

# 1 **Infected or informed? Social structure and the simultaneous**

## 2 **transmission of information and infectious disease**

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## 30 **Abstract**

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

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

48

## 49 **Introduction**

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

60           Social network analyses and modelling are increasingly used to study animal social  
61 behaviour, and these approaches have greatly benefitted research into disease and information  
62 transmission in wild animals. Interactions with more individuals result in increased opportunities for  
63 infectious disease spread, while simultaneously making it possible for information to proliferate  
64 within a group or population (Wey, Blumstein, Shen and Jordán 2008, White, Forester and Craft  
65 2017). However, studying the trade-off between efficient information transmission and rapid spread  
66 of disease is challenging due to the difficulty of combining data that reveal infection status, and data  
67 that indicate information transmission. Consequently, while studies have examined how social  
68 relationships impact the transmission of either infection or information separately, there has been  
69 little consideration of the interplay between information and infection spread within animal social  
70 networks.

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

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

81

## 82 **Infectious disease transmission in animal social networks**

83         The transmission of parasites and pathogens that cause infectious disease can happen  
84 directly via specific types of behavioural interaction or indirectly via the environment (e.g. an  
85 individual using a refuge that has been contaminated by an infectious individual) (White, Forester  
86 and Craft 2017). Many infections are endemic, persisting stably within a host population for a long  
87 period of time (Viana, Mancy, Biek, Cleaveland, Cross, Lloyd-Smith and Haydon 2014). Others are  
88 emergent, acquired either from a long-term environmental reservoir or spilling over from alternative  
89 host species (Daszak, Cunningham and Hyatt 2000). Networks of spatial associations and behavioural  
90 interactions are now known to be closely associated with infectious disease epidemiology in wild  
91 animal populations (e.g VanderWaal, Atwill, Isbell and McCowan 2014, Weber, Carter, Dall, Delahay,  
92 McDonald, Bearhop and McDonald 2013). Overall network structure is critical in determining how  
93 infectious disease spreads through populations. For example, the presence of distinct social  
94 communities can limit the spread of infection in animal groups or populations (Griffin and Nunn  
95 2012, Sah, Leu, Cross, Hudson and Bansal 2017). Social network analysis can also help identify  
96 potential routes of transmission (Silk, Croft, Delahay, Hodgson, Boots, Weber and McDonald 2017,  
97 White, Forester and Craft 2017), determines individual variation in transmission potential  
98 (VanderWaal and Ezenwa 2016) and predicts or explains how infection spreads through populations

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

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

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

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

134

## 135 **Information transmission in animal social networks**

136 Information can be acquired by sampling the environment (personal information; Dall,  
137 Giraldeau, Olsson, McNamara and Stephens 2005) or by observing or interacting with other  
138 individuals or their products (social information; Dall, Giraldeau, Olsson, McNamara and Stephens  
139 2005, Danchin, Giraldeau, Valone and Wagner 2004). Individuals can spread social information  
140 inadvertently or can choose to deliberately transmit information via signals. A receiver must then  
141 decide whether to act on this information or not (Dall, Giraldeau, Olsson, McNamara and Stephens  
142 2005, Schmidt, Dall and Van Gils 2010). The transfer of social information usually requires sensory  
143 contact between individuals and is therefore linked directly to spatial association and/or behavioural  
144 interactions. Consequently, as with infectious disease transmission, an individual's social network  
145 position causes variation in the probability and rate of receipt of information, and their contribution  
146 to the speed and quality of information transmission through a population (Lusseau 2003, Lusseau  
147 and Newman 2004, Modlmeier, Keiser, Watters, Sih and Pruitt 2014). Depending on the duration  
148 information is useful, an individual's network position will strongly influence how they can utilise this  
149 information. For example, information such as the discovery of a resource location (Aplin, Farine,

150 Morand-Ferron and Sheldon 2012, Blonder and Dornhaus 2011, Webster, Atton, Hoppitt and Laland  
151 2013) may only be accurate for a short time if a resource is ephemeral or is rapidly depleted. A  
152 central network position or high level of connectivity to the individual who initially discovers such  
153 resources will be highly beneficial to potential recipients, as demonstrated in several studies of the  
154 influence of network position on food patch discovery in flocks of songbirds (Aplin, Farine, Morand-  
155 Ferron and Sheldon 2012, Farine, Aplin, Sheldon and Hoppitt 2015, Jones, Aplin, Devost and  
156 Morand-Ferron 2017, Tóth, Tuliozi, Baldan, Hoi and Griggio 2017). Therefore, when information-  
157 gathering is beneficial, group members may be attracted to individuals who regularly provide  
158 information, changing their position in the social network. In ring tailed lemurs *Lemur catta*, for  
159 example, this led to informed individuals occupying more central network positions (Kulahci,  
160 Ghazanfar and Rubenstein 2018).

