Infected or informed? Social structure and the simultaneous transmission of information and infectious disease

- Julian C. Evans (Corresponding author)
  - ORCiD ID: 0000-0001-6810-199X
  - Address: Department of Evolutionary Biology and Environmental Studies, University of Zurich, Switzerland
  - E-mail: jevansbio@gmail.com
- Matthew J. Silk (Corresponding author)
  - ORCiD ID: 0000-0002-8318-5383
  - Address 1: Centre for Ecology and Conservation, University of Exeter Penryn Campus, UK
  - Address 2: Environment and Sustainability Institute, University of Exeter Penryn Campus, UK
  - E-mail: matthewsilk@outlook.com
- Neeltje Boogert
  - ORCiD ID: 0000-0002-1337-4365
  - Address: Centre for Ecology and Conservation, University of Exeter Penryn Campus, UK
  - E-mail: N.J.Boogert@exeter.ac.uk
- David J. Hodgson
  - ORCiD ID: 0000-0003-4220-2076
  - Address: Centre for Ecology and Conservation, University of Exeter Penryn Campus, UK
  - E-mail: D.J.Hodgson@exeter.ac.uk

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Abstract

Social interactions present opportunities for both information and infection to spread through populations. Social learning is often proposed as a key benefit of sociality, while disease epidemics are proposed as a major cost. Multiple empirical and theoretical studies have demonstrated the importance of social structure for either information or infectious disease, but rarely in combination. We provide an overview of relevant empirical studies, discuss differences in the transmission processes of infection and information, and review how these processes have been modelled. Finally, we highlight ways in which animal social network structure and dynamics might mediate the trade-off between the sharing of information and infection. We reveal how modular social network structures can promote the spread of information and mitigate against the spread of infection relative to other network structures. We discuss how the maintenance of long-term social bonds, clustering of social contacts in time, and adaptive plasticity in behavioural interactions, all play important roles in influencing the transmission of information and infection. We provide novel hypotheses and suggest new directions for research that quantifies the transmission of information and infection simultaneously across different network structures to help tease apart their influence on the evolution of social behaviour.

Key words: social network, epidemic, social learning, social evolution, group-living, dynamic network

Introduction

The diversity of social systems in animals is shaped by differences among populations in the costs and benefits of different types of social interaction (Krause and Ruxton 2002). The sharing of information provides one key benefit that improves fitness of more sociable individuals (Dall, Giraldeau, Olsson, McNamara and Stephens 2005, Danchin, Giraldeau, Valone and Wagner 2004), while the risk of acquiring infectious disease represents an important cost of sociality (Ezenwa, Ghai, McKay and Williams 2016, Ezenwa and Worsley-Tonks 2018, Krause and Ruxton 2002). An important challenge in evolutionary ecology is to identify whether, and how, natural selection might favour social structures that can simultaneously optimise information-sharing and reduce the spread of infection in social species. The solution to this evolutionary problem will depend on the differences in transmission dynamics between information and infection.

Social network analyses and modelling are increasingly used to study animal social behaviour, and these approaches have greatly benefitted research into disease and information transmission in wild animals. Interactions with more individuals result in increased opportunities for infectious disease spread, while simultaneously making it possible for information to proliferate within a group or population (Wey, Blumstein, Shen and Jordán 2008, White, Forester and Craft 2017). However, studying the trade-off between efficient information transmission and rapid spread of disease is challenging due to the difficulty of combining data that reveal infection status, and data that indicate information transmission. Consequently, while studies have examined how social relationships impact the transmission of either infection or information separately, there has been little consideration of the interplay between information and infection spread within animal social networks.

Here we synthesise knowledge on the role of social networks in transmission of information and infectious disease and develop hypotheses regarding how animal social systems may be adapted to reconcile the trade-off between acquiring information and contracting disease. We briefly review

studies examining the transmission of infectious disease and information in animal social networks. We then highlight potential differences between the transmission processes of information and infection that will mediate this trade-off. Finally, we discuss how social relationships may be adapted to optimise both types of transmission, integrating insights from the network modelling literature and through empirical work in natural populations. Throughout we emphasise new avenues of study into the flow of disease and information through animal social networks to promote a better understanding of how these two important ecological processes affect each other.

Infectious disease transmission in animal social networks

The transmission of parasites and pathogens that cause infectious disease can happen directly via specific types of behavioural interaction or indirectly via the environment (e.g. an individual using a refuge that has been contaminated by an infectious individual) (White, Forester and Craft 2017). Many infections are endemic, persisting stably within a host population for a long period of time (Viana, Mancy, Biek, Cleaveland, Cross, Lloyd-Smith and Haydon 2014). Others are emergent, acquired either from a long-term environmental reservoir or spilling over from alternative host species (Daszak, Cunningham and Hyatt 2000). Networks of spatial associations and behavioural interactions are now known to be closely associated with infectious disease epidemiology in wild animal populations (e.g VanderWaal, Atwill, Isbell and McCowan 2014, Weber, Carter, Dall, Delahay, McDonald, Bearhop and McDonald 2013). Overall network structure is critical in determining how infectious disease spreads through populations. For example, the presence of distinct social communities can limit the spread of infection in animal groups or populations (Griffin and Nunn 2012, Sah, Leu, Cross, Hudson and Bansal 2017). Social network analysis can also help identify potential routes of transmission (Silk, Croft, Delahay, Hodgson, Boots, Weber and McDonald 2017, White, Forester and Craft 2017), determines individual variation in transmission potential (VanderWaal and Ezenwa 2016) and predicts or explains how infection spreads through populations

(Craft 2015, Silk, Croft, Delahay, Hodgson, Weber, Boots and McDonald 2017). Network analyses have also revealed associations between individual phenotypes, infection and network position. For example, European badgers *Meles meles* that test positive for bovine tuberculosis tend to have fewer connections to their own social group and more social connections with neighbouring groups (Weber, Carter, Dall, Delahay, McDonald, Bearhop and McDonald 2013), and male-biased infection is associated with sex-differences in social network position (Silk, Weber, Steward, Hodgson, Boots, Croft, Delahay and McDonald 2018).

Fine-scale social networks can be used to identify if and how different types of social interaction generate transmission opportunities. The most important type of contact for transmission may vary among systems: in some species direct social contacts may be more important than shared space use (Blyton, Banks, Peakall, Lindenmayer and Gordon 2014, VanderWaal, Atwill, Isbell and McCowan 2014). More recently, the importance of cryptic contacts has been revealed in a mixed-species community of bats, with social networks based solely on the sharing of, or physical contact at, roosts not sufficient to capture fungal pathogen transmission dynamics (Hoyt, Langwig, White, Kaarakka, Redell, Kurta, DePue, Scullon, Parise, Foster, Frick and Kilpatrick 2018). Together these studies reveal that using social networks in disease ecology might help to identify potential transmission routes, but might be uninformative and potentially misleading if the types of social interactions modelled are not those that facilitate disease spread.

Infection may lead to temporal changes to network structure by changing patterns of social behaviour (Ezenwa, Archie, Craft, Hawley, Martin, Moore and White 2016). Pathogens and parasites often manipulate the host's social behaviour to facilitate further transmission (e.g. Berdoy, Webster and Macdonald 2000, Loot, Brosse, Lek and Guégan 2001, Randall, Marino, Haydon, Sillero-Zubiri, Knobel, Tallents, Macdonald and Laurenson 2006),while the social behaviour of the infected host and/or the individuals that interact with it might change to prevent spread, resulting in co-dynamics between disease spread and network structure (Silk, Croft, Delahay, Hodgson, Boots, Weber and McDonald 2017). In guppies *Poecilia reticulata* for example, infected individual are avoided by

uninfected fish, making sick individuals less well connected and causing the networks to become less clustered overall (Croft, Edenbrow, Darden, Ramnarine, van Oosterhout and Cable 2011). At a network-level these behaviour-infection co-dynamics can have a protective effect. In ants, for example, social networks of infected colonies become more modular and assortative, resulting in them becoming less efficient in terms of information transmission capacity but more effective at limiting the spread of infection (Stroeymeyt, Grasse, Crespi, Mersch, Cremer and Keller 2018). It is clear then that variation in connectivity among individuals, the resultant network structure, and changes in network dynamics following infection, all have important implications for the emergence, spread and persistence of wildlife disease.

