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 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 information is neither very common nor too rare. Lower sociality and higher virulence evolve when the prevalence patterns are reversed. When disease and 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; Weber et al. 2013; Aplin et al. 2015b) 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 or increased mating opportunities), which trade-off to determine whether populations evolve to be more or less social.

Intuitively, if the costs of sociality are greater than the benefits, then selection should favour greater sociality, and vice versa if the benefits are greater than the costs. If, for example, 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. 2020a) or a reduced risk of predation (Kenward 1978), but these do not necessarily depend on the sociality of the population as a whole. However, when the

costs and benefits are dynamic, as is the case with infectious diseases and social information, feedbacks may exist between the sociality of the population and the likelihood of realising an associated cost or benefit (Cantor et al. 2020b). 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. 2020) 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 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. 2020). 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. Survival-related 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 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 general mathematical model of infectious disease and social information dynamics, allowing sociality to evolve with no inherent costs or benefits. 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 information dynamics either occur on relatively fast or slow timescales, with the former allowing for a simple approximation to information prevalence. We show that selection favours increased sociality when information is neither very common nor too rare and disease is at either high or low levels. When information and disease are both at intermediate levels, selection may favour increased or decreased sociality depending on the relative costs and benefits of acquiring information and disease, the relative timescales of the transmission processes, as well as the current level of sociality in 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 information and disease ‘extinct’.

METHODS

We model the transmission of social information and infectious disease through a well-mixed animal population. Social information (e.g. foraging sites or predation risk) is beneficial and leads to a temporary reduction in the natural mortality rate, d, to ad where $0 \leq a < 1$, while infection is detrimental and leads to an increase in the mortality rate due to virulence, α. Individuals are classed as either susceptible (S) or infectious (I), and either unaware (U) or aware (A) of survival-related information. Infected individuals transmit disease to their susceptible social contacts at rate β, and recover from infection at rate γ, at which point they become susceptible again. Similarly, individuals in state A (aware) may transmit survival-related information to social contacts in state U (unaware) at rate τ, and the information remains beneficial for an average period of $1/\sigma$ (e.g. food sites are depleted, predators leave the area). Individuals in state A revert to state U at information expiry rate σ, at which point they may receive new survival-related information. The per-capita birth rate is equal to $(b - qN)$, where b is the maximum birth rate, q is the strength of density-dependence (resource competition), and N is the total population size.

The contact initiation rate (sociality), c, is assumed to be a quantitative trait under selection. Social contacts are ephemeral and random. Each pair of individuals with contact initiation rates c_i and c_j form a social contact at rate $c_i c_j / N$, during which time information or disease transmission may occur between them. In a homogeneous population, the overall contact rate is $c^2 N$. For simplicity, we assume that there are no costs associated with sociality other than the risk of infection. The dynamics of a homogenous (resident) population are described by the following set of ordinary differential equations:

$$\frac{dS_U}{dt} = (b - qN)N - \frac{c^2 S_U}{N}\big(\beta(I_U + I_A) + \tau(S_A + I_A)\big) - dS_U + \gamma I_U + \sigma S_A \quad (1a)$$

$$\frac{dS_A}{dt} = \frac{c^2}{N}\big(\tau S_U(S_A + I_A) - \beta S_A(I_U + I_A)\big) - adS_A + \gamma I_A - \sigma S_A \quad (1b)$$

$$\frac{dI_U}{dt} = \frac{c^2}{N}\big(\beta S_U(I_U + I_A) - \tau I_U(S_A + I_A)\big) - (d + \alpha + \gamma)I_U + \sigma I_A \quad (1c)$$

$$\frac{dI_A}{dt} = \frac{c^2}{N}\big(\beta S_A(I_U + I_A) + \tau I_U(S_A + I_A)\big) - (ad + \alpha + \gamma + \sigma)I_A \quad (1d)$$

where S_i and I_i refer to susceptible and infected individuals in information state $i \in \{U, A\}$. Terms with $\frac{c^2}{N}$ correspond to transmission of social information or disease.

We consider the invasion of a rare mutant with contact initiation rate $c_m \approx c$ into an established resident population at equilibrium (denoted by asterisks). The invasion dynamics of the rare mutant are given by:

$$\frac{dS_U^m}{dt} = (b - qN^*)N_m - \frac{c_m c S_U^m}{N^*}\big(\beta(I_U^* + I_A^*) + \tau(S_A^* + I_A^*)\big) - dS_U^m + \gamma I_U^m + \sigma S_A^m \quad (2a)$$

$$\frac{dS_A^m}{dt} = \frac{c_m c}{N^*}\big(\tau S_U^m(S_A^* + I_A^*) - \beta S_A^m(I_U^* + I_A^*)\big) - adS_A^m + \gamma I_A^m - \sigma S_A^m \quad (2b)$$

$$\frac{dI_U^m}{dt} = \frac{c_m c}{N^*}\big(\beta S_U^m(I_U^* + I_A^*) - \tau I_U^m(S_A^* + I_A^*)\big) - (d + \alpha + \gamma)I_U^m + \sigma I_A^m \quad (2c)$$

$$\frac{dI_A^m}{dt} = \frac{c_m c}{N^*}\big(\beta S_A^m(I_U^* + I_A^*) + \tau I_U^m(S_A^* + I_A^*)\big) - (ad + \alpha + \gamma + \sigma)I_A^m \quad (2d)$$