161 Social associations are also linked to the spread, through social learning, of behavioural  
162 innovations which can arise via trial and error learning (Allen, Weinrich, Hoppitt and Rendell 2013,  
163 Aplin, Farine, Morand-Ferron, Cockburn, Thornton and Sheldon 2014). Such innovations range from  
164 simply adopting a new foraging ground (Schakner, Petelle, Tennis, Leeuw, Stansell and Blumstein  
165 2017) to tool use (Coelho, Falótico, Izar, Mannu, Resende, Siqueira and Ottoni 2015, Hobaiter,  
166 Poisot, Zuberbühler, Hoppitt and Gruber 2014, Mann, Stanton, Patterson, Bienenstock and Singh  
167 2012, St Clair, Burns, Bettaney, Morrissey, Otis, Ryder, Fleischer, James and Rutz 2015) or novel  
168 foraging techniques (Aplin, Farine, Morand-Ferron, Cockburn, Thornton and Sheldon 2014, Boogert,  
169 Nightingale, Hoppitt and Laland 2014, Kendal, Custance, Kendal, Vale, Stoinski, Rakotomalala and  
170 Rasamimanana 2010). Innovations of long-term value can be transmitted to subsequent generations  
171 (Aplin, Farine, Morand-Ferron, Cockburn, Thornton and Sheldon 2014, Cantor and Whitehead) and  
172 impact long-term social structure, provided individuals alter their social interactions to maximise  
173 their chances of acquiring information (Coelho, Falótico, Izar, Mannu, Resende, Siqueira and Ottoni  
174 2015, Kulahci, Ghazanfar and Rubenstein 2018). One possible outcome is that long-term,  
175 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, Rieucan 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



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

208

## 209 **Differences between information and disease transmission**

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

215

216 Table 1: Summary of the general key differences in mechanisms and consequences of information and infectious disease  
 217 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.

218

219 Most importantly, information transmission will typically involve choice, sometimes for a  
 220 transmitter, who can choose when to transmit information and to whom, and always for the  
 221 receiver, who chooses whether to alter their behaviour based on the information. Choice by the  
 222 receiver means that the social transmission of information does not necessarily depend on a simple  
 223 probability of transmission associated with each interaction (Bakshy, Karrer and Adamic 2009,

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

240 Another key difference between information and infection is the effect that prior social  
241 associations can have on the likelihood of transmission. The current and previous social relationships  
242 of an individual can directly impact the probability of using information acquired through a particular  
243 social interaction. This phenomenon was first coined “directed social learning” (Coussi-Korbel and  
244 Fragasz 1995) and later described as one of many potential “social learning strategies”(Laland  
245 2004). Some of the clearest evidence of such a “Whom to learn from” social learning strategy comes  
246 from the importance of familiarity for the rate of social learning in many species (e.g. Kavaliers,  
247 Colwell and Choleris 2005, Swaney, Kendal, Capon, Brown and Laland 2001). Information from a  
248 familiar individual may result in an immediate change in behaviour, whereas an animal may require  
249 more exposures to a piece of information if the source is unfamiliar. Transmitter familiarity is one of

250 several relationship traits that might influence the decision to use a piece of information, with traits  
251 such as relatedness or social rank also potentially important (Boogert, Lachlan, Spencer, Templeton  
252 and Farine 2018, Evans, Jones and Morand-Ferron 2018, Farine, Spencer and Boogert 2015,  
253 Kavaliers, Colwell and Choleris 2005, Radford 2004, Valsecchi, Choleris, Moles, Guo and Mainardi  
254 1996). Relationship traits can also interact with the phenotype of the transmitter, such as their  
255 experience (McComb, Moss, Durant, Baker and Sayialel 2001) or obvious fitness cues (Toth and  
256 Griggio 2011), to shape the likelihood of information being used. Similarly, it is possible for prior  
257 social relationships with other group members to have a profound effect on the health of individuals  
258 in social species (Sapolsky 2005), and the social buffering hypothesis (Ezenwa, Ghai, McKay and  
259 Williams 2016) proposes that positive social relationships can increase resistance to, and tolerance  
260 of, infection in group-living species (e.g. Almborg, Cross, Dobson, Smith, Metz, Stahler and Hudson  
261 2015, Balasubramaniam, Beisner, Vandeleest, Atwill and McCowan 2016, Ezenwa and Worsley-Tonks  
262 2018, Scharf, Modlmeier, Beros and Foitzik 2012, Walker and Hughes 2009). However, unlike the  
263 spread of information, this is a general effect and specific prior relationships with infected  
264 individuals do not influence the transmission process in the same way that prior relationships with  
265 informed individuals do.

