



The role of social structure and dynamics in the maintenance of endemic disease

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Abstract

Social interactions are required for the direct transmission of infectious diseases. Consequently, the social network structure of populations plays a key role in shaping infectious disease dynamics. A huge research effort has examined how specific social network structures make populations more (or less) vulnerable to damaging epidemics. However, it can be just as important to understand how social networks can contribute to endemic disease dynamics, in which pathogens are maintained at stable levels for prolonged periods of time. Hosts that can maintain endemic disease may serve as keystone hosts for multi-host pathogens within an ecological community, and also have greater potential to act as key wildlife reservoirs of agricultural and zoonotic diseases. Here, we examine combinations of social and demographic processes that can foster endemic disease in hosts. We synthesise theoretical and empirical work to demonstrate the importance of both social structure and social dynamics in maintaining endemic disease. We also highlight the importance of distinguishing between the local and global persistence of infection and reveal how different social processes drive variation in the scale at which infectious diseases appear endemic. Our synthesis provides a framework by which to understand how sociality contributes to the long-term maintenance of infectious disease in wildlife hosts and provides a set of tools to unpick the social and demographic mechanisms involved in any given host–pathogen system.

Keywords Disease dynamics · Modular network · Cryptic epidemic · Host competence · Epidemiological trap · Infection avoidance behaviour

Introduction

Social and contact networks are integral to the transmission of directly transmissible infectious diseases through populations (Altmann et al. 1994; Keeling and Eames 2005; Volz and Meyers 2007; Miller and Volz 2013). Contact network structure shapes infectious disease outbreaks in humans (Rohani et al. 2010; Potter et al. 2012), livestock

(Kao et al. 2006; Volkova et al. 2010; Fielding et al. 2019) and wildlife (Godfrey 2013; VanderWaal and Ezenwa 2016; White et al. 2017). How individuals vary in their social behaviour and patterns of interactions drives variation in the emergent properties of these contact networks and consequently disease dynamics (Volz and Meyers 2007; Danon et al. 2011; Hock and Fefferman 2012). For example, variability in the number of other individuals interacted with is a key contributor to the *superspreader effect* (see *Glossary, Box 1*) that causes outbreaks to be more explosive when they occur (Lloyd-Smith et al. 2005b). In contrast, the tendency to share connections (or be connected to “friends of friends”) and so form more clustered social networks tends to slow down pathogen spread (Badham and Stocker 2010). Similarly, social dynamics (how network structure changes over time) can play a crucial role in the nature of disease dynamics in a host–pathogen system (Fefferman and Ng 2007; Bansal et al. 2010).

Box 1 Glossary

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Term	Definition
Cryptic epidemic	Apparent global endemic infection that instead consists of serial, localised, epidemic outbreaks
Demographic competence	The ability of host populations to sustain endemic disease as determined by life history traits, demographic parameters and compensatory population responses to disease
Epidemiological trap	When disease-induced mortality opens opportunity for immigration or behavioural change, but the change carries a high probability of infection and reduced fitness
Flatten the curve	To reduce the magnitude of peak disease incidence/prevalence by slowing the rate of transmission, thereby prolonging the duration (and potentially increasing the final size) of an outbreak
Host competence	The viability of an infected host in transmitting infection to an uninfected, susceptible host
Keystone host	A host species without which a disease will die out in an ecosystem
Modular network	A network including a set of social communities (modules, clusters or groups), each containing a greater density of connections within the community than externally to it
Reservoir host	A host species/population in which a disease may remain endemic, thereby acting as a potential seed for epidemic outbreaks in other, vulnerable host populations
Seasonal forcing	Extrinsic factors that cause seasonal variation in the transmission of infection (e.g. environmental conditions that enhance pathogen viability outside the host or increase social contact etc.)
Sickness behaviour	Disease-induced behaviours exhibited by infected individuals
Superspreader effect	When particular individuals are responsible for a disproportionate number of new infections within a network (e.g. due to increased numbers of contacts, greater pathogenic shedding, etc.)
Zoonotic disease	An infectious disease of humans that is the result of transmission from a non-human host

A key focus of network epidemiology has been to identify what makes populations vulnerable to epidemics—a sudden increase in the number of cases of disease above the levels expected (Anderson and May 1979; May and Anderson 1979). Understanding network properties that increase vulnerability to epidemic spread of disease can be crucial when implementing policy to mitigate human and livestock diseases (Natale et al. 2011; Valdano et al. 2015; Strona et al. 2018), or in preventing the disease-induced extinction of endangered wildlife populations (Silk et al. 2019). Concepts such as superspreading in static or slow-shifting networks, where a small number of individuals contribute disproportionately to transmission (Lloyd-Smith et al. 2005b; Fielding et al. 2020) causing more severe epidemics to occur (Lloyd-Smith et al. 2005b; Garske and Rhodes 2008), therefore receive considerable attention. However, the flip side to this is that social structure and dynamics will also play a central role in determining when epidemics burn-out, and both if and how infectious disease become endemic in a population, exhibiting stable prevalence over time (Del Genio and House 2013). Despite this, the role of sociality in maintaining endemic disease within host populations has largely remained understudied.

Here, it is important to note that, in the context of network-based epidemiology, endemic disease is not simply a successfully mitigated epidemic. In many cases, the social structure or dynamics of a population will cause the current reproduction number (R_t) of an infection to decrease below 1, leading to epidemic burn-out and disease eradication. The maintenance of endemic disease requires sufficient opportunities for transmission between susceptible and infected individuals to lead to the expected number of new cases caused by each current active infection (i.e. R_t) to remain close to or fluctuate around 1 (at which point the number of infections in the population remains constant over time). There are multiple routes to a basic reproductive rate fluctuating around 1 and so more than one path towards endemicity. Changes in behaviour over time can lead to stable oscillations around 1 that will often be interpreted as endemic disease. Such variations in outbreaks can also occur in space as an outcome of population structure, such that only part of the population experiences an epidemic-type outbreak at any one moment in time (Thrall and Burdon 1997). Alternatively, a reproductive rate fluctuating around (or remaining close to) 1 can be driven by demography through the recruitment of newly susceptible individuals by birth, immigration or the loss of immune memory (Hethcote and Tudor 1980; May and Anderson 1985). In each of these examples, the disease is not characterised by stable replacement values in infections (i.e. the simplest and most common definition of endemic), but would still lead to consistent low levels of infection, neither increasing to a population-wide outbreak nor decreasing to

disease eradication. Importantly, all of these mechanisms can be influenced in some way by social behaviour.