When the dynamics of social information are much faster than demographic ($\tau c^2, \sigma \gg b, d$) and epidemiological processes ($\tau, \sigma \gg \beta, \gamma$), the number of individuals in the aware state A for a given value of c, A_c, rapidly reaches equilibrium. Letting $A_c N = S_A + I_A$, we see that:

$$\frac{dA_c}{dt} = \frac{1}{N}\left(\frac{dS_A}{dt} + \frac{dI_A}{dt}\right)$$

$$\approx (\tau c^2(1 - A_c) - \sigma)A_c = 0 \qquad (3)$$

Thus, either no individuals are in information state A (aware), or $A_c^* = 1 - \frac{\sigma}{\tau c^2}$ when $\sigma < \tau c^2$. When information dynamics are fast, we can therefore make the following approximation to the full model:

$$\frac{dS}{dt} = (b - qN)N - \frac{\beta c^2 SI}{N} - d(1 - (1 - a)A_c^*)S + \gamma I \qquad (4a)$$

$$\frac{dI}{dt} = \frac{\beta c^2 SI}{N} - (d(1 - (1 - a)A_c^*) + \alpha + \gamma)I \qquad (4b)$$

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

For the invasion dynamics of a rare mutant, we let $A_c^m N_m = S_A^m + I_A^m$ so that:

$$\frac{dA_c^m}{dt} = \frac{1}{N_m}\left(\frac{dS_A^m}{dt} + \frac{dI_A^m}{dt}\right)$$

$$\approx (\, c_m c\tau(1 - A_c^m)A_c^* - \sigma A_c^m)N_m = 0 \qquad (5)$$

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

$$A_c^m = \max\left(\frac{c_m(\tau c^2 - \sigma)}{c^2 c_m \tau + \sigma(c - c_m)}, 0\right) \qquad (6)$$

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

$$\frac{dS_m}{dt} = (b - qN^*)N_m - \frac{\beta c_m c S_m I^*}{N^*} - d(1 - (1 - a)A_c^m)S_m + \gamma I_m \tag{7a}$$

$$\frac{dI_m}{dt} = \frac{\beta c_m c S_m I^*}{N^*} - (d(1 - (1 - a)A_c^m) + \alpha + \gamma)I_m \tag{7b}$$

We use evolutionary invasion analysis (Adaptive Dynamics) to determine the trait dynamics (Geritz et al. 1998). We calculate the invasion fitness, $w(c_m)$, of a rare mutant, c_m, when information dynamics are either slow (equation 2) or fast (equation 7) using the next generation method (Hurford et al. 2010). The fitness gradient is then given by $w'(c) = \left.\frac{dw}{dc_m}\right|_{c_m=c}$, and a *singular strategy*, c^*, occurs when $w'(c^*) = 0$. A singular strategy is *evolutionarily stable* if $E(c^*) = \left.\frac{d^2w}{dc_m^2}\right|_{c_m=c^*} < 0$ and is *convergence stable* if the fitness gradient is positive for $c < c^*$ and negative for $c > c^*$, with $c \approx c^*$. If c^* is both evolutionarily stable and convergence stable then it is an attracting fitness maximum, known as a *continuously stable strategy* (CSS). If c^* is neither evolutionarily stable nor convergence stable then it is a *repeller*. If the fitness gradient is zero for all $c < c^*$ because information and disease are not viable, but the fitness gradient is either negative or positive for $c > c^*$ with $c \approx c^*$, then we refer to this point as a *pseudo-CSS* or *pseudo-repeller*, respectively. These four classifications are the only ones that are possible in our model. We solve the model numerically as the expressions for the fitness gradient and evolutionary stability are intractable to algebraic manipulation. 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).

RESULTS

THRESHOLDS FOR SUSTAINED SOCIAL INFORMATION AND DISEASE TRANSMISSION

In the absence of disease or social information, the population has a non-trivial equilibrium at $N^* = (b-d)/q$, which exists and is asymptotically stable provided $b > d$ (hereafter I assume this condition is always satisfied). In the full model, the basic reproductive ratios – the average number of transmission events per informed/infected individual in an otherwise uninformed/susceptible population – for social information (R_0^I) and disease (R_0^D) are equal to the largest eigenvalues of their respective next-generation matrices (see *Supplementary Material*), with:

$$R_0^I = \frac{\tau c^2 \left(\Gamma_\gamma^I I_U + S_U(\Gamma_\alpha^I + \gamma)\right) N + \beta c^2 I_U (I_U + S_U)}{N\left((ad+\sigma)(\Gamma_\alpha^I + \gamma)N + \beta c^2 I_U \Gamma_\alpha^I\right)} \tag{8}$$

$$R_0^D = \frac{\beta c^2 \left((\Gamma_D + \sigma) S_A + S_U(\Gamma_\alpha^I + \gamma)\right) N + \tau c^2 S_A (S_U + S_A))}{N(\Gamma_D(\Gamma_\alpha^I + \gamma)N + \tau c^2 S_A \Gamma_\alpha^I)} \tag{9}$$

where $\Gamma_\alpha^I = ad + \alpha + \sigma$, $\Gamma_\gamma^I = ad + \gamma + \sigma$ and $\Gamma_D = d + \alpha + \gamma$. In the special cases where there is either no disease or no social information, the basic reproductive ratios reduce to:

$$\hat{R}_0^I = R_0^I(I_U = 0) = \frac{\tau c^2}{ad + \sigma} \tag{10}$$

$$\hat{R}_0^D = R_0^D(S_A = 0) = \frac{\beta c^2}{d + \alpha + \gamma} \tag{11}$$

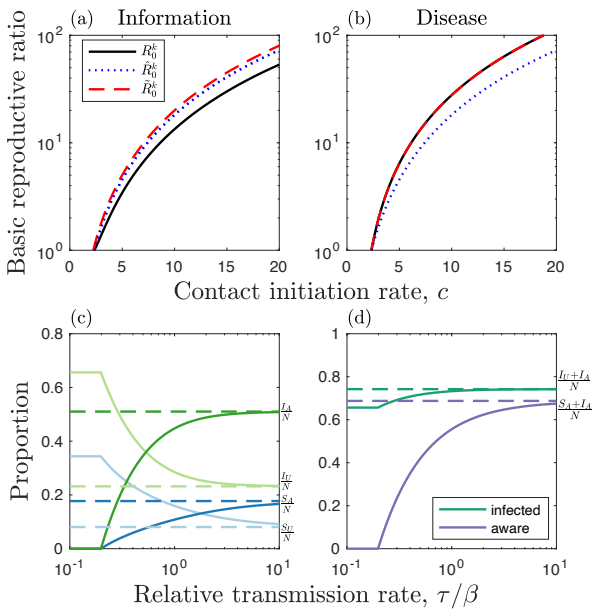
In the approximation for fast social information dynamics, the basic reproductive ratios are:

$$\tilde{R}_0^I = \frac{\tau c^2}{\sigma} \tag{12}$$

$$\tilde{R}_0^D = \frac{\beta c^2}{d(1-(1-a)A_c^*) + \alpha + \gamma} \tag{13}$$

with the latter reducing to $\tilde{R}_0^D = R_0^D(S_A = 0)$ when $A_c^* = 0$ ($\tilde{R}_0^I < 1$).