266 Another important consideration is *how* information and infection are transmitted. Social  
267 information can be transmitted in multiple ways (Blanchet, Clobert and Danchin 2010, Danchin,  
268 Giraldeau, Valone and Wagner 2004), which may require prolonged or close interactions (e.g. the  
269 waggle dance in bees; Von Frisch 1967), may be possible with much looser associations (e.g. auditory  
270 cues; Hollen and Radford 2009), or may be transmitted indirectly via environmental signals or cues  
271 (e.g. scent marking: Gosling and Roberts 2001). Conversely, infectious disease is likely to be  
272 transmitted through a different set of interactions, such as prolonged close contact that facilitates  
273 aerosol transmission (Delahay, Cheeseman and Clifton-Hadley 2001) ; shared use of environmental  
274 reservoirs of pathogens (Godfrey, Bull, James and Murray 2009); aggressive interactions or mating  
275 (Hamilton, Jones, Cameron, McCallum, Storfer, Hohenlohe and Hamede 2019) . The extent of the

276 overlap in the types of social interaction that expose individuals to either information or infection  
277 will be important in determining the costs and benefits of being central in different types of social  
278 network.

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

295

296           The outcome of these differences between the transmission of infection and information is  
297 that while infection can be considered a simple contagion process, the spread of information is  
298 increasingly considered a complex contagion (Centola 2010, Macy 1991) affected by many of the  
299 social learning rules described above. Consequently, the spread of infectious disease has normally  
300 been modelled as a simple contagion using cascade models (Moore and Newman 2000), in which the  
301 probability of infection increases with increasing absolute exposure to infected individuals. In

