SOCIAL INFORMATION USE SHAPES THE COEVOLUTION OF SOCIALITY AND VIRULENCE

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ABSTRACT

Social contacts can facilitate the spread of both survival-related information and infectious disease, but little is known about how these processes combine to shape host and parasite evolution. Here, we use a theoretical model that captures both transmission processes to investigate how host sociality and parasite virulence (co)evolve. We show how selection for sociality (and in turn, virulence) depends on both the intrinsic costs and benefits of social information and disease as well as their relative prevalence in the population. Specifically, greater sociality and lower virulence evolve when the risk of infection is either low or high and social information is neither very common nor too rare. Lower sociality and higher virulence evolve when the prevalence patterns are reversed. When disease and social information are both at moderate levels, the direction of selection depends on the relative costs and benefits of being infected or informed. We also show that sociality varies inversely with virulence, and that disease may be unable to constrain the evolution of sociality. Together, these findings provide new insights for our understanding of group living and how apparently opposing ecological processes can influence the evolution of sociality and virulence in a range of ways.

INTRODUCTION

Animals exhibit strong variation in sociality (contact rates), both within (Watters and Sih 2005; Pike et al. 2008; Croft et al. 2009; Tanner and Jackson 2012; Aplin et al. 2013, 2015b; Weber et al. 2013) and between (Sachser 1986; Barton et al. 1996) populations. Some individuals have relatively few, potentially stable associations (e.g. non-gregarious species or those that form pair bonds) while others have a greater number of interactions that may be more transient (e.g. herding behaviour or roaming individuals) (Couzin 2006; Silk et al. 2014; Farine et al. 2015b). Crucially, what constitutes a good social strategy under one set of ecological conditions may be a poor choice under different circumstances (Krause and Ruxton 2002), hence it is important to understand how changes in the environment shape social behaviour. Depending on the ecological circumstances, sociality may be associated with various costs (e.g. higher risk of disease or greater competition for resources) and benefits (e.g. information exchange, increased mating opportunities, or symbiont transmission), which trade-off to determine whether selection favours increased sociality.

Intuitively, if sociality confers diminishing benefits but accelerating costs, then the population will evolve to an evolutionarily stable level of sociality. This is straightforward when costs and benefits can be considered fixed, so that a given level of sociality always incurs the same cost or provides the same benefit. For example, living in a larger groups may lead to foraging benefits (Cantor et al. 2020) or a reduced risk of predation (Kenward 1978), but these do not necessarily depend on the sociality of other individuals in the population. However, when the costs and benefits are dynamic, as is the case with infectious diseases, social information and symbiont transmission, feedbacks may exist

between sociality and the likelihood of realising an associated cost or benefit (Cantor et al. 2021). For example, more social members of a population may experience greater exposure to disease (Lloyd-Smith et al. 2005; Eames and Keeling 2006; Ashby and Gupta 2013) and access to social information (Aplin et al. 2012, 2015a; Snijders et al. 2021) than those that form fewer contacts or have more stable associations (Evans et al. 2020). However, the sociality of the population will affect the prevalence of disease or social information in the population, and therefore the likelihood of incurring a cost or realising a benefit of sociality (Romano et al. 2020). Thus, while we should expect infectious disease and social information to influence the evolution of sociality, we must also consider how the evolution of sociality shapes the dynamics of these cost/benefit processes due to eco-evolutionary feedbacks. An outstanding question, which we address in the present study, is how dynamic costs (in terms of disease transfer) and benefits (in terms of information transfer) mediate the evolution of sociality.

Few theoretical studies consider how eco-evolutionary feedbacks mediate the evolution of sociality (see Cantor et al. 2020b for a review), but those that do have thus far focused on the role of infectious diseases as a dynamic cost. Bonds et al. (2005) showed that sociality (contact rates) is minimised for intermediate levels of disease, and therefore higher levels of disease can select for greater sociality. This is because there is little chance of avoiding disease when it is very common, and so the marginal costs of greater sociality are low. In a related study, Prado et al. (2009) used a stochastic individual-based network model to show that coevolution between host contact number and parasite virulence can lead to fluctuations in both traits, with the cost of sociality depending both on the virulence of the parasite and its prevalence in the population. These studies demonstrate the importance of

infectious diseases as dynamic costs in the evolution of sociality, but both assume that the benefits of sociality are fixed, so that each additional contact increases host fitness by a predetermined amount. Theoretical studies have yet to consider dynamic rather than fixed benefits of sociality.

One of the most prominent examples of a dynamic benefit of sociality is social information, which can provide individuals with knowledge about ephemeral resources or conditions, such as foraging or nesting sites, or predation risk (Doligez et al. 2002; Danchin et al. 2004; Valone 2007). This information can be transmitted through active signalling (Elgar 1986) or via inadvertent cues (Pöysä 1992; Galef and Giraldeau 2001), with highly social individuals likely to receive information sooner than poorly connected members of the population (Aplin et al. 2012, 2015a; Snijders et al. 2021). We should therefore expect sociality to increase access to survival-related information, but as is the case with disease, the prevalence of information in the population will depend on sociality, forming an eco-evolutionary feedback that causes the evolution of sociality to diverge from the fixed-benefit scenario.

While social information and infectious diseases are both transmission processes that share many similarities, their dynamics may occur over similar or very different timescales, which may affect selection on social strategies. Social information about predation or resource availability is expected to be of high value that rapidly decays over time (between seconds and days), whereas the risk of infectious disease may vary more slowly and over longer periods of time (weeks, months or years). For example, the life cycle of a parasite can de-couple the transmission from social contacts by several weeks (Grear et al. 2013). In general, we should expect social information dynamics to be relatively fast compared to

infectious disease dynamics, although it is possible that they may occur on similar timescales. The success of a particular social strategy is likely to depend on the relationship between these timescales, as well as wider contact patterns in the population, the value of social information and the prevalence and severity of disease. Environmental changes (e.g. food availability, pathogen transmissibility or virulence) therefore have the potential to shed light on how sociality evolves.

Here, we examine how social information mediates the evolution of sociality and virulence. We present a mathematical model of infectious disease and social information dynamics, allowing sociality to evolve with no inherent costs or benefits, and virulence to evolve subject to a trade-off with transmission. Evolution is determined purely by balancing access to social information, which reduces the mortality rate, and infectious disease, which increases the mortality rate. We consider two versions of the model, where social information dynamics occur on relatively fast or slow timescales, with the former allowing for a simple approximation to social information prevalence. We show that selection favours increased sociality when social information is neither very common nor too rare and disease is at either high or low levels. When social information and disease are both at intermediate levels, selection may favour increased or decreased sociality depending on the relative costs and benefits of acquiring social information and disease, the relative timescales of the transmission processes, as well as the sociality of other members of the population. As a result, we show how ecological conditions can determine if: (1) the population evolves towards an evolutionarily stable level of sociality; (2) disease is unable to constrain selection for sociality; or (3) if selection drives social information and disease ‘extinct’.

MODEL DESCRIPTION

FULL MODEL

We model the (co)evolution of sociality and virulence due to the transmission of social information and infectious disease (Fig. 1). We assume that the environment is constantly changing (e.g., food sites may be ephemeral, predation risk may vary over time and space) and that individuals can learn about the environment independently and through social cues. For simplicity, individuals either have poor (P) or good (G) information about the environment. Those with poor information have a baseline mortality rate of d (on average), and those with good information have mortality rate ad, where $0 \leq a < 1$. All individuals continuously learn about the environment at a constant (arbitrary) rate, but the extent to which an individual can learn independently is assumed to be limited, corresponding to a relatively poor information state (P). An individual in a poor information state can only improve to a good information state (G) through social cues from an individual who is better informed about the environment, which they do with probability τ given social contact. Individuals are poor at integrating information, such that multiple individuals with poor information cannot come together to produce good information—good information can only be socially learnt. For example, the location of a highly camouflaged predator may (almost never) be individually discovered, but is readily socially learnt, and that information is only valid while the individual is foraging nearby the predator. Since the environment is constantly changing, and since individual priorities may change (e.g., an individual may seek shelter once satiated, seek a different type of food, or simply leave the area), socially learned information loses value to an individual over time (but may still be valuable to

others). For simplicity, individuals in the good information state revert to the poor state of information at a constant rate, σ.