When $R_0^I, \tilde{R}_0^I > 1$ (equivalently, $R_0^D, \tilde{R}_0^D > 1$), social information (equivalently, disease) is able to spread in the population and reaches a stable equilibrium. The basic reproductive ratios for the full model and the approximation are very similar provided information dynamics are sufficiently fast ($\tau \gg \beta$; Fig. 1a-b). When information dynamics occur on slower timescales, then the approximation diverges from the full model (Fig. 1a-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. 1c-d).



225

226 **Figure 1. Ecological dynamics.** (a)-(b) Basic reproductive ratios for (a) social information ($k = I$) and (b)
227 disease ($k = D$) as a function of the contact initiation rate, c. Curves correspond to the basic reproductive
228 ratio in the full model, R_0^k (black, solid), the full model without disease or social information present, $\hat{R}_0^k$ (blue,
229 dotted), and the approximation for fast social information dynamics, $\tilde{R}_0^k$ (red, dashed). (c)-(d) Comparison of
230 the non-trivial equilibria in the full model (solid) and the approximation for fast social information (dashed). All
231 parameters except for τ and σ are fixed, with $\sigma/\tau = 5$ held constant. The full model approaches the
232 approximation asymptotically when social information dynamics are fast relative to other processes (i.e. in the
233 limit $\tau/\beta \to \infty$). (c) Proportion of individuals in each class. (d) Proportion of individuals that are infected
234 (green) or aware (purple). Fixed parameters (where applicable): $a = 0.2$, $b = 1$, $c = 4$, $d = 0.5$, $q = 10^{-3}$,
235 $\alpha = 0.4$, $\beta = 0.2$, $\gamma = 0.2$, $\sigma = 1$, $\tau = 0.2$.

236 **EVOLUTION OF HOST SOCIALITY**

237 We now consider the evolution of host sociality in terms of the contact initiation rate, c. We
238 first investigate how the fitness gradient varies with information and disease prevalence for
239 a fixed value of c. We then focus our analysis on the extent to which social information
240 reduces mortality (a), the cost of being infected (α), the information expiry rate relative to

the information transmission rate (σ/τ), and the information transmission rate relative to the disease transmission rate (τ/β). Derivations for host fitness, the fitness gradient, and evolutionary stability can be found in the *Supplementary Material.*

Fitness gradient

Fig. 2 shows how the fitness gradient of the host, $w'(c)$, varies with information and disease prevalence when the benefits of sociality are relatively high (Fig. 2a) or low (Fig. 2b) compared to the costs. Information dynamics are assumed to be fast, but the overall patterns hold for slow information dynamics. Note that here c is fixed, with σ and β modulating information and disease prevalence, but when c evolves (see *Evolutionary dynamics*) it will feedback to affect prevalence and hence selection for sociality. Nevertheless, considering how the fitness gradient varies when c is fixed allows us to determine how changes in information and disease prevalence affect the fitness gradient. For example, the fitness gradient is always positive when 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, when disease prevalence is low increased sociality is likely to be beneficial, but only if information is neither too rare nor very common. This is because greater sociality does not significantly increase the rate at which individuals obtain survival-related information if this information is readily available or unlikely to become accessible. Somewhat counter-intuitively, increased sociality may also be beneficial when disease prevalence is high (as shown in Bonds *et al.*, 2005). This is because most individuals are likely to be infected anyway, but increased sociality will improve access to social information with little increase to the risk of infection. Conversely, the fitness gradient is always negative when information is either rare

264 or very common and disease is at intermediate prevalence. This pattern mirrors the one
265 described above, but with the roles of information and disease switched due to their
266 opposing effects on the fitness gradient. Intuitively the fitness gradient is negative when
267 information prevalence is low and disease is common in the population, as greater sociality
268 increases the risk of infection but does not substantially increase the likelihood of obtaining
269 survival-related information. But perhaps counter-intuitively the fitness gradient is also
270 negative when information is widespread and disease is at moderate levels. This is because
271 most individuals are already likely to be informed, and so increased sociality will only
272 marginally improve access to social information but may substantially increase the risk of
273 infection.

274 When both information and disease are at intermediate prevalence, the sign of the fitness
275 gradient depends on the relative costs and benefits of being infected or informed. When
276 awareness of survival-related information is relatively more valuable than the cost of being
277 infected, the fitness gradient is positive whenever information prevalence is at an
278 intermediate level regardless of disease prevalence (Fig. 2a). Disease may therefore not
279 always be sufficient to curtail the evolution of sociality. In contrast, when awareness of
280 survival-related information is relatively less valuable, and the cost of being infected is
281 relatively high, the fitness gradient is negative whenever disease prevalence is at an
282 intermediate level regardless of information prevalence (Fig. 2b). This set of results suggests
283 that the host population may experience different evolutionary outcomes depending on the
284 initial conditions: if disease prevalence is sufficiently low the population may evolve towards
285 a stable level of sociality, but if disease is initially very common then the population may
286 experience runaway selection for greater sociality.

