. However, the occurrence of self-grooming was also linked to the occurrence of social grooming, larger numbers of female neighbours in relatively close proximity, as well as to lower dominance rank. These results therefore also support the hypothesis that, in addition to its original prophylactic function, self-grooming may act as a displacement activity that could potentially provide an escape from socially uncertain situations [11]. For instance, Japanese macaque social behaviour is highly biased towards kin so that individuals found often in proximity of each other are likely to be genetically related to a high degree [48]. Given that matrilines are rather small (between two and four adult females) and few (three) in the study group, larger numbers of female neighbours could be linked to the increased presence of non-kin in proximity which could be related to social uncertainty and bouts of self-grooming. Future studies could investigate the effect of the presence of kin versus non-kin in relation to SDB when possible.

A major factor positively associated with the occurrence of SDB was the occurrence of social grooming. Several studies have actually reported a decrease in SDB with the occurrence of grooming in accordance with its proposed role in tension reduction [10,16,22]. However, the occurrence of social grooming may intensify the expression of SDB, a pattern that is hypothesized to relate to the risk of aggression due to increased proximity (e.g. [60]), the uncertainty at the beginning or end of a grooming bout in terms of activity change or social situation (e.g. [10,23]), or behavioural transitions that could be facilitated by SDB (as displacement activities) (e.g. [61]). Other hypotheses for increased SDB in this context that have rarely been considered include the fact that animals may experience some kind of

behavioural contagion, simply copying the activities of others or wanting to prolong grooming (e.g. [44]), or they may still feel the touch of the grooming activity on their skin or a disturbance in hair arrangement (e.g. [61]). All these explanations remain speculative pending further investigation, but it is noteworthy that the relevant stimulus, a mild mechanical touch and/or a change of temperature (due to body contact or disturbance of the hair or feathers), has the potential to activate the same neural sensory afferent fibres (C fibres), i.e. those involved in the sensation of pain, temperature, touch and itch [62,63].

Interestingly, social and environmental factors that we investigated had less weight in explaining variation in SDB when compared to lice load. This is despite the fact that Japanese macaque society is governed by strict rules following dominance and kinship relationships where individuals are constrained in their behavioural options [48,64] and tightly linked to a seasonally changing environment impacting reproduction and sociality [34]. Although previous studies on primates have linked increased urinary cortisol levels (an indicator of unbalanced homeostasis or stress) and increased scratching rates to active reproductive state [39,40], and increased rates of SDB to challenging weather [9], those variables accounted for little to none of the variation in SDB in female macaques of Kōjima. One explanation could be that the measured variables are too coarse (either reproductively active or inactive over the season and average rain amount over 3 days) to detect any meaningful pattern. Concerning the apparent lack of effect of reproductive status on SDB, another explanation could be linked to the seasonality of reproduction. During the mating season, many females are cycling at the same time and many males get an opportunity to mate; as such, the degree of competition for reproduction can be considered moderate [65]. Thus, although females are more active than when they are not reproductively active, they may have means or opportunities to avoid stressful situations like male coercion, for example by isolating themselves from the group to copulate with a male of their choice [66].

It could thus be the case that animals scratch primarily because of an immune/stimulus itch triggered by ectoparasite bites/movements. Nevertheless, this primary explanation is not exclusive of the fact that animals can scratch because of an idiopathic non-immune itch, e.g. if they are anxious in a given situation or if the atmosphere is hot and humid. The endocrine system is implicated in the regulation of internal states and behaviours [67] and is linked to the immune system [68]. Long-term release of 'stress' hormones (glucocorticoids), whether linked to social or environmental factors, tempers immune function and decreases its efficacy, probably making animals more susceptible to infections from diverse parasites/pathogens [68]. Thus, an anxious animal or an animal in a challenging environment could also be a lousier animal because of a generally weakened state.

The prophylaxis/parasitic hypothesis can actually embody altogether several reasonable explanations for variation in SDB inasmuch as ectoparasites are often transferred from one host to the next through body contact between hosts [1,18]; they greatly depend—sometimes solely (e.g. louse)—on their hosts for reproduction and survival [1,18]; they are susceptible to seasonal changes due either to their own biology, that of the host or that of the environment [28,69]; and through their blood meal they may be sensitive to the physiological state of their hosts [19,70], which may in turn be dependent on environmental and social conditions [36–38,71]. Revisiting studies linking SDB changes to environmental or social changes taking into account ectoparasite loads could fill the gaps in our knowledge of mechanisms or functions that we are still unable to explain fully, for example considering the inconsistent results about the links between social grooming and scratching, or the so-far under-investigated difference between a stimulus and an idiopathic itch, or the inclusion of a broader range of ectoparasites such as ticks and fleas (e.g. [8,27,72,73]).

Previous research often examined each of the tested hypotheses separately. Our results attest to the fact that studies should not discount the importance of hygienic/prophylactic functions of behaviour, even when testing ideas linked to social processes. It is indeed more likely that a diversity of factors affects the behaviour of animals, sometimes synergistically, sometimes independently. Taking an integrative approach thus allows for a holistic view of animal behaviour. This is facilitated by the information-theory framework used in this paper and advocated by Burnham & Anderson and others [58,74–76]. In doing so, deeper integrative insights into an animal's biology are attained, which provides a basis for further investigation. Furthermore, the investigation and use of non-invasive indicators of ectoparasite infestation, like that used in this study, can bring about further understanding of wildlife epidemiology, infection risk and links between sociality and health.

Ethics. This research complied with the Guidelines for the Care and Use of Non-Human Primates of the Kyoto University Primate Research Institute and was approved by the Kyoto University Primate Research Centre ethics committee, the prefecture of Miyazaki and the city of Kushima.

Data accessibility. Data are provided in the electronic supplementary material.

Authors' contributions. J.D. designed the study, collected and analysed the data, and drafted the manuscript; V.R. collected data and helped draft the manuscript; C.S. and A.J.J.M. coordinated the study, provided financial and administrative support, participated in data analysis and helped draft the manuscript. All authors gave final approval for publication.

Competing interests. The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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One step at a time in investigating relationships between self-directed behaviours and parasitological, social and environmental variables

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We thank Norscia and Palagi for their insightful commentary on our article 'Scratch that itch: revisiting links between self-directed behaviour and parasitological, social and environmental factors in a free-ranging primate' [1]. We welcome such discussion because we think, as the authors themselves point out at the end of their commentary, that research needs to continue in this area. In general, we agree that different stressors may act at different time frames in triggering self-directed behaviours. As rightly pointed out by Norscia and Palagi, our analysis did not take into account the different time frames that would allow for separating the effects of acute and chronic stressors on self-directed behaviours. At the level of a behavioural observation of 15 min, we instead investigated whether the occurrence of scratching and self-grooming was linked to various factors such as lice load, social activities, neighbours in proximity and environmental conditions, together and/or separately. Our study was correlational and we, therefore, avoided claims of causality, although we did address potential causal mechanisms in the discussion.

That said, we would nonetheless like to respond to several points made by Norscia and Palagi. First, one of the main points of our study was to highlight biases in the investigation of certain research hypotheses, such as those involving self-directed behaviours. Studies in primatology have often, if perhaps inadvertently, assumed that the primary drivers of self-directed behaviour (SDB) are social, with parasite or abiotic factors being

secondary. Norscia and Palagi nonetheless state that '(...) the association between self-directed behaviours, and particularly scratching, with social, environmental and parasitological factors can be considered as more than just a hypothesis. Once established that the different factors are not alternative and that their relationship with scratching has been demonstrated, it is worth focusing on the role that each factor can have in relation to the time scale' (p. 2). We would fully agree with the logic here if the premise were true. While the association between parasitological factors and self-directed behaviour is extremely well-established in ungulates and birds [2–16], with also some evidence in insects [17–19], it has received surprisingly little attention in non-human primate research. Despite the fact that numerous earlier studies on the functional significance of self- and social grooming did mention the removal of ectoparasites [20–23], it has sometimes been dismissed groundlessly or ignored altogether in more recent studies [24–27]. There is no *a priori* or *a posteriori* reason to assume that what affects ungulates or birds does not affect primates when the system under study, in this case the ectoparasite–host system, is more or less identical. Along these lines, our study was an attempt to test multiple hypotheses simultaneously and objectively using the same comprehensive dataset. A multivariate approach perhaps provides the best opportunity to draw out the key factors influencing behaviour, and thereby contribute to advancing the field. All speculation aside, our study reveals that, among the candidate set of hypotheses tested (formulated as statistical models), parasite factors appear to best explain the occurrence of scratching, while parasite and social factors appear to do so for self-grooming. If future work can now tease out the impacts of these and other factors at distinct time scales while also accounting for alternative explanations, such work would be most welcome indeed.

Second, in our study, at the level of the aggregate dataset, the hypotheses put forward are indeed non-mutually exclusive in explaining general SDB patterns, as noted by Norscia and Palagi. However, at the level of an individual SDB event, each of the hypotheses is more likely to explain the behaviour independently than in concert, though we also acknowledge the possibility of additive or even synergistic effects here; note that our statistical models for self-grooming suggested that such additive effects were likely. Regardless, a single SDB may be caused by x, y or z, but seems less likely to occur because of all three simultaneously, so the use of the term 'alternative' is not necessarily incorrect. That said, contrary to the assertions of Norscia and Palagi, this does not imply that some relationships are secondary to others. We think this distinction is meaningless, and that is why we took an integrative approach in the first place. If we did not make that point clear enough in the original manuscript, then we reiterate it here.

Third, some of the arguments put forth by Norsica and Palagi involve generalizations that may not in fact be entirely supported. Essential facts concerning primates—and to some extent time scales—are omitted in their commentary. For instance, several studies have already demonstrated quite unambiguously that body parts estimated to have many louse eggs are generally inaccessible, cannot be self-groomed, and are socially groomed longer than other body parts [8,10,15,24,28,29]. Furthermore, lice loads estimated from nit-picking gestures during grooming were recently shown to vary seasonally in Japanese macaques [30], and variation in nit-picking activity during grooming, or louse-egg feeding, has been shown to influence grooming duration, frequency and reciprocity [28]. The findings in [29] and [28] are especially important, not only because they align what we know about primates with what we know about birds and ungulates, but also because they relate to the extent to which ectoparasites can mediate social interactions, a hypothesis that is rarely acknowledged in primate studies (e.g. [30,31]). The facts that treating animals against lice decreases grooming activity and that preventing animals from grooming or self-grooming dramatically increases ectoparasite load [8,15] speak volumes in favour of investigating the links between ectoparasites and SDB, in addition to further social processes also linked to hygienic practices, regardless of time scale. So, we would argue that before dissecting *when* or under what set of conditions a certain event is likely to occur, we need to first ensure that the event and these other conditions are indeed generally related. From our perspective, such an investigation has never been fully realized in taxa as socially complex as primates, and we therefore feel the approach taken in our original article is justified.

Finally, Norscia and Palagi state that 'The variation observed between time t_0 and t_1 cannot be linked to parasitological factors if the load is not significantly different between t_0 and t_1. There is no reason to believe that, in the absence of any other additional perturbing factor, the ectoparasite load varies significantly in the minutes immediately preceding and following the stressful event.' (p. 2) While this statement belies a lack of knowledge about louse behaviour (i.e. temporal patterns in feeding behaviour), to their credit the authors do later add that 'It may be questioned that in the short term a change in the parasite activity (e.g. in response to temperature, humidity or even solar radiation [18–22]), and not in the load, could possibly cause an increase in scratching levels. However, this aspect was not tested in

Duboscq *et al*. [1].' (p. 2) Louse-induced itch could indeed depend in the long term on louse load and in the short term on louse activity. A sudden change in a multitude of parameters might impact louse load and/or activity quickly if it creates disturbances in the hair/pelage/skin of the animals that constitutes the environment of the parasite. Some studies have shown that rabbit fleas respond to oestrogen blood concentration and adapt their reproductive activity to the reproductive activity of their host [32,33]. The variation in self-directed behaviours between t_0 and t_1 could therefore be linked to parasitological factors at time scales of minutes or even seconds, although we admit that we have no information about whether louse activity is likely to vary 'in the minutes immediately preceding and following the stressful event' (p. 2). Again, we have no intention here of asserting that all SDB events are related to lice, but the effect of variation in ectoparasite load and activity across time scales should be investigated in the future, and we think our study constitutes a step forward in that direction.

In conclusion, while we agree with most of the comments provided by Norscia and Palagi, we highlight that the aims of our study were not so much to exclude the role of social stressors in the production of SDB but instead to put SDB into the broader ecological framework under which they evolved. Like Norscia and Palagi, we look forward to future studies taking an integrative view of self-directed behaviours, accounting for various factors at different time scales in order to gain further insights into why animals scratch that itch.

Data accessibility. This article has no additional data.

Authors' contributions. All authors helped draft the manuscript and gave final approval for publication.

Competing interests. We have no competing interests.

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APPENDIX F: SUPPLEMENTARY MATERIAL FOR CHAPTER 1

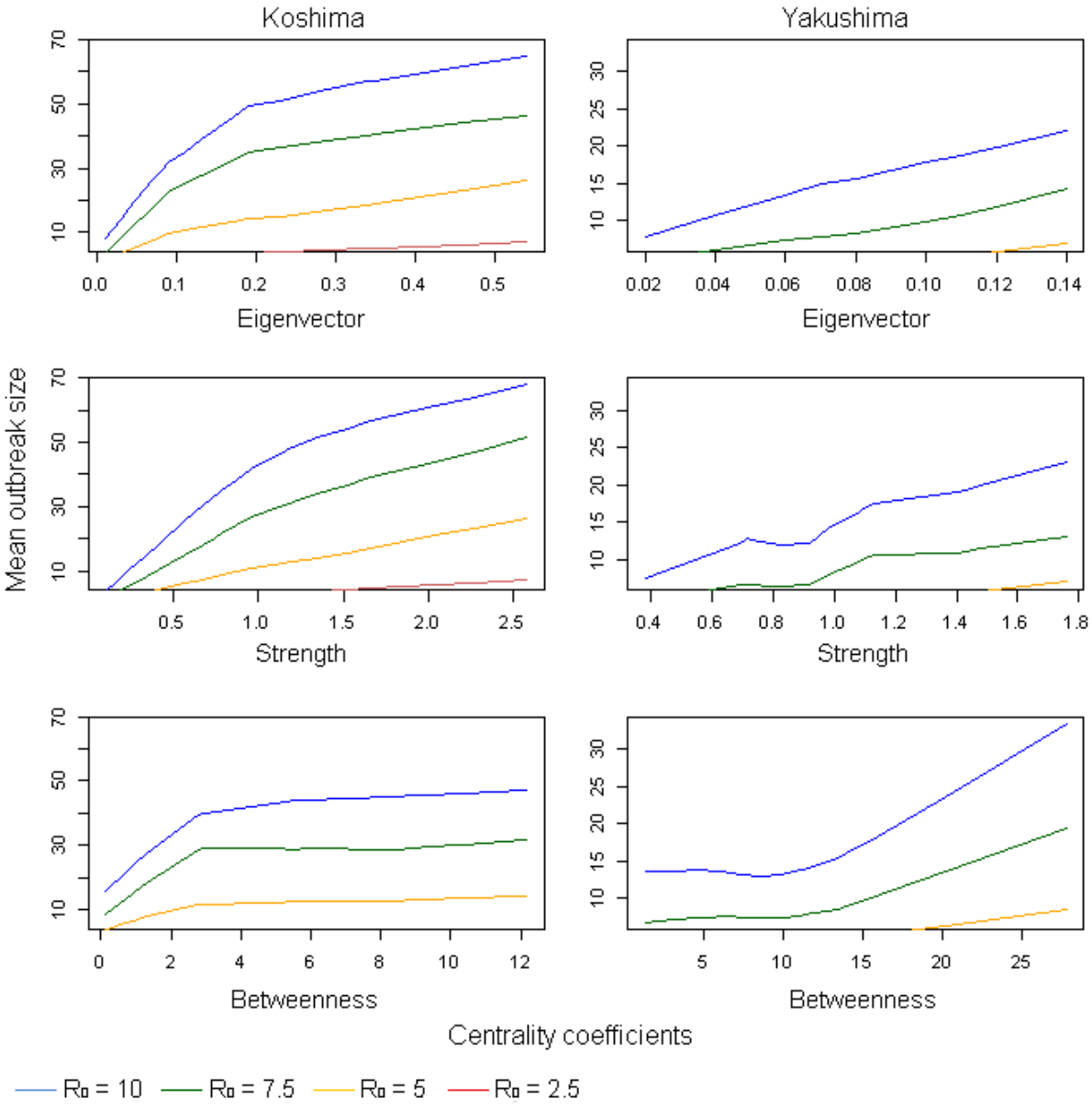

Figure FS1. Mean outbreak size across different values of pathogen infectiousness (R_0). Mean outbreak size is shown as a percentage of individuals infected in each group. The graphs indicate a consistent variation of contagion in Koshima and Yakushima, although the latter presents as a more moderate epidemic when compared to the former. Each line represents the outbreak size averaged across 10000 simulations for a given R_0. Definitions of network centralities are given in the chapter 1.