Host populations that are able to maintain endemic disease can act as *keystone host* for a pathogen by enabling its persistence within a wider ecological community. In addition, determining when pathogens are able to persist in a host population, and the reasons they do, can be important when taking a one health approach to disease management (Narrod et al. 2012; Cunningham et al. 2017). Nowhere is this more the case than in wildlife populations, in which identifying *reservoir hosts* of potentially *zoonotic diseases* is of great importance (Wilcox and Gubler 2005; Brisson et al. 2011; Viana et al. 2014). If contact network structure helps shape endemic disease dynamics over longer timeframes, then social traits of populations can be used to help predict key reservoirs of infection. In this review, we examine how social structure and dynamics, alongside demographic mechanisms, can promote the persistence of endemic disease in populations (Fig. 1). We demonstrate key features of animal social systems that may favour the stable prevalence of infection and influence how it persists over time. We then discuss some of the key considerations when investigating the role of social behaviour in shaping reservoir competence and identify the next steps for future research.

Social structure and the persistence of infection

Here, we use social structure to describe the emergent properties of a population social network over a given time period or at any one point in time (excluding temporal changes to network properties). We assume that the social network being studied is relevant to the transmission of the pathogen of interest; the use of networks constructed over timescales that are not epidemiologically relevant can lead to mischaracterisation of the epidemiology of the pathogen (Holme 2013a). Given that interactions within the social network represent key opportunities for pathogens to transmit between hosts, it is unsurprising that theoretical models have revealed the structure of contact networks to shape infectious disease dynamics (Lloyd-Smith et al. 2005b; Salathé and Jones 2010; Volz et al. 2011; Sah et al. 2017). One aspect of social structure that has an important influence on transmission that can slow the spread of infection is the presence of modules or communities. Social networks with modular structure are characterised by a set of densely interconnected regions, with many fewer links between these regions (Girvan and Newman 2002; Fig. 2). These types of networks are likely to be most closely

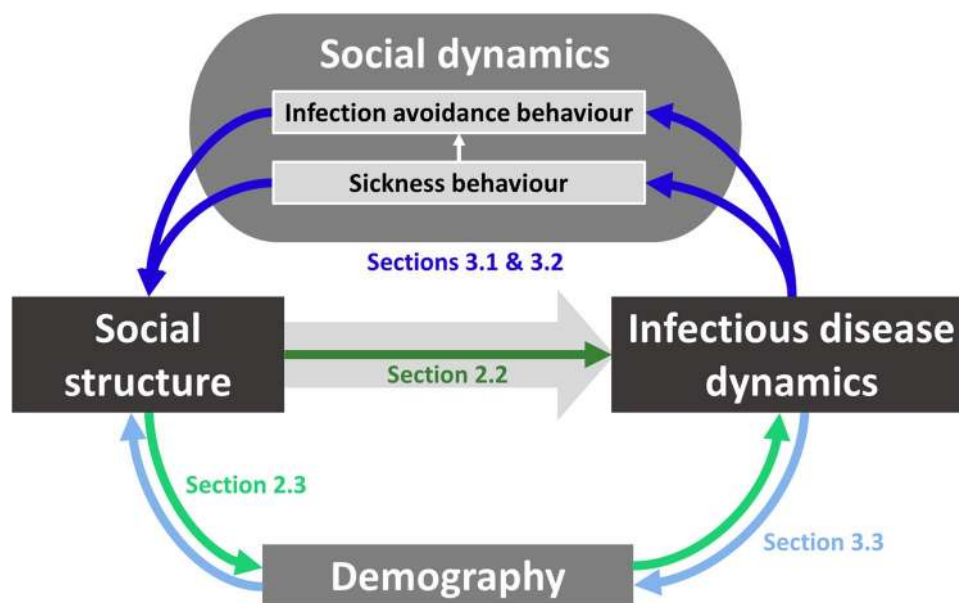


Fig. 1 A conceptual overview of the links between sociality, demography and disease dynamics examined in the paper. We show the pathways discussed in the paper only for clarity. Green arrows represent concepts discussed in the “Social structure and the persistence of infection” section of the paper and blue arrows represent concepts discussed in the “Social dynamics and the epidemic-endemic trade-

off” section. Dark arrows represent direction interactions between social behaviour and disease dynamics while light arrows indicate those effects mediated indirectly via demographic mechanisms (see “Modularity, synchronisation and cryptic epidemics” and “The role of social behaviour in disease-induced extinction of hosts” sections)