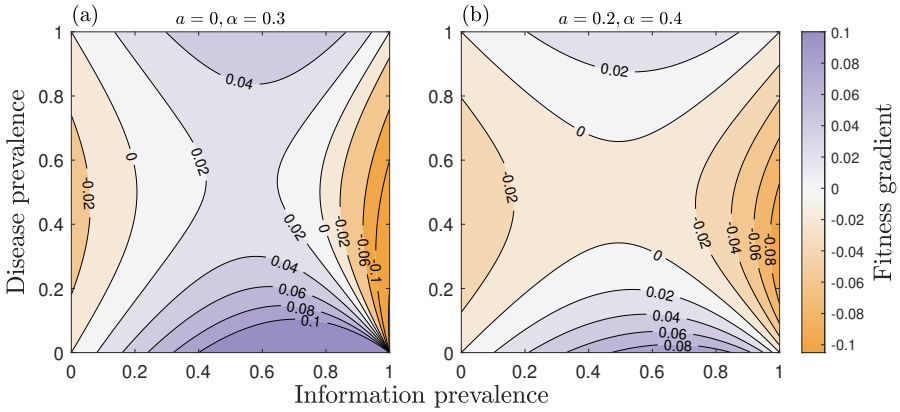


Fig. 2. Effects of information and disease prevalence on the fitness gradient of hosts. Contour plots of the fitness gradient, $w'(c)$, in the fast information approximation as a function of information and disease prevalence with: (a) higher $(a = 0, \alpha = 0.3)$ and (b) lower $(a = 0.2, \alpha = 0.4)$ 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 information and disease prevalence vary accordingly. In both cases, the fitness gradient is positive (bluer shading) when information prevalence is intermediate and there is either a high or low level of disease, and is negative (redder shading) when information prevalence is either at a high or low level and disease is at intermediate prevalence. Fixed parameters as in Fig. 1.

EVOLUTIONARY DYNAMICS

When information is at moderate to high prevalence (i.e. the information expiry rate, σ, is not too large relative to the information transmission rate, τ), there may exist a *continuously stable strategy* or CSS, which is locally attracting (dark blue curves in Fig. 3). Nearby trait values evolve towards the CSS by small mutations and remain there indefinitely (Fig. 4a-b). When the costs (α) that arise from being social are relatively low compared to the benefits of being informed (a), the CSS increases with σ/τ until there is no evolutionarily stable contact initiation rate, leading to runaway selection for sociality (Fig. 3a, d). When the costs of sociality are higher, the CSS peaks at intermediate values of σ/τ (e.g. Fig. 3b). This is because as σ/τ increases, information expires more quickly and so the overall prevalence of 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 (information awareness is at higher levels in the population) and higher costs of disease (α) or lower benefits of information (a), the CSS, c^*, is attracting for all values of $c > c^*$. This means that high initial levels of sociality will always evolve to a lower level. But for sufficiently large values of σ/τ (information awareness is at lower levels in the population) or low costs of disease (α) and high benefits of information (a), a *repeller* (dark red curves in Fig. 3) may exist above the CSS. Sociality evolves towards the CSS when the initial contact initiation rate is below the repeller, which reduces infection and information prevalence to a point where the costs and benefits of sociality are balanced (Fig. 4). There is runaway selection for greater sociality when the contact initiation rate is above the repeller, which increases infection and information prevalence (Fig. 4c). 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, as contact initiation rates 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 the evolution of sociality.