Information transmission in animal social networks

Information can be acquired by sampling the environment (personal information; Dall, Giraldeau, Olsson, McNamara and Stephens 2005) or by observing or interacting with other individuals or their products (social information; Dall, Giraldeau, Olsson, McNamara and Stephens 2005, Danchin, Giraldeau, Valone and Wagner 2004). Individuals can spread social information inadvertently or can choose to deliberately transmit information via signals. A receiver must then decide whether to act on this information or not (Dall, Giraldeau, Olsson, McNamara and Stephens 2005, Schmidt, Dall and Van Gils 2010). The transfer of social information usually requires sensory contact between individuals and is therefore linked directly to spatial association and/or behavioural interactions. Consequently, as with infectious disease transmission, an individual's social network position causes variation in the probability and rate of receipt of information, and their contribution to the speed and quality of information transmission through a population (Lusseau 2003, Lusseau and Newman 2004, Modlmeier, Keiser, Watters, Sih and Pruitt 2014). Depending on the duration information is useful, an individual's network position will strongly influence how they can utilise this information. For example, information such as the discovery of a resource location (Aplin, Farine,

Morand-Ferron and Sheldon 2012, Blonder and Dornhaus 2011, Webster, Atton, Hoppitt and Laland 2013) may only be accurate for a short time if a resource is ephemeral or is rapidly depleted. A central network position or high level of connectivity to the individual who initially discovers such resources will be highly beneficial to potential recipients, as demonstrated in several studies of the influence of network position on food patch discovery in flocks of songbirds (Aplin, Farine, Morand-Ferron and Sheldon 2012, Farine, Aplin, Sheldon and Hoppitt 2015, Jones, Aplin, Devost and Morand-Ferron 2017, Tóth, Tuliozi, Baldan, Hoi and Griggio 2017). Therefore, when information-gathering is beneficial, group members may be attracted to individuals who regularly provide information, changing their position in the social network. In ring tailed lemurs *Lemur catta*, for example, this led to informed individuals occupying more central network positions (Kulahci, Ghazanfar and Rubenstein 2018).

Social associations are also linked to the spread, through social learning, of behavioural innovations which can arise via trial and error learning (Allen, Weinrich, Hoppitt and Rendell 2013, Aplin, Farine, Morand-Ferron, Cockburn, Thornton and Sheldon 2014). Such innovations range from simply adopting a new foraging ground (Schakner, Petelle, Tennis, Leeuw, Stansell and Blumstein 2017) to tool use (Coelho, Falótico, Izar, Mannu, Resende, Siqueira and Ottoni 2015, Hobaiter, Poisot, Zuberbühler, Hoppitt and Gruber 2014, Mann, Stanton, Patterson, Bienenstock and Singh 2012, St Clair, Burns, Bettaney, Morrissey, Otis, Ryder, Fleischer, James and Rutz 2015) or novel foraging techniques (Aplin, Farine, Morand-Ferron, Cockburn, Thornton and Sheldon 2014, Boogert, Nightingale, Hoppitt and Laland 2014, Kendal, Custance, Kendal, Vale, Stoinski, Rakotomalala and Rasamimanana 2010). Innovations of long-term value can be transmitted to subsequent generations (Aplin, Farine, Morand-Ferron, Cockburn, Thornton and Sheldon 2014, Cantor and Whitehead) and impact long-term social structure, provided individuals alter their social interactions to maximise their chances of acquiring information (Coelho, Falótico, Izar, Mannu, Resende, Siqueira and Ottoni 2015, Kulahci, Ghazanfar and Rubenstein 2018). One possible outcome is that long-term, preferential associations with individuals who adopt the same behaviours (Mann, Stanton,

176 Patterson, Bienenstock and Singh 2012) will homogenise behavioural repertoires in any given group
177 and can establish “animal cultures” (Allen, Weinrich, Hoppitt and Rendell 2013, Aplin, Farine,
178 Morand-Ferron, Cockburn, Thornton and Sheldon 2014, Krützen, Mann, Heithaus, Connor, Bejder
179 and Sherwin 2005). For example, bottlenose dolphins *Tursiops spp.* using marine sponges as tools
180 during foraging have been shown to preferentially associate with other tool users (Krützen, Mann,
181 Heithaus, Connor, Bejder and Sherwin 2005, Mann, Stanton, Patterson, Bienenstock and Singh
182 2012). This behavioural homogenisation may, depending on initial network structure, increase
183 connectedness which can lead to the structure of networks becoming more random. Alternatively, if
184 networks are already divided into distinct social communities, these groups might become
185 increasingly isolated from each other (Cantor and Whitehead 2013, Morgan and Laland 2012).

186 Generally, the transmission of social information is considered to benefit the recipient
187 individuals. However, there is potential for information transmitted to be outdated, poor, corrupted
188 or misleading (Klein, Vogt, Unrein and Reineke 2018, Koops 2004, Preece and Beekman 2014,
189 Schmidt, Dall and Van Gils 2010). While such information might simply result in wasted time and
190 energy (Dall, Giraldeau, Olsson, McNamara and Stephens 2005, Giraldeau, Valone and Templeton
191 2002, Preece and Beekman 2014), more severe costs are possible depending on the value of
192 accurate information (Koops 2004, Nocera, Forbes and Giraldeau 2005, Rieucau and Giraldeau
193 2011). For example, inexperienced bobolinks *Dolichonyx oryzivorus* relying on social information to
194 make breeding habitat choices were found to settle in and defend sub-optimal territories in
195 response to misleading information (Nocera, Forbes and Giraldeau 2005). The spread of
196 misinformation through a network could have impacts on fitness that resemble the spread of
197 infectious disease (Laland and Williams 1998). When learning how to solve problems, individuals
198 commonly show strong preference for the first solution to which they are exposed (birds: Aplin,
199 Farine, Morand-Ferron, Cockburn, Thornton and Sheldon 2014, e.g. fish: Laland and Williams 1998).
200 In competitive situations, recipients of suboptimal information might lose out to better informed
201 individuals or successful innovators. While the spread of misinformation has not yet been the

subject of empirical study using network techniques, there is strong potential for it to be important in nature. Similar to infectious disease, misinformation may be more likely to spread through a population if an individual transmitting misinformation is highly central to the social network, as information from these individuals may be more likely to be utilised by others, and their central position provides more transmission opportunities (Pruitt, Wright, Keiser, DeMarco, Grobis and Pinter-Wollman 2016).

Differences between information and disease transmission

We have illustrated the importance of an individual's social connections both in their access to and sharing of information, and in their exposure to and onward transmission of infectious disease. This suggests that animal societies might suffer a direct trade-off between the transmission of information and infectious disease. However, there are important general (though not universal) distinctions between the two transmission processes (Table 1).

Table 1: Summary of the general key differences in mechanisms and consequences of information and infectious disease transmission.

	Infection	Information
Transmitter decisions	Individuals inadvertently infect others (though parasites might change host behaviour to facilitate infection).	Individuals can inadvertently inform others (e.g. through cues/eavesdropping) or choose to deliberately inform others (e.g. signals).
Receiver decisions	Recipients of infection cannot choose whether they become infected or not.	Individuals decide whether to alter their behaviour based on the information received.
Number of transmitters	The probability of infection depends directly on the absolute magnitude of exposure. The number of simultaneously infected associates does not affect per contact likelihood of infection.	The probability of accepting information can depend on the relative magnitude of exposure to transmitters and non-transmitters. The proportion and phenotypic traits of associates transmitting information can influence if an individual uses information received (social learning strategies).
Social relationships	Prior social relationships have no effect on the per-contact likelihood of infection.	Prior social relationships can influence whether an individual adopts information received.
Transmission vectors	Infection spreads mainly through direct physical contact or close proximity, or via shared use of environmental reservoirs.	Information spread does not tend to require physical contact and can potentially occur via long range sensory interactions.
Selection	Selection acts on both the host and the pathogens they transmit.	Selection acts on the information transmitter and receiver, but only indirectly on the information being transmitted.
Behavioural changes	Infected individuals are often avoided by group members, and become less well-connected in the social network. Infections might manipulate host behaviour to increase probabilities of onward transmission.	Informed individuals can be desirable to associate with, and become better connected in the social network.