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

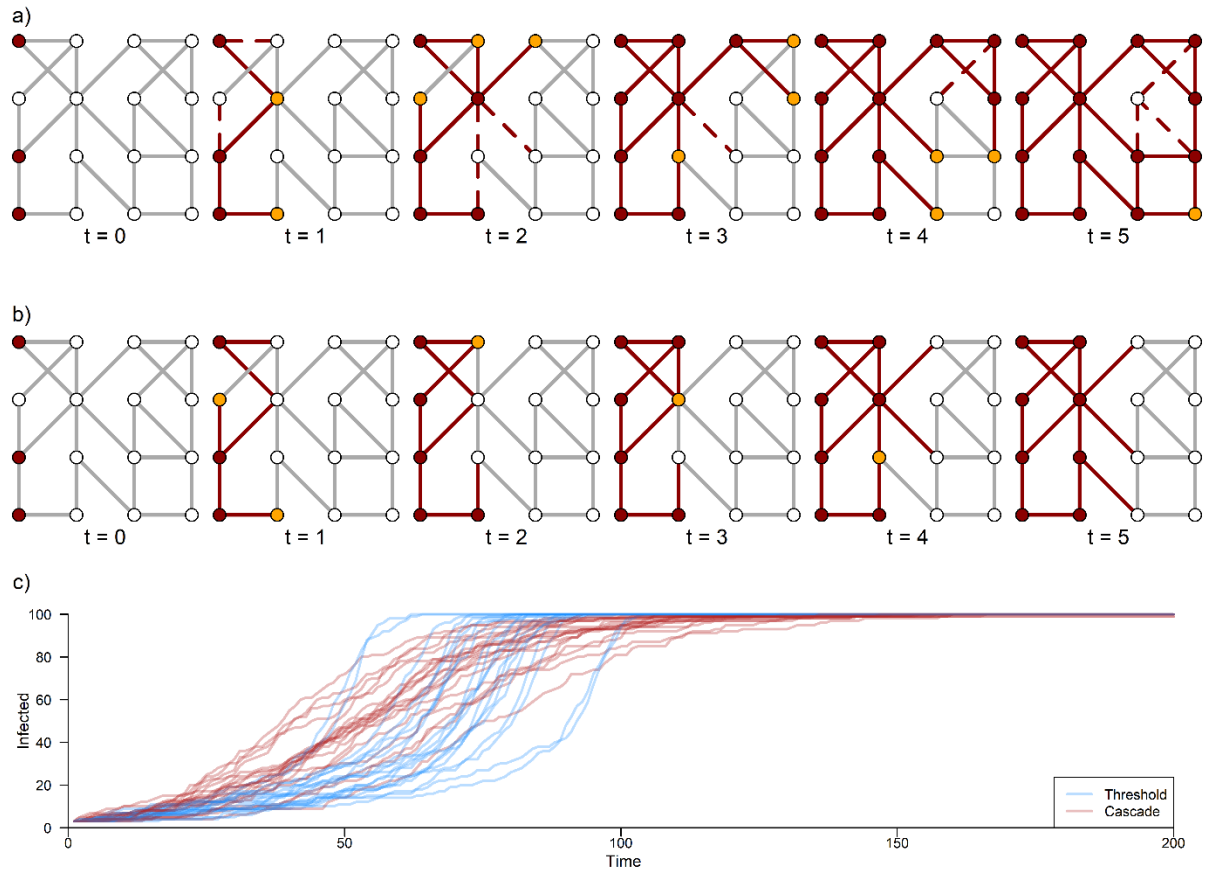
316

## 317 **Using models to capture the differences between** 318 **information and disease transmission**

319 The similarities and differences between information and disease transmission can be  
320 captured using dynamic computational modelling tools (Fig. 1), such as compartmental models.  
321 Compartmental models consider the transition of individuals between states, with individuals in  
322 each state assumed to have the same characteristics (Stattner and Vidot 2011). For example, a  
323 susceptible-infected-recovered (SIR) model (used commonly in epidemiology) contains three states:  
324 susceptible (or naïve) individuals; infected with a parasite (or exposed to and exploiting the  
325 information); and recovered individuals who are now immune to that infection (or who no longer

326 use the information to inform their behaviour). When applied to transmission through networks,  
327 compartmental models are typically applied as stochastic individual-based models, in which the  
328 transition of each individual between compartments is modelled separately and depends on the  
329 properties of their network connections. Such models are usually impossible to solve analytically  
330 (Craft 2015). These models avoid the assumption that populations mix freely, and hence that any  
331 individual will be able to infect any other individual in a population. General compartmental models  
332 applied to networks can be used to study infectious disease transmission, information flow, or both  
333 simultaneously (See supplementary table 1 for examples of simple compartmental models that can  
334 be applied to both information and disease and those more suitable for detailed models of particular  
335 transmission types).

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



347

348 Figure 1. a) Example of a cascade model of simple contagion acting on an unweighted network (all individuals have an  
 349 association strength of either 1 or 0) of 16 individuals over four time steps. Grey lines represent social associations, red  
 350 nodes represent infected/informed individuals while yellow nodes represent individuals who will become  
 351 infected/informed in the next time step. In this cascade model an infected/informed individual infects/informs each  
 352 uninfected neighbour with a probability of 0.5 per time step. Solid red lines indicate an infected/informed node  
 353 successfully infecting/informing a neighbour, while a dashed line represents a failure. If successful, the neighbour will  
 354 become infected/informed in the next time step. b) A conformist transmission model (here a true threshold model, but a  
 355 stochastic implementation would produce similar results) acting on the same network as a). Individuals become  
 356 infected/informed when 50% of their neighbours are infected/informed. In this simulation, spread stalls at timestep 4 as  
 357 there are not enough infected/informed individuals to result in transmission. c) Comparison between simple and  
 358 conformist contagion models in a random network of 100 individuals, showing the percentage of the population infected  
 359 over 200 arbitrary time-steps. For the simple contagion model there is a probability of 0.8% chance per time-step that  
 360 infection is transmitted through an edge between an infected and susceptible individual. In the conformist model a sigmoid  
 361 curve is fitted to the likelihood of an individual exploiting information with a baseline (asocial) individual learning rate of  
 362 0.2% per time-step, a maximum probability of learning of 30% per time-step and the threshold (pivot point of the sigmoid  
 363 function) occurring at 50% of connected individuals providing information. Full R code for the model is provided in the  
 364 Supplementary information.