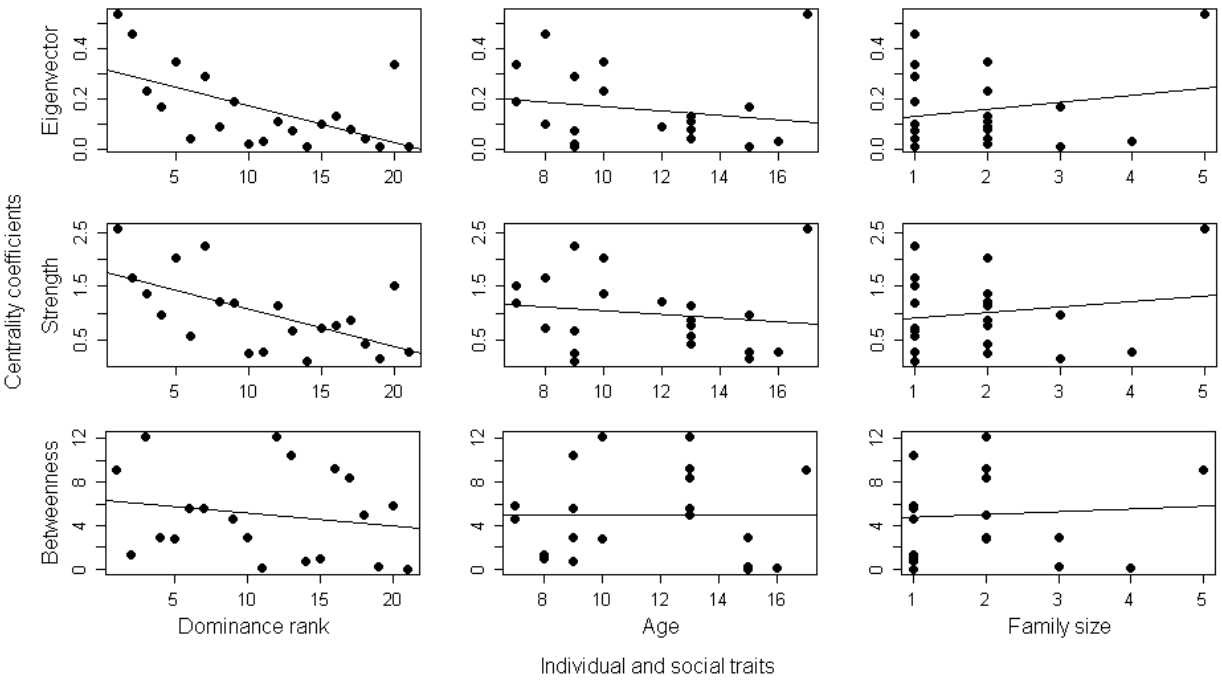

Figure FS2. Dispersion graphs showing individual/social factors and centrality coefficients in Koshima group. There is a clear relationship between dominance rank and centrality (eigenvector and strength) in Koshima while age marginally influenced eigenvector centrality. Family size had no effect on network position. Statistical tests and values are given in the chapter 1.

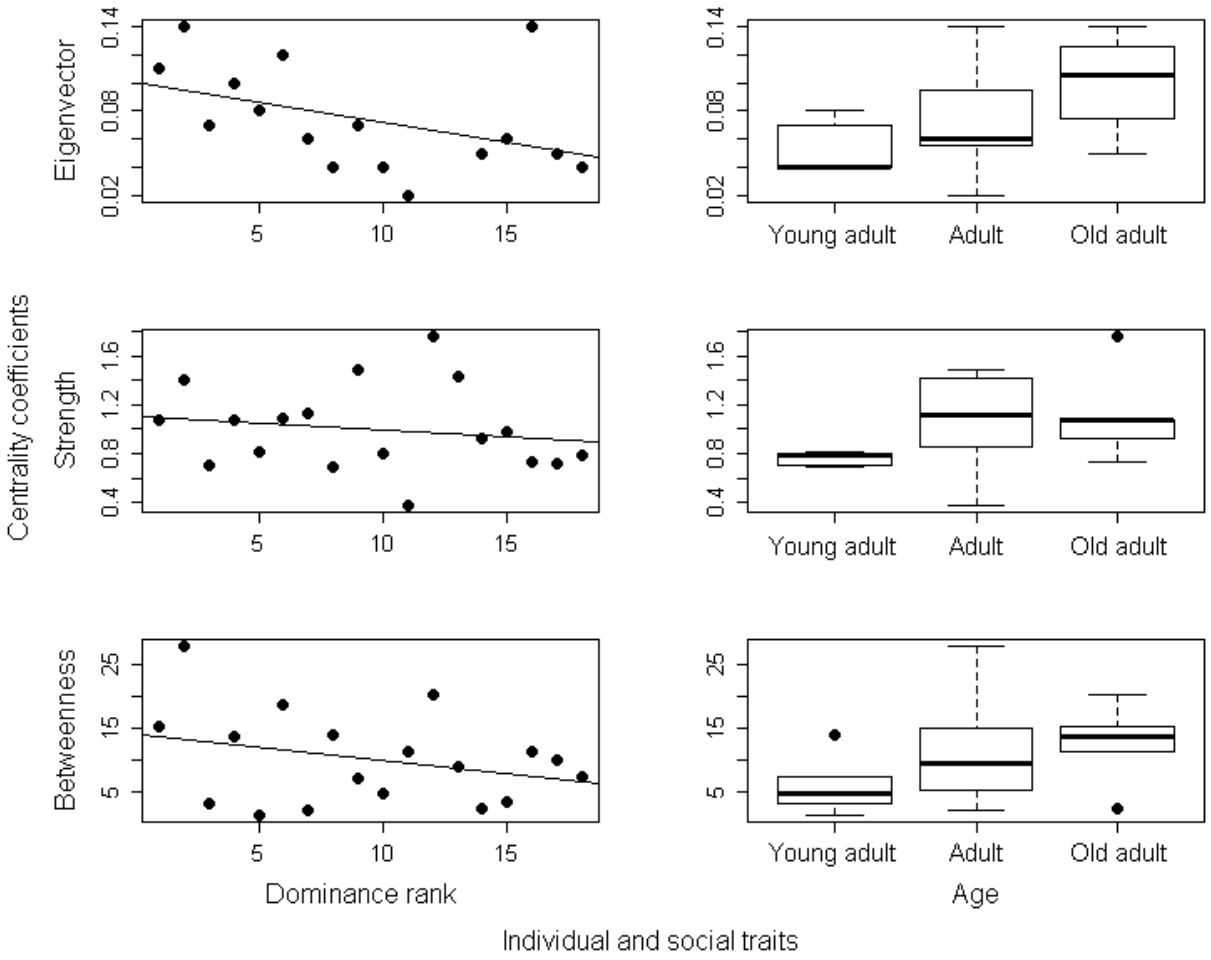

Figure FS3. Dispersion and boxplot graphs showing individual/social factors and centrality coefficients in Yakushima group. Age and to a lesser extent dominance rank influenced only eigenvector centrality in Yakushima group. As described in the main text, sexually-mature females were classified into three age classes (young adult ≥ 5 <10 yo, adult 10 – 14 yo, old adult >14). Statistical tests and values are given in the chapter 1.

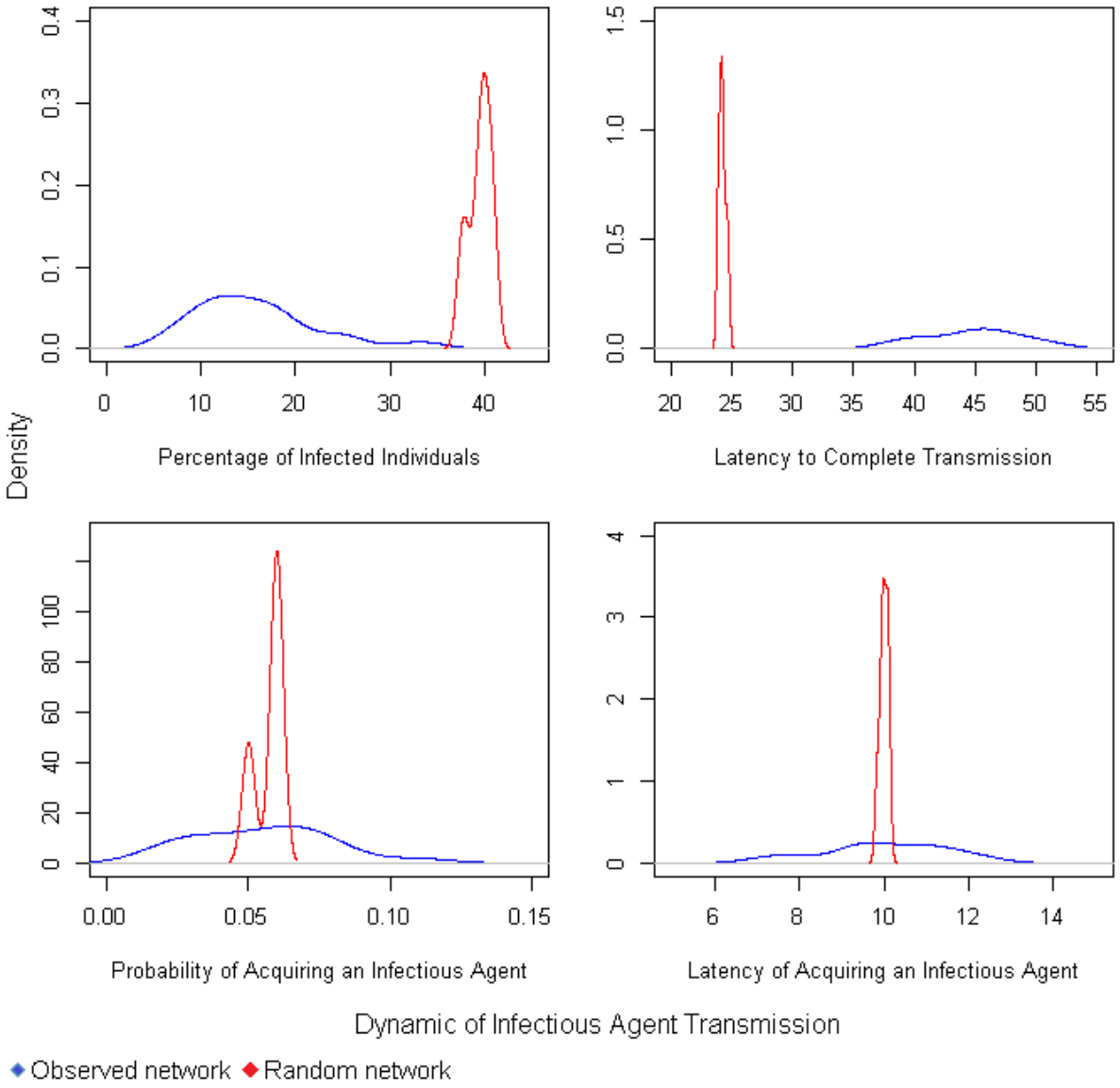

Figure FS4. Density probability plots of real and random distributions of infectious agent transmission in Yakushima group. Two of the four distributions from the simulated disease spread on the Yakushima network did not differ significantly from those expected of a random network (probability of acquiring an infectious agent: $D = 0.39$, $p = 0.2$; latency of acquiring an infectious agent: $D = 0.5$, $p = 0.08$). In contrast, the percentage of infected individuals ($D = 1$, $p < 0.001$) and latency to the whole group transmission ($D = 1$, $p < 0.001$) differed significantly between observed and random networks.

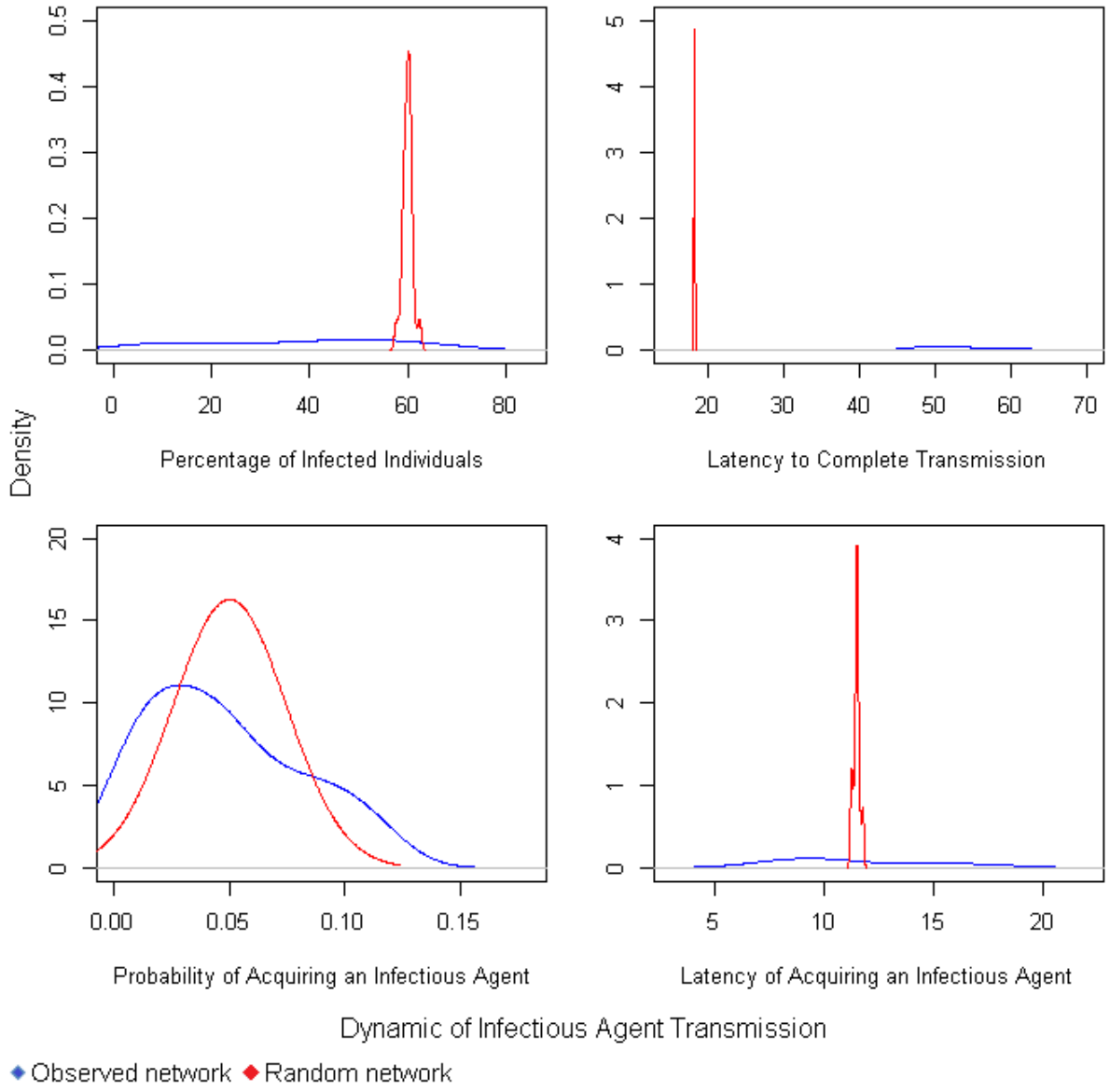

Figure FS5. Density probability plots of real and random distributions of infectious agent transmission in Koshima group. The probability of being infected (D = 0.52, p = 0.02), the percentage of infected individuals (D = 0.81, p < 0.001), latency to complete transmission (D = 1, p < 0.001) and latency of being infected (D = 0.62, p = 0.02) all significantly differed between observed and random networks.