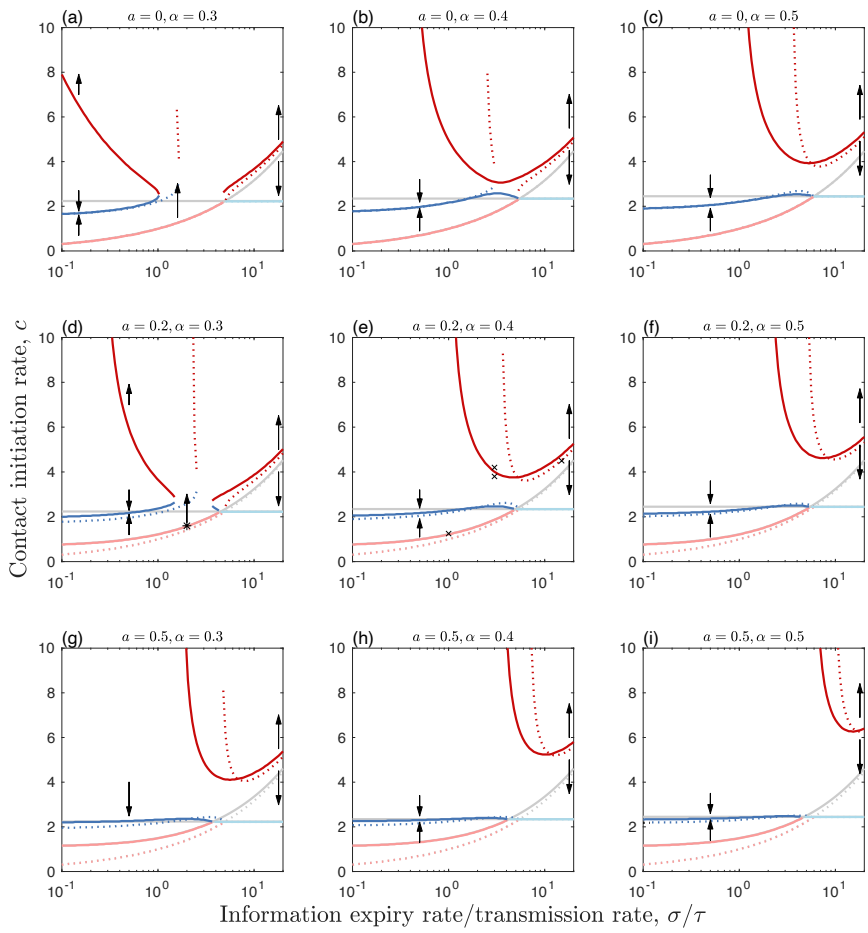


Fig. 3. The evolution of sociality arising from social information and disease transmission. Evolution of the contact initiation rate, c, as a function of information expiry/transmission, σ/τ, and for different values of the benefits of social information, a, and costs of disease, α. Results are shown for relatively slow ($\tau = \beta$; solid) and fast ($\tau = 10\beta$; dotted) information dynamics. Coloured curves correspond to singular strategies: repellers (red), continuously stable strategies (CSSs; dark blue), pseudo-repellers (pink), and pseudo-CSSs (light blue). For trait values above pseudo-repellers and pseudo-CSSs the dynamics are identical to repellers and CSSs, respectively, but the selection gradient is equal to zero below the curves due to disease and information being unviable. Arrows indicate the direction of selection when information dynamics are relatively slow. Grey curves do not indicate singular strategies but are continuations of the disease and information viability thresholds when $N = S_U$. Crosses in (e) correspond to simulations in Fig. 4 and the asterisk in (d) corresponds to simulations in Fig. 5. Fixed parameters as in Fig. 1.

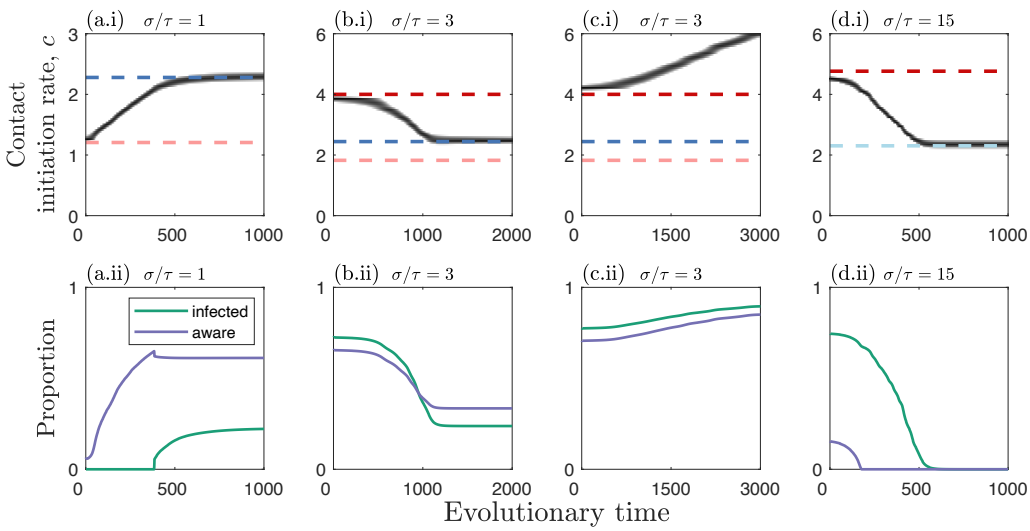


Fig. 4. Simulation trajectories when information and disease dynamics occur on similar timescales. Trajectories of simulations corresponding to crosses in Fig. 3e. Top row: evolutionary trajectories (solid black) and singular strategies (dashed): repellers (red), continuously stable strategies (CSSs; dark blue), pseudo-repellers (pink), and pseudo-CSSs (light blue). Bottom row: proportion of individuals that are infected (green) or aware (purple) in each simulation. Fixed parameters as in Fig. 1, with $\tau = \beta$.

For sufficiently large values of σ/τ, the threshold for sustained information transmission passes the threshold for sustained disease transmission ($R_0^D > R_0^I$ or $\tilde{R}_0^D > \tilde{R}_0^I$). In this region of the parameter space, there exists a *pseudo-CSS* (light blue curves in Fig. 3) at the point where the disease becomes unviable ($R_0^D = 1$ or $\tilde{R}_0^D = 1$). As with the pseudo-repeller, below this threshold neither information nor disease transmission can be sustained and so the fitness gradient is zero (i.e. no selection on the contact initiation rate). The pseudo-CSS is locally attracting for contact initiation rates that are not too large: the costs of disease (common) are greater than the benefits of social information (rare) and so sociality is selected against until the disease is driven extinct at the pseudo-CSS (Fig. 4d). For the reasons discussed above, a repeller may also exist in this region of parameter space, where for sufficiently high contact initiation rates, there is runaway selection for greater sociality.

Increasing the costs of disease (α) or decreasing the benefits of information (a) limits where the repeller exists to larger values of σ/τ and greater contact initiation rates. This has the effect of increasing the region of attraction for the CSS and pseudo-CSS. Conversely, decreasing the costs of disease or increasing the benefits of information may cause the repeller to collide with the CSS, leaving only the pseudo-repeller at intermediate values of σ/τ (Fig. 3a, 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 information and disease dynamics occur on similar timescales ($\tau \approx \beta$). When information dynamics occur on a much faster timescale ($\tau \gg \beta$), there are some notable differences (dotted curves in Fig. 3). First, the pseudo-CSS is shifted to a slightly lower contact initiation rate to reflect the lower threshold for information transmission. Second, the repeller curve for fast information dynamics is shifted to the right and down in Fig. 3, which may increase or decrease the domain of attraction for the CSS and pseudo-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 information (a). Finally, at low cost of disease, a CSS may exist where only a pseudo-repeller did before (e.g. Fig. 3d). This means that when information dynamics are relatively slow, there may be runaway selection for greater sociality, but when the information dynamics are relatively fast, sociality may evolve to a stable level (Fig. 5). The timescale of social information dynamics can therefore play an important role in determining the evolution of sociality.