## 365 Social structure and the infection-information trade-off

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



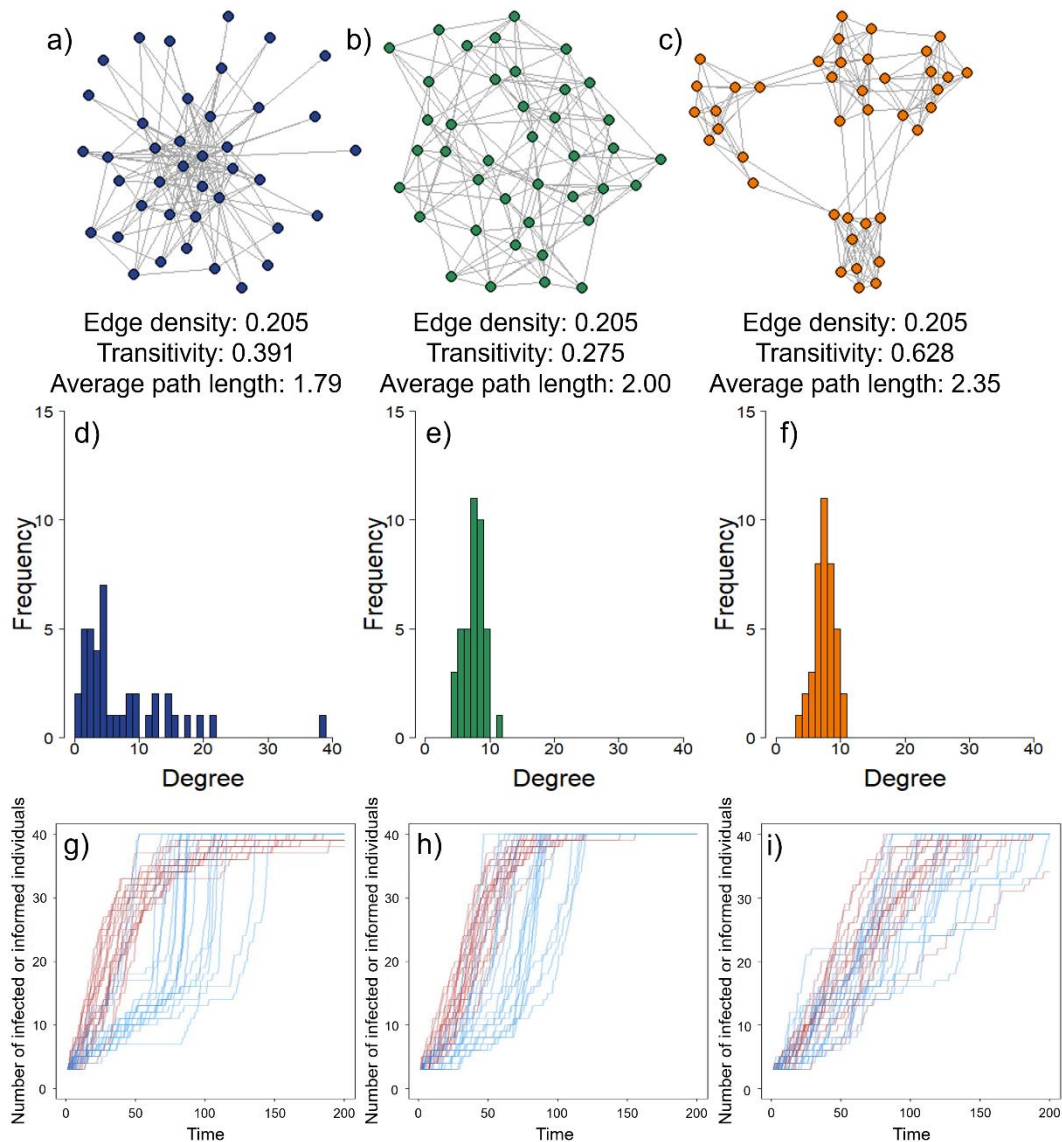
369 of structural heterogeneity in social networks, temporal heterogeneity in interactions, responsive  
370 changes in social interactions and the role of different types of interaction. We integrate the extent  
371 of our current knowledge of animal social systems with insights from compartmental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

373

#### 374 *Structural heterogeneity and transmission in animals*

375 The structure of contact networks is integral to transmission dynamics for cascade models  
376 (Moore and Newman 2000, Newman 2002) and threshold models (Alkemade and Castaldi 2005,  
377 Hodas and Lerman 2014). We focus on three aspects of social network structure that have received  
378 considerable research interest and have clear applications to the study of animal societies: i)  
379 variation in connectivity among individuals causing networks to possess heterogeneous degree  
380 distributions (the extreme case being networks with scale-free properties), ii) modular structure that  
381 is characterised by densely connected regions (called communities) with rather few connections  
382 between these communities, and iii) small-world structure, which is best envisaged as individuals (or  
383 “nodes”) being connected mostly with (spatial) neighbours, but possessing occasional contacts with  
384 much more distant nodes, resulting in transmission pathways through the network that are typically  
385 short compared to random or modular networks. We depict these different aspects of network  
386 structure in Fig. 2. Animal social structure is highly variable and can display one or multiple of the  
387 scale-free, small-world or modular properties introduced here (Wey, Blumstein, Shen and Jordán  
388 2008).

