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HIGHER DISEASE PREVALENCE CAN INDUCE GREATER SOCIALITY: A GAME THEORETIC COEVOLUTIONARY MODEL

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Abstract.—There is growing evidence that communicable diseases constitute a strong selective force on the evolution of social systems. It has been suggested that infectious diseases may determine upper limits of host sociality by, for example, inducing territoriality or early juvenile dispersal. Here we use game theory to model the evolution of host sociality in the context of communicable diseases. Our model is then augmented with the evolution of virulence to determine coevolutionarily stable strategies of host sociality and pathogen virulence. In contrast to a controversial hypothesis by Ewald (1994), our analysis indicates that pathogens may become more virulent when contact rates are low, and their prevalence can ultimately induce greater sociality.

Key words.—Coevolution, coevolutionarily stable strategy, evolution of virulence, game theory, sociality.

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It is well known that infectious diseases constitute a substantial source of morbidity and mortality in natural populations and are especially common among social organisms. Indeed, many communicable diseases are thought to require a minimum level of host interaction (e.g., mating, gregariousness, or population density) to avoid extinction. There is also growing evidence that animals have evolved behavioral responses to mitigate the risks of infection, such as lowering their level of contact (Hart 1990; Loehle 1995; Møller et al. 2001; Moore 2002; Altizer et al. 2003). For example, Freeland (1976, p. 12) presented his hypothesis that ''individual primates increase their fitness by patterning their behavior and social interaction so that they minimize the probability of acquiring new pathogens and minimize the pathogenicity of diseases they already harbor.'' It is therefore reasonable to suspect that diseases represent a selective force—specifically, a cost—on the evolution of social systems (Alexander 1974; Pulliam and Caraco 1984; Brown and Brown 1986; Lee 1994; Møller et al. 2001). Indeed, it has been suggested that infectious diseases may actually determine upper limits of host group size and contact levels (Freeland 1976, 1979; Moore 2002). But while theories on the evolution of sociality abound, formalizations of the role of infectious diseases are lacking. Here we present a theory of the evolution of host sociality in response to infectious diseases. We then augment this theory with the evolution of virulence and present coevolutionarily stable strategies of host sociality.

We find that for systems in which the benefits of social behavior are expressed in the form of lower mortality rates, such as decreased predation, pathogens become less virulent at high contact rates. This is in direct contrast to leading theories on pathogen evolution that have posited that greater transmission opportunities should either result in higher pathogen virulence (Ewald 1994; Massad 1996) or have no long-term effect on pathogen evolution (Bull 1994; Lipsitch and Nowak 1995; Frank 1996; Lipsitch 1997; Day 2002). Moreover, we find that increases in disease prevalence can ultimately induce greater host sociality.

THE EVOLUTION OF SOCIAL CONTACT IN THE CONTEXT OF INFECTIOUS DISEASES

The theoretical literature on the evolution of host contact in response to infectious diseases has mostly focused on sexually transmitted diseases, where such contacts are discrete and their benefits (reproduction) and costs (risk of sterilization or death through contracted disease) are explicit (Freeland 1976; Møller et al. 1993; Sheldon 1993; Antonovics and Thrall 1994; Thrall and Antonovics 1997; Boots and Knell 2002; Kokko et al. 2002). However, there has been significant theoretical exploration of the benefits of sociality generally, which often focuses on nonreproductive life-history traits such as greater survival from decreased predation (Pulliam et al. 1977; Caraco et al. 1980; Szekely et al. 1991). For

example, many social species of birds have been documented to benefit from early warning of predators (Pulliam 1973; Hoogland and Sherman 1976; Lazarus 1979; Caraco et al. 1980; Whitfield 2003; Beauchamp 2004). These same benefits have been hypothesized for many other social animals, from prairie dogs (Hoogland 1979) to squirrel monkeys (Boinski et al. 2003). Other advantages of grouping include enhanced defense, such as through mobbing of predators (Hoogland and Sherman 1976), and selfish herding (Hamilton 1971), where neighboring group members serve as alternative targets for predation. Of course, sociality is not without costs, and there has been much speculation on what factors limit social evolution. The most obvious candidate is intragroup competition for resources (Freeland 1976; Pulliam and Caraco 1984; Lee 1994), but there are also costs associated with predation. For example, larger group sizes may be more conspicuous to predators, leading to higher rates of attack per group member, in addition to possible greater attack efficiency that results from the prey density (Krebs 1971; Andersson and Wicklund 1978; Pulliam and Caraco 1984). Another proposed limiting factor of sociality—and the one that is the focus of this analysis—is the spread of infectious diseases (Freeland 1976; Pulliam and Caraco 1984; Brown and Brown 1986; Lee 1994; Møller et al. 2001; Moore 2002). Here we present a theory on the evolutionary relationship between nonreproductive social behavior and horizontally transmitted infectious diseases.

Model

We consider the evolution of social behavior of a homogeneous host population that reproduces asexually. Specifically, we imagine social behavior such as grouping, which confers both fitness benefits (early warning) and costs (group conspicuousness) in the form of individual survival. We assume that the relationship between survival and contact obeys a tradeoff, so that, in the absence of the disease, the hosts's contact rate or group size will evolve to some finite optimum, K.