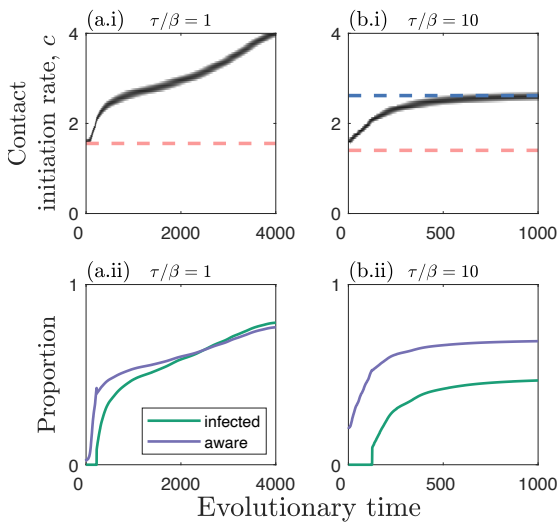


Fig. 5. Simulation trajectories when information dynamics occurs on a much faster timescales than disease dynamics. Simulations corresponding to the asterisk in Fig. 3d. Top row: evolutionary trajectories (solid black) and singular strategies (dashed): continuously stable strategies (CSSs; dark blue) and pseudo-repellers (pink). Bottom row: proportion of individuals that are infected (green) or aware (purple) in each simulation. Fixed parameters as in Fig. 1, with $\alpha = 0.3$, $\sigma = 2\tau$.

EVOLUTION OF PARASITE VIRULENCE

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 rate with diminishing returns, so that $\frac{d\beta}{d\alpha} > 0$ and $\frac{d^2\beta}{d\alpha^2} < 0$. For simplicity, first consider the case where information dynamics are fast. The invasion fitness of a rare mutant parasite with virulence α_m is proportional to its basic reproductive ratio:

$$\tilde{R}_0^D(\alpha_m) = \frac{\beta(\alpha_m)c^2}{d(1-(1-a)A_c^*) + \alpha_m + \gamma} \tag{14}$$

and so the singular strategies of the parasite are equivalent to maximisation of $\tilde{R}_0^D$. This guarantees that the parasite evolves towards the continuously stable strategy α^*, where $\left.\frac{d\tilde{R}_0^D}{d\alpha}\right|_{\alpha=\alpha^*} = 0$. The optimal level of virulence therefore occurs when:

$$\left.\frac{d\beta}{d\alpha}\right|_{\alpha=\alpha^*} = \frac{\beta(\alpha^*)c^2\tau}{c^2\tau(ad+\alpha^*+\gamma) + d\sigma(1-a)} = f(\alpha^*, c)\beta(\alpha^*) \tag{15}$$

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

$$\begin{aligned}\left.\frac{\partial y}{\partial \alpha}\right|_{\alpha=\alpha^*} &= \beta(\alpha^*)\left.\frac{df}{d\alpha}\right|_{\alpha=\alpha^*} + f(\alpha^*, c)\left.\frac{d\beta}{d\alpha}\right|_{\alpha=\alpha^*} - \left.\frac{d^2\beta}{d\alpha^2}\right|_{\alpha=\alpha^*} \\ &= -\left.\frac{d^2\beta}{d\alpha^2}\right|_{\alpha=\alpha^*} > 0 \end{aligned} \tag{16}$$

where $\left.\frac{d\beta}{d\alpha}\right|_{\alpha=\alpha^*} = f(\alpha^*, c)\beta(\alpha^*)$ from equation 15 and $\left.\frac{df}{d\alpha}\right|_{\alpha=\alpha^*} = -f^2(\alpha^*, c)$. This means that if sociality increases, the optimal level of virulence must decrease to balance equation 15 (Fig. 6). 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:

$$\left.\frac{d\beta}{d\alpha}\right|_{\alpha=\alpha^*} = \frac{\beta(\alpha^*)}{d+\alpha^*+\gamma} \tag{17}$$

which does not depend on the contact initiation rate, c. 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*.

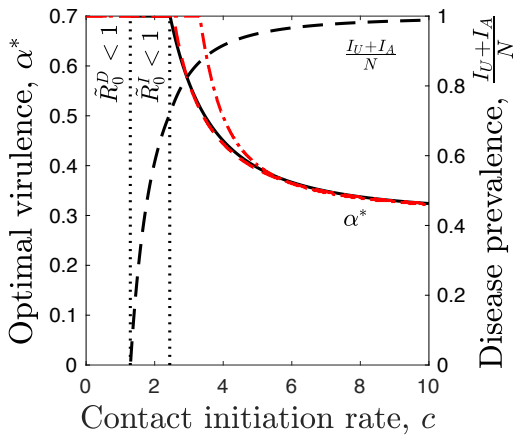


Fig. 6. The evolution of virulence. Optimal virulence, α^*, decreases with the contact initiation rate (solid black: fast information approximation; red: slow information, $\tau = 0.2$ (dashed) and $\tau = 0.02$ (dash-dot)). The black dashed curve indicates disease prevalence in the approximation for fast information dynamics. Vertical dotted lines indicate where disease ($\tilde{R}_0^D < 1$) or ($\tilde{R}_0^I < 1$) information transmission is not viable in the fast information approximation. Other parameters as in Fig. 1, with $\beta(\alpha) = \sqrt{\alpha}$.

CO-EVOLUTION OF HOST SOCIALITY AND PARASITE VIRULENCE

We combine our results from the previous two sections to analyse the effects of social information on the coevolution of sociality and virulence. For tractability, we make three simplifying assumptions: (1) information dynamics are fast (i.e. we use the approximation to the full model in equation 4); (2) the parasite evolves much faster than the host; and (3) the relationship between transmission and virulence is $\beta(\alpha) = \sqrt{\alpha}$. 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:

$$\alpha^* = \begin{cases} \dfrac{d\sigma(1-a)}{c^2\tau} + (ad+\gamma) & \text{for } \tilde{R}_0^I > 1 \\ d+\gamma & \text{for } \tilde{R}_0^I < 1 \end{cases} \tag{18}$$

from equations 15 and 17.