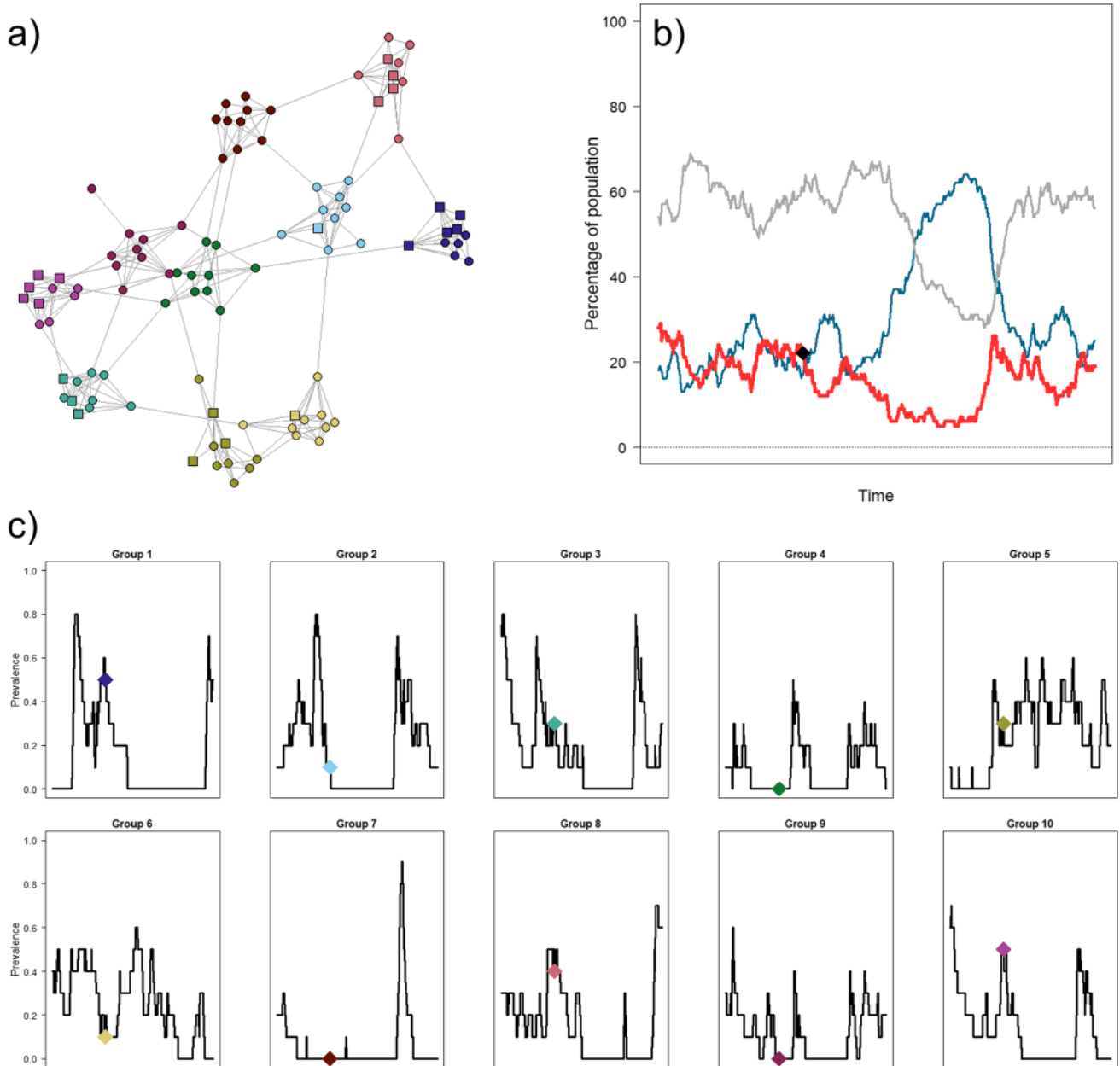


Fig. 2 Endemic disease in a modular network can persist as a series of cryptic epidemics. Here we show (a) the social network of the population with nodes coloured according to their social group membership; (b) the number of infected (red), susceptible (blue) and recovered (grey) individuals in the population over time from a stochastic susceptible-infected-recovered-susceptible (SIRS) network model; and (c) the prevalence of infection in each group over the same time period. Square nodes in the social network (a) indicate individu-

als infected at the time point marked by a diamond on the trajectories of infected individuals (b and c). An SIRS model could represent individual immune response waning over time or the replacement of recovered individuals with newly recruited susceptible individuals (with the same set of social relationships). Methodology for producing the figure, together with full code to generate the network and run the model, is provided in the supplementary material

associated with group-living species, and so be widespread in nature. Here, we discuss how these features of *modular networks*, especially when combined with demographic processes, can help increase the likelihood of infectious diseases remaining endemic.

Modular networks contain the spread of infection

Species that live in social groups, or even populations with strong spatial structure, frequently possess modular contact network structure (Weber et al. 2013; Shizuka et al. 2014; Weiss et al. 2020). Modular structure can also be present even

within social groups, especially in fission–fusion societies such as some primates (Kasper and Voelkl 2009; Griffin and Nunn 2012). The presence of modules or communities within a network (effectively the groups and/or subgroups in a population) can have important implications for pathogen transmission. For intermediate-high modularities, modelling work has shown the presence of modules can greatly slow the global spread of infection through a population (Salathé and Jones 2010; Sah et al. 2017), and this is especially striking for pathogens with lower transmissibility (Sah et al. 2017; Rozins et al. 2018). However, within each group, there tends to be a higher density of infection-relevant contacts and so local infection can be promoted (Sah et al. 2017). Therefore, the overall effect of modular networks is to trap infection within a particular part of a population, and high modularity reduces the frequency with which pathogens have an opportunity to spread between neighbouring subpopulations. Consequently, modular social networks have a substantial impact on infectious disease dynamics (Figs. 1 and 2), helping to contain outbreaks and limit the potential for population-wide epidemics. When infection leads to long-term immunity or death, containment can also promote local disease eradication (i.e. burn-out via localised herd immunity; Ferrari et al. 2006; Holme 2013b), eliminating the potential for further infection in the broader population. However, in many cases, especially with sufficient recruitment of susceptible individuals, modularity can also foster endemic disease. In the following paragraphs, we outline how, when combined with demographic processes, modularity can decrease the potential for both epidemic outbreaks and disease eradication to promote endemic stability at a variety of spatial scales.

Modularity, synchronisation and cryptic epidemics

One important effect of social group (or modular) structure is that infectious disease dynamics will be desynchronised at a global or population-level (Fig. 2). In a uniformly susceptible population without modular structure, in which a pathogen can easily spread globally, any pathogen with sufficient transmissibility will be capable of causing an epidemic. When individuals can recover from infection and are no longer susceptible (or are much less susceptible), the epidemic will peak and prevalence will decline over time until recovered individuals become more susceptible (e.g. if an immune response were to wane over time), or die and are replaced in the population by new susceptible individuals. After a sufficient length of time, a new epidemic could then occur. However, modular networks are one mechanism that can promote local transmission within social groups but reduce opportunities for infection to “jump” between groups (Sah et al. 2017). This can cause changes in the prevalence of infection in different groups to become desynchronised; an epidemic occurring in one group may not happen at the

same time as an epidemic in a second group (Fig. 2). Therefore, while individual groups or modules each experience epidemic-type disease dynamics (high local prevalence), overall prevalence in wild populations will remain lower and stable over time, promoting endemic persistence of disease (e.g. wolves *Canis lupus*: Brandell et al. 2021). In these situations, endemic disease can consist of a series of *cryptic epidemics* that each spread slowly to some other regions of the population. Each next module or region’s outbreak maintains a source of active infection while the previously impacted subpopulation can gradually replenish available susceptibles, allowing the disease to cycle back through previously affected network regions over longer timescales (Jiao and Fefferman 2021).

The role of modular networks in promoting persistence of infection in this way draws clear parallels with metapopulation models of infectious diseases (Grenfell and Harwood 1997; Earn et al. 1998; Keeling 2000; Ovaskainen and Hanski 2003; Keeling et al. 2004). These models have demonstrated that a lack of synchrony between subpopulations plays an important role in maintaining endemic disease (e.g. Earn et al. 1998; Lloyd and Jansen 2004). Strikingly, these studies also reveal the importance of accounting for synchronicity when designing management interventions. In the case of the Measles virus in humans in the UK, for example, continuous low levels of vaccination were shown to cause asynchronous outbreaks and promote persistence of infection (Bolker and Grenfell 1996), while the use of pulsed vaccinations could increase synchrony over large spatial scales and so make it easier to eradicate the disease (Earn et al. 1998). Similar examples have been revealed through subpopulation-specific investigations of disease in livestock and wildlife. For example, foot-and-mouth disease in agricultural animals in eastern Africa can easily be described as fluctuating endemicity, but analysis instead shows it to be comprised of a series of distinct outbreaks (Casey-Bryars et al. 2018; Wubshet et al. 2019) and similar studies have found similar patterns among wildlife populations in Western and Central Africa (Di Nardo et al. 2015). This body of research suggests that in wildlife populations where population-level endemic disease consists of a series of cryptic epidemics, management interventions will be more successful if they correctly account for synchronicity, especially when aiming to eliminate infection. However, it is important to note that relatively similar infections can respond in different ways to management. For vaccination, continuous low levels of vaccination against pertussis (Whooping cough) instead caused epidemics to become synchronous over wider spatial scales, albeit with a longer period (or gap between epidemics) (Rohani et al. 1999). Demographic and environmental stochasticity likely plays an important role in determining these differences, and stochasticity may be especially important in highly modular networks, given the scarcity

of opportunities for transmission between groups. Calls for vaccination protocols against foot-and-mouth disease therefore already include careful surveillance and inclusion of such factors, but inclusion is predicated on discovery of such cryptic dynamics (Munsey et al. 2019).