The system is described by a traditional susceptible-infected (S-I) model, in which we explicitly define the role of contact on host mortality and disease transmission, with each phenotype i corresponding to a rate of contact, C_i. The population dynamics of phenotype i can be described by the following differential equations:

$$\frac{dS_i}{dt} = (a - hN)(N_i) - \left[d + p(K - C_i)^2 + \frac{\beta_i(C_i)I}{N}\right]S_i \quad (1)$$

and

$$\frac{dI_i}{dt} = \frac{\beta_i(C_i)I}{N}S_i - [d + p(K - C_i)^2 + v]I_i. \quad (2)$$

The state variables, S_i and I_i, represent the number of susceptible and infected individuals of phenotype i. The density-dependent reproductive rate is $a - hN$, with $N = S + I$, $S = \Sigma_i S_i$, and $I = \Sigma_i I_i$. The parameter a represents the maximum per capita birth rate (as N approaches zero), and h represents the decrease in the birth rate that results from density dependence (e.g., through competition of resources). The death rate depends on the infection status of the host in addition to the contact rate. The death rate of susceptibles is the quadratic function $d + p(K - C_i)^2$, which in the absence of the disease can be minimized by a contact rate of $C_i = K$. The cost of deviating away from the disease-free optimum, K, is partially determined by the parameter p (i.e., if $p = 0$, then there are no advantages to sociality, and as p rises away from zero, the cost of avoidance rises). The pathogen is virulent, with infected individuals dying at the same rate as the susceptibles plus the rate of virulence, v. We assume that the hosts mix randomly, so that an individual's contact frequency can be viewed as the product of its own contact effort, E_i, and the average contact effort of the population, E_a, so that $C_i = E_iE_a$. The transmission rate is equal to the probability of infection per contact, ρ, times the contact frequency: $\beta_i(C_i) = \rho E_iE_a$.

Because the benefits (survival) and costs (disease transmission) of contact are determined not only by the contact effort of the individual but also by that of the rest of the population, the optimal contact effort, $\hat{E}$, can be considered the solution to an evolutionary game. The first step to solving this game is to maximize a fitness function (eq. 3) with respect to E_i. The fitness function equals the number of offspring in the lifetime of the host, which can be treated as a Markov process with three events: birth, infection, and death. The fitness function, ω_i, is therefore:

$$\omega_i \propto \frac{1}{d + p(K - E_iE_a)^2 + \rho E_iE_a\frac{I}{N}} + \frac{\rho E_iE_a\frac{I}{N}}{d + p(K - E_iE_a)^2 + \rho E_iE_a\frac{I}{N}}\,\frac{1}{d + p(K - E_iE_a)^2 + v}. \quad (3)$$

The first term, $1/[d + p(K - E_iE_a)^2 + \rho E_iE_aI/N]$, equals the time spent susceptible, $\rho E_iE_a(I/N)/[d + p(K - E_iE_a)^2 + \rho E_iE_aI/N]$ is the likelihood of reaching the infectious class (as opposed to dying), and $1/[d + p(K - E_iE_a)^2 + v]$ represents the time spent infected.

To obtain an expression for the optimal contact rate, we first define $\hat{E}_i = f_1(I/N, E_a)$ as the value of the contact effort, E_i, that maximizes equation (3). Because this game is symmetric (i.e., the rules are identical for all players), at the optimum, the contact efforts are equal to the Nash equilibrium level, $E_i = \hat{E}_i$ for all i. Therefore, to find the optimal contact effort, we set E_a equal to $\hat{E}_i$, generating $\hat{E} = f_2(I/N)$. The optimal contact rate is therefore, $\hat{C} = \hat{E}^2$. Figure 1 shows that, when the average contact effort equals $\hat{E}$, the fitness for any invading phenotype, $E_i \neq \hat{E}$, is less than that conferred from $\hat{E}$.

The Optimal Contact Responds to Disease Prevalence

To understand the evolutionary response of host sociality to changes in disease prevalence, we present the optimal contact rates and disease prevalence over a range of background mortality and transmission probability parameters (Fig. 2). To maintain consistency with the evolution of virulence literature as well as the developments in the next section, we specify the transmission probability, ρ, as a function of pathogen virulence: $\rho(v) = gv/(v + \xi)$, with g representing the

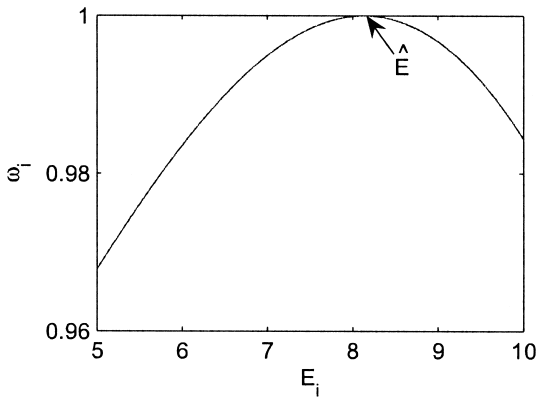

FIG. 1. The fitness of individual i, ω_i, defined as its lifetime reproductive success, is presented for values of invading contact efforts, E_i, when the population adopts the contact effort, $\hat{E}$. Some of the parameter values used (v, I/N, and ρ) are determined dynamically as state variables in the coevolutionary model in the Coevolution of Host Sociality and Infectious Disease section. These results therefore complement those presented in Figures 5 and 6; $v = 0.4$, $I/N = 0.7$, $p = 0.00005$, $K = 81$, $\rho = 0.03$, $d = 0.15$.

maximum probability of transmission as v approaches infinity, and ξ determining the rate at which that limit is arrived as virulence increases.