Most importantly, information transmission will typically involve choice, sometimes for a transmitter, who can choose when to transmit information and to whom, and always for the receiver, who chooses whether to alter their behaviour based on the information. Choice by the receiver means that the social transmission of information does not necessarily depend on a simple

probability of transmission associated with each interaction (Bakshy, Karrer and Adamic 2009, Jackson and López-Pintado 2013). Individuals may require multiple exposures to a transmitter, or require a certain proportion of social connections to be transmitting before choosing to utilise a piece of information (Bakshy, Karrer and Adamic 2009, Jackson and López-Pintado 2013). For example, chimpanzees were more likely to acquire a behaviour if it was demonstrated by three different individuals than when it was demonstrated three times by a single individual (Haun, Rekers and Tomasello (2012). Evidence for such social conformity, where naïve individuals disproportionally copy the behaviour demonstrated by the majority of conspecifics, has also been reported for mate-choice copying in fruit flies (Nöbel et al 2018) and great tits solving puzzle boxes in the wild (Aplin et al 2015). This information transmission process differs from disease transmission where a) the risk of acquiring infection rises monotonically with the duration and/or number of contacts with infected individuals, and b) having multiple infected social connections presents more opportunities for contact with infected individuals, but does not alter the per-interaction probability of infection. Therefore, in species showing conformist social learning strategies, the acceptance of information depends on the relative magnitude of exposure to transmitters and non-transmitters of information in a frequency-dependent manner, and similar non-linear changes in the likelihood of transmission can also occur for other social learning strategies.

Another key difference between information and infection is the effect that prior social associations can have on the likelihood of transmission. The current and previous social relationships of an individual can directly impact the probability of using information acquired through a particular social interaction. This phenomenon was first coined “directed social learning” (Coussi-Korbel and Fragaszy 1995) and later described as one of many potential “social learning strategies”(Laland 2004). Some of the clearest evidence of such a “Whom to learn from” social learning strategy comes from the importance of familiarity for the rate of social learning in many species (e.g. Kavaliers, Colwell and Choleris 2005, Swaney, Kendal, Capon, Brown and Laland 2001). Information from a familiar individual may result in an immediate change in behaviour, whereas an animal may require

more exposures to a piece of information if the source is unfamiliar. Transmitter familiarity is one of several relationship traits that might influence the decision to use a piece of information, with traits such as relatedness or social rank also potentially important (Boogert, Lachlan, Spencer, Templeton and Farine 2018, Evans, Jones and Morand-Ferron 2018, Farine, Spencer and Boogert 2015, Kavaliers, Colwell and Choleris 2005, Radford 2004, Valsecchi, Choleris, Moles, Guo and Mainardi 1996). Relationship traits can also interact with the phenotype of the transmitter, such as their experience (McComb, Moss, Durant, Baker and Sayialel 2001) or obvious fitness cues (Toth and Griggio 2011), to shape the likelihood of information being used. Similarly, it is possible for prior social relationships with other group members to have a profound effect on the health of individuals in social species (Sapolsky 2005), and the social buffering hypothesis (Ezenwa, Ghai, McKay and Williams 2016) proposes that positive social relationships can increase resistance to, and tolerance of, infection in group-living species (e.g. Almberg, Cross, Dobson, Smith, Metz, Stahler and Hudson 2015, Balasubramaniam, Beisner, Vandeleest, Atwill and McCowan 2016, Ezenwa and Worsley-Tonks 2018, Scharf, Modlmeier, Beros and Foitzik 2012, Walker and Hughes 2009). However, unlike the spread of information, this is a general effect and specific prior relationships with infected individuals do not influence the transmission process in the same way that prior relationships with informed individuals do.

Another important consideration is *how* information and infection are transmitted. Social information can be transmitted in multiple ways (Blanchet, Clobert and Danchin 2010, Danchin, Giraldeau, Valone and Wagner 2004), which may require prolonged or close interactions (e.g. the waggle dance in bees; Von Frisch 1967), may be possible with much looser associations (e.g, auditory cues; Hollen and Radford 2009), or may be transmitted indirectly via environmental signals or cues (e.g, scent marking: Gosling and Roberts 2001). Conversely, infectious disease is likely to be transmitted through a different set of interactions, such as prolonged close contact that facilitates aerosol transmission (Delahay, Cheeseman and Clifton-Hadley 2001) ; shared use of environmental reservoirs of pathogens (Godfrey, Bull, James and Murray 2009); aggressive interactions or mating

(Hamilton, Jones, Cameron, McCallum, Storfer, Hohenlohe and Hamede 2019) . The extent of the overlap in the types of social interaction that expose individuals to either information or infection will be important in determining the costs and benefits of being central in different types of social network.

A final important distinction between information and infectious disease transmission is that the former is the subject of selection only on the host population, while the latter depends on selection on both the host and the parasite being transmitted. For example, individuals that acquire novel social information may develop new social associations and become more central within a social network (Kulahci, Ghazanfar and Rubenstein 2018), which may benefit both themselves and other group members, especially in highly related groups. This is in direct contrast to transmission of infection, where group members would be expected to avoid contact with infected individuals (Croft, Edenbrow, Darden, Ramnarine, van Oosterhout and Cable 2011, Stephenson, Perkins and Cable 2018), which can lead *un*infected individuals to become more central as a result of infectious disease spread (Shaw and Schwartz 2008). A key component of this difference between infection and information is that there is often antagonistic selection on sickness behaviours between hosts and their parasites, whereby hosts will be selected to behave to avoid infecting (related) group members (Croft, Edenbrow, Darden, Ramnarine, van Oosterhout and Cable 2011, Lopes, Block and König 2016), while pathogens will be selected to cause host behaviours that maximise transmission (e.g. furious behaviour in rabid canines; Randall, Marino, Haydon, Sillero-Zubiri, Knobel, Tallents, Macdonald and Laurenson 2006)

The outcome of these differences between the transmission of infection and information is that while infection can be considered a simple contagion process, the spread of information is increasingly considered a complex contagion (Centola 2010, Macy 1991) affected by many of the social learning rules described above. Consequently, the spread of infectious disease has normally been modelled as a simple contagion using cascade models (Moore and Newman 2000), in which the

probability of infection increases with increasing absolute exposure to infected individuals. In contrast, the transmission of information could be either a simple or complex contagion depending on the social learning rules used by individuals. As a result, the spread of information has been modelled using a variety of dose-response models, including simple cascades, threshold models (Kempe, Kleinberg and Tardos 2003), and hybrid cascade-threshold models (de Kerchove, Krings, Lambiotte, Van Dooren and Blondel 2009) models (Fig. 1). The precise nature of the threshold, and whether it is a true threshold (deterministic), a stochastic transmission process with a threshold or a continuous dose-response curve, will depend on the social learning rules used. The measure of exposure used in these models might be relative exposure (conformist social learning in response to the prevalence of information among social contacts), absolute exposure (social learning in response to a minimum number of neighbours behaving in a particular way) or based on temporal rules (e.g. learning in response to a threshold number of interactions with informed individuals in a given time period). Variation in the status of informed individuals or their relationships to the focal individual could be key mechanisms which push the transmission process even further away from simple contagion.