Other features of a host–pathogen system will also help determine whether endemic disease can consist of a series of cryptic epidemics. Theoretical models have demonstrated that etiologically distinct subgroups (whether distinct due to life histories, age cohorts, differences in body condition, genetic predispositions to more severe outcomes, coinfections, etc.) can produce epidemiologically distinct groups, even if those groups are not distinct in social communities nor physically separated into groups or subpopulations (Fefferman and Naumova 2006). Seasonality represents another good example. *Seasonal forcing*, or seasonal variation in transmission opportunities, has been documented in a number of wildlife host–pathogen systems (Hosseini et al. 2004; Altizer et al. 2006; Duke-Sylvester et al. 2011), and could be driven by pulsed reproduction, migration or predictable changes in social behaviour, among other mechanisms. For Measles dynamics in humans, increased seasonal forcing of infection has been shown to promote greater synchrony in epidemics across a meta-population (Rohani et al. 1999). Therefore, the importance of the cryptic epidemic mechanism for the maintenance of endemic disease is likely to depend on the seasonality of host life history and behaviour. For example, data-driven models based on a diversity of mammal species demonstrate that hosts with strongly seasonal reproduction, and consequently pulses of new susceptible individuals being recruited, would be expected to have more synchronised epidemics and a higher chance of burn-out and pathogen extinction (Peel et al. 2014).

Social groups as persistent sources of infection

The role of modular network structure in promoting long-term persistence of infection can be enhanced further by demographic processes (Fig. 1). Pathogen persistence requires sufficient turnover of the host population, especially for pathogens that kill their host or trigger a long-term immune response (Anderson and May 1981; Thrall et al. 1993). Therefore, the rate of recruitment through reproduction and immigration into the host population is integral to longer term disease dynamics. Even for virulent diseases, it is possible for some host populations to compensate through density-dependent increases in the rate of recruitment (e.g. McDonald et al. 2016). Group living can shape the importance of demographic turnover for the maintenance of endemic disease by changing the spatial scale at which these compensatory demographic mechanisms occur. A good example of this is provided

by empirical research on bovine tuberculosis (bTB) in European badgers *Meles meles* (McDonald et al. 2018). bTB causes substantial disease-induced mortality in high-density badger populations (Graham et al. 2013), meaning that mortality rates increase substantially in infected groups. However, because badger populations are regulated at a social group level, density dependence in recruitment into the group can compensate for the losses caused by bTB (McDonald et al. 2016). The effect of this is to allow infection to persist for long periods of time within particular groups (Delahay et al. 2000), which can then act as sources of infection to other groups. Thus, when behaviour and demography are considered together, it is possible for modular networks to promote not just global endemic disease (within the population as a whole) but also local endemic disease within particular regions of the population network.

The role of demography alongside network structure would be expected to be most important when the spatial scales of population regulation and transmission are most closely coupled. Other important requirements are that the life history of the host allows sufficient compensation for disease-induced mortality—*demographic competence* (Silk and Hodgson 2021; Valenzuela-Sánchez et al. 2021)—and that the life history of the pathogen allows the host population to compensate by being neither too transmissible nor too virulent (Silk and Hodgson 2021). Naturally, modelling work shows that both age- and disease-related mortality can also compromise emergent community structure (Gallos and Fefferman 2015a), but also increase path distance between remaining modules, leading to complicated dynamics between modularity, density and vulnerability to epidemic spread (Ferrari et al. 2006).

When social groups can maintain endemic disease in this way, there are important ecological and management consequences. First, it means that some groups or parts of a population can act as an *epidemiological trap* (Leach et al. 2016; Lilley et al. 2018; Hopkins et al. 2021; Fig. 3). Disease-induced mortality will create apparent opportunities for individuals without territories or breeding opportunities elsewhere. However, immigration into the infected group will risk a substantial shortening of their lifespan which thereby also risks reduced fitness (although a fitness reduction may not occur if they are able to reproduce before succumbing to disease or if there are no opportunities for reproduction elsewhere in the population within that individual's potential lifespan). Recruitment through dispersal in this way might be important in allowing groups to remain persistent sources of infection, especially in species with slow life histories where reproductive queuing is more likely (Hatchwell and Komdeur 2000). From a management perspective, populations in which endemic disease is maintained locally might be

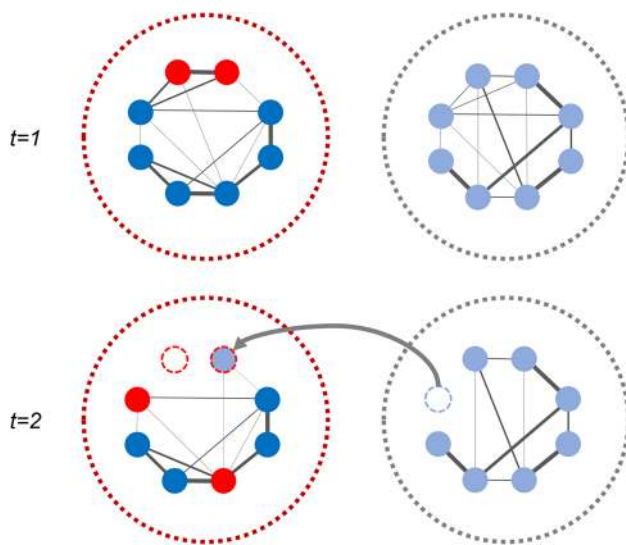


Fig. 3 Dispersal into an infected group can promote persistence of disease but also act as an epidemiological trap. Here, we show two groups, one infected (red, dashed circle) and one not (grey, dashed circle). Individuals interact as indicated by the within-group social networks. Uninfected individuals are represented by blue nodes (dark blue for the infected group and light blue for the uninfected group) and infected individuals by red nodes. At time step 1 two individuals in the first group are infected. By time step two these individuals have died (empty red nodes) but transmitted infection to two of their contacts. An individual from the second group has detected a perceived opportunity created by the death of these individuals and dispersed. By doing so, this individual contributes to the persistence of infection but may also have fallen into an epidemiological trap and reduced its own fitness by putting itself at risk of being infected

especially vulnerable to negative impacts of social perturbation (McDonald et al. 2008; Jones et al. 2019). Forms of management that might increase dispersal between groups (Carter et al. 2007) or breakdown territorial boundaries (Woodroffe et al. 2006a) may allow a pathogen to become established in other groups. Therefore, social perturbation would be expected to be especially important when endemic disease is maintained locally (although may also increase the severity of cryptic epidemics).