In addition to their information status, individuals are classed as either susceptible (S) or infectious (I). Infected hosts transmit disease to susceptible social contacts with probability β per social contact. Infected hosts recover at rate γ, at which point they become susceptible again. Infection is detrimental and leads to an increase in the mortality rate due to virulence, α.

We model social contacts following Bonds et al. (2005). Social contacts are assumed to be ephemeral, random, and occur based on contact 'effort'. Each individual, i, has a contact effort, E_i, which is assumed to be a heritable quantitative trait. An individual's contact frequency, $C_i = E_i\bar{E}$, is the product of its own contact effort and the average contact effort of the population $\left(\bar{E} = \frac{1}{N}\sum_{j=1}^{N} E_j\right)$. We assume that the only benefits and costs associated with contact effort (sociality) are access to survival-related information and the risk of infection, respectively.

In a monomorphic population with $E_i = E$ there are four classes (S_P, S_G, I_P, I_G), where S_i and I_i refer to susceptible and infected individuals in information state $i \in \{P, G\}$ and the total population size is $N = S_P + S_G + I_P + I_G$. The per-capita birth rate is equal to $(b - qN)$, where b is the maximum birth rate and q is the strength of density-dependence (resource competition). The per-capita information transmission rate is equal to the product of the information transmission probability per contact, τ, the population contact frequency, E^2, and the proportion of individuals in state G, $\frac{1}{N}(S_G + I_G)$. Similarly, the disease transmission rate is equal to the product of the disease transmission probability per

contact β, the population contact frequency, E^2, and the proportion of individuals who are infected, $\frac{1}{N}(I_P + I_G)$. The population dynamics are described by the following set of ordinary differential equations (ODEs):

$$\frac{dS_P}{dt} = (b - qN)N - \frac{E^2 S_P}{N}\big(\beta(I_P + I_G) + \tau(S_G + I_G)\big) - dS_P + \gamma I_P + \sigma S_G \quad (1a)$$

$$\frac{dS_G}{dt} = \frac{E^2}{N}\big(\tau S_P(S_G + I_G) - \beta S_G(I_P + I_G)\big) - adS_G + \gamma I_G - \sigma S_G \quad (1b)$$

$$\frac{dI_P}{dt} = \frac{E^2}{N}\big(\beta S_P(I_P + I_G) - \tau I_G(S_G + I_G)\big) - (d + \alpha + \gamma)I_P + \sigma I_G \quad (1c)$$

$$\frac{dI_G}{dt} = \frac{E^2}{N}\big(\beta S_G(I_P + I_G) + \tau I_G(S_G + I_G)\big) - (ad + \alpha + \gamma + \sigma)I_G \quad (1d)$$

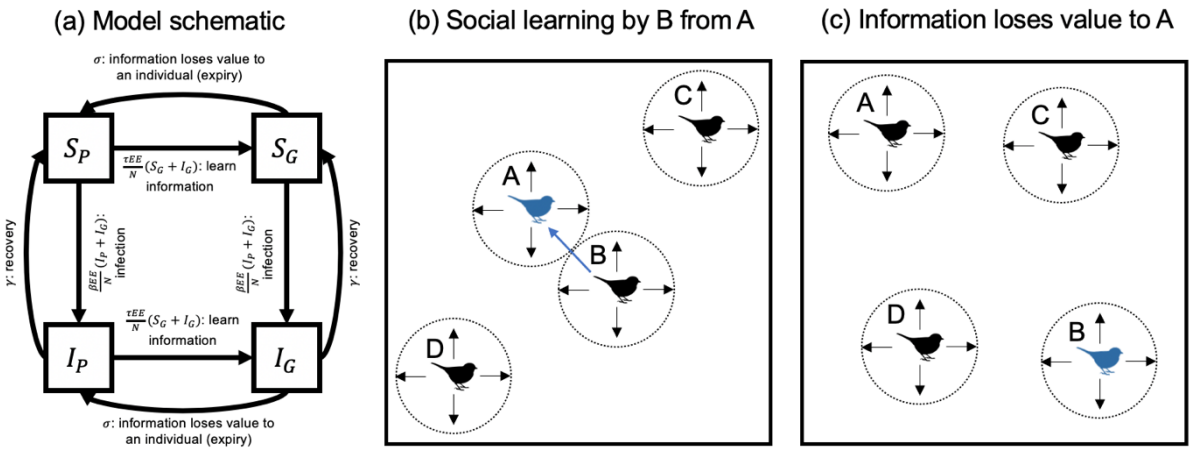

Figure 1. Model schematic and illustration of social information dynamics. (a) Transition diagram with per-capita rates of transition between states, as described in the main text. (b-c) Illustration of social information dynamics. (b) An individual, B, in the "poor" information state (black) learns socially transmitted information about the environment from individual A, who is better informed (i.e., in the "good" information state, blue). (c) Information loses value to individual A (expires), but remains valuable to individual B (e.g., individual A may be satiated or may leave the area).

FAST SOCIAL INFORMATION APPROXIMATION

When the dynamics of social information are much faster than demographic ($\tau E^2, \sigma \gg b, d$) and epidemiological processes ($\tau E^2 \gg \beta, \sigma \gg \gamma$), the proportion of individuals in the good information state for a given value of E, G_E, rapidly reaches equilibrium. Letting $G_E = \frac{1}{\mathrm{N}}(S_G + I_G)$, we see that:

$$\frac{dG_E}{dt} = \frac{1}{N}\left(\frac{dS_G}{dt} + \frac{dI_G}{dt}\right)$$

$$\approx (\tau E^2(1 - G_E) - \sigma)G_E = 0 \qquad (2)$$

Thus, either no individuals are in information state G, or $G_E^* = 1 - \frac{\sigma}{\tau E^2}$ when $\sigma < \tau E^2$. We can therefore make the following approximation to the full model when social information dynamics are relatively fast:

$$\frac{dS}{dt} = (b - qN)N - \frac{\beta E^2 SI}{N} - d(1 - (1 - a)G_E^*)S + \gamma I \qquad (3a)$$

$$\frac{dI}{dt} = \frac{\beta E^2 SI}{N} - (d(1 - (1 - a)G_E^*) + \alpha + \gamma)I \qquad (3b)$$

where $N = S + I$ and $G_E^* = \max\left(1 - \frac{\sigma}{\tau E^2}, 0\right)$ is the proportion of individuals in information state G. Note that G_E^* is an increasing, concave downward function of E, which means that there are diminishing effects of sociality on mortality.

ANALYSIS

We use evolutionary invasion analysis (adaptive dynamics; Geritz et al. 1998) to determine how the host and parasite traits (co)evolve. For the full model (equation 1), we derive host fitness analytically (see *Supplementary Material* for details), but solve the system numerically, as it is not possible to find an expression for the coexistence equilibrium where disease and social information are both present. For the fast social information approximation (equation 3), it is possible to find an expression for the coexistence equilibrium, and in turn, expressions for fitness and fitness gradients. However, we omit these expressions as they are lengthy and provide no analytical insights, and so we present numerical results instead. We illustrate the different dynamics using evolutionary simulations as described in previous work (Ashby and Bruns 2018). Briefly, every T time units of the deterministic ODE solver, a rare mutant is introduced which is phenotypically similar to a randomly chosen member of the population. Phenotypes which fall below an

arbitrary frequency, ϵ, are removed from the population, and the process is repeated. The simulations relax the adaptive dynamics assumptions of continuous traits and a complete separation of ecological and evolutionary time scales (source code available in the *Supplementary Material* and at github.com/ecoevogroup/Ashby_and_Farine_2020).