Using models to capture the differences between information and disease transmission

The similarities and differences between information and disease transmission can be captured using dynamic computational modelling tools (Fig. 1), such as compartmental models. Compartmental models consider the transition of individuals between states, with individuals in each state assumed to have the same characteristics (Stattner and Vidot 2011). For example, a susceptible-infected-recovered (SIR) model (used commonly in epidemiology) contains three states: susceptible (or naïve) individuals; infected with a parasite (or exposed to and exploiting the

information); and recovered individuals who are now immune to that infection (or who no longer use the information to inform their behaviour). When applied to transmission through networks, compartmental models are typically applied as stochastic individual-based models, in which the transition of each individual between compartments is modelled separately and depends on the properties of their network connections. Such models are usually impossible to solve analytically (Craft 2015). These models avoid the assumption that populations mix freely, and hence that any individual will be able to infect any other individual in a population. General compartmental models applied to networks can be used to study infectious disease transmission, information flow, or both simultaneously (See supplementary table 1 for examples of simple compartmental models that can be applied to both information and disease and those more suitable for detailed models of particular transmission types).

Cascade, threshold and hybrid compartmental models can all be adapted to capture system-specific nuances regarding the importance of transmitter identity, social history, and behaviours that change in response to exposure (Fig. 1). Cascade models are typically implemented as stochastic models, with each additional unit of social interaction associated with a linear increase in the risk of infection (Fig. 1a). True threshold models are deterministic with individuals moving between states following fixed rules that are determined by the states of their neighbours, and can be used to model strictly conformist social learning, for example. Hybrid models can be used to mix properties of either model, for example by introducing stochasticity to the threshold model or incorporating continuous dose-response curves. For example, the latter might be applicable to studying imperfect conformist social learning where changes in state are governed by the states of neighbouring individuals according to a sigmoidal function rather than a strict threshold (e.g. Fig. 1c).

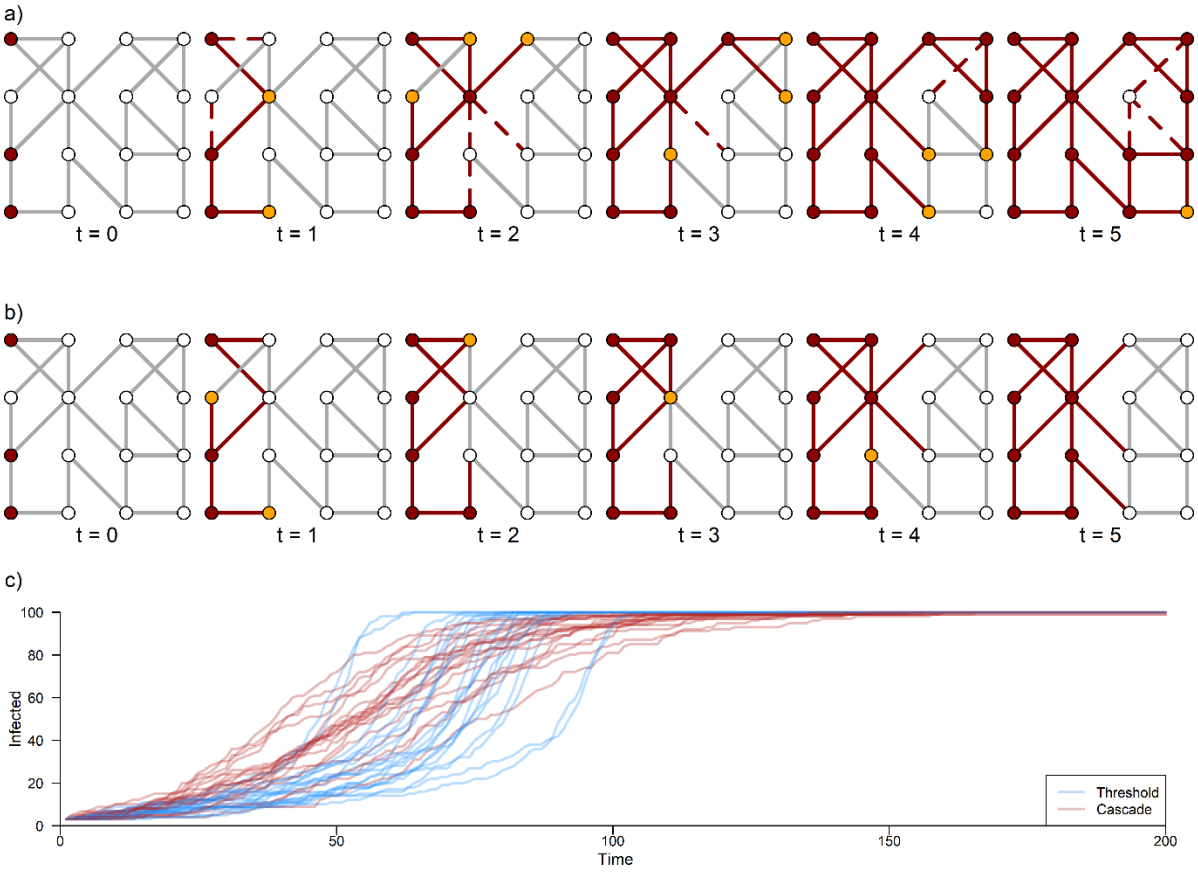

Figure 1. a) Example of a cascade model of simple contagion acting on an unweighted network (all individuals have an association strength of either 1 or 0) of 16 individuals over four time steps. Grey lines represent social associations, red nodes represent infected/informed individuals while yellow nodes represent individuals who will become infected/informed in the next time step. In this cascade model an infected/informed individual infects/informs each uninfected neighbour with a probability of 0.5 per time step. Solid red lines indicate an infected/informed node successfully infecting/informing a neighbour, while a dashed line represents a failure. If successful, the neighbour will become infected/informed in the next time step. b) A conformist transmission model (here a true threshold model, but a stochastic implementation would produce similar results) acting on the same network as a). Individuals become infected/informed when 50% of their neighbours are infected/informed. In this simulation, spread stalls at timestep 4 as there are not enough infected/informed individuals to result in transmission. c) Comparison between simple and conformist contagion models in a random network of 100 individuals, showing the percentage of the population infected over 200 arbitrary time-steps. For the simple contagion model there is a probability of 0.8% chance per time-step that infection is transmitted through an edge between an infected and susceptible individual. In the conformist model a sigmoid curve is fitted to the likelihood of an individual exploiting information with a baseline (asocial) individual learning rate of 0.2% per time-step, a maximum probability of learning of 30% per time-step and the threshold (pivot point of the sigmoid function) occurring at 50% of connected individuals providing information. Full R code for the model is provided in the Supplementary information.

Social structure and the infection-information trade-off

In the following sections, we highlight ways in which animal social networks might reflect the outcome of selection acting on individuals to maximise their acquisition of beneficial information and minimise their risk of being infected by parasites or pathogens. Specifically, we focus on the role

of structural heterogeneity in social networks, temporal heterogeneity in interactions, responsive changes in social interactions and the role of different types of interaction. We integrate the extent of our current knowledge of animal social systems with insights from compartmental network models (SI table 1) applied to theoretical and data-driven network structures in other disciplines.

Structural heterogeneity and transmission in animals

The structure of contact networks is integral to transmission dynamics for cascade models (Moore and Newman 2000, Newman 2002) and threshold models (Alkemade and Castaldi 2005, Hodas and Lerman 2014). We focus on three aspects of social network structure that have received considerable research interest and have clear applications to the study of animal societies: i) variation in connectivity among individuals causing networks to possess heterogeneous degree distributions (the extreme case being networks with scale-free properties), ii) modular structure that is characterised by densely connected regions (called communities) with rather few connections between these communities, and iii) small-world structure, which is best envisaged as individuals (or "nodes") being connected mostly with (spatial) neighbours, but possessing occasional contacts with much more distant nodes, resulting in transmission pathways through the network that are typically short compared to random or modular networks. We depict these different aspects of network structure in Fig. 2. Animal social structure is highly variable and can display one or multiple of the scale-free, small-world or modular properties introduced here (Wey, Blumstein, Shen and Jordán 2008).