Social dynamics and the epidemic-endemic trade-off

It is not only the structure of social networks that shapes infectious disease dynamics; how interaction patterns change over time is also important (Bansal et al. 2010). Two separate forms of network dynamics are important here: changes in network structure that occur independently from the spread of infection, and co-dynamics of disease-related behaviour and infection (Fig. 1).

For disease-independent behaviour, more dynamic networks, with faster turnover of social connections, can cause greater mixing and so reduce the importance of network structure in regulating infectious disease spread (Volz and Meyers 2007; Springer et al. 2016). These changes will have the strongest effect when they change the structural form of the network. Consequently, when individuals do not interact consistently with each other over time, especially if they interact with individuals in different groups or vary the frequency of between-group contacts, potential transmission routes change through time and the importance modular structure for disease dynamics is reduced even when the modularity of any single snapshot of the network is unchanged (Masuda and Holme 2017). Similarly, if contact patterns change substantially between seasons (e.g. chacma baboon *Papio ursinus*: Henzi et al. 2009; European badger: Silk et al. 2017; raccoon *Procyon lotor*: Hirsch et al. 2016), then with sufficient overall contact density, the disease dynamics may be dominated by seasonal forcing. As described previously, synchronisation of outbreaks through seasonal forcing may reduce the stability of disease dynamics by limiting the possibility of cryptic epidemics (“Modularity, synchronisation and cryptic epidemics” section).

Here, we focus predominantly on the co-dynamics of infection and behaviour (Ezenwa et al. 2016; Townsend et al. 2020; Stockmaier et al. 2021) as a rich source of mechanisms that can promote endemic disease dynamics in wild populations. Changes in the behaviour of individuals in response to infection, termed behavioural or social immunity in some systems (Cremer et al. 2007; De Roode and Lefèvre 2012), can *flatten the curve* of epidemics, reducing the peak of infection and prolonging the duration of an outbreak. As a result, behavioural responses to disease will either cause local pathogen extinction and epidemic burn-out or naturally push a system towards more stable, endemic disease dynamics. Infection dynamics and behaviour can also covary indirectly via demographic mechanisms and we touch on the role of these types of behavioural dynamics in the final part of this section. Changes in social interaction patterns as a population declines can play a key role in determining the likelihood of disease-induced extinction of hosts (De Castro and Bolker 2005; McCallum 2012), and consequently increasing the stability of a host–pathogen system (see below).

Sickness, infection avoidance and changes in interaction patterns

In this section, we focus on changes in behaviour that result directly from the spread of infection. There are two relevant categories of behaviours, the sickness behaviour of infected individuals and the behaviour shown by uninfected individuals towards those with symptoms of infection (including

both infection avoidance and increased exposure through provision of support/care).

Sickness behaviour is a joint phenotype, influenced by both the host and the pathogen. Consequently, it may be difficult to predict its role in disease dynamics as the evolutionary interests of hosts and their pathogens are often not aligned. In many cases, sickness behaviour can cause lethargic behaviour and a decrease in social connectivity (e.g. house mice *Mus musculus*: Lopes et al. 2016; common vampire bats *Desmodus rotundus*: Stockmaier et al. 2018, 2020). Typically, this would be expected to contribute to flattening the curve (Colman et al. 2018); however, in some environments, lethargy can increase contact rates and therefore opportunities for transmission (Franz et al. 2018). Some other sickness behaviours will clearly promote onward transmission, e.g. in three-spined sticklebacks *Gasterosteus aculeatus* (Demandt et al. 2018).

Infection avoidance behaviour is likely to be of greater importance in driving the co-dynamics of infection and behaviour. Avoidance behaviour requires individuals to be able to perceive cues of disease, and so its role will

vary among host–pathogen systems (Hawley et al. 2021; Stockmaier et al. 2021). There is increasing evidence of infection avoidance in a wide range of taxa (Stockmaier et al. 2021). Changes in behaviour can encompass avoidance of particular individuals (Caribbean spiny lobsters *Panulirus argus*: Behringer et al. 2006; mandrills *Mandrillus sphinx*: Poirotte et al. 2017; guppies *Poecilia reticulata*: Stephenson et al. 2018; Stephenson 2019), through general responses to the presence of infection within a group (e.g. black garden ants *Lasius niger*: Stroeymeyt et al. 2018; dampwood termites *Zootermopsis angusticollis*: Rosengaus et al. 1999; western lowland gorilla *Gorilla gorilla gorilla*: Baudouin et al. 2019). For example, guppies have been shown to detect diseased conspecifics using multiple sensory modalities and modulate their behavioural response to avoid particular individuals according to the risk of infection (Stephenson et al. 2018; Stephenson 2019). In contrast, in dampwood termites, individuals exposed to fungal infections communicate disease risks to unexposed nestmates, which then withdraw from the region of the nest indicated, drastically reducing the potential for local