ECOLOGICAL DYNAMICS

In the full model, the host population has a trivial equilibrium at $S_P^* = N^* = (b - d)/q$ in the absence of disease or social information, which exists provided $b > d$ (hereafter we assume this condition is always satisfied). This equilibrium is stable if:

$$\hat{R}_0^I = \frac{\tau E^2}{ad + \sigma} < 1 \tag{4}$$

$$\hat{R}_0^D = \frac{\beta E^2}{d + \alpha + \gamma} < 1 \tag{5}$$

where $\hat{R}_0^I$ and $\hat{R}_0^D$ are the basic reproductive ratios for information and disease at the trivial equilibrium, respectively. The basic reproductive ratios are the average number of transmission events per informed or infected individual in an otherwise uninformed or susceptible population. If $\hat{R}_0^I > 1$ then social information can spread in the absence of disease and if $\hat{R}_0^D > 1$ then disease can spread in the absence of social information.

The full model also has three non-trivial equilibria: (i) information-only (viable if $\hat{R}_0^I > 1$), (ii) disease-only (viable if $\hat{R}_0^D > 1$), and (iii) both information and disease present (coexistence equilibrium). The latter is viable if $R_0^I > 1$ at the disease-only equilibrium and $R_0^D > 1$ at the

information-only equilibrium, where R_0^I and R_0^D are the full basic reproductive ratios for information and disease:

$$R_0^I = \frac{\tau E^2 \left(\Gamma_\gamma^I I_P + S_P(\Gamma_\alpha^I + \gamma)\right) N + \beta E^2 I_P (I_P + S_P)}{N\left((ad + \sigma)(\Gamma_\alpha^I + \gamma)N + \beta E^2 I_P \Gamma_\alpha^I\right)} \tag{6}$$

$$R_0^D = \frac{\beta E^2 \left((\Gamma_D + \sigma)S_G + S_P(\Gamma_\alpha^I + \gamma)\right)N + \tau E^2 S_G (S_P + S_G))}{N(\Gamma_D(\Gamma_\alpha^I + \gamma)N + \tau E^2 S_G \Gamma_\alpha^I)} \tag{7}$$

with $\Gamma_\alpha^I = ad + \alpha + \sigma$, $\Gamma_\gamma^I = ad + \gamma + \sigma$ and $\Gamma_D = d + \alpha + \gamma$ (see *Supplementary Material* for derivation). Note that in the special case where there is either no disease or no social information, equations 6-7 reduce to equations 4-5. Intuitively, for the coexistence equilibrium to be stable, we require $R_0^I > 1$ at the disease-only equilibrium and $R_0^D > 1$ at the information-only equilibrium (i.e. social information and disease can always spread when rare). While it is not possible to obtain an analytical expression for the coexistence equilibrium, a numerical parameter sweep indicates that it is always stable when it is viable.

The fast social information approximation has two equilibria. There is a disease-free equilibrium at $S^* = \frac{b - d(1-(1-a)G_E^*)}{q}$ and a coexistence equilibrium at:

$$\frac{S^*}{N^*} = \frac{d(1 - (1-a)G_E^*) + \alpha + \gamma}{E^2 \beta} \tag{8}$$

$$\frac{I^*}{N^*} = \frac{E^2\beta - d(1 - (1-a)G_E^*) - \alpha - \gamma}{E^2 \beta} \tag{9}$$

where

$$N^* = \frac{\alpha^2 + \alpha(d(1 - (1-a)G_E^*) + \gamma - E^2\beta) + E^2\beta(b - d(1 - (1-a)G_E^*))}{E^2 \beta q} \tag{10}$$

The corresponding basic reproductive ratios for the fast social information approximation are:

$$\tilde{R}_0^I = \frac{\tau E^2}{\sigma} \tag{11}$$

$$\tilde{R}_0^D = \frac{\beta E^2}{d(1-(1-a)G_E^*) + \alpha + \gamma} \tag{12}$$

(see *Supplementary Material*). The coexistence equilibrium is stable when $\tilde{R}_0^D > 1$ and is unstable when $\tilde{R}_0^D < 1$ (the opposite is true for the disease-free equilibrium). Note that when $\tilde{R}_0^I < 1$ we must have $G_E^* = 0$ (social information transmission cannot be sustained), and hence $\tilde{R}_0^D = \hat{R}_0^D$.

The basic reproductive ratios for the full model and the approximation are very similar provided social information dynamics are sufficiently fast ($\tau \gg \beta$; Fig. 2a-b). When information dynamics occur on slower timescales, then the approximation diverges from the full model (Fig. 2a-b). Likewise, as the information dynamics become increasingly fast relative to the disease dynamics, the equilibrium population distributions in the full model approach that of the approximation (Fig. 2c-d).

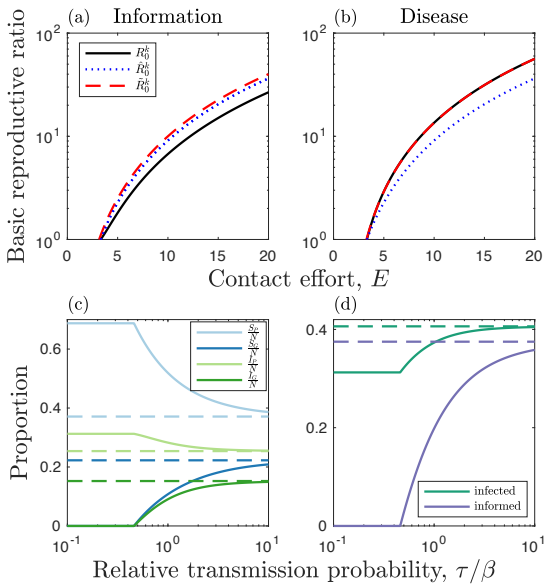

Figure 2. Ecological dynamics. (a)-(b) Basic reproductive ratios for (a) social information ($k = I$) and (b) disease ($k = D$) as a function of the contact effort, E. Curves correspond to the basic reproductive ratio in the full model, R_0^k (black, solid), the full model without disease or social information present, $\hat{R}_0^k$ (blue, dotted), and the approximation for fast social information dynamics, $\tilde{R}_0^k$ (red, dashed). (c)-(d) Comparison of the non-trivial equilibria in the full model (solid) and the approximation for fast social information (dashed). All parameters except for τ and σ are fixed, with $\sigma/\tau = 10$ held constant. The full model approaches the approximation when social information dynamics are fast relative to other processes. (c) Proportion of individuals in each class. (d) Proportion of individuals that are infected (green) or are in the good information state (purple). Fixed parameters (where applicable): $a = 0.2$, $b = 1$, $d = 0.5$, $E = 4$, $q = 10^{-3}$, $\alpha = 0.4$, $\beta = 0.1$, $\gamma = 0.2$, $\sigma = 1$, $\tau = 0.1$.

EVOLUTION OF SOCIALITY

We explore the evolution of host sociality in terms of the contact effort, E. We first investigate how the fitness gradient varies for a fixed value of E to illustrate how social information and disease prevalence influence the direction of selection. We then focus our analysis on the extent to which social information reduces mortality (a), the cost of being infected (α), the rate at which social information loses value to an individual (expires)

relative to its transmission probability (σ/τ), and the relative transmission probabilities of social information and disease (τ/β).