AF1. Source code used in chapters 1 and 2.

```
breed [nodes node]
nodes-own [node-id activity]
breed [links link]
links-own [strength]
patches-own [area] ;; 0 : repos; 1 : fourragement
globals [links-list clock simulations adhesion-time mimetic-coefficient]
```

```
to setup
ca
set simulations 1
ask patches [
if ((distancexy (-0.8 * max-pxcor) (0.8 * max-pycor)) < 5)
  [ set area 1
   set pcolor green]
if ((distancexy 0 0) < 5)
  [ set area 0
   set pcolor brown]
 ]
end


to simulate
ct
clear-plot
import-network
set clock 0
set adhesion-time 0
ask nodes [facexy -28 28
  set activity 0]
output-write "sim " + simulations
ifelse mimetism?
[dependent]
[independent]
output-print " "
ifelse simulations < simulations-number
  [set simulations simulations + 1
    simulate]
  [stop simulate]
end

to dependent
set mimetic-coefficient 0.002
ask nodes
[if activity = 0
  [ifelse aff?
    [ifelse any? nodes with [activity != 0]
      [if random-float 1 <= (((1 / resting-time)/(number-of-nodes-present)) + ( Cfoll * mimetic-
coefficient * follower))
         [fd 1
         set activity 1
         do-plot
```

```
        output-write node-id
        set adhesion-time 0]]
      [if random-float 1 <= (size * ((1 / resting-time)/ number-of-nodes-present) * (resting-
nodes))
        [fd 1
        set activity 1
        do-plot
        output-write node-id ;; for the first individual
        set adhesion-time 0]
        ]
   ]
   [ifelse any? nodes with [activity = 1]
     [if random-float 1 <= (((1 / resting-time) + (mimetic-coefficient * follower))/(number-of-
nodes-present))
       [fd 1
       set activity 1
       do-plot
       output-write node-id
       set adhesion-time 0]
     ]
     [if random-float 1 <= (size * (1 / resting-time)/ number-of-nodes-present) * (resting-
nodes)
        [fd 1
        set activity 1
        do-plot
        output-write node-id
        set adhesion-time 0]
      ]
   ]
   ]
if activity = 1
  [ifelse patch-here = patch -28 28
    [fd 0
    set activity 2]
    [fd 1]
    ]
]
 set clock clock + 1
 set adhesion-time adhesion-time + 1

ifelse resting-nodes < number-of-nodes-present
[if resting-nodes >= 1
  [ifelse adhesion-limit?
    [if adhesion-time <= time-of-adhesion-limit
```

```
    [dependent]
    ]
    [dependent]
  ]]
[dependent]
end

to independent
ask nodes
[if activity = 0
  [if random-float 1 <= ((1 / resting-time)/ number-of-nodes-present) * (resting-nodes)
    [fd 1
     set activity 1
     do-plot
     output-write adhesion-time
     set adhesion-time 0]
   ]
 if activity = 1
   [ifelse patch-here = patch -28 28
     [fd 0
     set activity 2]
     [fd 1]
   ]
 ]
set clock clock + 1
set adhesion-time adhesion-time + 1
ifelse resting-nodes < number-of-nodes-present
[if resting-nodes >= 1
  [ifelse adhesion-limit?
    [if adhesion-time <= time-of-adhesion-limit
      [independent]
    ]
    [independent]
  ]]
 [independent]
end

to-report number-of-nodes-present
report count nodes
end

to-report resting-nodes
report count nodes-at 0 0
end
```

```
to-report follower
report count nodes with [activity != 0]
end

to-report Cfoll
report ((mean values-from __my-in-links with [activity-of __other-end != 0][label])/ 0.11)
end

to-report Caff
report mean values-from __my-in-links [label]
end

to do-plot
set-current-plot "adhesion-time"
set-current-plot-pen "adhesion-time"
plot adhesion-time
end

to import-network
  set-default-shape nodes "monkey"
  import-attributes
  import-links
end

;; This procedure reads in a files that contains node-specific attributes including an unique
;;identification number to import-attributes.
  ;; use CAREFULLY to ensure the file is
  ;; closed if there is an error and to notify
  ;; user of the error
  carefully [
    ;; This opens the file, so we can use it.
    file-open "attributes.txt"
    ;; Read in all the data in the file
    ;; data on the line is in this order: node-id attribute1 attribute2
    while [not file-at-end?]
    [
      ;; this reads a single line into a three-item list
      let items read-from-string (word "[" file-read-line "]")
      create-custom-nodes 1 [
        set node-id item 0 items
        set size    item 1 items
        set color   item 2 items
      ]
```

```
    ]
  ]
  ;; this is the error handling block of the carefully command
  [
    user-message (word "Error reading attributes.txt: " error-message)
  ]
  file-close
end

;; This procedure reads in a file that contains all the links. The file is simply 3 columns
;;separated by spaces. The first column contains the node-id of the node originating the link.
;; The second column the node-id of the node on the other end of the link. The third column is
;; the strength of the link.

to import-links
  carefully
  [
    ;; This opens the file, so we can use it.
    file-open "links.txt"
    ;; Read in all the data in the file
    while [not file-at-end?]
    [
      ;; this reads a single line into a three-item list
      let items read-from-string (word "[" file-read-line "]")
      ask get-node (item 0 items)
      [
        __create-link-to get-node (item 1 items)
          [ set label item 2 items ]
      ]
    ]
  ]
  ;; this is the error handling block of the carefully command
  [
    user-message (word "Error reading links.txt: " error-message)
  ]
  file-close
end

;; Helper procedure for looking up a node by node-id.
to-report get-node [id]
  report one-of nodes with [node-id = id]
end
```

APPENDIX G: SUPPLEMENTARY MATERIAL FOR CHAPTER 2

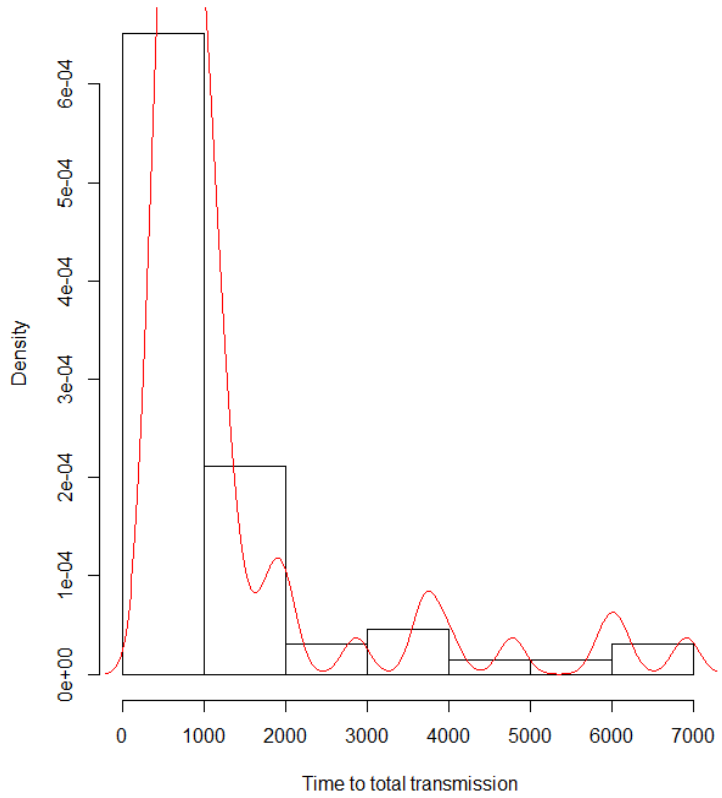

Figure GS1. Observed (black line) and density (red line) distribution functions of latency to total pathogen transmission in 40 primate networks.

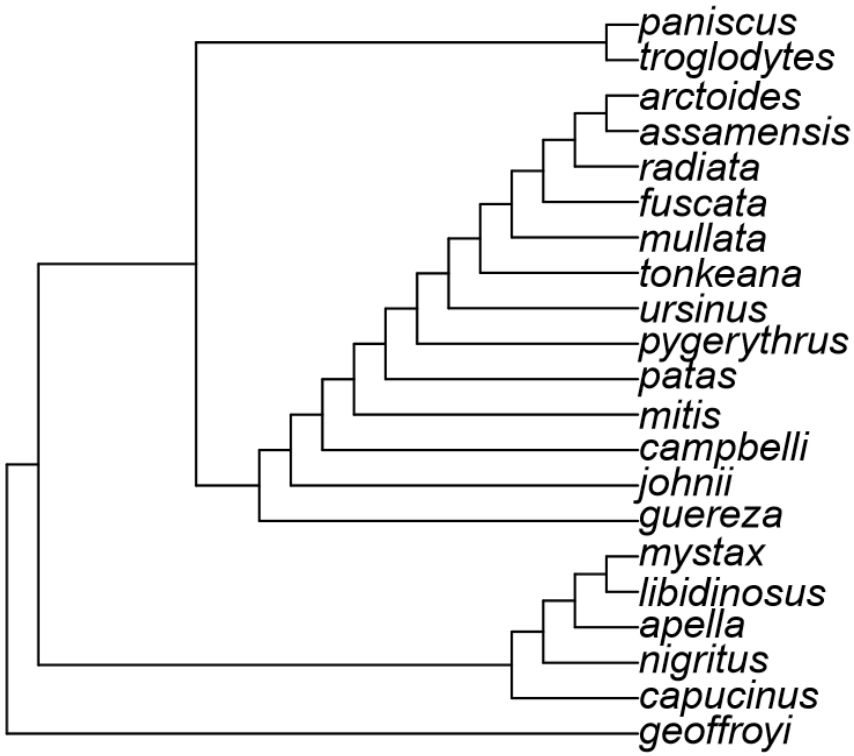

Figure GS2. Phylogenetic tree used for comparative tests.

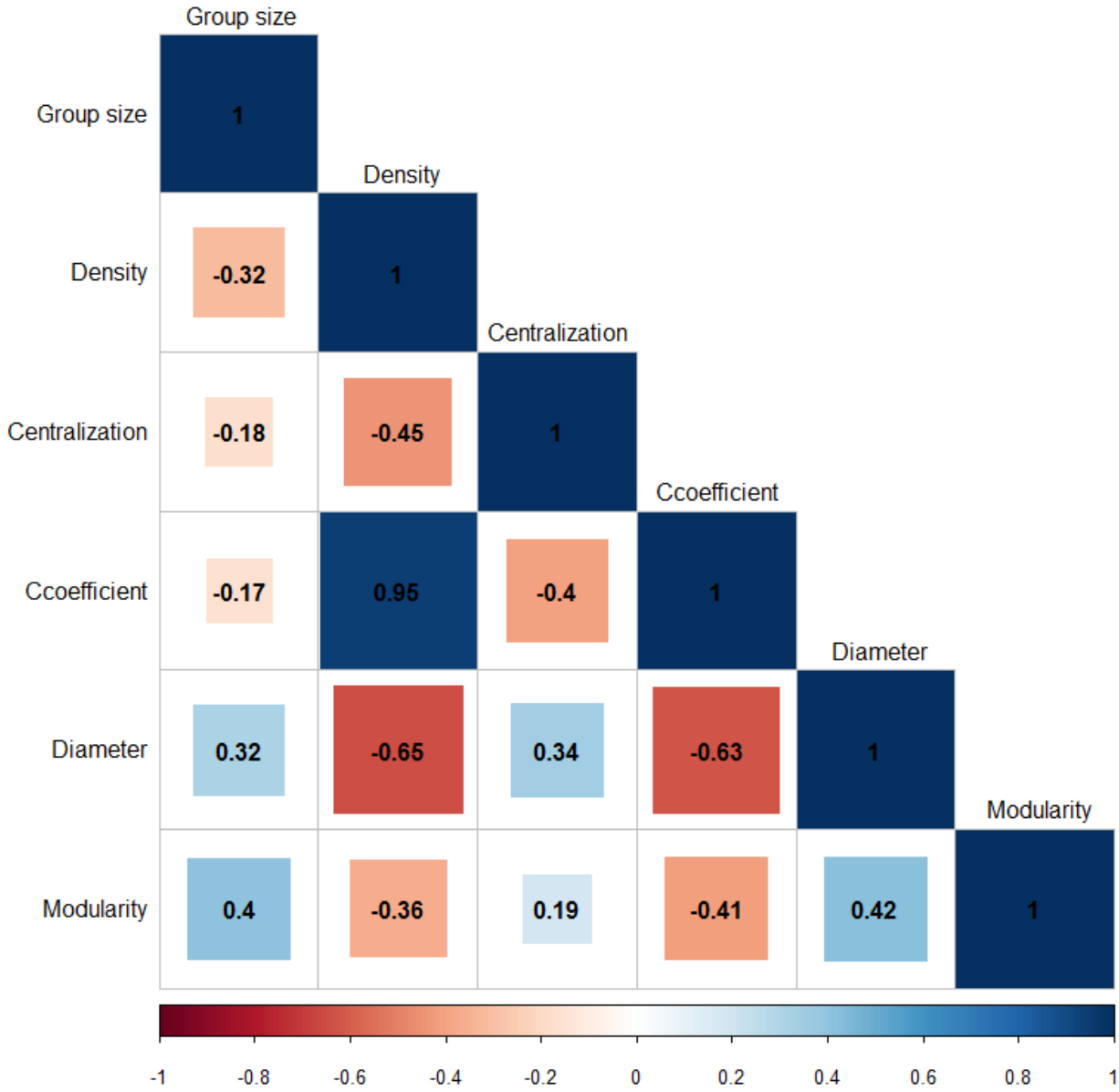

Figure GS3. Correlation matrix between group size, density, eigenvector centralization, clustering coefficient (Ccoefficient), diameter and modularity. The stronger the coefficient of correlation, the darker its blue (positive correlation) and the darker its red (negative correlation).

Table GS1. Details from the dataset used in chapter 2, including information on sources of data and group size.

Species	Common name	Sources of data (literature* or co-authors)	Group size
Ateles geoffroyi	Spider monkey	Ahumada 1992	15
Cebus capucinus	White-faced capuchin	Dr. Linda Fedigan Dr. Mackenzie L. Bergstrom Dr. Eugenia P. di Sorrentino	5
Cebus capucinus	White-faced capuchin	Perry 1996	6
Cebus capucinus	White-faced capuchin	Dr. Linda Fedigan Dr. Mackenzie L. Bergstrom Dr. Eugenia P. di Sorrentino	7
Cebus capucinus	White-faced capuchin	Dr. Linda Fedigan Dr. Mackenzie L. Bergstrom Dr. Eugenia P. di Sorrentino	10
Cebus capucinus	White-faced capuchin	Dr. Margaret Crofoot	10
Cebus capucinus	White-faced capuchin	Dr. Margaret Crofoot	12
Cebus capucinus	White-faced capuchin	Dr. Margaret Crofoot	12
Cercopithecus campbelli	Campbell's monkey	Hunkeler et al. 1972	15
Cercopithecus mitis	Blue monkey	Rowell et al. 1991	17
Chlorocebus pygerythrus	Vervet monkey	Dr. Erica van de Waal M.Sc. Christèle Borgeaud	25
Chlorocebus pygerythrus	Vervet monkey	Dr. Erica van de Waal M.Sc. Christèle Borgeaud	26
Chlorocebus pygerythrus	Vervet monkey	Dr. Erica van de Waal M.Sc. Christèle Borgeaud	37
Colobus guereza	Black-and-white colobus	Dunbar & Dunbar 1976	8
Erythrocebus patas	Patas monkeys	Nakagawa 1992	8
Erythrocebus patas	Patas monkeys	Kaplan & Zucker 1980	19

Macaca arctoides	Stump-tailed macaque	Estrada et al. 1977	19
Macaca assamensis	Assamese macaques	Cooper et al. 2005	19
Macaca fuscata	Japanese macaque	Takahashi & Furuichi 1998	21
Macaca fuscata	Japanese macaque	Dr. Andrew MacIntosh	21
Macaca fuscata	Japanese macaque	Dr. Andrew MacIntosh	31
Macaca mullata	Rhesus macaque	Sade 1972	16
Macaca radiata	Bonnet macaque	Sugiyama 1971	16
Macaca radiata	Bonnet macaque	Koyama 1973	23
Macaca tonkeana	Tonkean macaque	Nunn et al. 2015	25
Pan paniscus	Bonobo	Nunn et al. 2015	19
Pan troglodytes	Chimpanzee	Sugiyama & Koman 1979	12
Pan troglodytes	Chimpanzee	Sugiyama 1969	14
Pan troglodytes	Chimpanzee	Nunn et al. 2015	24
Papio ursinus	Chacma baboon	Dr. Andrew King	15
Saguinus mystax	Moustached tamarins	Nunn et al. 2015	6
Sapajus apella	Tufted capuchin	Izawa 1980	5
Sapajus apella	Tufted capuchin	Dr. Barbara Tiddi Dr. Eugenia P. di Sorrentino	8
Sapajus apella	Tufted capuchin	Dr. Barbara Tiddi Dr. Eugenia P. di Sorrentino	11
Sapajus apella	Tufted capuchin	Izawa 1980	12
Sapajus apella	Tufted capuchin	Dr. Barbara Tiddi Dr. Eugenia P. di Sorrentino	13
Sapajus libidinosus	Black-striped capuchin	Dr. Patrícia Izar	10
Sapajus nigritus	Black-horned capuchin	Dr. Patrícia Izar	7
Sapajus nigritus	Black-horned capuchin	Dr. Patrícia Izar	10
Trachypithecus johnii	Nilgiri langur	Poirier 1969	10

* Literature used to extract the dataset:

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List GS1. Candidate models for the spread of a moderately contagious pathogen (N= 40). Term codes are the same to all stages of the outbreak.

```
Term codes:
scale(-ccoef)     1
scale(cent)       2
scale(dia)        3
scale(gsize)      4
scale(mod)        5
scale(-ccoef):scale(gsize)     6
scale(cent):scale(gsize)       7
scale(dia):scale(gsize)        8
scale(gsize):scale(mod)        9
```

Stage 1

Component models:

	df	logLik	AICc	delta	weight
247	7	29.21	-40.93	0.00	0.14
1247	8	30.54	-40.44	0.49	0.11
2457	8	30.28	-39.91	1.01	0.09
459	7	28.70	-39.90	1.03	0.09
2347	8	29.58	-38.51	2.42	0.04
45	6	26.52	-38.50	2.43	0.04
24579	9	31.19	-38.37	2.56	0.04
12457	9	31.12	-38.23	2.69	0.04
1459	8	29.27	-37.89	3.04	0.03
3459	8	28.92	-37.19	3.74	0.02
12467	9	30.56	-37.11	3.82	0.02
12347	9	30.54	-37.09	3.84	0.02
2459	8	28.83	-37.02	3.91	0.02
23457	9	30.45	-36.91	4.02	0.02
146	7	27.19	-36.89	4.04	0.02
124579	10	32.21	-36.83	4.10	0.02
145	7	27.06	-36.62	4.31	0.02
14	6	25.40	-36.26	4.67	0.01
1456	8	28.41	-36.17	4.75	0.01
4	5	23.91	-36.05	4.88	0.01
345	7	26.68	-35.87	5.06	0.01
12459	9	29.78	-35.56	5.37	0.01
245	7	26.52	-35.54	5.39	0.01
34589	9	29.76	-35.52	5.40	0.01
23478	9	29.74	-35.47	5.46	0.01
234579	10	31.46	-35.33	5.59	0.01
2345789	11	33.24	-35.05	5.88	0.01
34	6	24.75	-34.96	5.96	0.01
124567	10	31.14	-34.69	6.24	0.01
123457	10	31.12	-34.65	6.28	0.01
14569	9	29.30	-34.60	6.33	0.01
13459	9	29.27	-34.54	6.39	0.01
23459	9	29.26	-34.52	6.41	0.01
1245679	11	32.85	-34.27	6.66	0.01
1346	8	27.39	-34.13	6.80	0.00
1246	8	27.35	-34.05	6.87	0.00
24	6	24.09	-33.63	7.30	0.00
234578	10	30.56	-33.53	7.40	0.00
123467	10	30.56	-33.53	7.40	0.00
123478	10	30.55	-33.51	7.42	0.00
1345	8	27.06	-33.48	7.45	0.00
12456	9	28.74	-33.47	7.46	0.00
134	7	25.46	-33.41	7.52	0.00
234589	10	30.35	-33.12	7.81	0.00
1234579	11	32.21	-32.99	7.94	0.00
3458	8	26.81	-32.97	7.96	0.00
13456	9	28.48	-32.97	7.96	0.00
2345	8	26.80	-32.95	7.98	0.00
348	7	24.87	-32.24	8.68	0.00

123459	10	29.82	-32.06	8.87	0.00
134589	10	29.81	-32.03	8.90	0.00
234	7	24.75	-32.01	8.92	0.00
124569	10	29.80	-32.01	8.92	0.00
12346	9	27.64	-31.28	9.65	0.00
12345789	12	33.36	-31.17	9.76	0.00
13468	9	27.55	-31.11	9.82	0.00
134569	10	29.31	-31.04	9.89	0.00

Stage 2

Component models:

	df	logLik	AICc	delta	weight
124579	10	-14.84	57.27	0.00	0.23
1245679	11	-12.99	57.41	0.15	0.22
12457	9	-17.12	58.24	0.98	0.14
1234579	11	-14.22	59.86	2.59	0.06
234579	10	-16.73	61.05	3.79	0.03
123457	10	-16.74	61.06	3.80	0.03
12345679	12	-12.88	61.31	4.05	0.03
12459	9	-18.76	61.52	4.25	0.03
23457	9	-18.89	61.78	4.51	0.02
124567	10	-17.11	61.82	4.55	0.02
1459	8	-20.88	62.40	5.14	0.02
1234578	11	-15.54	62.50	5.24	0.02
2457	8	-20.96	62.57	5.30	0.02
23459	9	-19.33	62.67	5.40	0.02
24579	9	-19.46	62.92	5.65	0.01
123459	10	-17.75	63.08	5.81	0.01
13459	9	-19.56	63.13	5.86	0.01
12345789	12	-14.21	63.98	6.71	0.01
2345789	11	-16.34	64.12	6.85	0.01
1234567	11	-16.65	64.73	7.47	0.01
234578	10	-18.67	64.93	7.67	0.01
3459	8	-22.15	64.94	7.67	0.01
124569	10	-18.76	65.10	7.83	0.00
123456789	13	-12.66	65.32	8.05	0.00
234589	10	-19.05	65.70	8.43	0.00
14569	9	-20.86	65.72	8.46	0.00
12345678	12	-15.25	66.05	8.78	0.00
2459	8	-22.75	66.14	8.88	0.00
12456	9	-21.14	66.27	9.01	0.00
134589	10	-19.53	66.65	9.39	0.00
134569	10	-19.54	66.66	9.40	0.00
1234569	11	-17.73	66.90	9.63	0.00
1234589	11	-17.74	66.91	9.64	0.00
123458	10	-19.79	67.16	9.90	0.00

Stage 3

Component models:

	df	logLik	AICc	delta	weight
459	7	-111.74	240.98	0.00	0.28
1459	8	-111.41	243.46	2.48	0.08
2459	8	-111.45	243.54	2.57	0.08
1348	8	-111.48	243.60	2.62	0.08
3459	8	-111.68	244.00	3.03	0.06
348	7	-113.52	244.54	3.56	0.05
3458	8	-112.44	245.52	4.54	0.03
34589	9	-110.83	245.66	4.68	0.03
13458	9	-110.90	245.80	4.82	0.03
2348	8	-112.59	245.82	4.84	0.02
24579	9	-110.93	245.86	4.89	0.02
12348	9	-111.17	246.35	5.37	0.02
12459	9	-111.31	246.63	5.65	0.02
14569	9	-111.34	246.67	5.69	0.02
13459	9	-111.40	246.81	5.83	0.02
23459	9	-111.45	246.90	5.92	0.01
146	7	-114.72	246.95	5.97	0.01

13468	9	-111.47	246.95	5.97	0.01
4	5	-117.80	247.37	6.39	0.01
23478	9	-111.69	247.38	6.40	0.01
134589	10	-109.92	247.42	6.44	0.01
23458	9	-111.77	247.54	6.56	0.01
123478	10	-110.39	248.36	7.38	0.01
234589	10	-110.40	248.39	7.41	0.01
24	6	-116.94	248.44	7.46	0.01
45	6	-117.11	248.77	7.80	0.01
123458	10	-110.69	248.96	7.98	0.01
124579	10	-110.73	249.06	8.08	0.00
14	6	-117.29	249.13	8.15	0.00
134568	10	-110.86	249.31	8.33	0.00
1246	8	-114.35	249.35	8.38	0.00
234579	10	-110.89	249.37	8.39	0.00
34	6	-117.45	249.45	8.48	0.00
234578	10	-111.04	249.67	8.69	0.00
1456	8	-114.55	249.75	8.77	0.00
123468	10	-111.17	249.93	8.95	0.00
1346	8	-114.68	250.00	9.02	0.00
124569	10	-111.22	250.02	9.04	0.00
12467	9	-113.05	250.11	9.13	0.00
123459	10	-111.30	250.19	9.21	0.00
134569	10	-111.34	250.26	9.28	0.00
2345789	11	-109.47	250.37	9.39	0.00
1234578	11	-109.67	250.77	9.79	0.00
1345689	11	-109.69	250.81	9.83	0.00
245	7	-116.73	250.95	9.98	0.00
1234589	11	-109.77	250.97	9.99	0.00

Stage 4

Component models:

	df	logLik	AICc	delta	weight
459	7	-158.09	333.69	0.00	0.21
34	6	-160.20	334.95	1.26	0.11
247	7	-158.89	335.28	1.59	0.10
245	7	-159.11	335.72	2.03	0.08
124	7	-159.11	335.72	2.03	0.08
234	7	-159.33	336.17	2.48	0.06
1459	8	-158.09	336.83	3.14	0.04
1247	8	-158.22	337.09	3.40	0.04
2347	8	-158.50	337.65	3.96	0.03
24579	9	-156.83	337.66	3.97	0.03
2457	8	-158.51	337.67	3.99	0.03
1245	8	-158.66	337.97	4.29	0.02
2345	8	-158.80	338.24	4.56	0.02
23478	9	-157.19	338.37	4.68	0.02
1234	8	-159.00	338.65	4.96	0.02
1246	8	-159.11	338.87	5.18	0.02
12467	9	-157.66	339.32	5.63	0.01
12457	9	-158.05	340.10	6.41	0.01
12347	9	-158.17	340.34	6.65	0.01
23458	9	-158.24	340.48	6.79	0.01
124579	10	-156.46	340.50	6.81	0.01
23457	9	-158.25	340.51	6.82	0.01
234579	10	-156.70	340.99	7.31	0.01
12345	9	-158.59	341.19	7.50	0.00
12456	9	-158.64	341.28	7.60	0.00
234578	10	-157.02	341.62	7.94	0.00
123478	10	-157.15	341.89	8.20	0.00
12346	9	-159.00	342.00	8.31	0.00
14569	9	-159.02	342.04	8.35	0.00
124567	10	-157.48	342.55	8.87	0.00
124569	10	-157.52	342.62	8.94	0.00
(Null)	4	-166.84	342.81	9.13	0.00
123467	10	-157.66	342.90	9.21	0.00
5	5	-165.61	342.98	9.30	0.00
34589	9	-159.51	343.02	9.34	0.00
3	5	-165.81	343.38	9.70	0.00

123457	10	-158.01	343.61	9.92	0.00

Stage 5

Component models:

	df	logLik	AICc	delta	weight
345	7	-142.67	302.84	0.00	0.18
34	6	-144.92	304.38	1.54	0.08
4	5	-146.61	304.98	2.14	0.06
3458	8	-142.29	305.22	2.38	0.05
2345	8	-142.57	305.79	2.95	0.04
1345	8	-142.66	305.97	3.13	0.04
3459	8	-142.67	305.98	3.14	0.04
45	6	-145.84	306.23	3.39	0.03
35	6	-145.99	306.53	3.69	0.03
348	7	-144.61	306.73	3.89	0.03
14	6	-146.27	307.09	4.25	0.02
145	7	-144.80	307.10	4.26	0.02
24	6	-146.29	307.14	4.30	0.02
(Null)	4	-149.03	307.19	4.35	0.02
134	7	-144.85	307.20	4.36	0.02
234	7	-144.92	307.34	4.50	0.02
245	7	-145.06	307.63	4.79	0.02
5	5	-147.94	307.64	4.80	0.02
25	6	-146.64	307.83	4.99	0.01
34589	9	-142.03	308.05	5.21	0.01
23458	9	-142.17	308.34	5.50	0.01
235	7	-145.43	308.36	5.52	0.01
13458	9	-142.19	308.37	5.53	0.01
13456	9	-142.20	308.40	5.56	0.01
15	6	-146.97	308.48	5.64	0.01
3	5	-148.37	308.51	5.67	0.01
23457	9	-142.35	308.71	5.87	0.01
2347	8	-144.11	308.87	6.03	0.01
23459	9	-142.57	309.14	6.30	0.01
12345	9	-142.57	309.14	6.30	0.01
247	7	-145.83	309.16	6.32	0.01
459	7	-145.84	309.18	6.34	0.01
135	7	-145.89	309.27	6.43	0.01
2	5	-148.76	309.28	6.44	0.01
13459	9	-142.66	309.32	6.48	0.01
1	5	-148.78	309.33	6.49	0.01
1245	8	-144.49	309.62	6.78	0.01
124	7	-146.15	309.80	6.96	0.01
1348	8	-144.61	309.86	7.02	0.01
2348	8	-144.61	309.87	7.03	0.01
146	7	-146.27	310.04	7.20	0.00
1456	8	-144.70	310.05	7.21	0.00
125	7	-146.29	310.09	7.25	0.00
1346	8	-144.76	310.16	7.32	0.00
1459	8	-144.80	310.25	7.41	0.00
1234	8	-144.84	310.33	7.49	0.00
23478	9	-143.24	310.47	7.63	0.00
2457	8	-144.95	310.55	7.71	0.00
2459	8	-145.06	310.76	7.92	0.00
234578	10	-141.62	310.82	7.98	0.00
134569	10	-141.74	311.06	8.22	0.00
134589	10	-141.76	311.10	8.26	0.00
23	6	-148.33	311.21	8.37	0.00
234589	10	-141.83	311.24	8.40	0.00
13	6	-148.37	311.29	8.45	0.00
1235	8	-145.42	311.49	8.65	0.00
123458	10	-142.10	311.79	8.95	0.00
134568	10	-142.10	311.80	8.96	0.00
123456	10	-142.13	311.85	9.01	0.00
1247	8	-145.62	311.90	9.06	0.00
12	6	-148.68	311.90	9.06	0.00
12347	9	-144.04	312.08	9.24	0.00
234579	10	-142.31	312.20	9.36	0.00
123457	10	-142.35	312.29	9.45	0.00

```
123459 10 -142.57 312.72  9.88   0.00
12457   9 -144.38 312.75  9.91   0.00
12456   9 -144.40 312.80  9.96   0.00
```

List GS2. Candidate models for the spread of a highly contagious pathogen (N= 40). Term codes are the same to all stages of the outbreak.

```
Term codes:
scale(-ccoef)     1
scale(cent)       2
scale(dia)        3
scale(gsize)      4
scale(mod)        5
scale(-ccoef):scale(gsize)     6
scale(cent):scale(gsize)       7
scale(dia):scale(gsize)        8
scale(gsize):scale(mod)        9
```

Stage 1

```
Component models:
         df logLik   AICc delta weight
1459      8 -70.17 160.98  0.00   0.24
12459     9 -69.12 162.24  1.26   0.13
2459      8 -71.19 163.03  2.05   0.09
134589   10 -67.77 163.13  2.15   0.08
13459     9 -70.05 164.11  3.13   0.05
14569     9 -70.17 164.34  3.35   0.05
1234589  11 -66.55 164.54  3.56   0.04
13458     9 -70.36 164.72  3.74   0.04
1345689  11 -67.06 165.55  4.57   0.02
124579   10 -68.98 165.55  4.57   0.02
23459     9 -70.80 165.61  4.63   0.02
123458   10 -69.06 165.71  4.73   0.02
124569   10 -69.09 165.77  4.79   0.02
123459   10 -69.11 165.80  4.82   0.02
3459      8 -72.71 166.07  5.08   0.02
24579     9 -71.14 166.29  5.31   0.02
459       7 -74.47 166.45  5.47   0.02
234589   10 -69.82 167.22  6.24   0.01
12345689 12 -65.94 167.44  6.46   0.01
134568   10 -69.95 167.49  6.51   0.01
12345789 12 -65.99 167.53  6.55   0.01
134569   10 -70.05 167.69  6.70   0.01
34589     9 -72.24 168.48  7.50   0.01
1234578  11 -68.63 168.69  7.71   0.01
234579   10 -70.67 168.92  7.94   0.00
1245679  11 -68.80 169.03  8.05   0.00
12348     9 -72.59 169.19  8.21   0.00
1234579  11 -68.95 169.33  8.35   0.00
1348      8 -74.37 169.39  8.41   0.00
1234569  11 -69.07 169.58  8.59   0.00
2345789  11 -69.55 170.52  9.54   0.00
```

Stage 2

```
Component models:
        df  logLik   AICc delta weight
459      7 -130.69 278.88  0.00   0.19
348      7 -130.96 279.42  0.54   0.15
1348     8 -130.17 280.99  2.11   0.07
23478    9 -128.77 281.54  2.66   0.05
2348     8 -130.46 281.57  2.69   0.05
4        5 -135.01 281.79  2.90   0.05
2459     8 -130.60 281.85  2.96   0.04
```