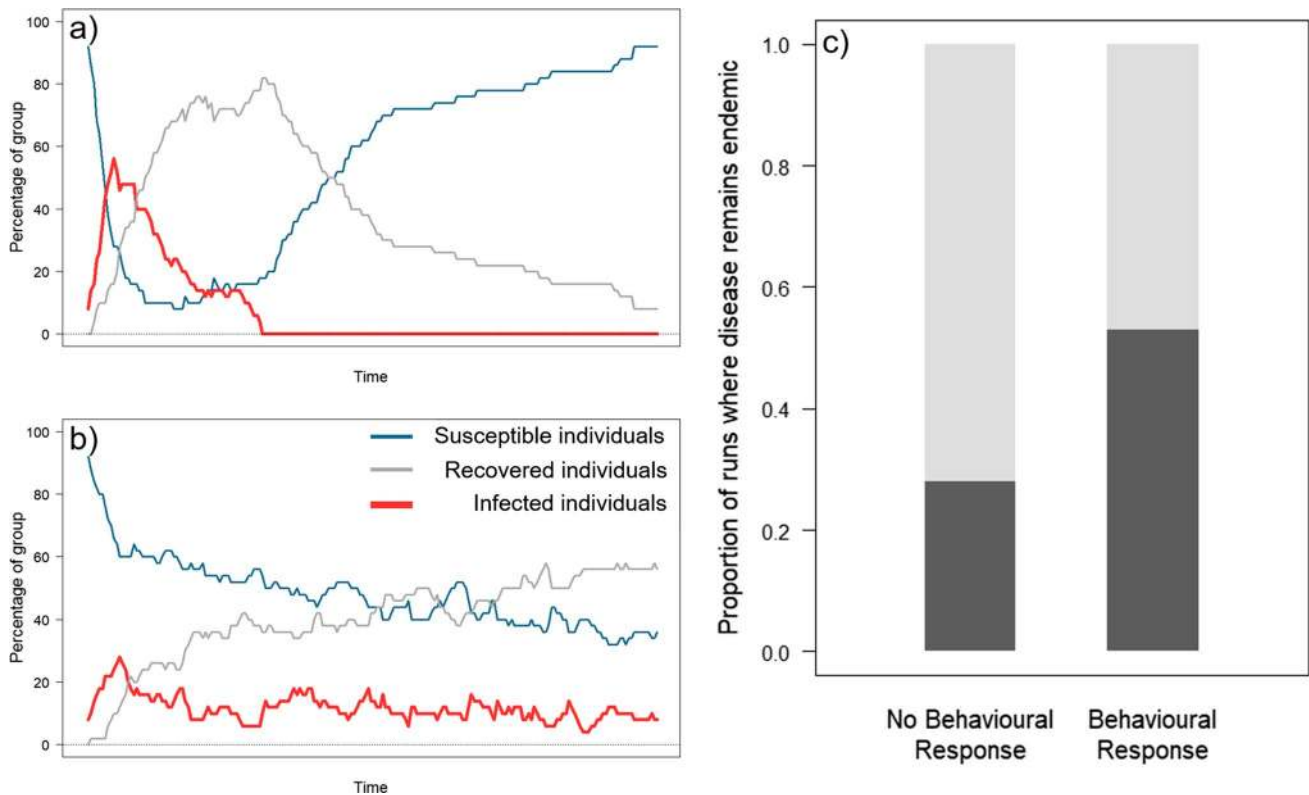


Fig. 4 Behavioural responses to infection can promote endemic disease dynamics. Here, we illustrate runs of a stochastic, network-based susceptible-infected-recovered-susceptible (SIRS) model in a group of 50 individuals when (a) there is no behavioural response to infection and (b) individuals cut 75% of their social contacts when the prevalence of infection exceeds 10% (i.e. there is non-negligible

prevalence of infection in the group). (c) When we run each version of the model 100 times, the behavioural response to infection means that disease will persist for the entire time series modelled more frequently. Methodology for producing the figure, together with full code to generate the network and run the model, is provided in the supplementary material

contact with all other individuals present in the exposed area (Rosengaus et al. 1999). We illustrate a simple example of the general changes in the sociality of a group in response to the presence of infection in Fig. 4. While the simulation is designed as an illustrative tool only, similarly abrupt changes in behaviour are feasible in the wild (e.g. only 7% of infected spiny lobsters occupied shared dens compared to 56% of uninfected individuals; Behringer et al. 2006).

Changes in contact patterns caused by infection avoidance behaviour take two main forms: individuals may cut their social interactions to reduce their frequency of transmission-relevant contacts (Fig. 4), or individuals may change who they associate or interact with to maintain the benefits of sociality while reducing their risk of becoming infected. Theoretical models suggest that either behavioural response will typically mitigate epidemics (Shaw and Schwartz 2008; Funk et al. 2009; Van Segbroeck et al. 2010; Tunc and Shaw 2014; Just et al. 2018), although in some circumstances where information about infection is limited or delayed, rewiring to form new social connections can exacerbate it (Zhou and Xia 2014). In some cases, infection avoidance behaviours can lead to epidemic burn-out and pathogen extinction. However, by delaying epidemic spread of disease, these behaviours can also maintain a higher number of susceptible individuals in a population and can therefore foster endemic disease (Fig. 4).

Importantly, theoretical models have revealed that infection avoidance behaviour will also cause changes in social network structure. When individuals “swap” their social relationships with diseased individuals to healthy individuals, the average distance through the social network from each healthy individual to a diseased individual will increase (Shaw and Schwartz 2008), with individuals that are still susceptible to infection becoming assorted within the network (Gross et al. 2006). If there is no upper limit to connectivity, then remaining susceptible individuals will also often have more social connections, and there will be greater heterogeneity in degree distribution (Shaw and Schwartz 2008), increasing the likelihood of superspreading if a pathogen re-emerges. Finally, in modular networks, infection avoidance behaviour can lead to fewer between-group social contacts and cause the modularity of the network to increase (Yang et al. 2012). Higher network modularity can increase the likelihood of infection being trapped in particular groups, resulting in (a) pathogen extinction being more likely or (b) some groups remaining highly vulnerable to disease re-emergence if it has not been eradicated (see also “[Modular networks contain the spread of infection](#)” and “[Modularity, synchronisation and cryptic epidemics](#)” sections). As a result, both the disease dynamics and changes in network structure that result from infection avoidance behaviour can leave a population vulnerable to re-emergence or prolonged outbreaks (Gross et al. 2006; Zhou et al. 2012).

In contrast to infection avoidance, some social species exhibit care for diseased conspecifics (whether initiated due to infection or otherwise). General increases in care that are distributed broadly among group mates or the recruitment of increased contact (and therefore potential transmission) can increase exposure risk and alter disease dynamics within a group or population, as demonstrated empirically in ants (Ugelvig et al. 2010; Reber et al. 2011). Modelling has demonstrated that, for care to have evolved, the expected individual fitness benefit associated with receipt of care must outweigh the potential fitness costs of infection derived from providing care to others (Hock et al. 2010). Some species have even been shown to have evolved physiological differentiation in those providing care to others that helps prevent the spread of infection throughout the population (e.g. honeybees *Apis mellifera*: Cini et al. 2020). However, the implications of social care for the endemicity of infections remain unclear.