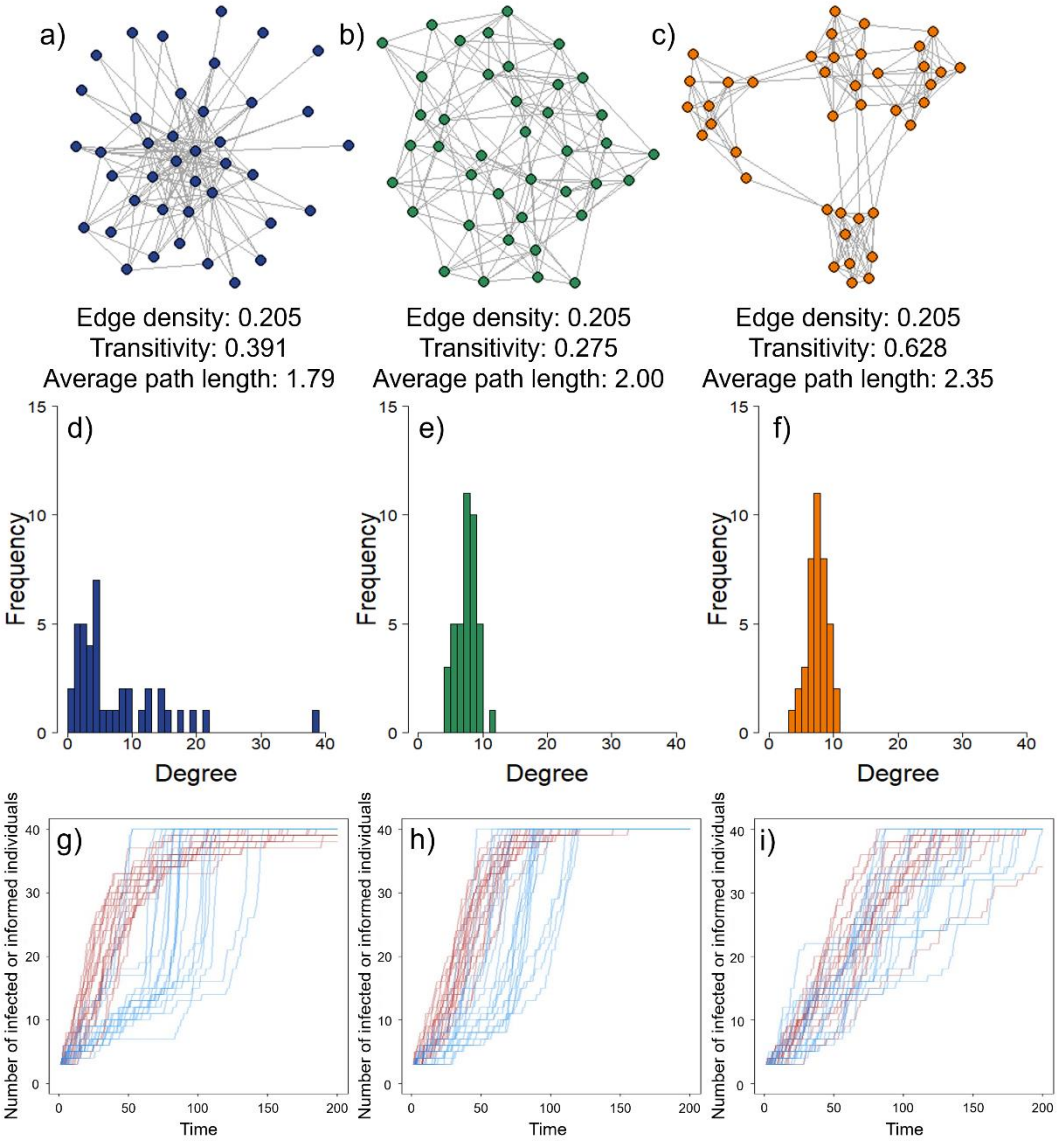

Figure 2. Demonstration of three key types of network structure with important implications for transmission. *The network structure (a-c), degree distribution (d-f) and transmission dynamics (g-i) of a simple contagion model for infection (red) and conformist contagion model for information (blue) are illustrated. All networks plotted here have the same edge densities (proportion of potential edges that are connected). Scale-free (or approximately scale-free) networks (a,d,g) have highly heterogeneous degree distributions (i.e. high variation in connectivity) with some high-degree (very well connected) individuals acting as "hubs", causing average path lengths to be short and resulting in very rapid spread of disease but slower spread of information. In small-world networks (b,e,h) most connections are to neighbours, but occasional long-range contacts act as "bridges", maintaining short average path lengths and enabling more rapid diffusion than random networks, and permitting faster spread via the cascade than the threshold model. In modular networks (c,f,i) most connections are to individuals in the same social community or module, resulting in high transitivity (or clustering of connections to 'friends of friends') and high average path lengths. Modular networks can have mixed effects on transmission speed that can depend on whether transmission follows a simple or conformist contagion dynamics. In this example, infection and information are able to spread at similar speeds through the modular network (i) but infection spreads more rapidly through scale-free (g) and small-world (h) network structures. Code for generating and plotting the networks and running the stochastic models is provided in the Supplementary Information.*

Heterogeneous degree distributions

Many animal social networks have highly heterogeneous degree distributions, with certain highly connected individuals acting as "hubs". Taking these differences in connectivity into account is important to understand transmission dynamics. For infectious disease spread, models show that more heterogeneous degree distributions increase the speed of epidemic spread and result in a higher prevalence of epidemic peaks due to the presence of highly connected superspreader individuals (Barthélemy, Barrat, Pastor-Satorras and Vespignani 2004, Lloyd-Smith, Schreiber, Kopp and Getz 2005), but reduce the frequency of epidemics (Lloyd-Smith, Schreiber, Kopp and Getz 2005). In more extreme situations where networks are truly scale-free, epidemics can spread almost instantaneously through populations (Barthélemy, Barrat, Pastor-Satorras and Vespignani 2004), making them especially vulnerable to infectious disease spread. For information transmission, the role of degree heterogeneity is more complex. In some contexts, individuals occupying globally central roles in a network are more likely to acquire information (Aplin, Farine, Morand-Ferron and Sheldon 2012, Jones, Aplin, Devost and Morand-Ferron 2017). However, when considering information transmission as complex contagion (as might be appropriate when individuals have conformity biases and accept information based on relative exposure), it is possible that individuals with many social connections might require stronger signals to distinguish a piece of information from the general "noise" received from their many associates (Hodas and Lerman 2012, Hodas and Lerman 2014). Conversely, lower-degree individuals may be more likely to utilise information sooner, as having a smaller number of ties means that fewer transmitting associates are required to achieve conformist transmission (González-Avella, Eguíluz, Marsili, Vega-Redondo and San Miguel 2011). Differences in the nature of transmission between information (when considered to spread through complex contagion) and infection may generate differences in the "most susceptible" network position between the two types of transmission that will reduce the intensity of any trade-off between the acquisition of information and infectious disease.

Networks with highly heterogeneous degree distributions will allow the rapid spread of infection and (often) information through populations. However, we hypothesise that information will spread more slowly than infection through these types of network when conformist social learning strategies are used. Individuals with a larger number of connections will require a larger proportion number of their associates to transmit the information in order to achieve the same relative magnitude of exposure, compared to less centrally positioned individuals. Hubs may therefore be slower to respond to information than less well-connected individuals. This will drive differences in which network positions are most likely to acquire information and those which are most likely to become infected. Being highly connected may be disproportionately risky in terms of the risk of infection per unit of social information acquired (and used), while being embedded within a network region (i.e. sharing contacts with your associates) will minimise the risk of becoming infected per unit of social information gathered.