1459	8	-130.67	281.99	3.11	0.04
3459	8	-130.69	282.03	3.14	0.04
3458	8	-130.90	282.45	3.57	0.03
24579	9	-129.62	283.25	4.36	0.02
34589	9	-129.77	283.54	4.65	0.02
12348	9	-129.98	283.95	5.07	0.02
13468	9	-130.00	284.01	5.12	0.01
123478	10	-128.24	284.07	5.19	0.01
146	7	-133.29	284.08	5.20	0.01
24	6	-134.78	284.10	5.22	0.01
13458	9	-130.17	284.34	5.46	0.01
14	6	-135.00	284.56	5.67	0.01
45	6	-135.01	284.56	5.68	0.01
34	6	-135.01	284.57	5.68	0.01
23458	9	-130.46	284.91	6.03	0.01
234578	10	-128.73	285.05	6.17	0.01
23459	9	-130.58	285.15	6.27	0.01
12459	9	-130.60	285.20	6.32	0.01
14569	9	-130.64	285.27	6.39	0.01
13459	9	-130.66	285.33	6.44	0.01
247	7	-134.37	286.23	7.35	0.00
134589	10	-129.42	286.42	7.54	0.00
12467	9	-131.25	286.49	7.61	0.00
234589	10	-129.52	286.62	7.74	0.00
234579	10	-129.62	286.82	7.94	0.00
124579	10	-129.62	286.83	7.95	0.00
1246	8	-133.12	286.88	8.00	0.00
245	7	-134.74	286.97	8.09	0.00
234	7	-134.74	286.99	8.10	0.00
2345789	11	-127.78	286.99	8.11	0.00
124	7	-134.75	286.99	8.11	0.00
1456	8	-133.25	287.14	8.26	0.00
123468	10	-129.79	287.17	8.29	0.00
1346	8	-133.29	287.23	8.34	0.00
134568	10	-129.96	287.50	8.62	0.00
145	7	-135.00	287.51	8.62	0.00
134	7	-135.00	287.51	8.63	0.00
345	7	-135.01	287.51	8.63	0.00
123458	10	-129.97	287.54	8.65	0.00
1234678	11	-128.21	287.84	8.96	0.00
1234578	11	-128.24	287.90	9.02	0.00
124569	10	-130.55	288.69	9.81	0.00
123459	10	-130.57	288.73	9.85	0.00
134569	10	-130.63	288.85	9.97	0.00

Stage 3

Component models:

	df	logLik	AICc	delta	weight
24579	9	-148.34	320.69	0.00	0.21
459	7	-151.87	321.25	0.56	0.16
245	7	-152.52	322.54	1.85	0.08
247	7	-153.05	323.60	2.92	0.05
124579	10	-148.04	323.67	2.98	0.05
23478	9	-149.88	323.76	3.07	0.05
2457	8	-151.64	323.93	3.25	0.04
3459	8	-151.74	324.12	3.43	0.04
234579	10	-148.33	324.24	3.55	0.04
1459	8	-151.87	324.39	3.71	0.03
1245	8	-152.13	324.91	4.22	0.03
1247	8	-152.23	325.10	4.42	0.02
234578	10	-148.79	325.16	4.48	0.02
23459	9	-150.75	325.49	4.81	0.02
12467	9	-150.77	325.53	4.85	0.02
2345	8	-152.49	325.62	4.94	0.02
12457	9	-151.23	326.46	5.78	0.01
2347	8	-152.94	326.54	5.85	0.01
2345789	11	-147.72	326.86	6.17	0.01

124567	10	-149.73	327.05	6.37	0.01
1234579	11	-147.82	327.07	6.39	0.01
123478	10	-149.81	327.20	6.51	0.01
14569	9	-151.62	327.24	6.55	0.01
23457	9	-151.63	327.26	6.58	0.01
34589	9	-151.68	327.36	6.67	0.01
1245679	11	-148.03	327.49	6.81	0.01
12456	9	-151.96	327.92	7.23	0.01
12345	9	-152.11	328.21	7.53	0.00
123467	10	-150.37	328.33	7.65	0.00
12347	9	-152.20	328.39	7.71	0.00
1234578	11	-148.78	328.98	8.30	0.00
1234567	11	-149.24	329.92	9.23	0.00
123457	10	-151.17	329.93	9.24	0.00
1234678	11	-149.54	330.50	9.82	0.00
12345789	12	-147.54	330.64	9.95	0.00

Stage 4

Component models:

	df	logLik	AICc	delta	weight
247	7	-132.58	282.66	0.00	0.11
2347	8	-131.70	284.05	1.39	0.06
24	6	-134.77	284.08	1.42	0.06
4	5	-136.39	284.54	1.88	0.04
34	6	-135.18	284.91	2.25	0.04
1247	8	-132.16	284.97	2.31	0.04
2457	8	-132.18	285.00	2.33	0.03
14	6	-135.26	285.06	2.40	0.03
245	7	-133.81	285.13	2.47	0.03
2	5	-136.70	285.16	2.50	0.03
(Null)	4	-138.16	285.46	2.80	0.03
25	6	-135.47	285.48	2.82	0.03
23457	9	-130.88	285.76	3.10	0.02
146	7	-134.26	286.02	3.36	0.02
234	7	-134.32	286.15	3.49	0.02
345	7	-134.37	286.24	3.57	0.02
2345	8	-132.84	286.33	3.66	0.02
124	7	-134.45	286.39	3.73	0.02
1	5	-137.32	286.41	3.75	0.02
145	7	-134.48	286.45	3.79	0.02
1245	8	-133.09	286.83	4.17	0.01
12457	9	-131.49	286.99	4.33	0.01
45	6	-136.23	287.01	4.35	0.01
234579	10	-129.78	287.14	4.48	0.01
24579	9	-131.58	287.17	4.51	0.01
15	6	-136.33	287.21	4.55	0.01
125	7	-134.88	287.27	4.61	0.01
348	7	-134.89	287.28	4.62	0.01
12347	9	-131.67	287.33	4.67	0.01
134	7	-134.92	287.35	4.68	0.01
3	5	-137.79	287.35	4.69	0.01
23478	9	-131.68	287.36	4.70	0.01
1246	8	-133.36	287.37	4.71	0.01
5	5	-137.87	287.51	4.85	0.01
12	6	-136.52	287.58	4.92	0.01
235	7	-135.18	287.85	5.19	0.01
23	6	-136.67	287.89	5.23	0.01
1456	8	-133.81	288.28	5.61	0.01
2459	8	-133.81	288.28	5.61	0.01
12467	9	-132.16	288.31	5.65	0.01
1345	8	-133.86	288.36	5.70	0.01
35	6	-137.04	288.63	5.96	0.01
2348	8	-134.08	288.81	6.15	0.01
3458	8	-134.10	288.85	6.19	0.01
1346	8	-134.12	288.88	6.22	0.00
12456	9	-132.46	288.91	6.25	0.00
1234	8	-134.26	289.16	6.50	0.00
123457	10	-130.79	289.16	6.50	0.00
13	6	-137.32	289.18	6.52	0.00

23458	9	-132.65	289.29	6.63	0.00
12345	9	-132.65	289.30	6.64	0.00
234578	10	-130.87	289.32	6.66	0.00
3459	8	-134.35	289.35	6.69	0.00
1459	8	-134.48	289.60	6.94	0.00
23459	9	-132.83	289.66	7.00	0.00
124579	10	-131.04	289.67	7.01	0.00
135	7	-136.23	289.96	7.29	0.00
459	7	-136.23	289.96	7.29	0.00
1348	8	-134.77	290.18	7.52	0.00
12459	9	-133.09	290.19	7.52	0.00
14569	9	-133.10	290.20	7.53	0.00
1235	8	-134.84	290.33	7.66	0.00
2345789	11	-129.46	290.35	7.69	0.00
123	7	-136.52	290.53	7.87	0.00
124567	10	-131.49	290.57	7.91	0.00
12346	9	-133.31	290.63	7.97	0.00
123467	10	-131.61	290.81	8.15	0.00
123478	10	-131.62	290.83	8.17	0.00
13456	9	-133.46	290.92	8.26	0.00
1234579	11	-129.76	290.95	8.29	0.00
124569	10	-131.76	291.12	8.45	0.00
34589	9	-133.70	291.39	8.73	0.00
13458	9	-133.77	291.54	8.88	0.00
13459	9	-133.85	291.69	9.03	0.00
13468	9	-134.01	292.02	9.36	0.00
123456	10	-132.24	292.06	9.39	0.00
12348	9	-134.07	292.14	9.48	0.00
234589	10	-132.35	292.29	9.62	0.00
1234567	11	-130.61	292.65	9.99	0.00

Stage 5

Component models:

	df	logLik	AICc	delta	weight
4	5	-102.04	215.84	0.00	0.15
24	6	-101.05	216.65	0.82	0.10
14	6	-101.17	216.89	1.05	0.09
34	6	-101.30	217.14	1.30	0.08
348	7	-99.94	217.38	1.55	0.07
45	6	-101.70	217.95	2.11	0.05
146	7	-100.43	218.36	2.52	0.04
459	7	-100.67	218.83	3.00	0.03
124	7	-100.75	219.00	3.16	0.03
234	7	-100.82	219.13	3.30	0.03
245	7	-100.96	219.42	3.59	0.02
134	7	-101.03	219.57	3.73	0.02
247	7	-101.04	219.57	3.74	0.02
145	7	-101.11	219.72	3.88	0.02
2348	8	-99.55	219.74	3.90	0.02
345	7	-101.20	219.89	4.06	0.02
2459	8	-99.77	220.19	4.35	0.02
3458	8	-99.83	220.31	4.48	0.02
1348	8	-99.92	220.49	4.66	0.01
1246	8	-99.96	220.57	4.73	0.01
1459	8	-100.13	220.90	5.06	0.01
3459	8	-100.23	221.11	5.28	0.01
1456	8	-100.26	221.16	5.33	0.01
1346	8	-100.38	221.41	5.57	0.01
1234	8	-100.70	222.04	6.20	0.01
1247	8	-100.72	222.08	6.24	0.01
1245	8	-100.73	222.10	6.26	0.01
2347	8	-100.77	222.19	6.36	0.01
2345	8	-100.78	222.20	6.37	0.01
2457	8	-100.89	222.43	6.59	0.01
1345	8	-101.00	222.64	6.80	0.00
23458	9	-99.50	223.00	7.16	0.00
23478	9	-99.51	223.02	7.18	0.00

```
12348  9  -99.55 223.09  7.25   0.00
12459  9  -99.61 223.22  7.38   0.00
23459  9  -99.66 223.33  7.49   0.00
24579  9  -99.75 223.50  7.67   0.00
34589  9  -99.76 223.52  7.68   0.00
12467  9  -99.77 223.54  7.70   0.00
13458  9  -99.83 223.66  7.83   0.00
12456  9  -99.86 223.72  7.89   0.00
13468  9  -99.91 223.83  7.99   0.00
12346  9  -99.96 223.92  8.08   0.00
14569  9 -100.04 224.07  8.24   0.00
13459  9 -100.04 224.08  8.24   0.00
13456  9 -100.24 224.47  8.64   0.00
12347  9 -100.65 225.30  9.47   0.00
12457  9 -100.66 225.33  9.49   0.00
12345  9 -100.68 225.36  9.53   0.00
23457  9 -100.70 225.39  9.56   0.00
```

APPENDIX H: SUPPLEMENTARY MATERIAL FOR CHAPTER 3

H.1: Code to create truncated networks and to estimate global network metrics in R v. 3.0.1

```
library(igraph)
library(erer)
library(brainwaver)

ctr <- function(A){
        # formula eigenvector centralization
    ls<-apply(A,1,sum)
    cmax<-max(ls)
    n<-length(ls)
    C<-sum(cmax-ls)/((n-1)*(n-2))
    C
    }

prop.mySF <- function(n,power=2){
  # a list shows all the properties and matrix etc. for n with m going from 1 to n-1
            ls<-create.netSF(n,power=1)
            mylist<-list()
            length(mylist)<-n-1
            for (i in 1:(n-1)) {
mylist[[i]]<-data.frame(
"Global.Efficiency"=round(2*n/(sum(as_adjacency_matrix(ls[[i]],sparse=F))*diameter(ls[[
i]])),3)
"Group.Size"=n,
"Density"=round(graph.density(ls[[i]]),1),
            "Centralization"=round(ctr(as_adjacency_matrix(ls[[i]],sparse=F)),1),
"Modularity"=round(cluster_leading_eigen(ls[[i]],membership=
membership(ls[[i]],sparse=F)),1),
"Adjacency.Matrix"=as_adjacency_matrix(ls[[i]],sparse=F))
                    }
mylist
            }

create.netSF<-function(n,power=2){
            # create n-1 truncated networks
            mv<-c(1:(n-1))  #vector of m for a group size n
            list.graph<-list()
            length(list.graph)<-(n-1)
            for (i in 1:(n-1)) {
            list.graph[[i]]<-sample_pa(n,power,m=mv[i],directed=F)
            # create n-1 truncated networks for n nodes, m from 1 to n-1
```

```
                }
        list.graph
        }

test<-prop.mySF(8,power = 2) # a list of n = 8, m = 1:n-1
# a list combining the metrics estimated for n and m from 1 to n-1 of random networks
write.list(test,file="name.csv",row.names = FALSE)
#write the list to a csv file named      "name.csv"
```

Table HS1. Published empirical data on global efficiency and modularity originally measured for 68 primate social groups of 21 species extracted from the Supplementary Material of Pasquareta et al. 2014*.

Species	Family	Interaction	Origin	Group size	Modularity	Global Efficiency
Alouatta palliata	Aotidae	Proximities	Wild	17	0.254	0.1
Cebus capucinus	Cebidae	Body contacts	Wild	7	0.214	0.1842
Cebus capucinus	Cebidae	Body contacts	Wild	12	0.415	0.25
Cebus capucinus	Cebidae	Body contacts	Wild	14	0.489	0.1591
Cebus capucinus	Cebidae	Body contacts	Wild	13	0.357	0.2063
Cebus capucinus	Cebidae	Body contacts	Wild	5	0.208	0.5
Cebus capucinus	Cebidae	Body contacts	Wild	6	0.197	0.4
Cebus capucinus	Cebidae	Body contacts	Wild	10	0.31	0.1136
Saimiri sciureus	Cebidae	Proximities	Captive	8	0.499	0.1667
Saimiri sciureus	Cebidae	Proximities	Captive	10	0.603	0.1389
Saimiri sciureus	Cebidae	Body contacts	Captive	12	0.339	0.1818
Saimiri sciureus	Cebidae	Proximities	Captive	9	0.748	0.1406
Sapajus apella	Cebidae	Body contacts	Wild	11	0.382	0.1111
Sapajus apella	Cebidae	Body contacts	Wild	10	0.001	0.1333
Sapajus apella	Cebidae	Body contacts	Wild	10	0.41	0.1351
Sapajus apella	Cebidae	Body contacts	Wild	8	0.412	0.2051
Sapajus apella	Cebidae	Body contacts	Wild	12	0.362	0.1818
Sapajus apella	Cebidae	Body contacts	Captive	7	0.237	0.3333
Sapajus apella	Cebidae	Body contacts	Wild	5	0.362	0.2778
Sapajus apella	Cebidae	Body contacts	Wild	8	0.491	0.2353
Cercopithecus diana	Cercopithecidae	Body contacts	Captive	7	0.373	0.1842

Cercopithecus mitis	Cercopithecidae	Body contacts	Wild	17	0.197	0.0833
Chlorocebus pygerythrus	Cercopithecidae	Body contacts	Wild	38	0.195	0.0413
Chlorocebus pygerythrus	Cercopithecidae	Body contacts	Wild	25	0.189	0.0576
Chlorocebus pygerythrus	Cercopithecidae	Body contacts	Wild	26	0.178	0.0335
Erythrocebus patas	Cercopithecidae	Body contacts	Wild	8	0.372	0.2857
Macaca arctoides	Cercopithecidae	Body contacts	Wild	21	0.295	0.1313
Macaca arctoides	Cercopithecidae	Body contacts	Captive	14	0.22	0.0778
Macaca fuscata	Cercopithecidae	Body contacts	Wild	25	0.227	0.0504
Macaca fuscata	Cercopithecidae	Body contacts	Wild	31	0.392	0.0662
Macaca fuscata	Cercopithecidae	Proximities	Captive	14	0.322	0.0787
Macaca fuscata	Cercopithecidae	Body contacts	Wild	21	0.489	0.1094
Macaca fuscata	Cercopithecidae	Body contacts	Captive	22	0.435	0.088
Macaca mulatta	Cercopithecidae	Body contacts	Captive	9	0.251	0.1324
Macaca mulatta	Cercopithecidae	Body contacts	Captive	10	0.399	0.1563
Macaca radiata	Cercopithecidae	Body contacts	Wild	23	0.228	0.071
Macaca tonkeana	Cercopithecidae	Body contacts	Captive	10	0.256	0.1351
Macaca tonkeana	Cercopithecidae	Body contacts	Captive	25	0.255	0.0541
Macaca tonkeana	Cercopithecidae	Body contacts	Captive	18	0.208	0.06
Macaca tonkeana	Cercopithecidae	Body contacts	Captive	10	0.212	0.2222
Mandrillus sphinx	Cercopithecidae	Body contacts	Captive	18	0.367	0.15
Homo sapiens	Hominidae	Proximities	NA	22	0.483	0.1128
Homo sapiens	Hominidae	Proximities	NA	34	0.44	0.1236
Homo sapiens	Hominidae	Proximities	NA	29	0.185	0.0604
Homo sapiens	Hominidae	Proximities	NA	11	0.293	0.1196
Pan paniscus	Hominidae	Proximities	Captive	5	0.066	0.2778
Pan troglodytes	Hominidae	Body contacts	Wild	12	0.331	0.12
Pan troglodytes	Hominidae	Body contacts	Captive	9	0.174	0.1452
Pan troglodytes	Hominidae	Body contacts	Captive	5	0.19	0.5
Pan troglodytes	Hominidae	Body contacts	Captive	11	0.134	0.2
Pan troglodytes	Hominidae	Proximities	Captive	7	0.204	0.1842
Pan troglodytes	Hominidae	Proximities	Captive	7	0.191	0.3333

Pan troglodytes	Hominidae	Body contacts	Captive	9	0.176	0.5
Pan troglodytes	Hominidae	Proximities	Captive	6	0.255	0.2727
Pan troglodytes	Hominidae	Proximities	Captive	6	0.195	0.4
Pan troglodytes	Hominidae	Proximities	Captive	8	0.145	0.1538
Pan troglodytes	Hominidae	Body contacts	Captive	8	0.153	0.2857
Pan troglodytes	Hominidae	Body contacts	Captive	10	0.108	0.2222
Pan troglodytes	Hominidae	Proximities	Wild	21	0.366	0.0972
Pan troglodytes	Hominidae	Proximities	Captive	17	0.204	0.0634
Pongo pygmaeus	Hominidae	Proximities	Captive	5	0.535	0.2778
Eulemur catta	Lemuridae	Proximities	Wild	12	0.367	0.1091
Eulemur catta	Lemuridae	Proximities	Wild	12	0.427	0.1017
Eulemur catta	Lemuridae	Proximities	Wild	11	0.55	0.131
Eulemur fulvus	Lemuridae	Body contacts	Captive	11	0.463	0.1146
Eulemur rufifrons	Lemuridae	Proximities	Wild	12	0.312	0.1429
Eulemur rufifrons	Lemuridae	Proximities	Wild	11	0.367	0.1667
Eulemur rufifrons	Lemuridae	Proximities	Wild	14	0.441	0.1037

*Pasquareta et al. (2014). *Sci. Rep.* 4: 7600. (doi:10.1038/srep07600).