389



390

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

406

407 *Heterogeneous degree distributions*

408           Many animal social networks have highly heterogeneous degree distributions, with certain  
409 highly connected individuals acting as “hubs”. Taking these differences in connectivity into account is  
410 important to understand transmission dynamics. For infectious disease spread, models show that  
411 more heterogeneous degree distributions increase the speed of epidemic spread and result in a  
412 higher prevalence of epidemic peaks due to the presence of highly connected superspreader  
413 individuals (Barthélemy, Barrat, Pastor-Satorras and Vespignani 2004, Lloyd-Smith, Schreiber, Kopp  
414 and Getz 2005), but reduce the frequency of epidemics (Lloyd-Smith, Schreiber, Kopp and Getz  
415 2005). In more extreme situations where networks are truly scale-free, epidemics can spread almost  
416 instantaneously through populations (Barthélemy, Barrat, Pastor-Satorras and Vespignani 2004),  
417 making them especially vulnerable to infectious disease spread. For information transmission, the  
418 role of degree heterogeneity is more complex. In some contexts, individuals occupying globally  
419 central roles in a network are more likely to acquire information (Aplin, Farine, Morand-Ferron and  
420 Sheldon 2012, Jones, Aplin, Devost and Morand-Ferron 2017). However, when considering  
421 information transmission as complex contagion (as might be appropriate when individuals have  
422 conformity biases and accept information based on relative exposure), it is possible that individuals  
423 with many social connections might require stronger signals to distinguish a piece of information  
424 from the general “noise” received from their many associates (Hodas and Lerman 2012, Hodas and  
425 Lerman 2014). Conversely, lower-degree individuals may be more likely to utilise information  
426 sooner, as having a smaller number of ties means that fewer transmitting associates are required to  
427 achieve conformist transmission (González-Avella, Eguíluz, Marsili, Vega-Redondo and San Miguel  
428 2011). Differences in the nature of transmission between information (when considered to spread  
429 through complex contagion) and infection may generate differences in the “most susceptible”  
430 network position between the two types of transmission that will reduce the intensity of any trade-  
431 off between the acquisition of information and infectious disease.

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

444

#### 445 *Small-world networks*

446           Small-world networks can arise as a result of the majority of social associations or  
447 interactions occurring mainly with close neighbours within groups, but with infrequent longer-range  
448 connections acting as “bridges” between regions of the network. It is easy to see how small-world  
449 properties might arise in animal networks through behaviours such as territoriality with occasional  
450 extra-territorial forays. For example, in African Lions *Panthera leo* contacts between prides are  
451 normally driven by space use, with prides from neighbouring territories coming into contact much  
452 more frequently. However, occasional contacts between prides that are normally spatially well-  
453 separated do occur, resulting in a network with small-world properties (Craft, Volz, Packer and  
454 Meyers 2009). In small-world networks the epidemic threshold (i.e. the transmission probability at  
455 which epidemics become possible) decreases considerably as the likelihood of long-range  
456 connections in the network increases (Moore and Newman 2000). For example, in a territorial,  
457 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***

483 ***“bridge” individuals or changes to their interactions will reduce the rate of global transmission of***  
484 ***infection and information.***

485

#### 486 *Modular network structure*

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

505 In contrast, models suggest that modularity may not interfere with information diffusion in  
506 the same way. In some scenarios a modular network structure may actually increase global diffusion  
507 by enhancing within-community spreading. For example, Nematzadeh, Ferrara, Flammini and Ahn  
508 (2014) used a threshold model to show that conformist social learning strategies could lead to

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

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

523

#### 524 *Different types of associations - Multilayer relationships*

525 As outlined in the previous section, different types of interaction will not all be equivalent  
526 for the transmission of infection or information. Considering how transmission dynamics vary for  
527 different types of interactions is therefore critical in understanding how animal societies might be  
528 adapted for efficient information transmission and minimal disease spread. Multilayer networks  
529 allow multiple interaction types to be incorporated within a single conceptual framework (Kivelä,  
530 Arenas, Barthelemy, Gleeson, Moreno and Porter 2014). A layer can denote different types of  
531 behavioural interaction between the same (or similar) set(s) of individuals, such as one layer for  
532 affiliative interactions and another for agonistic interactions (Finn, Silk, Porter and Pinter-Wollman  
533 2019, Silk, Finn, Porter and Pinter-Wollman 2018). Layers can also consist of interactions between  
534 different types of individuals, such as different sexes (Silk, Weber, Steward, Hodgson, Boots, Croft,