The range of qualitative outcomes for coevolution are the same as for one-sided adaptation in the host (Fig. 7). The host and parasite may tend to a joint stable or pseudo-stable level of sociality and virulence, respectively, or they may experience runaway selection for higher sociality and lower virulence. However, note that in the case of runaway selection virulence asymptotes to a lower bound of $\alpha^* = ad + \gamma$ (from equation 18). Under coevolution, the evolutionary stable level of virulence increases with the ratio of the information expiry rate to transmission rate (σ/τ), until information dynamics are no longer viable ($\tilde{R}_0^I < 1$).

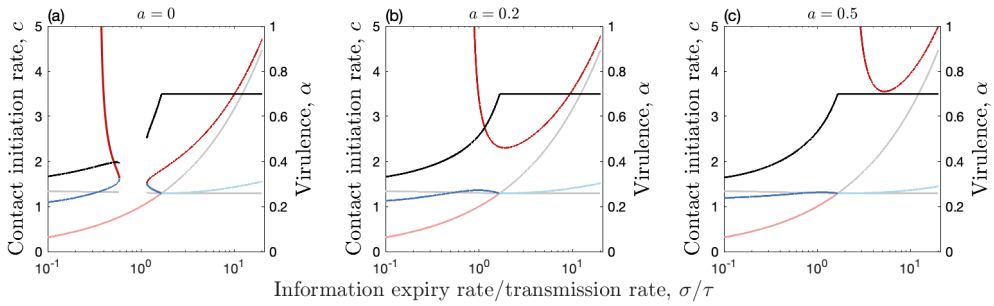


Fig. 7. Coevolution arising from social information and disease transmission dynamics. Coevolution of host sociality (contact initiation rate, c) and parasite virulence (α) as a function of information expiry/transmission, σ/τ, and for different values of the benefits of social information, a (fast information approximation). Black curves show the evolutionary stable level of virulence that corresponds to the stable (dark blue) or pseudo-stable (light blue) level of host sociality. All other curves as described in Fig. 3: repellers (red); pseudo-repeller (pink); information viability threshold (grey, increasing); disease viability threshold (grey, horizontal). Other parameters as in Fig. 1, with $\beta(\alpha) = \sqrt{\alpha}$.

DISCUSSION

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

Our model shows how contrasting transmission processes can trade-off to mediate the evolution of sociality and virulence. Crucially, we have shown that selection for sociality (and hence virulence) depends non-monotonically on the prevalence of both information and disease (Fig. 2). Specifically, selection usually favours greater sociality (and lower virulence) when information is neither too common nor too rare and disease risk is either low or high. Conversely, selection against sociality (and for greater virulence) typically occurs when information is either rare or very common and disease is at moderate prevalence. The fact that these two processes mirror each other is not surprising: the transmission processes are fundamentally the same in the model, but information is

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

475 In general, conditions on information prevalence that select for greater sociality (and lower
476 virulence) are the same conditions on disease prevalence that select against sociality (and
477 for greater virulence). Naively, one might assume that selection for sociality should increase
478 with information prevalence and decrease with disease prevalence. But when information
479 prevalence is high, the marginal benefit of increasing sociality is low, and similarly when
480 disease prevalence is high, the marginal cost of being more social is low. Slightly higher or
481 lower sociality therefore makes little difference to the rate of exposure to information or
482 disease when prevalence is high. The effects of disease prevalence on the evolution of
483 sociality have previously been observed by Bonds et al. (2005), but the effects of social
484 information prevalence on the evolution of sociality have not previously been documented.
485 Bonds et al. (2005) likened the relationship between disease prevalence and selection for
486 sociality to optimal host investment in an immune response, which is predicted to peak
487 when the risk of infection is neither too low, nor too high. In fact, this pattern is quite
488 general and occurs for a variety of behavioural and physiological defences against

489 parasitism, including resistance (Haldane 1949), recovery (van Baalen 1998), mate choice
490 (Ashby and Boots 2015) and sexual reproduction (Ashby and King 2015).

491 The patterns described above for high/low prevalence are quite robust, but the picture is
492 more complicated when information and disease are both at intermediate prevalence.
493 Selection may increase or decrease sociality and virulence depending on the relative costs
494 and benefits of being infected or informed, in addition to the likelihood of exposure. As a
495 consequence, it is possible for evolutionary trajectories to exist where the sign of the fitness
496 gradient is invariant to disease or information prevalence, leading to runaway selection for
497 higher sociality/lower virulence or lower sociality/higher virulence (Fig. 2, 7). Note that,
498 while our model allowed for runaway selection for sociality, in reality one would expect
499 other factors to eventually constrain evolution (e.g. physiological constraints, additional
500 costs). We omitted additional constraints from our model so that we could determine when
501 disease alone is sufficient to constrain the evolution of sociality. The results for "runaway
502 selection" should therefore be interpreted in this light.

503 It is also possible for the initial conditions to determine the long-term evolutionary outcome
504 (stable level of sociality and virulence, or runaway selection for increased sociality and
505 decreased virulence) due to the existence of evolutionary repellers (Fig. 3, 7). The possibility
506 of more than one (co)evolutionary outcome suggests that different populations might
507 evolve very different social strategies for coping with infectious disease, and that variable
508 ecological conditions can lead to sudden shifts in sociality and virulence. How evolutionary
509 outcomes play out in a given population is likely to also be affected by the structure of the
510 social contacts. Our model assumed a well-mixed population, which can represent dynamics
511 in one small patch. However, at a broader scale, it is now clear that almost all animal

512 populations exhibit some structure (Cantor et al. 2020b), and that population structure can
513 shape both transmission dynamics and evolution of diseases (Eames and Keeling 2006; Lion
514 and Boots 2010; Ashby and Gupta 2013). Structure can arise due to individual preferences
515 for certain relationships (Alberts 2019) or because the environment induces limitations on
516 who can come into contact with whom (He et al. 2019; Silk et al. 2019; Albery et al. 2020).
517 Some mammal (Grueter et al. 2020) and birds (Papageorgiou et al. 2019) also exhibit
518 multiple levels of social connections – named multilevel societies – with social contacts
519 being expressed within and across social units. Further, it is now becoming well-established
520 that individuals can change their social behaviours once they become infected (reviewed in
521 Romano et al. 2020), which can introduce a more rapid feedback between disease state and
522 social behaviour. Thus, future research should aim to determine how specific social
523 strategies might evolve under different environmental conditions (e.g. (Udiani and
524 Fefferman 2020).