Small-world networks

Small-world networks can arise as a result of the majority of social associations or interactions occurring mainly with close neighbours within groups, but with infrequent longer-range connections acting as "bridges" between regions of the network. It is easy to see how small-world properties might arise in animal networks through behaviours such as territoriality with occasional extra-territorial forays. For example, in African Lions *Panthera leo* contacts between prides are normally driven by space use, with prides from neighbouring territories coming into contact much more frequently. However, occasional contacts between prides that are normally spatially well-separated do occur, resulting in a network with small-world properties (Craft, Volz, Packer and Meyers 2009). In small-world networks the epidemic threshold (i.e. the transmission probability at which epidemics become possible) decreases considerably as the likelihood of long-range connections in the network increases (Moore and Newman 2000). For example, in a territorial, monogamous animal this would equate to epidemics of a sexually-transmitted infection becoming

458 more likely as extra-pair copulations occurred over greater distances rather than happening only
459 among neighbouring territories.

460 As with models of disease transmission, theoretical models predict that information flow will
461 be faster in small-world networks than random networks (Alkemade and Castaldi 2005, de Kerchove,
462 Krings, Lambiotte, Van Dooren and Blondel 2009, Delre, Jager and Janssen 2007, Nekovee, Moreno,
463 Bianconi and Marsili 2007) and the importance of multiple social contacts in enabling transmission
464 will be increased (de Kerchove, Krings, Lambiotte, Van Dooren and Blondel 2009). The findings of de
465 Kerchove, Krings, Lambiotte, Van Dooren and Blondel (2009) suggest that to spread information
466 effectively, an individual with long-range connections must have somewhat stable associations to
467 "seed" individuals within the region of the network it is connected to, as a single interaction may be
468 insufficient to enable transmission. If relative exposure to new information is important (i.e. it is
469 necessary for a threshold proportion of contacts to be informed before an individual accepts
470 information) then we can speculate that these "seed" individuals are more likely to be low-degree
471 individuals who adopt information more rapidly due to their small number of other connections. The
472 exception to this will occur if highly central individuals in the naïve group have a low information-use
473 threshold (i.e. they require few demonstrators to transmit the information before adopting it
474 themselves), which may be the case if individuals that acquire useful information tend to become
475 more central in networks or if the bridging individuals are extremely influential due to their social
476 status (or another trait).

477 ***Small-world networks are characterised by the importance of occasional long-range social***
478 ***connections involving small number of individuals. We suggest that the importance of these***
479 ***"bridge" individuals is easier to predict for infectious disease transmission, while for threshold***
480 ***models of information transmission their role will depend to a greater extent on the social learning***
481 ***rules of the individuals they are connected to, and therefore be more variable. We also predict that***
482 ***social dynamics will play a greater role in these small-world networks as the death of these***

"bridge" individuals or changes to their interactions will reduce the rate of global transmission of infection and information.

Modular network structure

Social networks with distinct social community structure are widespread in animals, especially in species living in stable social groups (Drewe, Madden and Pearce 2009, Weber, Carter, Dall, Delahay, McDonald, Bearhop and McDonald 2013, Weinrich 1991). Both community structure and transitivity (the tendency to be connected to 'friends of friends') reduce the size of infectious disease outbreaks but can lower the epidemic threshold. This makes it easier for less transmissible infections to spread, as the presence of many connections among the same set of individuals increases the probability of local spread, but these local connections reduce the probability of large-scale epidemics (Newman 2003, Sah, Leu, Cross, Hudson and Bansal 2017, Salathé and Jones 2010). The effect of modular structure is greater when interactions between individuals in different social communities are more infrequent so that sub-divisions between them are stronger (Sah, Leu, Cross, Hudson and Bansal 2017, Salathé and Jones 2010). A meta-analysis of animal social networks has shown that the impact of modularity on the spread of disease is limited except when there are very few connections between communities (Sah, Leu, Cross, Hudson and Bansal 2017). The impact of modularity will also depend on the transmissibility of the pathogen. For example, (Rozins, Silk, Croft, Delahay, Hodgson, McDonald, Weber and Boots 2018) demonstrated that the effect of the modular structure of empirically-derived European badger contact networks was greatest for simulated pathogens with intermediate transmissibility (i.e. infectious enough to cause an outbreak but not so infectious that it can spread easily between social groups).

In contrast, models suggest that modularity may not interfere with information diffusion in the same way. In some scenarios a modular network structure may actually increase global diffusion by enhancing within-community spreading. For example, Nematzadeh, Ferrara, Flammini and Ahn (2014) used a threshold model to show that conformist social learning strategies could lead to

information being transmitted most quickly in networks of intermediate modularity. The networks of species living in stable groups would therefore be expected to have reduced epidemic spreading, and potentially enhanced (or unchanged) information diffusion, as an outcome of being highly modular (Fig. 2). In this way, structural heterogeneity in animal social networks may mediate the trade-off between the transmission of information and infection, especially for group-living or fission-fusion social systems with more modular social networks.

We therefore suggest that a modular network structure may be critical in mediating the trade-off between minimising the spread of disease and maximising the spread of information. Community structure can promote the spread of social information when individuals follow conformist social learning strategies, while trapping infection within particular regions of the network. We predict that the dual selection pressures imposed by the access to information and the risk of acquiring disease will lead to natural selection generating modular network structures. The modularity of these structures will depend on the relative benefits information and costs of infectious disease o individuals, and the social learning strategies that they use.

Different types of associations - Multilayer relationships

As outlined in the previous section, different types of interaction will not all be equivalent for the transmission of infection or information. Considering how transmission dynamics vary for different types of interactions is therefore critical in understanding how animal societies might be adapted for efficient information transmission and minimal disease spread. Multilayer networks allow multiple interaction types to be incorporated within a single conceptual framework (Kivelä, Arenas, Barthelemy, Gleeson, Moreno and Porter 2014). A layer can denote different types of behavioural interaction between the same (or similar) set(s) of individuals, such as one layer for affiliative interactions and another for agonistic interactions (Finn, Silk, Porter and Pinter-Wollman 2019, Silk, Finn, Porter and Pinter-Wollman 2018). Layers can also consist of interactions between different types of individuals, such as different sexes (Silk, Weber, Steward, Hodgson, Boots, Croft,

Delahay and McDonald 2018) or species (Silk, Drewe, Delahay, Weber, Steward, Wilson-Aggarwal, Boots, Hodgson, Croft and McDonald 2018), with edges between layers representing interactions between those types of individuals.

Theoretical models using multilayer networks have been valuable in understanding the spread of a single pathogen or piece of information through multiple types of interaction, and the consequences of multiple spreading processes occurring across the same set of individuals (for example, multiple information types: Liu et al. 2018a, multiple pathogens: e.g. Azimi-Tafreshi 2016, or infection and information together: e.g. Funk et al. 2009; Granell, Gómez & Arenas 2013; Granell, Gómez & Arenas 2014; Guo et al. 2016, Funk and Jansen 2010, Marceau, Noël, Hébert-Dufresne, Allard and Dubé 2011, Zhao, Zheng and Liu 2014). Applying these approaches to animal behaviour research (Finn, Silk, Porter and Pinter-Wollman 2019, Silk, Finn, Porter and Pinter-Wollman 2018) requires data on multiple types of social connections simultaneously (e.g. Franz, Altmann and Alberts 2015, Gazda, Iyer, Killingback, Connor and Brault 2015), and quantification of the importance of these different social connections for transmission (e.g. Farine, Aplin, Sheldon and Hoppitt 2015).

Taking a multilayer approach also enables the integration of the indirect effect of positive and negative social relationships on transmission processes. Theoretical models on multilayer networks consider the effects of these different types of social relationships by modelling them as a type of transmission through the network, alongside infection and/or information. For example, one type of model considers the flow of social support that improves recovery rate from infection (which could, for example, represent the strength of affiliative relationships) on a second layer and has been used to show that social support can supress disease outbreaks, but that the effect is dependent on network structure and the correlation between the layers (Chen, Wang, Tang, Cai, Stanley and Braunstein 2018, Chen, Wang, Cai, Stanley and Braunstein 2018). Using models such as these enables the impact of social buffering to be integrated into network models, to determine how it may shape the trade-off between encountering useful information and risking infection. At its

simplest, if well-connected individuals are healthy and capable of resisting infection, then they do not face a trade-off at all.