Mitigating epidemics can come at the cost of promoting persistence

A key consequence of changes to disease dynamics and network structure that result from infection avoidance behaviour is that collectively they can foster endemic disease if mitigation does not lead to pathogen extinction (Gross et al. 2006). When disease spreads rapidly and generates high densities of infected individuals in a population or group, avoidance behaviour can often lead to densely connected clusters of susceptible individuals, which can facilitate onward transmission of infection (Gross et al. 2006). Additionally, changes in the patterns of social interactions may change the effectiveness of disease management interventions. For example, infection avoidance behaviour that causes highly connected individuals to be more likely to be connected with each other (a positive degree correlation) would reduce the effectiveness of network-targeted vaccination (Gross et al. 2006). Consequently, behaviour that mitigates epidemics (“flattens the curve”) may promote endemic disease if not fully successful, generating an epidemic-endemic trade-off.

An important aspect of this relationship between mitigated epidemics and promoted persistence is that, beyond its impact within single host-single pathogen systems, it can be important across a pathogen community (Chen and Preciado 2014). Behaviour that mitigates against the rapid spread of a newly (re-)emerged pathogen could lead to a structural rearrangement of a group or population’s social network that facilitates the endemic persistence of other, existing pathogens. Alternatively, changes in behaviour in response to a novel pathogen could cause large reductions in the prevalence of other pathogens with similar transmission

mechanisms (e.g. Lei et al. 2020; Soo et al. 2020). This could be beneficial to a host population by causing local extinction of the existing pathogen, but in the longer term could lead to a buildup of susceptible individuals and so destabilise disease dynamics and make a future, severe epidemic more likely. In essence, host behaviour meaningfully affects the landscape for entire ecological communities of pathogens/parasites. As a result, behavioural responses to infection may play a key role in determining how changes in parasitism driven by environmental change will influence the stability of existing host–pathogen systems.

The role of social behaviour in disease-induced extinction of hosts

Infectious disease can also impact social behaviour indirectly through its effect on population dynamics (Fig. 1). Diseases with sufficient mortality rates will cause host populations to decline, especially when that host population is unable to compensate for the deaths of diseased individuals by reduced mortality of others or increased recruitment into the population (Lloyd-Smith et al. 2005a; Silk and Hodgson 2021; Valenzuela-Sánchez et al. 2021). The loss of individuals from a population, and subsequent reduction in population density, would be expected to have a substantial influence on the movement and interactions of remaining individuals (e.g. great tits *Parus major*: Firth et al. 2017; house mice: Evans et al. 2020). Any changes in behaviour can then influence disease dynamics by altering how transmission-relevant contacts occur. If behaviour changes little and contact rates decline with population density, then disease begins to spread more slowly through the remaining population. Conversely, if individuals change their behaviour and contact rates remain the same, then disease will still spread equally fast as before. This captures the distinction between density-dependent and frequency-dependent transmission in epidemiological models (Silk et al. 2019; Hopkins et al. 2020). It is pathogens with frequency-dependent transmission that are much more likely to drive host populations extinct (De Castro and Bolker 2005; McCallum 2012), while those with density-dependent transmission in which host–parasite coexistence is more likely.

Importantly, the precise nature of how contacts change with population density will depend on the social system of the host. Few host populations will perfectly follow a frequency-dependent or density-dependent model meaning that the relationship between population density and transmission rate will more often be somewhere between these two extremes (Hopkins et al. 2020). Some social systems may naturally lend themselves to maintaining contact rates even as the number of individuals to interact with declines. For example, if a territorial species were simply to expand its territory when there was an opportunity to do so (e.g. European badgers: Woodroffe et al. 2006a), then contact rates

would change relatively little. In contrast, if territory size is not limited by social interactions and each territory contains sufficient resources, then individuals may not expand their territories and contact rates would decline with population density. In group-living species, the relationship may become more complicated still. Data from wild systems on how social networks respond to the loss or removal of individuals remains relatively scarce (Shizuka and Johnson 2020). Naively, declines in group size would be expected to lead to reduced contact rates if individuals did not change their social or spatial behaviour. However, in multiple wild systems, individuals have been shown to adjust their social behaviour to compensate for the loss of social connections (e.g. great tits: Firth et al. 2017; house mice: Evans et al. 2020; yellow baboons *Papio cynocephalus*: Franz et al. 2015). Under some circumstances, changes in social behaviour may lead to recruitment into the group or even to the merger of different groups, meaning contact rates can even increase in the short term. For example, culling in European badger populations can cause more dispersal between groups (Carter et al. 2007) and greater overlap between the territories of neighbouring groups (Woodroffe et al. 2006a) leading to increased incidence of disease (Woodroffe et al. 2006b). Theoretical models have shown that the formation of new connections to others already in the population can increase the density of social contacts among those who remain and provide a mechanism for increased transmission (Gallos and Fefferman 2015b). Consequently, social dynamics play a key role in determining whether a pathogen can push a host population to extinction (De Castro and Bolker 2005; McCallum 2012), and so contribute to (de)stabilising host–pathogen dynamics indirectly as well as directly.

Key considerations

Many other characteristics of a host–pathogen system will not only shape whether a disease will become endemic, but also influence the role that social behaviour has in facilitating pathogen persistence. We briefly discuss several of the most important here, but this is by no means an exhaustive list and it is important to take a holistic approach to identifying what makes specific host populations competent reservoirs of infectious disease.

Physical environment

The physical environment and social interaction patterns are intrinsically linked by spatial behaviour. How individuals move around a landscape will be critical in influencing infectious disease dynamics (Dougherty et al. 2018; Albery et al. 2021), with a large part of this tied to how the spatial and

social behaviour of individuals are interlinked. Landscape connectivity, for example, might be a key factor shaping the modularity of population contact networks or determine if and how contact rates change as populations decline. Similarly, a limiting resource may contribute to the formation of epidemiological traps by increasing the likelihood of dispersal into a diseased group or territory. Empirical research has revealed that for some pathogens the physical environment may also change the types of social contact that can result in transmission (Godfrey et al. 2009; Blyton et al. 2014). Therefore, placing the role of social structure and dynamics in a wider landscape context will be important in truly unpicking the drivers of endemic disease dynamics.

Immune ecology

Immune ecology will also play a central role in shaping how sociality and demography contribute to the maintenance of endemic disease. Much of the discussion in this paper assumes individuals recover from infection and then remain immune (susceptible-infected-recovered or SIR dynamics in an epidemiological model). Some of the key network modelling studies examining adaptive network dynamics model SIS (susceptible-infected-susceptible) dynamics (e.g. Gross et al. 2006; Shaw and Schwartz 2008) and therefore assume that individuals can be re-infected once they recover, making endemic states much more likely. In real-world systems, the immune system of the host will be integral to determining, for example, whether a host resists or tolerates a pathogen (Read et al. 2008), the length of the infectious period (Hawley and Altizer 2011) and whether and how fast immunity wanes over time leading to future re-infection (Miller et al. 2007), all of which will influence the role of social behaviour in promoting endemic disease. In addition, host immune performance may be tied to social relationships (Sapolsky 2005), meaning that social interactions can influence immunity and *host competence* as well as infection risk. Therefore, understanding the eco-evolutionary dynamics of host immune and social ecology will be central to understanding the role of social systems in maintaining endemic disease.