525 An important consideration when modelling the benefits of sociality dynamically in terms of
526 survival-related information, as opposed to fixed benefits (Bonds et al. 2005; Prado et al.
527 2009), is the timescale of social information dynamics relative to other processes. We have
528 shown that for the most part, evolutionary outcomes are qualitatively and quantitatively
529 similar whether information dynamics are relatively fast or on a similar timescale to
530 demographic and epidemiological processes. However, this is not always the case, as shown
531 in Fig. 5, where increasing the speed of information dynamics relative to other processes
532 has a stabilising effect on the evolution of sociality. In general, faster information dynamics
533 tend to have a stabilising effect, reducing parameter ranges for runaway selection. Hence,
534 while one can often approximate information dynamics by assuming the population rapidly

535 reaches a stable distribution, this assumption does not always hold and can fundamentally
536 change evolutionary outcomes.

537 One of the key predictions from this study and previous work (Bonds et al. 2005) is that
538 sociality should vary inversely with virulence, which may appear at odds with classical
539 predictions for virulence evolution (Ewald 1994). The contradiction is resolved by noticing
540 that: (1) our model considers contact rates in a randomly mixing population rather than
541 contact number in a structured population; and (2) higher contact rates do not directly
542 select for lower virulence, but do so indirectly through the spread of survival-related
543 information, which reduces host mortality and increases the infectious period. Hence,
544 greater sociality as modelled here is not equivalent to population mixing in a structured
545 model, and the effects on virulence evolution are mediated via host mortality rather than
546 contact rates.

547 In previous studies, the fitness benefits of sociality were fixed rather than dynamic (Bonds et
548 al. 2005; Prado et al. 2009). While some studies have explored how social information and
549 infectious disease affect the contact structure of a population, this has been in the form of
550 host plasticity during an epidemic, rather than an evolutionary response (Funk et al. 2010),
551 or where the outcomes are compared across populations with different fixed social
552 strategies (Udiani and Fefferman 2020). Here, the two processes operate as independent
553 forces of selection. Our findings are closely linked to those of Bonds et al. (2005), where
554 sociality and virulence are shown to coevolve to stable levels. In contrast, Prado et al. (2009)
555 have shown that coevolutionary cycling (fluctuating selection) may occur between sociality
556 and virulence (Prado et al. 2009). It is not clear why Prado et al. (2009) found cycles, yet
557 Bonds et al. (2005) and the present study have not, but part of the reason may be due to

558 model structure. Prado et al. (2009) modelled disease dynamics using a stochastic
559 individual-based network, whereas Bonds et al. (2005) and the present study employed
560 deterministic models with randomly-mixing populations. A priority for future research in
561 this area is therefore to establish precisely when coevolutionary cycling of sociality and
562 virulence occurs, and whether this is driven by population structure and/or stochasticity.

563 The present study is relevant to empirical research on the sociality of animal populations
564 (Dall et al. 2004; Sih et al. 2004; Dingemanse and Réale 2005; Evans et al. 2020). In
565 particular, a number of studies have shown that variation in sociality influences the spread
566 of social information (Aplin et al. 2012; Allen et al. 2013; Farine et al. 2015a; Snijders et al.
567 2020). For example, the social network attributes of wild great tits (*Parus major*) can be
568 used to predict the discovery rate of novel food patches (Aplin et al. 2012). As in previous
569 theoretical studies (Bonds et al. 2005; Prado et al. 2009), we did not observe coexistence of
570 different social strategies. However, several empirical studies have highlighted the potential
571 importance of maintaining mixed strategies in social groups or local populations. For
572 example, Aplin et al. (2014) suggested that variation in sociality could increase group-level
573 efficiency in exploiting patches. Similarly, in ants, mixed colonies (in terms of aggressive
574 type) have more efficient task allocation and are more successful at capturing prey
575 (Modlmeier et al. 2012). A potential reason for the lack of mixed social strategies in current
576 theoretical studies is that they do not include assortative mixing (Farine 2014). Assortativity
577 has been shown to facilitate the coexistence of multiple parasites (Eames and Keeling 2006),
578 so it is possible that mixed social strategies could also evolve in the presence of non-random
579 mixing – a process that could be either actively generated through social preferences or
580 passively through exogenous drivers of social structure. It is also possible that mixed social

581 strategies could evolve in fluctuating environments, especially if individuals value
582 information differently (e.g. due to personality traits, Aplin et al. 2014), or if individuals vary
583 in their propensity to form social relationships (e.g. due to developmental conditions,
584 Boogert et al. 2014). A challenge for future theoretical research is to establish whether
585 selection arising from disease and information-use dynamics can drive the evolution of
586 mixed social strategies. Our results suggest that a number of potential mechanisms (e.g.
587 variation in the perceived value of information) could facilitate this process.

588 In summary, our study suggests that not only does the sociality of the population affect
589 information and disease dynamics, but also that these processes are likely to influence the
590 evolution of sociality and virulence. Crucially, we predict that: (1) selection for sociality and
591 virulence will vary non-monotonically with information and disease prevalence; and (2) if
592 social information increases host survival, then increased sociality may correlate with
593 decreased virulence. The context-dependent relationships that we have shown to exist
594 between the ecology of information/disease dynamics and the evolution of
595 sociality/virulence highlights the need to understand host and parasite evolution in the
596 context of multiple ecological processes.

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DATA AVAILABILITY

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

COMPETING INTERESTS

The authors have no competing interests to declare.

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