Multiple spreading processes can also interact to promote or interfere with each other. For example, Liu, Wang, Cai, Tang and Lai (2018), when modelling the synergistic spread of multiple pieces of information transmitted simultaneously, showed that individuals having adopted one piece of information were subsequently more likely to adopt the other piece of information, one enhancing the other. A similar scenario in animal societies may be choosing to follow a particular individual's migratory route leading to an increased likelihood of socially learning a more efficient version of that route (Berdahl, Kao, Flack, Westley, Codling, Couzin, Dell and Biro 2018). . Alternatively, different types of information might compete, with one piece of information overriding/displacing the other (Kostka, Oswald and Wattenhofer 2008, Trpevski, Tang and Kocarev 2010). This could be important if the two pieces of information differ in their accuracy, or represent alternative strategies. Similarly, models suggest that multiple pathogens spreading on a multilayer network can promote (Azimi-Tafreshi 2016) or inhibit (Funk and Jansen 2010) each other's spread, and so are likely to be beneficial in understanding patterns of co-infection. When considering infection and information spread together, transmission models that integrate different transmission processes can provide fascinating insights (e.g. Funk, Gilad, Watkins and Jansen 2009, Granell, Gómez and Arenas 2013, Granell, Gómez and Arenas 2014, Guo, Lei, Jiang, Ma, Huo and Zheng 2016). For example, Funk, Gilad, Watkins and Jansen (2009) showed that information diffusing across a second network layer could slow epidemics, or even prevent the spread of infection across the first network layer, and that the impact of the information layer was amplified if it overlapped with the infection layer (i.e. had more similar patterns of interactions), or if the networks on each layer were highly clustered. These findings would suggest that if information about an infection can be spread through an animal social network via similar types of interaction as the infection itself, then infection avoidance behaviour can be much more effective in preventing the spread of disease. Social insect colonies offer a perfect candidate system through combining the feasibility of

experimental approaches, well documented roles for networks in information sharing (Preece and Beekman 2014), and evidence for adaptive changes to network structure in response to infection (Stroeymeyt, Grasse, Crespi, Mersch, Cremer and Keller 2018).

We predict that animal social systems will have evolved such that different network structures for different types of interactions can help facilitate rapid acquisition of information while minimising the risk of infection. Multilayer network analysis may provide a valuable tool in modelling the combined spread of different pathogens and/or different types of information. We expect that taking into account the full complexity of animal social systems using this approach will i) provide important new insights into transmission dynamics of both infection and disease and ii) reveal crucial information as to when trade-offs between the gathering of information and avoidance of infection actually arise (and when they do not), and iii) be critical in revealing how this balance can be mediated.

Social dynamics and the infection-information trade-off

Temporal heterogeneity and transmission in animals

Most animal social networks are dynamic, varying predictably over time (Hirsch, Reynolds, Gehrt and Craft 2016) or across different contexts (Smith, Gamboa, Spencer, Travenick, Ortiz, Hunter and Sih 2018). Accounting for these temporal changes can change how we understand transmission in animal social systems (e.g. Hirsch, Reynolds, Gehrt and Craft 2016, Springer, Kappeler and Nunn 2017). If social associations change faster than transmission occurs, then considering contacts as dynamic is important when using models to understand transmission through a network (Taylor, Taylor and Kiss 2012, Volz and Meyers 2007). While the presence of temporal changes to network structure in animals is well-established, very few empirical studies have considered temporal heterogeneity, or burstiness, in contact dynamics. “Bursty” contact dynamics consist of many contact events with a short gap between them, and occasional much longer gaps between contacts

(Fig. 3), such as the clustering of heterospecific associations around a watering hole in an arid environment. "bursty" contact dynamics cause temporal clustering of interactions, which can impede the transmission of infection as compared to a scenario where contacts are distributed more uniformly through time, because such clustered repeated exposure can result in connections that redundant from a transmission perspective. In contrast, bursty contact dynamics may enhance the spread of information for some social learning strategies, as repeated exposures to novel information in quick succession might enhance learning opportunities by passing the information "acceptance threshold" (see Karsai, Kivelä, Pan, Kaski, Kertész, Barabási and Saramäki 2011; Min, Goh & Kim 2013; c.f. Rocha, Liljeros & Holme 2011; Rocha & Blondel 2013). Consequently, varied temporal patterns of interactions could mitigate the potential trade-off between acquiring information and avoiding infection, especially for more "risky" interactions, such as between-group interactions in group-living species, which are more likely to be "bursty". Temporally clustered interactions with new individuals will disproportionately increase the likelihood of acquiring information relative to becoming infected. Recent theoretical models have incorporated both temporal heterogeneity and structural heterogeneity (e.g. community structure), demonstrating that regulation of spread is typically dominated by one or the other (Delvenne, Lambiotte and Rocha 2015). This suggests that the importance of heterogeneous contact dynamics for transmission in animal societies may vary systematically with other aspects of the social system, such as the stability of social groups.

We recommend research that focuses on the implications of temporal clustering of interactions and/or contacts for the spread of information and infectious disease, providing valuable insights into how individuals balance the costs and benefits of their social interactions. We predict that "bursty" contact dynamics could enhance the transmission of some types of information (depending on the social learning strategies of individuals), while having no effect or even reducing the risk of disease transmission – a good example may be lek mating systems. It would be possible to test these predictions in established experimental systems and then scale the

findings to a population or network level using stochastic models. We also expect that accounting for the effects of heterogeneous contact dynamics will be most important for species living in highly fluid societies rather than more stable social groups.

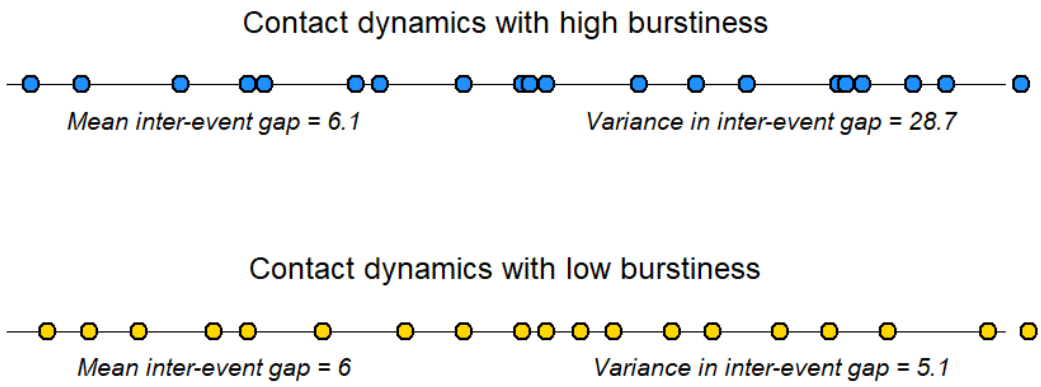

Figure 3. An illustration of differences in the burstiness of contact dynamics*. When contact dynamics are "bursty", there is a high variance in the gaps between contact events, resulting in clusters of contacts with occasional longer gaps. Bursty dynamics may promote the transmission of some types of information whilst reducing the risk of infection.*