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

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

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



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

562 Multiple spreading processes can also interact to promote or interfere with each other. For  
563 example, Liu, Wang, Cai, Tang and Lai (2018), when modelling the synergistic spread of multiple  
564 pieces of information transmitted simultaneously, showed that individuals having adopted one piece  
565 of information were subsequently more likely to adopt the other piece of information, one  
566 enhancing the other. A similar scenario in animal societies may be choosing to follow a particular  
567 individual's migratory route leading to an increased likelihood of socially learning a more efficient  
568 version of that route (Berdahl, Kao, Flack, Westley, Codling, Couzin, Dell and Biro 2018).  
569 Alternatively, different types of information might compete, with one piece of information  
570 overriding/displacing the other (Kostka, Oswald and Wattenhofer 2008, Trpevski, Tang and Kocarev  
571 2010). This could be important if the two pieces of information differ in their accuracy, or represent  
572 alternative strategies. Similarly, models suggest that multiple pathogens spreading on a multilayer  
573 network can promote (Azimi-Tafreshi 2016) or inhibit (Funk and Jansen 2010) each other's spread,  
574 and so are likely to be beneficial in understanding patterns of co-infection. When considering  
575 infection and information spread together, transmission models that integrate different  
576 transmission processes can provide fascinating insights (e.g. Funk, Gilad, Watkins and Jansen 2009,  
577 Granell, Gómez and Arenas 2013, Granell, Gómez and Arenas 2014, Guo, Lei, Jiang, Ma, Huo and  
578 Zheng 2016). For example, Funk, Gilad, Watkins and Jansen (2009) showed that information diffusing  
579 across a second network layer could slow epidemics, or even prevent the spread of infection across  
580 the first network layer, and that the impact of the information layer was amplified if it overlapped  
581 with the infection layer (i.e. had more similar patterns of interactions), or if the networks on each  
582 layer were highly clustered. These findings would suggest that if information about an infection can  
583 be spread through an animal social network via similar types of interaction as the infection itself,  
584 then infection avoidance behaviour can be much more effective in preventing the spread of disease.  
585 Social insect colonies offer a perfect candidate system through combining the feasibility of

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

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

598

## 599 **Social dynamics and the infection-information trade-off**

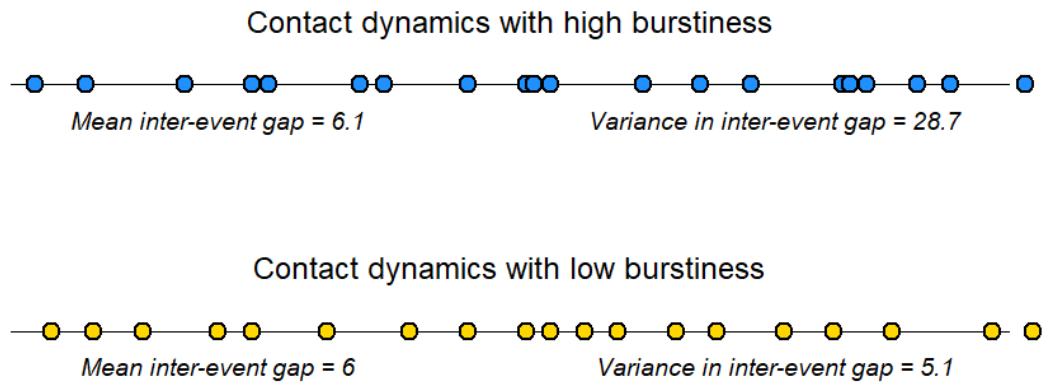
### 600 **Temporal heterogeneity and transmission in animals**