Pathogen and parasite traits

Traits of the pathogen itself are also a key part of the puzzle. Many pathogens will spread via multiple transmission modes. Pathogens for which indirect (or environmental) transmission is important will be impacted less (and in different ways) by social structure than pathogens that can only be transmitted through specific types of close contact. Even among directly transmitted pathogens, different transmission modes will also alter the likely social structure and dynamics of host contact networks. Different types of social contact in wild populations are structured in different ways in animal

groups (e.g. Castles et al. 2014), meaning that pathogens spread through different types of behaviour (e.g. sharing refuges: Leu et al. 2010; aggressive interactions: Jenkins et al. 2012; Hamede et al. 2013 or reproductive behaviour: Nyari et al. 2017) will experience very different contact networks, with consequences for their epidemiology that it is important to account for.

For directly transmitted pathogens, host social structure does not influence the spread of pathogens that are highly transmissible in the same way that it does less transmissible pathogens. Infection avoidance behaviour also typically requires cues that an individual is diseased (Hawley et al. 2021; Stockmaier et al. 2021). For pathogens with pre-symptomatic transmission or that cause “hidden” pathologies, then social dynamics may not be sufficient to slow epidemic spread, and in some cases can exacerbate it when individuals change social partners before displaying symptoms (Zhou and Xia 2014). In a similar vein, for pathogens that are highly virulent and cause high mortality rates, a behavioural response to the loss of diseased individuals may be insufficient to prevent host extinction, at least at a local level (De Castro and Bolker 2005). As a result, social structure and dynamics will play a more important role in stabilising disease dynamics of some pathogens than others.

Parasitic infections complicate socio-epidemiological dynamics further. Ectoparasites, specifically, can have infection-severity-dependent transmission (i.e. the parasite only searches for a new host when the current host becomes over-parasitised) or can instead rely on life-cycle dynamics for the transmission of infection (Mullen and Durden 2009). Social structure can also directly influence the infectiousness of contacts, either because the contact process itself affects the severity of infection for the parasite source (e.g. allo-grooming) or because the density of exposures dilutes the number of parasites likely to colonise subsequent social partners (shown empirically in ants by Theis et al. 2015 and modelled by Wilson et al. 2020). Finally, infection avoidance behaviours may depend on cues or signals that are not well matched to ectoparasite transmission risk (Kavaliers et al. 2003; Sarabian et al. 2018). For example, the highest parasite transmission risk may precede pathology (i.e. cues signalling illness from parasitic infection only become detectable after parasite populations have begun to dwindle due to decreased host viability).

Next steps

Explanations for what makes a competent reservoir of endemic disease will be multi-faceted and depend on a suite of traits of both the host and the parasites/pathogens being considered. However, given the importance of social behaviour to the transmission of many pathogens,

differences in social systems are likely to play a key role in driving variation in the persistence of infectious disease in natural populations. A key outstanding question, therefore, is whether host social structure can be used as a trait to identify key reservoirs of zoonotic or agricultural diseases. Beyond this, it would be valuable to identify the role of hosts with social group structure more generally, to determine if and how they promote the persistence of pathogens in wider ecological communities. We might predict for example, that hosts with social group structure are more likely to act as keystone hosts in this regard. Finally, social and demographic process act in concert to stabilise (or destabilise) disease dynamics, and it will be important to identify how this shapes the competence of wild host populations. Doing so will enhance our ability to forecast disease dynamics and spillover risk at finer spatial and temporal scales across a range of host–pathogen study systems.

A key initial step will be designing network-based epidemiological models that examine the vulnerability of different social systems to the long-term persistence of infection. An important component of many of these models may be the integration of contact networks and demographic processes given the strong interdependency in the impacts of social structure and population turnover (Silk et al. 2019). In many cases, meta-population models (Cross et al. 2005) or explicitly spatial models (White et al. 2018a, b) may be sufficient in the place of models that explicitly model social networks. An important modelling challenge will be to identify regions of “host parameter space” (combinations of life-history, demographic and social traits) where infection is able to persist locally (though potentially only transiently) and regions where global persistence is favoured. Models that simultaneously consider the spread of multiple pathogens within a combined network-demographic framework are also likely to be valuable, particularly in determining the knock-on impacts of infection avoidance behaviour across a pathogen community.

Alongside the rapid development of modelling approaches, there is an increasing wealth of social network data from wild populations (e.g. Sah et al. 2019). These newly available datasets will enable the use of comparative approaches (e.g. Sah et al. 2017, 2018) to answer key questions, especially if combined with relevant disease data. In addition, these data repositories facilitate data-driven modelling on a scale that has not been feasible previously. Applying previously developed theoretical models to real datasets will help ground them in realistic scenarios and can provide insights specific to particular social or host–pathogen systems. Important insights into disease dynamics have been provided by long-term studies of wild populations (Barroso et al. 2020) and through models applied to these systems (e.g. Hamede et al. 2012; Rozins et al. 2018; Weiss et al.

2020). Detailed data-driven models that provide system-specific insights can also be important in addressing some of hypotheses identified in this review. Tracking technology is increasingly being used to study the social and spatial behaviour of individual animals in these well-studied host–pathogen systems (e.g. Hamede et al. 2009; Weber et al. 2013; Hirsch et al. 2016; Silk et al. 2018; Stroeymeyt et al. 2018), and these sources of high-resolution data are likely to be particularly valuable in quantifying the extent of infection avoidance behaviour and determining its importance in stabilising host–pathogen dynamics.

Conclusions

Social systems are likely to form an integral part of what make some species competent hosts of endemic diseases. Quantifying the role of sociality in the maintenance of endemic disease can also be integral in determining how infection is able to persist and can help guide how (or, perhaps more importantly, how not) to manage host–pathogen systems. More broadly, we can use the insights provided by better understanding how social structure and dynamics make populations vulnerable to endemic disease to help guide the search for wildlife reservoirs of zoonotic (or agricultural) diseases by targeting surveillance based (in part) on characteristics of host social systems.

Data and materials availability

Not applicable.

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Author contribution MJS and NHF developed the idea for the paper. MJS wrote the initial draft and run models to produce Figs. 2 and 4 with input from NHF. NHF edited the initial draft and wrote additional text for multiple sections. Both authors approve the final version of the paper.

Code availability R code to produce Figs. 2 and 4 is provided as supplementary files.

Declarations

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