To explore host evolution, we consider the invasion of rare host mutant with contact effort $E_m \approx E$ into an established resident population at equilibrium (denoted by asterisks). In the full model, the invasion dynamics of the rare host mutant are given by:

$$\frac{dS_P^m}{dt} = (b - qN^*)N_m - \frac{E_m E S_P^m}{N^*}\left(\beta(I_P^* + I_G^*) + \tau(S_G^* + I_G^*)\right) - dS_P^m + \gamma I_P^m + \sigma S_G^m \quad (13a)$$

$$\frac{dS_G^m}{dt} = \frac{E_m E}{N^*}\left(\tau S_P^m(S_G^* + I_G^*) - \beta S_G^m(I_P^* + I_G^*)\right) - adS_G^m + \gamma I_G^m - \sigma S_G^m \quad (13b)$$

$$\frac{dI_P^m}{dt} = \frac{E_m E}{N^*}\left(\beta S_P^m(I_P^* + I_G^*) - \tau I_P^m(S_G^* + I_G^*)\right) - (d + \alpha + \gamma)I_P^m + \sigma I_G^m \quad (13c)$$

$$\frac{dI_G^m}{dt} = \frac{E_m E}{N^*}\left(\beta S_G^m(I_P^* + I_G^*) + \tau I_P^m(S_G^* + I_G^*)\right) - (ad + \alpha + \gamma + \sigma)I_G^m \quad (13d)$$

For the fast social information approximation, we let $G_E^m = \frac{1}{N_m}(S_G^m + I_G^m)$ so that:

$$\frac{dG_E^m}{dt} = \frac{1}{N_m}\left(\frac{dS_G^m}{dt} + \frac{dI_G^m}{dt}\right)$$

$$\approx (E_m E\tau(1 - G_E^m)G_E^* - \sigma G_E^m)N_m = 0 \quad (14)$$

Rearranging and substituting $G_E^* = 1 - \frac{\sigma}{\tau E^2}$ gives:

$$G_E^m = \max\left(\frac{E_m(\tau E^2 - \sigma)}{E^2 E_m \tau + \sigma(E - E_m)}, 0\right) \quad (15)$$

We can therefore approximate the invasion dynamics of a rare mutant when social information dynamics are fast by:

$$\frac{dS_m}{dt} = (b - qN^*)N_m - \frac{\beta E_m E S_m I^*}{N^*} - d(1 - (1 - a)G_E^m)S_m + \gamma I_m \tag{16a}$$

$$\frac{dI_m}{dt} = \frac{\beta E_m E S_m I^*}{N^*} - (d(1 - (1 - a)G_E^m) + \alpha + \gamma)I_m \tag{16b}$$

In the *Supplementary Material*, we show the derivation for invasion fitness in both versions of the model.

HOST FITNESS GRADIENT

Fig. 3 shows how the host fitness gradient varies with social information and disease prevalence when the benefits of sociality are relatively high (Fig. 3a) or low (Fig. 3b) compared to the costs. Social information dynamics are assumed to be fast, but the overall patterns hold for slow information dynamics. Note that here E is fixed, with σ and β modulating social information and disease prevalence, but when E evolves (see *Host evolutionary dynamics*) it will feedback to affect prevalence and hence selection for sociality. Nevertheless, considering how the fitness gradient varies when E is fixed allows us to qualitatively see how changes in social information and disease prevalence affect the fitness gradient, all else being equal.

The fitness gradient is always positive when social information prevalence is intermediate and disease is either rare or common, regardless of whether the benefits of sociality are relatively high or low compared to the costs. Intuitively, increased sociality is likely to be beneficial when disease prevalence is low, but only if social information is neither too rare nor very common. This is because greater sociality does not significantly increase the rate at which individuals obtain social information if it is readily available or unlikely to become

309 accessible. Somewhat counter-intuitively, increased sociality may also be beneficial when
310 disease prevalence is high (as shown in Bonds *et al.*, 2005). This is because most individuals
311 are likely to be infected anyway, but increased sociality will improve access to social
312 information with little increase to the risk of infection. Conversely, the fitness gradient is
313 always negative when social information is either rare or very common and disease is at
314 intermediate prevalence. This pattern mirrors the one described above, but with the roles
315 of social information and disease switched due to their opposing effects on the fitness
316 gradient. Intuitively the fitness gradient is negative when social information prevalence is
317 low and disease is common in the population, as greater sociality increases the risk of
318 infection but does not substantially increase the likelihood of obtaining social information.
319 But perhaps counter-intuitively the fitness gradient is also negative when social information
320 is widespread and disease is at moderate levels. This is because most individuals are well-
321 informed, and so increased sociality will only marginally improve access to social
322 information but may substantially increase the risk of infection.

323 When both social information and disease are at intermediate prevalence, the sign of the
324 fitness gradient depends on the relative costs and benefits of being infected or informed.
325 When awareness of social information is relatively more valuable than the cost of being
326 infected, the fitness gradient is positive whenever social information prevalence is at an
327 intermediate level regardless of disease prevalence (Fig. 3a). Disease may therefore not
328 always be sufficient to curtail the evolution of sociality. In contrast, when awareness of
329 social information is less valuable, and the cost of being infected is relatively high, the
330 fitness gradient is negative whenever disease prevalence is at an intermediate level
331 regardless of social information prevalence (Fig. 3b). Our qualitative analysis of the fitness

gradient suggests that the host population may experience different evolutionary outcomes depending on the initial conditions: if disease prevalence is sufficiently low the population may evolve towards a stable level of sociality, but if disease is initially very common then the population may experience runaway selection for greater sociality.

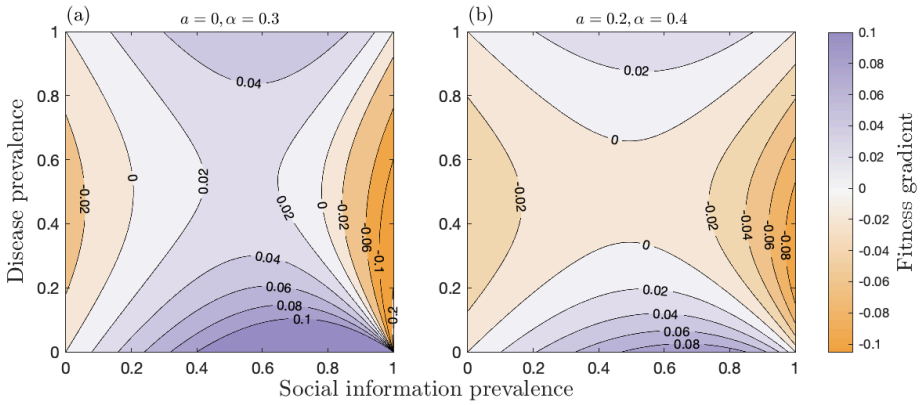

Figure 3. Effects of social information and disease prevalence on the host fitness gradient. Contour plots of the host fitness gradient, in the fast social information approximation as a function of social information and disease prevalence with: (a) higher ($a = 0, \alpha = 0.3$) and (b) lower ($a = 0.2, \alpha = 0.4$) benefits of sociality relative to costs. All parameters except for σ and β are fixed within each panel, with values of σ and β chosen so that social information and disease prevalence vary accordingly. In both cases, the host fitness gradient is positive (bluer shading) when social information prevalence is intermediate and there is either a high or low level of disease, and is negative (redder shading) when social information prevalence is either at a high or low level and disease is at intermediate prevalence. Fixed parameters as in Fig. 2, except $\beta = 0.2$.

HOST EVOLUTIONARY DYNAMICS

When the rate at which social information loses value to an individual (the expiry rate), σ, is not too large relative to the information transmission probability, τ, there may exist a *continuously stable strategy* or CSS, which is locally attracting (dark blue curves in Fig. 4). Nearby trait values evolve towards the CSS by small mutations and remain there indefinitely (Fig. 5a-b). When the costs (α) that arise from being social are relatively low compared to the benefits of being better informed (a), the CSS increases with σ/τ until there is no

evolutionarily stable contact effort, leading to runaway selection for higher values of E (Fig. 4a, d). When the costs of sociality are higher, the CSS peaks at intermediate values of σ/τ (e.g. Fig. 4b). This is because as σ/τ increases, social information expires more quickly and so the overall prevalence of social information in the population falls, which increases selection for sociality. However, as sociality increases so too does disease prevalence, which reduces selection for sociality. Thus, if the costs of being more social are relatively low, the population may experience runaway selection, but as the costs increase due to higher disease prevalence, sociality is curtailed at a stable intermediate level.