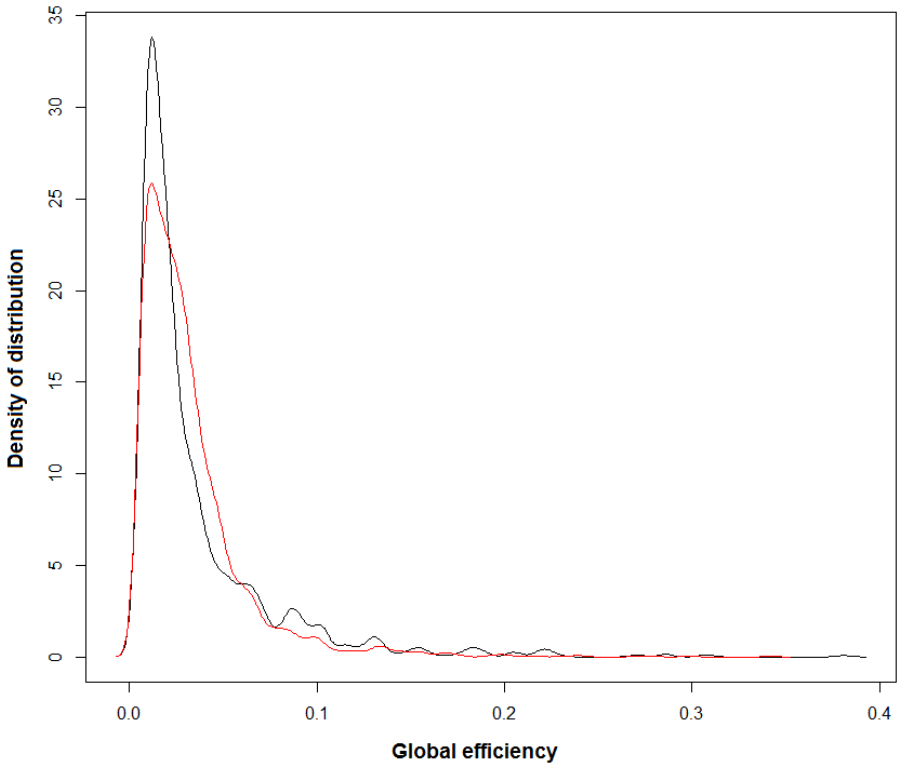

Figure HS1. Observed (black line) and gamma (red line) distribution functions of global efficiency. The distribution of our response variable (global efficiency) deviated from the Gaussian case and performed better as a gamma distribution.

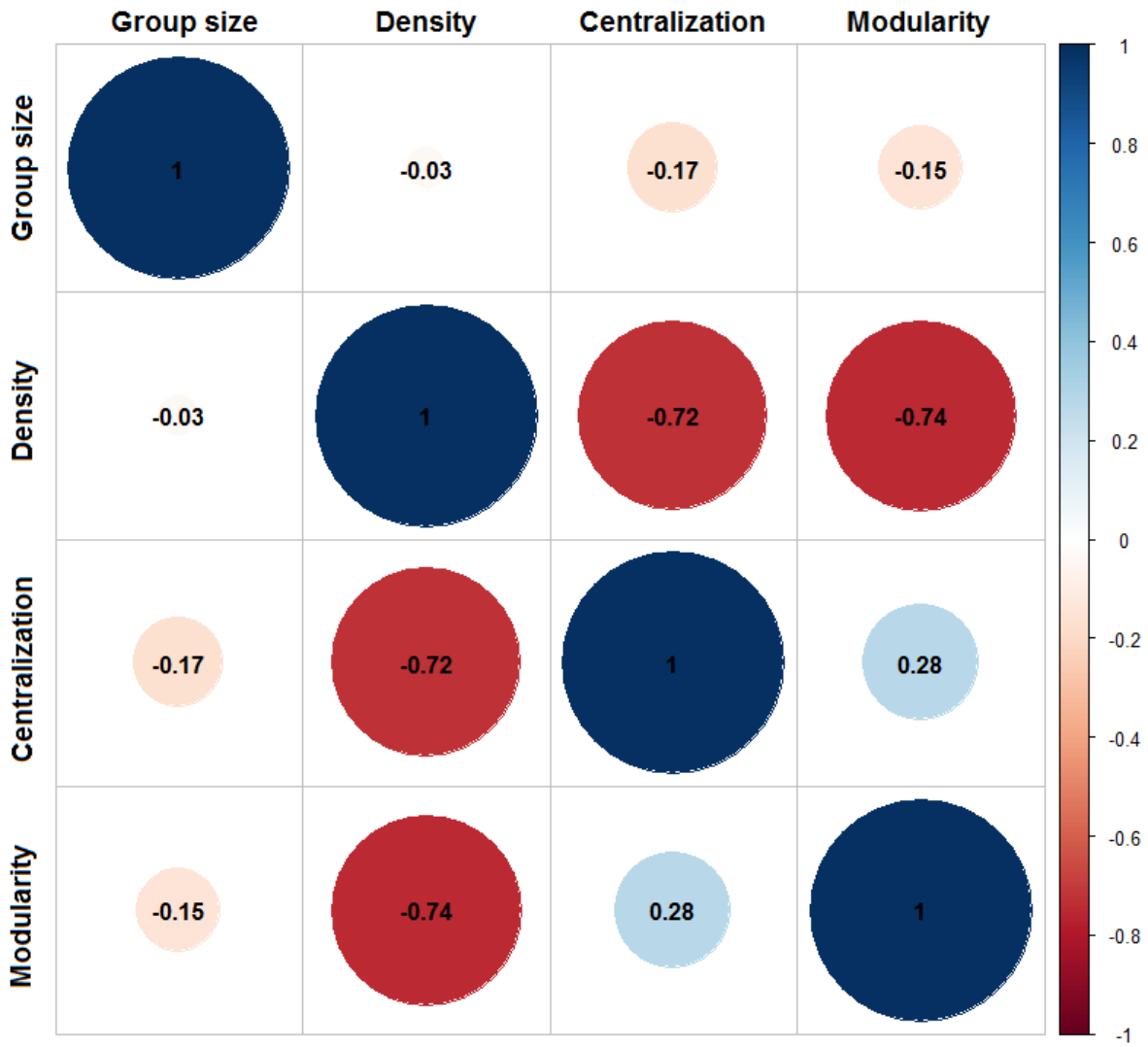

Figure HS2. Correlation matrix among network properties and group size. The stronger the coefficient of correlation, the darker the blue (positive correlation) and the darker the red (negative correlation).

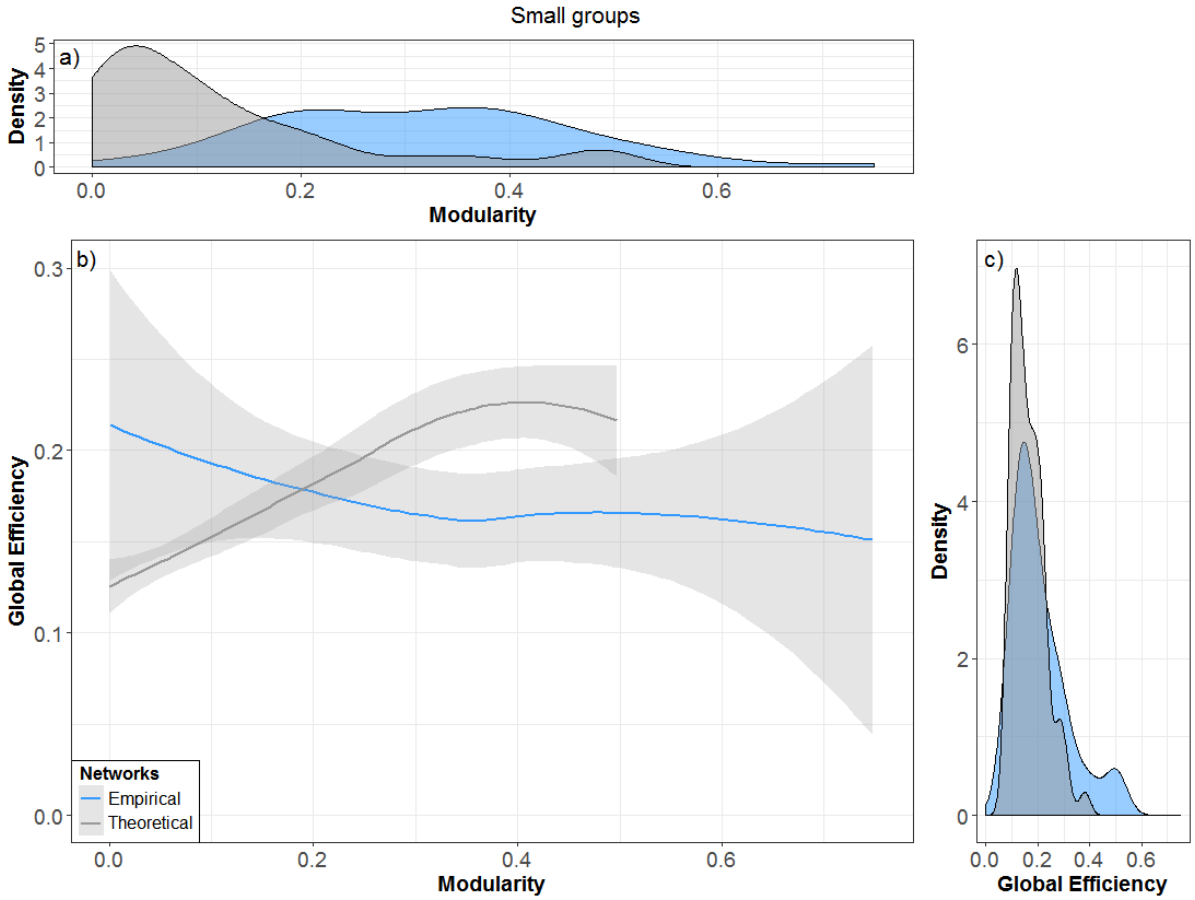

Figure HS3. Theoretical (shaded grey) and empirical (shaded blue) distribution functions of modularity (a) and global efficiency (c) for small-sized groups. The distribution of both variables differs between the theoretical and empirical networks (Modularity: D = 0.63, p = 1.09^{e-12}; Global efficiency: D = 0.24, p = 0.04). Figure b shows the relationship between modularity and global efficiency for theoretical and empirical networks. Our results suggest that the slope between them are statistically different (p = 1.47^{e-08}). Intercepts are at 0.21 in empirical and 0.15 in theoretical networks.

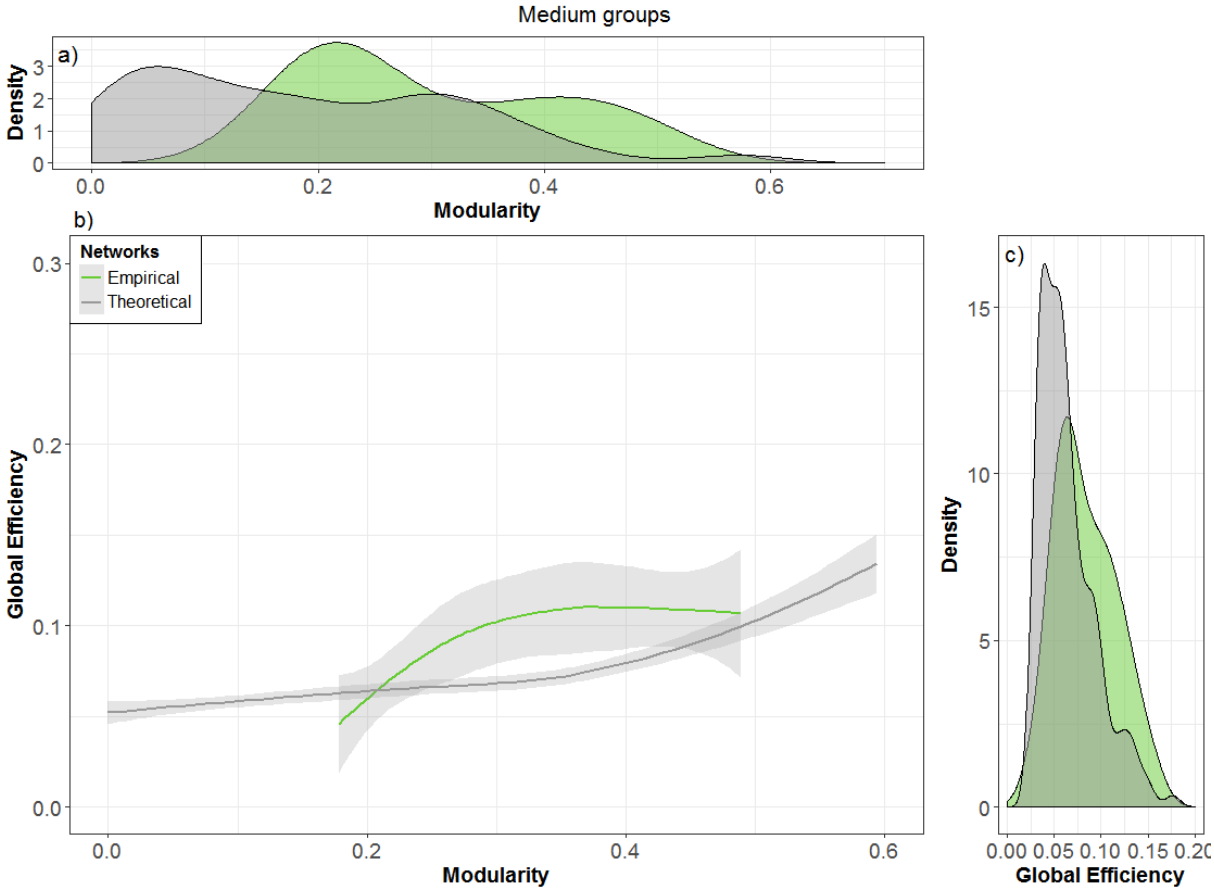

Figure HS4. Theoretical (shaded gray) and empirical (shaded green) distribution functions of modularity (a) and global efficiency (c) for medium-sized groups. The distribution of both variables differs between the theoretical and empirical networks (Modularity: D = 0.52, p = 0.0002; Global efficiency: D = 0.35, p = 0.03). Figure b shows the relationship between modularity and global efficiency for theoretical and empirical networks. Our results suggest that the slope between them are not statistically different (p = 0.10). Intercepts are at 0.08 in empirical and 0.05 in theoretical networks.

APPENDIX I: SUPPLEMENTARY MATERIAL FOR CHAPTER 4

I.1: The ODD PROTOCOL

The model description follows the ODD (Overview, Design concepts, Details) protocol for describing individual-based models (Grimm et al. 2006; Grimm et al. 2010).