601 Most animal social networks are dynamic, varying predictably over time (Hirsch, Reynolds,  
602 Gehrt and Craft 2016) or across different contexts (Smith, Gamboa, Spencer, Travenick, Ortiz, Hunter  
603 and Sih 2018). Accounting for these temporal changes can change how we understand transmission  
604 in animal social systems (e.g. Hirsch, Reynolds, Gehrt and Craft 2016, Springer, Kappeler and Nunn  
605 2017). If social associations change faster than transmission occurs, then considering contacts as  
606 dynamic is important when using models to understand transmission through a network (Taylor,  
607 Taylor and Kiss 2012, Volz and Meyers 2007). While the presence of temporal changes to network  
608 structure in animals is well-established, very few empirical studies have considered temporal  
609 heterogeneity, or burstiness, in contact dynamics. “Bursty” contact dynamics consist of many  
610 contact events with a short gap between them, and occasional much longer gaps between contacts

611 (Fig. 3), such as the clustering of heterospecific associations around a watering hole in an arid  
612 environment. “bursty” contact dynamics cause temporal clustering of interactions, which can  
613 impede the transmission of infection as compared to a scenario where contacts are distributed more  
614 uniformly through time, because such clustered repeated exposure can result in connections that  
615 redundant from a transmission perspective. In contrast, bursty contact dynamics may enhance the  
616 spread of information for some social learning strategies, as repeated exposures to novel  
617 information in quick succession might enhance learning opportunities by passing the information  
618 “acceptance threshold” (see Karsai, Kivelä, Pan, Kaski, Kertész, Barabási and Saramäki 2011; Min,  
619 Goh & Kim 2013; c.f. Rocha, Liljeros & Holme 2011; Rocha & Blondel 2013). Consequently, varied  
620 temporal patterns of interactions could mitigate the potential trade-off between acquiring  
621 information and avoiding infection, especially for more “risky” interactions, such as between-group  
622 interactions in group-living species, which are more likely to be “bursty”. Temporally clustered  
623 interactions with new individuals will disproportionately increase the likelihood of acquiring  
624 information relative to becoming infected. Recent theoretical models have incorporated both  
625 temporal heterogeneity and structural heterogeneity (e.g. community structure), demonstrating  
626 that regulation of spread is typically dominated by one or the other (Delvenne, Lambiotte and Rocha  
627 2015). This suggests that the importance of heterogeneous contact dynamics for transmission in  
628 animal societies may vary systematically with other aspects of the social system, such as the stability  
629 of social groups.

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

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



640

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

644

### 645 **Responsive network dynamics and transmission in animals**

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

658 individuals display infection-avoidance behaviour by either losing or reducing the strength of  
659 connections with infected individuals (e.g. Van Segbroeck, Santos and Pacheco 2010) or by replacing  
660 them with connections to other non-infected individuals (e.g. Shaw and Schwartz 2008). In the case  
661 of infectious disease, computational models indicate that adaptive networks typically have higher  
662 epidemic thresholds, delaying outbreaks and reducing peak prevalences (e.g. Gross, D’Lima and  
663 Blasius 2006, Shaw and Schwartz 2008, Van Segbroeck, Santos and Pacheco 2010). These  
664 behavioural responses to infection also frequently impact aspects of the network structure, for  
665 example by increasing variation in the connectivity of susceptible individuals and causing infected  
666 individuals to be much more poorly connected (Shaw and Schwartz 2008) or by increasing  
667 community structure with community membership assorted by infection state (Yang, Tang and  
668 Zhang 2012). While these changes reduce the impact of the current epidemic, they may make  
669 endemic disease more likely (Gross, D’Lima and Blasius 2006, Shaw and Schwartz 2008) or even  
670 result in long-term epidemic re-emergence (Zhou, Xiao, Cheong, Fu, Wong, Ma and Cheng 2012),  
671 which may have important implications for longer-term eco-evolutionary dynamics in animal  
672 populations. It is also possible for infection avoidance behaviour to exacerbate epidemics if  
673 individuals switch their connections from infected to susceptible individuals subsequent to being  
674 infected, although this remains relatively poorly explored (but see (Zhang, Small, Fu, Sun and Wang  
675 2012)).

676           Many adaptive network models have previously assumed perfect knowledge about the  
677 infection status of other individuals, and this is unlikely to be the case in many natural host-  
678 pathogen systems. Identifying when information is available about the infection status of individuals  
679 relative to when the infection is most transmissible (Stephenson, Perkins and Cable 2018) will be  
680 crucial to understanding how “adaptive” changes to network structure can mediate the trade-off  
681 between information and infectious disease transmission. It may also be important to consider  
682 changes to the behaviour of infected individuals; sickness behaviour. Sickness behaviours in  
683 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

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

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

728

## 729 **Future research priorities**

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

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

## 741 **Conclusions**

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

758 Crucially, these differences in the nature of transmission and the types of interactions that  
759 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.

769

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