Two possibilities exist for initial trait values above the CSS. For sufficiently low values of σ/τ (social information is common) and higher costs of disease (α) or lower benefits of social information (a), the CSS, E^*, is attracting for all values of $E > E^*$. This means that high initial levels of sociality will always evolve to a lower level. But for sufficiently large values of σ/τ (social information is rare) or low costs of disease (α) and high benefits of social information (a), a *repeller* (dark red curves in Fig. 4) may exist above the CSS. Contact effort evolves towards the CSS when the initial value is below the repeller, which reduces infection and social information prevalence to a point where the costs and benefits of sociality are balanced (Fig. 5). There is runaway selection for greater sociality when the contact effort starts above the repeller, which increases infection and social information prevalence (Fig. 5c). Runaway selection occurs because most individuals in the population are infected, and so there is little cost to increased sociality. Runaway selection for sociality is unrealistic, however, as contact efforts would be constrained by other factors (e.g. non-disease associated costs or physical constraints). Instead, runaway selection for sociality in the model should be interpreted as disease being insufficient to constrain sociality.

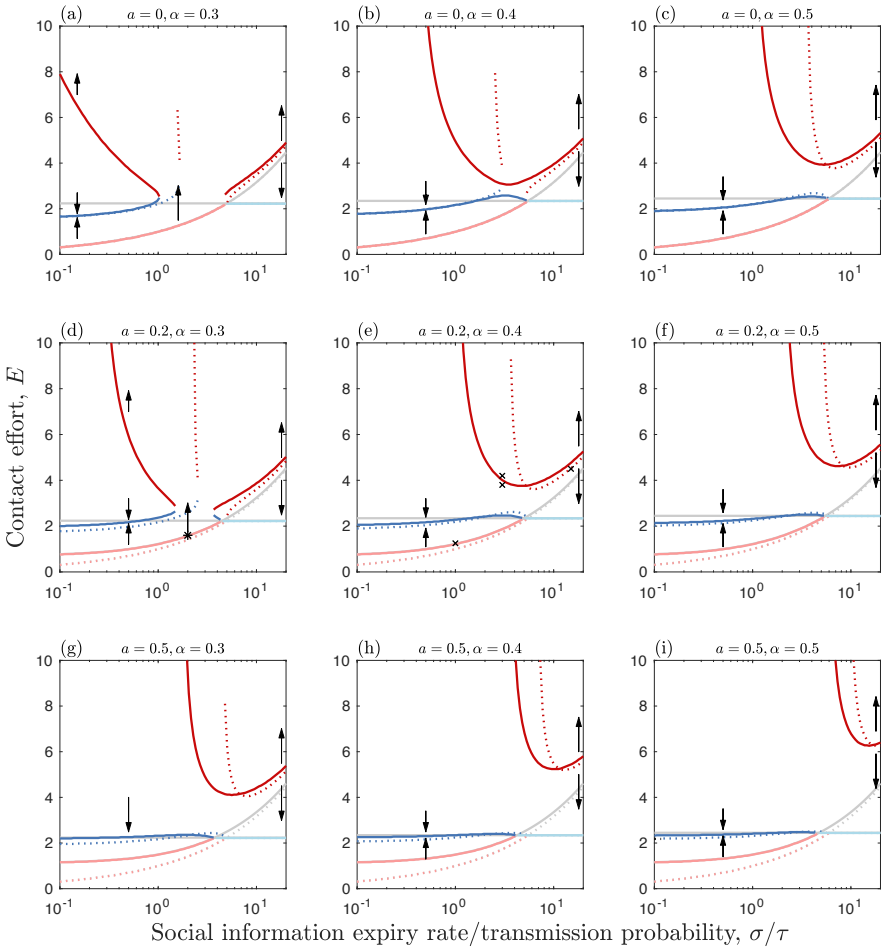

Figure 4. The evolution of sociality arising from social information and disease transmission. Evolution of contact effort, E, as a function of the social information expiry rate divided by the transmission probability, σ/τ, and for different values of the benefits of social information, a, and costs of disease, α. Results are shown for relatively slow (solid) and fast (dotted) social information dynamics. Red and dark blue curves correspond to repellers and continuously stable strategies (CSSs), respectively. Pink and light blue curves show thresholds below which neither social information nor disease transmission is viable. Above the pink curve, sociality increases and above the light blue curve sociality decreases. Arrows indicate the direction of selection when social information dynamics are relatively slow. Grey curves do not indicate singular strategies but are continuations of the disease and social information viability thresholds when $N = S_P$. Crosses in (e) correspond to simulations in Fig. 5 and the asterisk in (d) corresponds to simulations in Fig. 6. Fixed parameters as in Fig. 2, except $\beta = 0.2$.

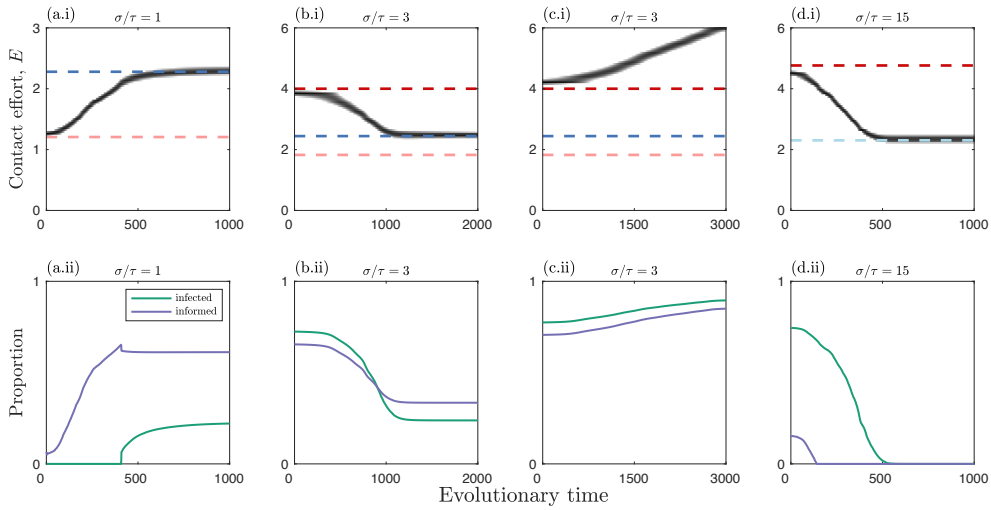

Figure 5. Simulation trajectories when social information and disease dynamics occur on similar timescales. Trajectories of simulations corresponding to crosses in Fig. 4e. Top row: evolutionary trajectories (solid black); singular strategies: repellers (red) and continuously stable strategies (CSSs; dark blue); and thresholds below which neither social information nor disease transmission is viable (pink and light blue). Above the pink dashed line, sociality increases, and above the light blue line sociality decreases. Bottom row: proportion of individuals that are infected (green) or are in the good information state (purple) in each simulation. Fixed parameters as in Fig. 2, except $\tau = \beta = 0.2$.

For sufficiently large values of σ/τ, the threshold for sustained social information transmission passes the threshold for sustained disease transmission ($R_0^D > R_0^I$ or $\tilde{R}_0^D > \tilde{R}_0^I$). Below these thresholds neither social information nor disease can be sustained and so the fitness gradient is zero (i.e., no selection on the contact effort). For the reasons discussed above, a repeller may also exist in this region of parameter space, where for sufficiently high contact efforts, there is runaway selection for greater sociality. Increasing the costs of disease (α) or decreasing the benefits of social information (a) limits where the repeller exists to larger values of σ/τ and greater contact efforts. This has the effect of increasing the region of attraction for the CSS. Conversely, decreasing the costs of disease or increasing

the benefits of social information may cause the repeller to collide with the CSS at intermediate values of σ/τ (Fig. 4a, d, g). In this region, disease is less costly and information more beneficial, so as soon as social information is able to spread there is runaway selection for greater sociality.