Purpose

Our model, named the Optimal Relationship Model, aims to identify the type of social network structure that arises from relationships that maximize interactions with informed individuals and minimize interactions with infected individuals.

Entities, state variables, and scales

- *Agents/entities:* The model has one type of agent, called an individual. Individuals are provided with three state variables: 1) a unique *identification number* is provided to each individual and remains constant through time; 2) a value for *My-Information*, which is the probability of an interaction partner perceiving the agent as being informed during a social interaction; and, 3) a value for *My-pathogen*, which is the probability of an interaction partner perceiving the agent as being infected during a social interaction.

- *Environment:* The model has two global variables, called *social-increase* and *social-decrease*. These control the degree to which an individual either increases or decreases its interactions with a given partner following a 'positive' (information perceived) or 'negative' (pathogen perceived) interaction.

- *Temporal scale:* One simulation comprises 10000-time steps set arbitrarily to provide a large number of possible interactions among group members.

- *Collectives:* not applied.

- *Spatial units:* not applied.

Process overview and scheduling

Included in chapter 4.

Design concepts

Basic principles: Acquiring information or pathogens via social transmission may deeply affect an individual's fitness. In animal societies, the network properties optimizing the spreading of information should also increase the pathogen transmission rate, creating a trade-off between information transmission and infection risk. We aimed to explore this potential trade-off by examining social network properties and investigating which structure arise when individuals maximize contact with conspecifics providing high values of information but low values of pathogen.

Emergence: The structure of the network is dependent upon the perceptions that individuals have of other group members with which they have interacted, i.e. as informed or infected. As they interact with others, they increase their probabilities of interacting with individuals characterized by low cost-benefit ratios (more information than pathogens). Predictions for each condition can be found in the introduction of chapter 4.

Adaptation: Individuals will interact with those providing more information than pathogens. The more beneficial the relationship, the stronger the weight of the relationship becomes, and the higher the probability of interaction.

Objectives: Individuals create and reinforce social bonds with group members that provide them with a higher probability of acquiring information and a lower probability of acquiring pathogens.

Learning: There is not a learning function in the model. However, learning is implicit in the way that individuals update the weights of their relationships after each interaction. In our model, the terms 'learn' and 'perceive' can be used interchangeably, though we recognize that,

in reality, these are very different concepts.

Prediction: Individuals predict the outcome of future interactions based on their previous interactions with each individual.

Sensing: not applied.

Interaction: Individuals directly interact with each other, but the interaction does not change the status of an individual, i.e. its own values of *My-Information* and *My-Pathogen* do not change. At the end of each interaction, individual *I* simply updates the weight of its relationship with individual *j*.

Stochasticity: Several processes in the model are stochastic:

1. Probability of selecting a given interaction partner: after an individual is activated, it chooses an interaction partner. Which individual is chosen depends on the weights of the relationships between itself and all other group members. As the simulation progress, individuals with higher weights have higher chances of being selected as interaction partners.

2. Probability of perceiving group members as informed, infected, both or neither: after a social interaction, an individual perceives its interaction partner as being informed, infected, both or neither. The probability of perceiving any of these four possible outcomes is given by the value of *My-information* and *My-pathogen* of the interaction partner (see section 1 for more information). All probabilities add to 1.

Collectives: not applied.

Observation: From the model, we collected data on each individual's interaction partners, the values of *My-information* and *My-pathogen* of each individual, the types of interaction observed (if individual could perceive only information, only pathogen, both information and pathogen or neither), and the initial and updated values of the relationship weights for each dyad. At the end of each simulation, a csv.file, containing the records of these data is created.

Initialization

The relationships between all individuals are set with an initial weight, determined by the ratio derived from the maximum possible weight (1) divided by the number of individuals in the group (N) minus 1. Thus, at the beginning of the simulation, all individuals have the same probability of being selected as interaction partners. Values of *My-information* and *My-pathogen* set at the initialization settings do not vary among simulations.

Input data

Any data is necessary to be included prior to each simulation.

Sub-models

The model has no sub-models.

I.2. Source code of the Optimal Relationships Model

```
extensions [ nw
        matrix
        r
       ]

breed [ individuals individual ]

individuals-own [
  My-information
  My-pathogen   ]

directed-link-breed [ connections connection ]

connections-own [
  weight
           ]

globals[
  simulations
  Group-matrix
  IDActor
  IDReceiver
```

```
  Interaction
  Type-of-interaction
  Old-weight
  New-Weight
  social-increase
  social-decrease
        ]

;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;;;; SETUP PROCEDURES ;;;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

to setup
  clear-all
  ask patches [ set pcolor 92 ]
  set-default-shape individuals "circle"
  ; number of individuals in the group controlled by the slider group-size
  create-individuals group-size
  [
    ; sets initial position of individuals in the patch
    setxy (random-xcor * 0.50) (random-ycor * 0.50)
    ; links are created between individuals in the group
    create-connections-to other individuals
    set color blue
  ]
 ask individual 0 [set My-information 0 set My-pathogen 1]
 ask individual 1 [set My-information 0 set My-pathogen 0.34867844]
 ask individual 2    [set My-information 0 set My-pathogen 0.107374182]
 ask individual 3    [set My-information 0 set My-pathogen 0.028247525]
 ask individual 4    [set My-information 0 set My-pathogen 0.006046618]
 ask individual 5    [set My-information 0 set My-pathogen 0.000976563]
 ask individual 6    [set My-information 0 set My-pathogen 0.000104858]
 ask individual 7    [set My-information 0 set My-pathogen 0.0000059]
 ask individual 8    [set My-information 0 set My-pathogen 0.000000102]
 ask individual 9    [set My-information 0 set My-pathogen 0.0000000001]
; individuals have the same probability for interacting at the first-time activation.
ask connections [ set weight (1 / (group-size - 1)) ]
; creating a matrix to record the final number of interactions among individuals
set Group-matrix matrix:make-constant group-size group-size 0
set-default-shape connections "default"
data-collection-reset
reset-ticks
end

to setup2
  clear-ticks clear-turtles clear-patches clear-drawing clear-all-plots
  ask patches [ set pcolor 92 ]
  set-default-shape individuals "circle"
  create-individuals group-size
  [
```

```
    setxy (random-xcor * 0.50) (random-ycor * 0.50)
    create-connections-to other individuals
    set color blue

  ]
 ask individual 0    [set My-information 0 set My-pathogen 1]
 ask individual 1    [set My-information 0 set My-pathogen 0.34867844]
 ask individual 2    [set My-information 0 set My-pathogen 0.107374182]
 ask individual 3    [set My-information 0 set My-pathogen 0.028247525]
 ask individual 4    [set My-information 0 set My-pathogen 0.006046618]
 ask individual 5    [set My-information 0 set My-pathogen 0.000976563]
 ask individual 6    [set My-information 0 set My-pathogen 0.000104858]
 ask individual 7    [set My-information 0 set My-pathogen 0.0000059]
 ask individual 8    [set My-information 0 set My-pathogen 0.000000102]
 ask individual 9    [set My-information 0 set My-pathogen 0.0000000001]
 ask connections [ set weight (1 / (group-size - 1)) ]
 set Group-matrix matrix:make-constant group-size group-size 0
 set-default-shape connections "default"
 data-collection-reset
 reset-ticks
end


;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;
;; GO PROCEDURES ;;
;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;;

to go
  set simulations 0
  while [simulations < simulations-number]
       [
       data-collection-reset
       setup2
       let simulation-over FALSE
       create-title-data-collection
       while [simulation-over = FALSE] [
                                  interact
                                  tick-advance 1
                                  update-plots
                                  if ticks >= Max-number-interactions [set simulation-over TRUE]
                                  ]
  set simulations simulations + 1
  ]
end


to  create-title-data-collection
  let File_name word "Simulation" (word simulations ".csv")
  ;file-delete File_name
  file-open File_name
```

```
    file-type "IDActor"
    file-type ","
    file-type "IDReceiver"
    file-type ","
    file-type "Interaction"
    file-type ","
    file-type "Type-of-interaction"
    file-type ","
    file-type "Old-weight"
    file-type ","
    file-type "New-Weight"
    file-type ","
    file-type "My-information_Actor"
    file-type ","
    file-type "My-pathogen_Actor"
    file-type ","
    file-type "My-information_Receiver"
    file-type ","
    file-type "My-pathogen_Receiver"
    file-type ","
    file-print ""
    file-close
  end


  to interact
    ask individuals [ ;activate all individuals to interact at the same time step
    data-collection-reset
    let ME self ;to call individual that is activated
    let IDActiveAgent [who] of ME ;gets the identity of the Activated Individual
    let InteractionPartner individual (select-partner IDActiveAgent)
    let IDInteractionPartner [who] of InteractionPartner ;show the ID of interaction partner
    if IDInteractionPartner = -1 [ error "Interaction Partner = -1" ]
    set IDActor (IDActiveAgent + 1) ;provide identities to individuals starting from 1 and not from 0, as set in NetLogo. Fill the csv file.
    set IDReceiver (IDInteractionPartner + 1)
    set Old-weight [weight] of connection IDActiveAgent IDInteractionPartner
    matrix:set Group-matrix IDActiveAgent IDInteractionPartner (matrix:get Group-matrix IDActiveAgent IDInteractionPartner + 1)
    ;create a matrix that records the number of interactions
    set Interaction 1
    ;set the probability of outcome based on the values of My-information and My-pathogen.
    ;The higher the value of information, the higher the probability of interaction and the same for pathogen.
    let Pinf [My-information] of InteractionPartner * (1 - [My-pathogen] of InteractionPartner) ;Probability of acquiring only information
    let Ppat [My-pathogen] of InteractionPartner * (1 - [My-information] of InteractionPartner) ;Probability of acquiring only pathogen
    let Pinfpat [My-information] of InteractionPartner * [My-pathogen] of InteractionPartner ;Probability of acquiring information and pathogen
```

```
    let Pnone (1 - [My-information] of InteractionPartner) * (1 - [My-pathogen] of
InteractionPartner) ;Probability of acquiring nothing
    let Prob random-float 1
    ifelse Prob < Pinf [

   ;INDIVIDUAL GETS INFORMATION
                        set Type-of-interaction 1 ;to be recorded in the output file
                        ;output-print "Type-of-interaction" output-print Type-of-interaction
                        let delta 0 ;set the initial increment of weight = 0
                        set social-increase random-normal 0.20 0.05
                        set social-decrease random-normal 0.20 0.05
                        foreach n-values group-size [ [?1] -> ?1 ] [ [?1] -> if IDActiveAgent
!= ?1 and IDInteractionPartner != ?1 [
                            ;if the picked individual is different from the Actived Agent and
the Interaction Partner,
                            ;set the increment to be equivalent to 20%±5% of the weight
between the Activated Agent and the picked individual.
                            ;it means that once one individual gets information from the
Interaction Partner, the others group members
                            ;proportionally loose 20% of their weight to the Activated Agent
                                                set delta delta + ( [weight] of
connection IDActiveAgent ?1 * social-increase)
                                                ;and then, decrease 20% of the weight
between the Activated Agent and the picked individual
                                                ask connection IDActiveAgent ?1
[let OldW [weight] of connection IDActiveAgent ?1

set weight weight - ( weight * social-increase )

]] ]
                        ask connection IDActiveAgent IDInteractionPartner [set weight
weight + delta] ;increase weight of connection by 20%
          ;check if the sum of relationships is different of 1
                        let sam 0
                        foreach n-values group-size [ [?1] -> ?1 ] [ [?1] -> if IDActiveAgent
!= ?1 [ set sam sam + ( [weight] of connection
                                                IDActiveAgent ?1 )] ]
                    ]

        [ifelse Prob < (Pinf + Ppat) [

    ;INDIVIDUAL GETS PATHOGEN
                        set Type-of-interaction 2
                        let weight-diference 0 ;represents the total weight of the Activated
Individual and the others individuals in the group,
          ;except the Interaction Partner
                        set social-increase random-normal 0.20 0.05
                        set social-decrease random-normal 0.20 0.05
                        foreach n-values group-size [ [?1] -> ?1 ] [ [?1] -> if IDActiveAgent
!= ?1 and IDInteractionPartner != ?1 [
```

```
                                                                    set weight-
diference weight-diference + ( [weight] of
                                                                    connection IDActiveAgent
?1 )] ]
                        let delta ([weight] of connection IDActiveAgent
IDInteractionPartner * social-decrease)
                        foreach n-values group-size [ [?1] -> ?1 ] [ [?1] -> if IDActiveAgent
!= ?1 and IDInteractionPartner != ?1
                    [ask connection IDActiveAgent ?1  ;update the connection between the
Activated Agent and the group member
                                                    [ let OldW [weight] of connection
IDActiveAgent ?1
                                                      set weight weight + (delta * (weight /
weight-diference)) ;proportional for each weight
                                                      ] ;its is add to their connection a relative
proportion of the weight decreased between the
                ;Activated Agent and the Interaction Partner
                                                    ] ]
                        ask connection IDActiveAgent IDInteractionPartner [set weight
weight - delta]
                        let sam 0
                        foreach n-values group-size [ [?1] -> ?1 ] [ [?1] -> if IDActiveAgent
!= ?1 [ set sam sam + ( [weight] of connection
                                                                    IDActiveAgent ?1 )]
]
                    ]

        [ifelse Prob < (Pinf + Ppat + Pinfpat) [

    ;INDIVIDUAL GETS INFORMATION AND PATHOGEN

                            let diference Pinf - Ppat
                            set social-increase random-normal 0.20 0.05
                            set social-decrease random-normal 0.20 0.05
                            ifelse diference > 0
                            [
    ;INDIVIDUAL GETS MORE INFORMATION THAN PATHOGEN
                            set Type-of-interaction 3
                            let delta 0
                            foreach n-values group-size [ [?1] -> ?1 ] [ [?1] -> if
IDActiveAgent != ?1 and IDInteractionPartner != ?1
                                                    [
                                                    set delta delta + ( [weight] of connection
IDActiveAgent ?1 * (social-increase * abs diference))
                                                    ask connection IDActiveAgent ?1 [let OldW
[weight] of connection IDActiveAgent ?1
                                                    set weight weight - ( weight * (social-
increase * abs diference))
                                                                    ]]
                                                     ]
```

```
                                        ask connection IDActiveAgent IDInteractionPartner [set
weight weight + delta]
                                        let sam 0
                                        foreach n-values group-size [ [?1] -> ?1 ] [ [?1] -> if
IDActiveAgent != ?1 [ set sam sam + ( [weight] of
                                                                connection IDActiveAgent
?1 )] ]
                                    ]
                                        ;INDIVIDUAL GETS MORE PATHOGEN THAN
INFORMATION
                                        [
                                        set Type-of-interaction 4
                                        let weight-diference 0 ; the sum of weight from
remanining partners
                                        foreach n-values group-size [ [?1] -> ?1 ] [ [?1] -> if
IDActiveAgent != ?1 and IDInteractionPartner != ?1
                [set weight-diference weight-diference + ( [weight] of connection
IDActiveAgent ?1 )] ]
                                        let delta ([weight] of connection IDActiveAgent
IDInteractionPartner * (social-decrease * abs diference))
                                        foreach n-values group-size [ [?1] -> ?1 ] [ [?1] -> if
IDActiveAgent != ?1 and IDInteractionPartner != ?1
                                                        [ask connection IDActiveAgent ?1
                                                        [ set weight weight + (delta *
(weight / weight-diference))]
                                                        ] ]
                                        ask connection IDActiveAgent IDInteractionPartner [set
weight weight - delta]                                                  let
sam 0
                                        foreach n-values group-size [ [?1] -> ?1 ] [ [?1] -> if
IDActiveAgent != ?1 [ set sam sam + ( [weight] of
                                                                connection IDActiveAgent
?1 )] ]
                                    ]
                                    ]

                                [if Prob < (Pinf + Ppat + Pinfpat + Pnone) [
 ;INDIVIDUAL GETS NEITHER INFORMATION NOR PATHOGEN = NO UPDATE OF
WEIGHT
                                                set Type-of-interaction 5
                                                let sam 0
                                                foreach n-values group-size [ [?1] -> ?1 ] [ [?1]
-> if IDActiveAgent != ?1 [ set sam sam + ( [weight] of connection IDActiveAgent ?1 )] ]
                                                ]
                    ]
            ]
        ]
    set New-weight [weight] of connection IDActiveAgent IDInteractionPartner
    data-collection
        ]
```

```
 end


to-report select-partner [IDActiveAgent]
  let Prob random-float 1.0      ;probability of interacting or not with a given group member
  let IDPartner -1               ;error-message. For ex: no individual is selected.
  let InteractionProbPartner 0
  foreach n-values group-size [ [?1] -> ?1 ] ; call all possible individuals to interact until its
InteractionProbPartner > Prob
  [ [?1] ->                      ; selection of first interaction partner
  if IDActiveAgent != ?1 [set InteractionProbPartner InteractionProbPartner + ([weight] of
connection IDActiveAgent ?1)]
  if InteractionProbPartner >= Prob [
  set IDPartner ?1
   report IDPartner]
  ]
let sam 0
foreach n-values group-size [ [?1] -> ?1 ] [ [?1] -> if IDActiveAgent != ?1 [ set sam sam +
( [weight] of connection IDActiveAgent ?1 )] ]
error "No interaction partner"
report -1 ; error-message
end


to update-information-rate
  ask individuals [set My-information random-float 1]
end


to update-pathogen-rate
  ask individuals [set My-pathogen random-float 1]
end


to data-collection
  let File_name word "Simulation" (word simulations ".csv")
  file-open File_name
  file-type IDActor
  file-type ","
  file-type IDReceiver
  file-type ","
  file-type Interaction
  file-type ","
  file-type Type-of-interaction
  file-type ","
  file-type Old-weight
  file-type ","
  file-type New-Weight
  file-type ","
  file-type [My-information] of turtle (IDActor - 1)
```

```
  file-type ","
  file-type [My-pathogen] of turtle (IDActor - 1)
  file-type ","
  file-type [My-information] of turtle (IDReceiver - 1)
  file-type ","
  file-type [My-pathogen] of turtle (IDReceiver - 1)
  file-type ","
  file-print ""
  file-close
end


to data-collection-reset
  set IDActor -1
  set IDReceiver -1
  set Interaction -1
  set Type-of-interaction -1
  set Old-weight -1
  set New-Weight -1
en
```

APPENDIX J: RÉSUMÉ DE LA THÈSE DE DOCTORAT

UNIVERSITÉ DE STRASBOURG

Discipline: Sciences du vivant

Spécialité: Ecologie et Ethologie

Présentée par: ROMANO DE PAULA Valéria

Titre: Les réseaux sociaux comme compromis entre une transmission d'information efficace et une réduction de la transmission des pathogènes.