Responsive network dynamics and transmission in animals

Animal social networks can change in response to the spread of infection (Croft, Edenbrow, Darden, Ramnarine, van Oosterhout and Cable 2011, Stroeymeyt, Grasse, Crespi, Mersch, Cremer and Keller 2018) and information (Kulahci, Ghazanfar and Rubenstein 2018) with a key difference between infection and information being that changes to network structure during the spread of infection may be determined by the phenotype of both the hosts and their parasites/pathogens (Franz, Kramer-Schadt, Greenwood and Courtiol 2018), while any adaptive changes to network structure in response to the acquisition of information are solely an outcome of selection on the "host", or "hosts" in the case of heterospecific transmission (Table 1). Theoretical models can provide some useful predictions as to how this affects transmission dynamics. Models where network connections can be altered in response to infection or information are referred to as adaptive network models (Bansal, Read, Pourbohloul and Meyers 2010, Funk, Bansal, Bauch, Eames, Edmunds, Galvani and Klepac 2015). The most common assumptions in disease modelling are that

individuals display infection-avoidance behaviour by either losing or reducing the strength of connections with infected individuals (e.g. Van Segbroeck, Santos and Pacheco 2010) or by replacing them with connections to other non-infected individuals (e.g. Shaw and Schwartz 2008). In the case of infectious disease, computational models indicate that adaptive networks typically have higher epidemic thresholds, delaying outbreaks and reducing peak prevalences (e.g. Gross, D'Lima and Blasius 2006, Shaw and Schwartz 2008, Van Segbroeck, Santos and Pacheco 2010). These behavioural responses to infection also frequently impact aspects of the network structure, for example by increasing variation in the connectivity of susceptible individuals and causing infected individuals to be much more poorly connected (Shaw and Schwartz 2008) or by increasing community structure with community membership assorted by infection state (Yang, Tang and Zhang 2012). While these changes reduce the impact of the current epidemic, they may make endemic disease more likely (Gross, D'Lima and Blasius 2006, Shaw and Schwartz 2008) or even result in long-term epidemic re-emergence (Zhou, Xiao, Cheong, Fu, Wong, Ma and Cheng 2012), which may have important implications for longer-term eco-evolutionary dynamics in animal populations. It is also possible for infection avoidance behaviour to exacerbate epidemics if individuals switch their connections from infected to susceptible individuals subsequent to being infected, although this remains relatively poorly explored (but see (Zhang, Small, Fu, Sun and Wang 2012)).

Many adaptive network models have previously assumed perfect knowledge about the infection status of other individuals, and this is unlikely to be the case in many natural host-pathogen systems. Identifying when information is available about the infection status of individuals relative to when the infection is most transmissible (Stephenson, Perkins and Cable 2018) will be crucial to understanding how "adaptive" changes to network structure can mediate the trade-off between information and infectious disease transmission. It may also be important to consider changes to the behaviour of infected individuals; sickness behaviour. Sickness behaviours in particular could be influenced by selection on hosts or parasites/pathogens. At times, sickness

684 behaviour could be favoured by both host and parasite (e.g. dispersal away from a highly related
685 group; Iritani and Iwasa 2014), but at other times optimal outcomes may be directly opposed (e.g.
686 reduction in number of contacts; Lopes, Block and König 2016) and generate antagonistic co-
687 evolution between the host and pathogen.

688 The results from equivalent models of information transmission are more diverse. One
689 model suggests that individuals may be more likely to cluster with those who are more inclined to
690 use information they are deliberately transmitting (Jackson and López-Pintado 2013), while another
691 model suggests individuals will break ties with those who do not use the information they
692 deliberately transmit (Zhang, Zhang, Weissing, Perc, Xie and Wang 2012). In some species of animal,
693 individuals may preferentially associate with those who will accept foraging information from them,
694 so as to maximise the likelihood of gaining benefits from recruiting others to feed (Wright, Stone and
695 Brown 2003). Similarly, a male displaying within a lek will attempt to maximise the number of
696 individuals who receive their signals, while also choosing to give up and stop transmitting to those
697 who are unlikely to mate with them (Patricelli and Krakauer 2009). In a similar manner to signalling
698 individuals manipulating their physical environment (e.g. birds singing from prominent perches),
699 individuals may also dynamically alter their social interactions so as to maximise their chances of
700 transmitting information to less informed node (Liu and Zhang 2014) if it is beneficial for them to do
701 so (e.g. in highly related groups). In contrast to the avoidance behaviour expected in response to the
702 spread of disease, “adaptive” behaviours that favour the acquisition of useful information while
703 minimising exposure to misinformation would be expected (Kulahci and Quinn 2019), depending on
704 previous interactions between the individuals involved. An individual who produces useful
705 information may be more likely to have others use that information in the future, while an individual
706 that frequently provides inaccurate information may be ignored (refractory behaviour). An
707 important caveat to this idea is that an individual who has previously produced useful information
708 may subsequently be more likely to cause misinformation to be transmitted (Pruitt, Wright, Keiser,
709 DeMarco, Grobis and Pinter-Wollman 2016), especially if the value of information changes over time

(e.g. by becoming outdated). This can be exploited by individuals aiming to transmit misinformation to manipulate the receivers' behaviour to their advantage, as is the case in fork-tailed drongos (*Dicrurus adsimilis*) who mimic other species' alarm calls to steal food from meerkats (Flower, Gribble and Ridley 2014). Whether drongos flexibly change their social associations with heterospecifics once they have been identified as cheats by the local meerkat group remains to be determined.

Considering behavioural dynamics alongside transmission dynamics is important to our understanding of how individuals may resolve the conflict between the acquisition and transmission of information and infection. Obvious signs of infection or regular transmission of misinformation can result in individuals becoming less well connected in a network while transmission of useful information can lead to the opposite pattern. We predict that behavioural plasticity that causes patterns of social interactions to be modified in the presence of infection or innovations will therefore be a key mechanism by which this balance between the costs and benefits of being highly socially connected is mediated and expect that these behavioural dynamics are much more widespread than previously described. Behavioural dynamics are also likely to be closely interlinked with network structure, and we predict that behavioural responses to infectious disease and information will co-vary with social structure (especially group dynamics and modularity) between populations.

Future research priorities

Our review highlights several key priorities for future research. First, it is essential that we continue to build on our understanding of how infection and information are transmitted through natural populations. In particular, discovering how widespread the use of complex social learning strategies is in animals will be critical in revealing whether particular social network structures, and particular social network positions within them, favour the transfer or acquisition of information

over and above that of infectious disease. Second, a renewed effort to consider the dynamic and multilayer structure of animal social networks is also crucial to understanding differences between these two ecological processes, especially when they spread through different but overlapping sets of interactions. Third, the development of data-based evolutionary models will be required to fully understand the implications of these differences in the transmission of infection and information for the evolution of animal social systems.

Conclusions

Social network structure is fundamental to both the transmission of information and infectious disease through populations. Both represent important selection pressures on how individuals structure their social interactions. Individuals face a trade-off to maximise the acquisition of reliable information while minimising the risk of becoming infected with parasites and pathogens. However, our understanding of this trade-off is complicated by how these processes depend on social network structure in different ways. The risk of acquiring infection typically increases monotonically with the frequency and duration of interaction with infectious individuals. In contrast, information acquisition is more complex, with the likelihood of accepting information often depending on exposure to that information in a non-linear fashion. For example, empirical evidence from some animal social networks suggests that acquisition of information might often be a threshold trait. A receiver's threshold of exposure could be determined by the proportion of associates demonstrating the behaviour, or could be determined more broadly by the identity, influence or traits of transmitters (e.g. social learning directed by dominance, familiarity, relatedness). Information transmission is also complicated by the sharing of both good and bad (or out-dated) information, and by "refractory" behaviours among recipients that result in the acquisition of information not affecting the behaviour of all individuals in the same way.

Crucially, these differences in the nature of transmission and the types of interactions that result in transmission can mediate the apparent trade-off between acquiring information and

760 infection in social systems. Furthermore, plasticity of social behaviour can generate changes to social
761 structures that can protect against the spread of disease or promote the spread of information. In
762 this way, behavioural plasticity is likely critical in regulating infection risk and information benefits
763 obtained by social animals. Information transmission is often integral to behavioural responses to
764 avoid infection, making quantifying differences in how information and infection are transmitted and
765 their different routes of transmission even more important. Consequently, our understanding of the
766 interplay between information and infection in shaping animal social systems requires a better grasp
767 of how transmission is affected by the structural, temporal and multi-layered heterogeneities that
768 are inherent to animal social networks.
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