Thus far, we have only discussed the evolution of sociality when social information and disease dynamics occur on similar timescales ($\sigma \approx \gamma$). When social information dynamics occur on a much faster timescale ($\sigma \gg \gamma$), there are some notable differences (dotted curves in Fig. 4). First, the viability thresholds (pink and light blue curves) for disease and social information are shifted to slightly lower contact efforts. Second, the repeller curve for fast social information dynamics is shifted to the right and down in Fig. 4, which may increase or decrease the domain of attraction for the CSS depending on the value of σ/τ. Third, the collision of the CSS and repeller occurs for larger costs of disease (α) and smaller benefits of social information (a). Finally, at low cost of disease, a CSS may exist which is absent in the full model (e.g. Fig. 4d). This means that when social information dynamics are relatively slow, there may be runaway selection for greater sociality, but when the social information dynamics are relatively fast, sociality may evolve to a stable level (Fig. 6). The timescale of social information dynamics can therefore play an important role in determining the evolution of sociality.

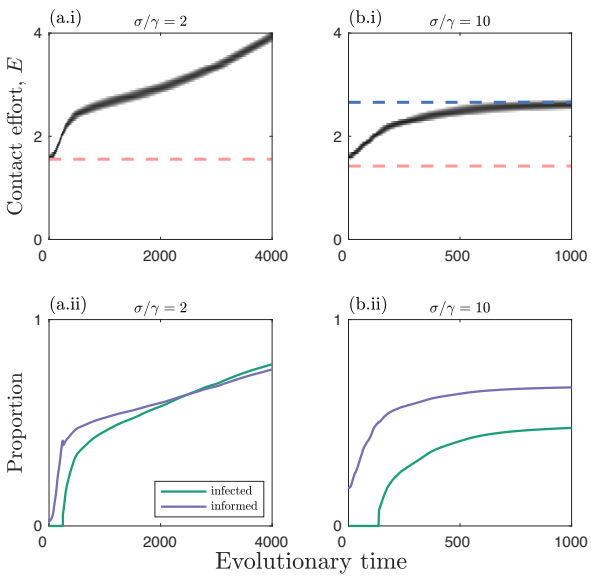

Figure 6. Simulation trajectories when social information dynamics occurs on similar (a) or faster (b) timescales than disease dynamics. Simulations corresponding to the asterisk in Fig. 4d. Top row: evolutionary trajectories (solid black); continuously stable strategies (CSSs; dark blue); and viability threshold for social information (pink). Bottom row: proportion of individuals that are infected (green) or are informed by social information (purple) in each simulation. Fixed parameters as in Fig. 2, except $\alpha = 0.3$, $\beta = 0.2$, $\sigma/\tau = 2$.

EVOLUTION OF VIRULENCE

To explore parasite evolution, we assume there is a trade-off between transmission and virulence such that $\beta = \beta(\alpha)$ (i.e. more transmissible parasites are more virulent). We consider the invasion of rare parasite mutant with virulence $\alpha_M \approx \alpha$ into an established resident population at equilibrium (denoted by asterisks). For simplicity we assume that social information dynamics are fast (i.e. we use the approximation to the full model). The invasion dynamics of the rare parasite mutant are then given by:

$$\frac{dI_M}{dt} = \frac{\beta(\alpha_M)E^2S^*I_M}{N^*} - (d(1-(1-a)G_E^*) + \alpha_M + \gamma)I_M \qquad (17)$$

In the *Supplementary Material*, we derive the invasion fitness and show that evolution maximises the basic reproductive ratio, $\tilde{R}_0^D(\alpha)$:

$$\tilde{R}_0^D(\alpha) = \frac{\beta(\alpha)E^2}{d(1-(1-a)G_E^*) + \alpha + \gamma} \tag{18}$$

We now consider how the evolution of sociality might feedback to affect the evolution of parasite virulence. In the absence of a trade-off with another life-history trait, the parasite will evolve to the lowest level of virulence possible, as this increases the infectious period. We therefore assume, as is common in the literature (Alizon 2008), that there is a positive relationship between virulence and the transmission probability with diminishing returns, so that $\frac{d\beta}{d\alpha} > 0$ and $\frac{d^2\beta}{d\alpha^2} < 0$. The optimal level of virulence, α^*, occurs when:

$$\left.\frac{d\beta}{d\alpha}\right|_{\alpha=\alpha^*} = \frac{\beta(\alpha^*)E^2\tau}{E^2\tau(ad+\alpha^*+\gamma) + d\sigma(1-a)} = g(\alpha^*, E)\beta(\alpha^*) \tag{19}$$

To determine what happens to the optimal level of virulence when sociality changes, consider $y(\alpha, E) = g(\alpha, E)\beta(\alpha) - \frac{d\beta}{d\alpha}$ and suppose $\alpha = \alpha^*$, so that $y(\alpha^*, E) = 0$. Note that $g(\alpha, E)$ is a decreasing function of α and is an increasing function of E provided $0 \leq a < 1$. Hence, $y(\alpha, E)$ is also an increasing function of E. Now consider the derivative of y with respect to α at $\alpha = \alpha^*$:

$$\begin{aligned}\left.\frac{\partial y}{\partial \alpha}\right|_{\alpha=\alpha^*} &= \beta(\alpha^*)\left.\frac{dg}{d\alpha}\right|_{\alpha=\alpha^*} + g(\alpha^*, E)\left.\frac{d\beta}{d\alpha}\right|_{\alpha=\alpha^*} - \left.\frac{d^2\beta}{d\alpha^2}\right|_{\alpha=\alpha^*} \\ &= -\left.\frac{d^2\beta}{d\alpha^2}\right|_{\alpha=\alpha^*} > 0 \end{aligned} \tag{20}$$

where $\left.\frac{d\beta}{d\alpha}\right|_{\alpha=\alpha^*} = g(\alpha^*, E)\beta(\alpha^*)$ from equation 19 and $\left.\frac{dg}{d\alpha}\right|_{\alpha=\alpha^*} = -g^2(\alpha^*, E)$. This means that if sociality increases, the optimal level of virulence must decrease (Fig. 7). This may appear to contradict classical predictions on population mixing and the evolution of virulence (Ewald 1994), but the difference occurs due to the lack of population structure in the present model. Note that if the host mortality rate is independent of sociality ($a = 1$), then it follows that the optimal level of virulence occurs when:

$$\left.\frac{d\beta}{d\alpha}\right|_{\alpha=\alpha^*} = \frac{\beta(\alpha^*)}{d + \alpha^* + \gamma} \tag{21}$$

which does not depend on the contact effort, E. Thus, the reason optimal virulence is inversely related to sociality in the present model is due to effects of sociality on the mortality rate of the host (and hence the infectious period), rather than effects on population mixing, *per se*.

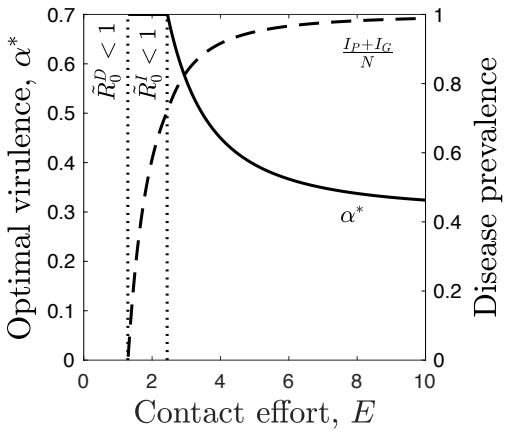

Figure 7. The evolution of virulence in the fast social information approximation. Optimal virulence, α^*, decreases with the contact effort (solid) and disease prevalence (dashed) increases. Vertical dotted lines indicate where disease ($\tilde{R}_0^D < 1$) and ($\tilde{R}_0^I < 1$) social information transmission are not viable. Other parameters as in Fig. 2, with $\beta(\alpha) = \sqrt{\alpha}$ and $0 \leq \alpha \leq 1$.