Unité de Recherche: UMR7178 - Institut Pluridisciplinaire Hubert Curien (IPHC) - Département Ecologie, Physiologie et Ethologie (DEPE)

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☐ ED - Sciences de l'Homme et des sociétés ☐ ED 99 – Humanités ☐ ED 101 – Droit, sciences politique et histoire ☐ ED 182 – Physique et chimie physique ☐ ED 221 – Augustin Cournot ☐ ED 222 - Sciences chimiques	☐ ED 269 - Mathématiques, sciences de l'information et de l'ingénieur ☐ ED 270 – Théologie et sciences religieuses ☐ ED 413 – Sciences de la terre, de l'univers et de l'environnement X ED 414 – Sciences de la vie et de la santé

Contexte scientifique

Comprendre le lien entre le comportement individuel et l'organisation et le fonctionnement d'une population a longtemps été crucial en Ecologie et en Biologie Evolutive. La structure sociale d'un groupe animal ou d'une population peut théoriquement réguler la transmission d'information et les risques de pathogènes via les contacts ou la proximité sociale. L'étude des sociétés humaines de chasseurs-cueilleurs a, par exemple, montré que les individus à haute centralité sociale – ceux le plus interconnectés dans le groupe – ont une meilleure valeur sélective que leurs congénères mais aussi plus de maladies. En outre, la façon dont les animaux interagissent et développent des relations peut profondément affecter la dynamique et la chaine de transmission sociale. Ceci créerait donc un compromis entre le risque d'infection et le flux d'information, nous conduisant à la question suivante:

Comment la forme des réseaux sociaux influence la transmission de l'information et des parasites dans les sociétés animales?

Afin de répondre à cette question, j'ai combiné des approches empirique[1] et théorique[2] afin de comprendre l'influence de la structure sociale sur la transmission. J'ai commencé par étudier le rôle de la centralité individuelle et des propriétés globales du réseau (tels que le niveau de modularité, la centralisation et la densité) en lien avec une approche épidémiologique appliquée aux primates non-humains.

[1] Prise de données comportementales de macaques japonais à Koshima pendant 9 mois et collaboration avec des primatologues fournissant des données comportementales de 20 autres espèces de primates

[2] Approches théoriques d'analyses des réseaux sociaux et de systèmes multi-agents.

J'ai finalisé la thèse en utilisant une approche théorique où j'ai à la fois estimé l'efficience des réseaux selon le niveau de modularité et prédit un scénario de transmission sociale optimale où les individus maximisent leurs chances d'acquérir des informations mais minimisent le risque d'être infectés. J'ai donc organisé ma thèse en quatre études:

Etude 1 – Modéliser la transmission des pathogènes dans les réseaux de primates afin de prédire les risques liés à la centralité individuelle (publication 1 dans la liste de publications*).

***** Cette étude a été mise en avant par la Société Américaine de Primatologie

Dans cette première étude de mon doctorat, j'ai adressé la question de l'influence de la centralité individuelle sur la chaine de transmission dans les réseaux de primates. J'ai associé l'analyse des réseaux sociaux à la modélisation individus-centrée pour prédire la transmission d'agents infectieux théoriques chez deux groupes sauvages de macaques japonais. Mes collaborateurs et moi-même avons collecté des données de macaques femelles adultes vivant sur les îles de Koshima et de Yakushima. Au Japon, durant respectivement huit et seize mois. Les identités des macaques ainsi que les réseaux de toilettage ont été implémentés dans des simulation de Markov à base de réseaux (Markov graph-based simulation). Dans ce modèle, la probabilité qu'un individu transmette un agent pathogène dépend de la force de ses relations sociales avec les autres membres du groupe. De la même façon, la probabilité qu'il soit infecté dépend de ses relations avec les membres du groupe déjà infectés. J'ai ensuite testé les corrélations entre les centralités individuelles (eigenvector, force et betweenness) avec (i) le pourcentage d'individu infectés durant un temps donné, (ii) la latence moyenne d'infection complète (à tout le groupe), (iii) la probabilité qu'un individu soit infecté en premier, (iv) le rang moyen d'infection de chaque individu dans la chaine de transmission. Les résultats de cette étude soutiennent l'hypothèse que les individus les plus centraux

socialement transmettent un pathogène en un temps plus court et sont plus sujets à être infectés et plus rapidement que leurs conspécifiques moins centraux. Cependant, j'ai aussi observé que la transmission des agents infectieux du réseau de macaques de Yakushima ne différait pas, pour toutes les mesures testées, d'une transmission dans des réseaux aléatoires. La généralisation de l'influence des réseaux dans la transmission des pathogènes doit donc être faite avec précaution, puisque l'influence des caractéristiques individuelles dans certains réseaux réels semblent être moins pertinente par rapport à d'autres pour prédire les épidémies. Cette étude a donc mis en lumière que l'utilisation de stratégie de vaccination basée sur les caractéristiques individuelles – qui est censée améliorer l'efficacité des interventions en ciblant les individus clés (« super-diffuseurs ») - pouvait donc être moins efficace que suggérée.

Etude 2 - La diffusion des pathogènes et l'effet de la variation de la connectivité sociale: une évaluation à travers la vitesse de contagion (publication 10 dans la liste de publications)

Dans cette seconde étude, je suis passé des mesures individuelles (étude 1) aux propriétés globales des réseaux, avec l'objectif de comprendre les mécanismes qui sous-tendent les épidémies dans les réseaux sociaux de primates. Comme les maladies infectieuses sont considérées comme une des menaces majeures pour la survie des espèces, plusieurs études se sont focalisées sur le rôle du réseau social au stade final d'une épidémie (nombre d'infectés, rapidité d'infection). Cependant, aucune étude n'a jamais été faire sur l'influence de ce réseau à différents moments de l'épidémie, du début à la fin de cette dernière. Dans cette étude, j'ai utilisé une approche comparative. Les données de 40 groupes sauvages de 21 espèces de primates non humains ont été utilisées pour comprendre comment la centralisation du réseau, sa densité, son diamètre, ou la modularité interagissent avec la taille de groupe

pour optimiser ou contraindre la diffusion d'agents infectieux, à cinq moments différents d'une épidémie. J'ai implémenté les données empiriques des contacts sociaux dans un modèle stochastique dynamique considérant la force des interactions entre les membres du groupe et j'ai examiné la dynamique des pathogènes via le pourcentage moyen d'individus infectés à chacun des cinq moments de l'épidémie. Les résultats montrent que la prévalence d'une épidémie est plus grande proportionnellement quand la taille des groupes augmente, mais la centralisation et la modularité, et d'une moindre importance le coefficient de clustering et le diamètre, influencent le nombre d'individus infectés. Cependant cette influence dépend du moment de l'épidémie et de la virulence du pathogène. Ces résultats soutiennent l'hypothèse de l'engorgement social selon laquelle un nombre accru de sous-groupes sociaux réduit le coût des relations sociales dont la diffusion des pathogènes. Cette étude aide à mieux comprendre quelle propriété du réseau pourrait être ciblée selon le moment d'une épidémie afin de stopper la progression de cette dernière.

Etude 3 – Des niveaux intermédiaires de sous-divisions de groupes sociaux favorisent la transmission sociale (publication 8 dans la liste de publications)

La troisième étude de ma thèse concerne l'application d'une approche théorique pour comprendre la relation entre la modularité des groupes sociaux et l'efficience de la transmission sociale. Parmi les propriétés des réseaux, il a été avancé qu'une modularité accrue, c'est-à-dire un nombre de sous-divisions important des groupes, est une contribution majeure à l'évolution des réseaux biologiques tels que les réseaux protéiques, les réseaux neuronaux ou les réseaux métaboliques bactériens. Tout au long de ma thèse, des indices variés suggérèrent que les structures modulaires peuvent diminuer la vitesse de la transmission sociale. Alors qu'une relation linéaire peut être envisagée entre la vitesse de propagation et la modularité, aucune preuve ne permet encore de déterminer à quel degré

l'efficience des réseaux, proxy de la transmission sociale, est modularité dépendant. Dans cette étude, j'ai créé 2798 réseaux invariants d'échelle (« scale-free ») et différents en taille de groupe afin de tester comment le réseau - en terme de densité, de modularité et de centralisation - et la taille du groupe affectent l'efficience de transmission. J'ai également utilisé 68 réseaux sociaux de primates – captifs et sauvages – afin de déterminer si les résultats trouvés dans nos conditions théoriques étaient vérifiés via ces données empiriques. Les résultats de cette étude montrent une relation non linéaire entre la modularité et l'efficience globale avec des niveaux d'efficience maximaux atteints à des niveaux intermédiaires de modularité, à la fois dans les données théoriques et les données empiriques. L'identification de ce phénomène améliore notre compréhension sur la variation des réseaux sociaux, en lien avec la performance des transmissions sociales, et en plus de fournir les bases de discussion de l'évolution des systèmes complexes incluant les sociétés animales.

Etude 4 – Investiguer le compromis entre une transmission optimale de l'information et un risqué réduit de pathogènes dans les sociétés animales via un modèle multi-agents (publication 9 dans la liste de publications)

Dans cette dernière partie de ma thèse, j'ai créé un modèle multi-agent appelé le Modèle de Relations Optimales (Optimal Relationship Model) afin de simuler les conditions optimales à la transmission de l'information et des parasites et ceci, dans l'objectif ultime de comprendre les patrons émergents des structures sociales. Cette approche apporte une contribution importante pour prédire et comprendre quelles propriétés du réseau interagissent pour maximiser les bénéfices de la socialité tout en diminuant les coûts liés à la transmission des pathogènes. Dans cette étude, j'ai contrôlé la quantité d'information et de pathogène de chaque individu, totalisant vingt conditions différentes, puis j'ai observé l'évolution de réseaux sociaux variant en taille (10, 30, 70). Le modèle théorique stipule que les individus ne

sont pas conscients de la quantité d'information et de pathogènes de leur conspécifiques mais en interagissant avec eux, ils apprennent quels individus sont informés et lesquels sont parasités. Ils ajustent donc leurs interactions sociales en fonctions de ces deux quantités afin d'être connectés aux individus les plus informés mais les moins parasités. Les simulations de cette étude permettent de déterminer que les réseaux sont plus centralisés et moins denses quand les valeurs de pathogènes et d'information varient entre les individus. Comme attendu, les individus développent de fortes relations avec les individus les plus informés. Les préférences sociales observées dans le modèle indiquent que les individus, selon les règles du modèle, optimisent leurs relations pour réduire le nombre de connections (augmentant l'isolation relative) et en étant en contact avec ceux fournissant les bénéfices les plus importants. Cette approche innovante apporte de nouvelles questions et hypothèses à tester quant à l'évolution des relations et des structures sociales chez les animaux.

En conclusion, les principaux objectifs de cette thèse étaient d'étudier comment les réseaux sociaux façonnent la transmission sociale, soit au niveau individuel, soit au niveau global et finalement comprendre comment les individus équilibrent leurs relations entre les coûts et les bénéfices de la vie en groupe. Les résultats de ma thèse ne fournissent pas seulement une meilleure compréhension quant aux mécanismes de la transmission sociale dans un groupe, mais également quant aux variations et à la dynamique des réseaux en fonction de différents facteurs comme la taille de groupe et les pressions écologiques. Ceci permet de conclure sur l'évolution du comportement social en fonction des compromis de la vie en groupe.

Des études complémentaires sont nécessaires pour étudier le lien entre la complexité du comportement et l'influence environnementale telle que la disponibilité des ressources ainsi que la dynamique des populations tels que l'immigration, les

naissances ou les décés des membres du groupe, et ceci dans le but de complexifier les modèles multi-agents développés ici et d'avoir une vision plus approfondie de l'évolution des systèmes sociaux.

Publications

1. **Romano V.**, Duboscq J., Sarabian C., Thomas E., Sueur C., MacIntosh AJJ. (2016) Modelling infection transmission in primate networks to predict centrality-based risk. *American Journal of Primatology*. 78:767-779 (doi: 10.1002/ajp.22542). *Featured article by the American Society of Primatology: https://www.asp.org/

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9. **Romano V.**, Puga-Gonzalez I., MacIntosh AJJ., Sueur C. Investigating the trade-off between information access and infection avoidance in animal societies: an individual-based model. *Soumission hiver 2017/2018.*
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2017 **Romano V.,** Sueur C., MacIntosh AJJ. *Social behavior and infectious disease: investigating contagious risk in primate networks*. Seminar on Ecology and Social Behavior. Primate Research Institute of Kyoto University. Inuyama, Japan.

2016 **Romano V.,** Sueur C., MacIntosh AJJ. *From individuals to groups: the network science behind information and pathogen transmission*. Interdisciplinary Seminar on Primatology. Primate Research Institute of Kyoto University. Inuyama, Japan.

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A souvenir from the two cities I have lived during the PhD:

Strasbourg, France

Inuyama, Japan

UNIVERSITÉ DE STRASBOURG

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Social networks as a trade-off between optimal information transmission and reduced disease transmission

Résumé

La structure sociale d'un groupe peut théoriquement réguler la transmission des informations et le risques de maladies via les contacts sociaux et la proximité. En théorie, les mêmes propriétés de réseau qui favorisent la transmission d'information favorisent également la transmission de pathogènes, créant de fait un potentiel compromis entre eux. Dans ma thèse, j'ai utilisé des données empiriques, des analyses de réseaux et modèle de simulation individuel afin de comprendre l'influence des structures sociales sur la transmission sociale chez les primates et dans des réseaux théoriques. Mes études ont montré que i) les macaques japonais centraux dans le groupe transmettent les pathogènes plus rapidement mais sont également plus susceptibles d'être infectés; ii) le nombre d'individus infectés dans 40 groupes de primates est dépendant des propriétés globales du réseau et de l'étape de l'infection: iii) un pic d'efficacité de réseau à des valeurs intermédiaires de sous-structure de groupe dans des réseaux empiriques et théoriques; et iv) des variations dans les propriétés de réseaux sont la conséquence de décisions individuelles en fonction de compromise entre la collecte d'information et l'évitement de l'infection. Ainsi, ma thèse a démontré les mécanismes de transmission social et indiqué que les propriétés de réseau pourrait réflecter un compromise entre transmission de l'information et transmission de pathogène.

Abstract

Social structure can theoretically regulate information transmission and disease risk via social contact or proximity. In theory, the same network properties that favor information transmission also favor pathogen transmission creating a potential trade-off between them. In my thesis, I used empirical data, network analysis and individual-based modelling to understand the influence of social structure on social transmission in primate and theoretical networks. My studies show that i) central Japanese macaques transmit disease faster but are also more prone to acquiring infectious agents; ii) the number of infected individuals in 40 wild primate groups is dependent on global network properties and epidemic time; iii) network efficiency peaks with intermediate values of group substructure in theoretical and empirical networks; and, iv) variation in the network properties is a consequence of individual decisions given the trade-offs between collecting information and avoiding infection. Altogether, my thesis reveals the mechanisms of social transmission and indicates that network properties might reflect a trade-off between information and pathogen transmission.