COEVOLUTION OF SOCIALITY AND VIRULENCE

We combine our results from the previous sections to analyse the effects of social information on the coevolution of sociality and virulence. For tractability, we make three simplifying assumptions: (1) social information dynamics are fast (i.e. we use the approximation to the full model); (2) the parasite evolves much faster than the host; and (3) the relationship between transmission and virulence is $\beta(\alpha) = \sqrt{\alpha}$ and $0 \leq \alpha \leq 1$. Simulations indicate that relaxing these assumptions does not qualitatively affect our results. The second assumption allows us to substitute $\alpha = \alpha^*$ into the host fitness gradient, where:

$$\alpha^* = \begin{cases} \dfrac{d\sigma(1-a)}{E^2\tau} + (ad+\gamma) & \text{for } \tilde{R}_0^I > 1 \\ d+\gamma & \text{for } \tilde{R}_0^I < 1 \end{cases} \tag{22}$$

The range of qualitative outcomes for coevolution are the same as for one-sided adaptation in the host (Fig. 8). The host and parasite may tend to a joint stable level of sociality and virulence, respectively, or they may experience runaway selection for higher sociality and lower virulence, or disease or social information may be unviable. However, note that in the case of runaway selection virulence asymptotes to a lower bound of $\alpha^* = ad + \gamma$ (from equation 22). Under coevolution, the evolutionary stable level of virulence increases with the ratio of the information expiry rate to transmissibility (σ/τ), until social information dynamics are no longer viable ($\tilde{R}_0^I < 1$).

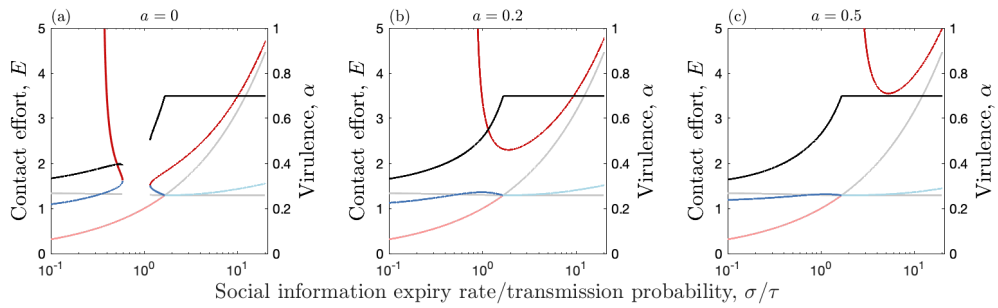

Figure 8. Coevolution arising from social information and disease transmission dynamics. Coevolution of host sociality (contact effort, E) and parasite virulence (α) as a function of the social information expiry rate divided by the transmission probability, σ/τ, and for different values of the benefits of social information, a (fast social information approximation). Black curves show the evolutionary stable level of virulence that corresponds to the stable (dark blue) level of host sociality (or the threshold below which disease is driven extinct, light blue). All other curves as described in Fig. 4. Other parameters as in Fig. 2, with $\tau = 1$, $\beta(\alpha) = \sqrt{\alpha}$ and $0 \leq \alpha \leq 1$.

Scenario	Variable	Description of results	Figure(s)
Host evolution	Disease prevalence	Selection against sociality at high/low disease prevalence	Fig. 3
	Social information prevalence	Selection for sociality at high/low social information prevalence	Fig. 3
	Social information expiry rate	Slow expiry: sociality either evolves to a stable level or increases without bound; fast expiry: sociality either increases without bound or decreases until disease is driven extinct	Fig. 4
	Social information timescale	Faster social information dynamics generally have a stabilising effect (prevent runaway selection for sociality)	Fig. 4, 6
Parasite evolution	Contact effort	Virulence decreases with higher contact efforts	Fig. 7
Coevolution	Social information expiry rate	Virulence increases with the social information expiry rate until social information is no longer viable	Fig. 8

Table 1 – Summary of key results.

493 ## DISCUSSION

494 Sociality entails both risks and benefits, but there is currently little understanding of how
495 dynamic ecological processes influence the evolution of sociality. Here, we explored a model
496 of social information and disease transmission to determine their effects on the evolution of
497 host sociality and parasite virulence. Although previous studies have examined the role of
498 parasitism in the evolution of sociality, ours is the first that captures both dynamic costs and
499 benefits of different social tendencies. This means that the fitness benefits of sociality, in
500 terms of increased access to social information, and fitness costs, in terms of increased
501 exposure to infection, are not imposed on the system, but instead are emergent properties
502 that are subject to eco-evolutionary feedbacks. Hence, changes in sociality at the individual
503 level affect the properties of transmission at the population level, and these population-
504 level properties, in turn, affect both the costs and benefits of sociality at the level of the
505 individual.

506 Our model shows how contrasting transmission processes can trade-off to mediate the
507 evolution of sociality and virulence. Crucially, we have shown that selection for sociality
508 (and hence virulence) depends non-monotonically on the prevalence of both social
509 information and disease (Fig. 3). Specifically, selection usually favours greater sociality (and
510 lower virulence) when social information is neither too common nor too rare and disease
511 risk is either low or high. Conversely, selection against sociality (and for greater virulence)
512 typically occurs when social information is either rare or very common and disease is at
513 moderate prevalence. The fact that these two processes mirror each other is not surprising:
514 the transmission processes are fundamentally the same in the model, but social information

515 is beneficial and disease is costly. We note, however, that in real populations transmission
516 processes for social information do not always mirror those of disease. In terms of social
517 information, individuals can use different learning rules, for example if they are conformist
518 (e.g. having a disproportionate preference for copying a more common behaviour, (Aplin et
519 al. 2015a) or if they require passing a certain threshold number of informed contacts before
520 becoming informed (Rosenthal et al. 2015), which can modify how transmission pathways
521 emerge from social contacts. From the perspective of diseases, the life cycle of pathogens,
522 such as the reliance of an intermediate host, can also impact how social contacts translate
523 to transmission events (Grear et al. 2013; Farine 2017).

524 In general, conditions on social information prevalence that select for greater sociality (and
525 lower virulence) are the same conditions on disease prevalence that select against sociality
526 (and for greater virulence). Naively, one might assume that selection for sociality should
527 increase with social information prevalence and decrease with disease prevalence. But
528 when social information prevalence is high, the marginal benefit of increasing sociality is
529 low, and similarly when disease prevalence is high, the marginal cost of being more social is
530 low. Slightly higher or lower sociality therefore makes little difference to the rate of
531 exposure to social information or disease when prevalence is high. The effects of disease
532 prevalence on the evolution of sociality have previously been observed by Bonds et al.
533 (2005), but the effects of social information prevalence on the evolution of sociality have
534 not previously been documented. Bonds et al. (2005) likened the relationship between
535 disease prevalence and selection for sociality to optimal host investment in an immune
536 response, which is predicted to peak when the risk of infection is neither too low, nor too
537 high. In fact, this pattern is quite general and occurs for a variety of behavioural and

538 physiological defences against parasitism, including resistance (Haldane 1949), recovery
539 (van Baalen 1998), mate choice (Ashby and Boots 2015) and sexual reproduction (Ashby and
540 King 2015).

541 The patterns described above for high/low prevalence are quite robust, but the picture is
542 more complicated when social information and disease are both at intermediate
543 prevalence. Selection may increase or decrease sociality and virulence depending on the
544 relative costs and benefits of being infected or informed, in addition to the likelihood of
545 exposure. As a consequence, it is possible for evolutionary trajectories to exist where the
546 sign of the fitness gradient is invariant to disease or social information prevalence, leading
547 to runaway selection for higher sociality/lower virulence or lower sociality/higher virulence
548 (Fig. 4, 8). Note that, while our model allowed for runaway selection for sociality, in reality
549 one would expect other factors to eventually constrain evolution (e.g., physiological
550 constraints, additional costs). We omitted additional constraints from our model so that we
551 could determine when disease alone is sufficient to constrain the evolution of sociality. The
552 results for "runaway selection" should therefore be interpreted in this light.

553 It is also possible for the initial conditions to determine the long-term evolutionary outcome
554 (stable level of sociality and virulence, or runaway selection for increased sociality and
555 decreased virulence) due to the existence of evolutionary repellers (Fig. 4, 8). The possibility
556 of more than one (co)evolutionary outcome suggests that different populations might
557 evolve very different social strategies for coping with infectious disease, and that variable
558 ecological conditions can lead to sudden shifts in sociality and virulence. How evolutionary
559 outcomes play out in a given population is likely to also be affected by the structure of the
560 social contacts. Our model assumed a well-mixed population, which can represent dynamics

561 in one small patch. However, at a broader scale, it is now clear that almost all animal
562 populations exhibit some structure (Cantor et al. 2021), and that population structure can
563 shape both transmission dynamics and evolution of diseases (Eames and Keeling 2006; Lion
564 and Boots 2010; Ashby and Gupta 2013). Structure can arise due to individual preferences
565 for certain relationships (Alberts 2019) or because the environment induces limitations on
566 who can come into contact with whom (He et al. 2019; Silk et al. 2019; Albery et al. 2021).
567 For example, recent work has suggested that—all else being equal—some landscapes may
568 be more predisposed to facilitating disease transmission than other landscapes (He et al.
569 2021). Some mammal (Grueter et al. 2020) and birds (Papageorgiou et al. 2019) also exhibit
570 multiple levels of social connections – named multilevel societies – with social contacts
571 being expressed within and across social units. Further, it is now becoming well-established
572 that individuals can change their social behaviours once they become infected (reviewed in
573 Romano et al. 2020; Stockmaier et al. 2021), which can introduce a more rapid feedback
574 between disease state and social behaviour. Thus, future research should aim to determine
575 how specific social strategies might evolve under different environmental conditions (e.g.
576 Udiani and Fefferman 2020).

577 An important consideration when modelling the benefits of sociality dynamically in terms of
578 social information, as opposed to fixed benefits (Bonds et al. 2005; Prado et al. 2009), is the
579 timescale of information dynamics relative to other processes. We have shown that for the
580 most part, evolutionary outcomes are qualitatively and quantitatively similar whether social
581 information dynamics are relatively fast or on a similar timescale to demographic and
582 epidemiological processes. However, this is not always the case. For example, increasing the
583 speed of social information dynamics relative to other processes has a stabilising effect on

584 the evolution of sociality (Fig. 6). In general, faster social information dynamics tend to have
585 a stabilising effect, reducing parameter ranges for runaway selection. Hence, while one can
586 often approximate social information dynamics by assuming the population rapidly reaches
587 a stable distribution, this assumption does not always hold and can fundamentally change
588 evolutionary outcomes.

589 One of the key predictions from this study and previous work (Bonds et al. 2005) is that
590 sociality should vary inversely with virulence, which may appear at odds with classical
591 predictions for virulence evolution (Ewald 1994). The contradiction is resolved by noticing
592 that: (1) our model considers contact rates in a randomly mixing population rather than
593 contact number in a structured population; and (2) higher contact rates do not directly
594 select for lower virulence, but do so indirectly through the spread of social information,
595 which reduces host mortality and increases the infectious period. Hence, greater sociality as
596 modelled here is not equivalent to population mixing in a structured model, and the effects
597 on virulence evolution are mediated via host mortality rather than contact rates. Based on
598 these insights, we therefore predict that sociality should covary inversely with virulence in
599 populations where social or spatial structuring is weak but should covary positively with
600 virulence when structuring is strong.

601 In previous studies, the fitness benefits of sociality were fixed rather than dynamic (Bonds et
602 al. 2005; Prado et al. 2009). While some studies have explored how social information and
603 infectious disease affect the contact structure of a population, this has been in the form of
604 host plasticity during an epidemic, rather than an evolutionary response (Funk et al. 2010),
605 or where the outcomes are compared across populations with different fixed social
606 strategies (Udiani and Fefferman 2020). Here, the two processes operate as independent

607 forces of selection. Our findings are closely linked to those of Bonds et al. (2005), where
608 sociality and virulence are shown to coevolve to stable levels. In contrast, Prado et al. (2009)
609 have shown that coevolutionary cycling (fluctuating selection) may occur between sociality
610 and virulence (Prado et al. 2009). It is not clear why Prado et al. (2009) found cycles, yet
611 Bonds et al. (2005) and the present study have not, but part of the reason may be due to
612 model structure. Prado et al. (2009) modelled disease dynamics using a stochastic
613 individual-based network, whereas Bonds et al. (2005) and the present study employed
614 deterministic models with randomly-mixing populations. A priority for future research in
615 this area is therefore to establish precisely when coevolutionary cycling of sociality and
616 virulence occurs, and whether this is driven by population structure and/or stochasticity.

617 The present study is relevant to empirical research on the sociality of animal populations
618 (Dall et al. 2004; Sih et al. 2004; Dingemanse and Réale 2005; Evans et al. 2020). In
619 particular, a number of studies have shown that variation in sociality influences the spread
620 of social information (Aplin et al. 2012; Allen et al. 2013; Farine et al. 2015a; Snijders et al.
621 2021). For example, the social network attributes of wild great tits (*Parus major*) can be
622 used to predict the discovery rate of novel food patches (Aplin et al. 2012). As in previous
623 theoretical studies (Bonds et al. 2005; Prado et al. 2009), we did not observe coexistence of
624 different social strategies. However, several empirical studies have highlighted the potential
625 importance of maintaining mixed strategies in social groups or local populations. For
626 example, Aplin et al. (2014) suggested that variation in sociality could increase group-level
627 efficiency in exploiting patches. Similarly, in ants, mixed colonies (in terms of aggressive
628 type) have more efficient task allocation and are more successful at capturing prey
629 (Modlmeier et al. 2012). A potential reason for the lack of mixed social strategies in current

630 theoretical studies is that they do not include assortative mixing (Farine 2014). Assortativity
631 has been shown to facilitate the coexistence of multiple parasites (Eames and Keeling 2006),
632 so it is possible that mixed social strategies could also evolve in the presence of non-random
633 mixing – a process that could be either actively generated through social preferences or
634 passively through exogenous drivers of social structure. It is also possible that mixed social
635 strategies could evolve in fluctuating environments, especially if individuals value
636 information differently (e.g. due to personality traits, Aplin et al. 2014), or if individuals vary
637 in their propensity to form social relationships (e.g. due to developmental conditions,
638 Boogert et al. 2014). A challenge for future theoretical research is to establish whether
639 selection arising from disease and information-use dynamics can drive the evolution of
640 mixed social strategies. Our results suggest that a number of potential mechanisms (e.g.
641 variation in the perceived value of information) could facilitate this process.

642 In summary, our study suggests that not only does the sociality of the population affect
643 social information and disease dynamics, but also that these processes are likely to
644 influence the evolution of sociality and virulence. Crucially, we predict that: (1) selection for
645 sociality and virulence will vary non-monotonically with both social information and disease
646 prevalence; and (2) if social information increases host survival, then increased sociality may
647 correlate with decreased virulence. The context-dependent relationships that we have
648 shown to exist between the ecology of information/disease dynamics and the evolution of
649 sociality/virulence highlights the need to understand host and parasite evolution in the
650 context of multiple ecological processes.

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660 ## DATA AVAILABILITY

661 Source code is available in the *Supplementary Material* and at the following Github
662 repository: https://github.com/ecoevogroup/Ashby_and_Farine_2020.

663 ## COMPETING INTERESTS

664 The authors have no competing interests to declare.

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