As expected, we find that as disease prevalence rises away from zero the optimal contact rate falls from its disease-free optimum, K. What is more surprising however, is that after some threshold level of prevalence the optimal contact rate rises for a large range of d and g values (this phenomenon was also observed with an alternative mortality function; see Supplementary Material available online only at http://dx.doi.org/10.1554/05-028.1.s1). This is because when the disease prevalence is high the benefits of disease avoidance, in terms of decreased likelihood of acquiring the disease, are negated by the survival advantages conferred from higher contact rates. This is directly analogous to the finding by van Baalen (1998) that the optimal host investment in an immune response (which, similar to disease avoidance, is assumed to have negative effects on host survival) is a nonmonotonic function of the force of infection. Specifically, optimal immune investment is highest at intermediate probabilities of infection and is lowest at high and low probabilities. Note that in our system we are assuming there remain benefits of contact even after infection. So, the benefits of lowering contact come in the form of a delayed timing of infection, where the cost of lowering contact is suffered throughout the course of the host's life. This is why at high transmission probabilities the optimal contact rates start rising with respect to disease prevalence. As van Baalen (1998) noted, the host makes ''the best of a bad job.'' This is also why, as the pathogen becomes increasingly pathogenic and the benefits of contact after infection fall (because the host dies sooner), the relationship between contact and disease prevalence becomes monotonic (Figure 3).

The important point to draw from Figures 1–3 is that there is no a priori reason to expect host sociality to generally decrease with increases in disease prevalence. Indeed, while the contact never rises above its disease-free optimum, K, the ability of the disease to induce contact rates below K is weakest when the prevalence is both low and high. As a result, when the prevalence increases above some threshold level, so too may the optimal contact rate.

Evolutionarily Stable Contact and Disease Prevalence Respond to Each Other

The analysis in the previous section is important for a partial understanding of the interactions between host evolution in response to communicable diseases but it is nevertheless superficial in that it unrealistically treats disease prevalence as exogenous. In reality, host contact is not only a function of the pathogen prevalence, but the prevalence is also a function of host contact. Solving for the fitness-maximizing contact rate and equilibrium disease prevalence simultaneously results in the evolutionarily stable (ES) contact rate, $C^* = f_3(I^*/N^*)$ (note that we use the hat [$\wedge$] notation to denote the optimal value when prevalence is not in equilibrium with contact and the star [*] notation to denote the ES value when prevalence is in equilibrium with contact). In Figure 4, we present corresponding values of C^* and I^*/N^* over a range of background mortality and transmission probabilities.

In Figure 4, we see a discernible trend in the general relationship between equilibrium disease prevalence and ES

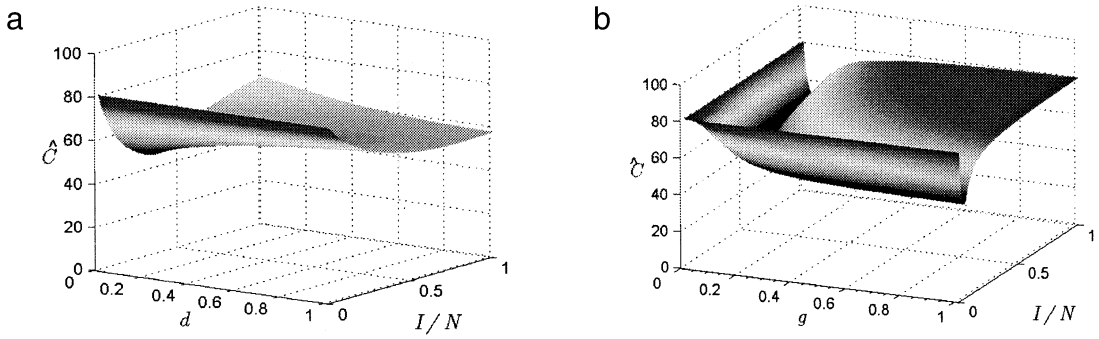

FIG. 2. (a) The optimal contact rate is presented over a range of exogenously determined rates of mortality and disease prevalence; $K = 81$, $\rho = gv/(v + \xi)$, $g = 0.1$, $v = 0.5$, $\xi = 1$, $p = 0.00005$ (the value for p corresponds to more than a 50% increase in the host mortality rate when the contact rate is reduced by 50%). (b) The optimal contact rate is presented over a range of exogenously determined transmission probabilities and disease prevalence; $d = 0.15$, $K = 81$; $\rho = gv/(v + \xi)$, $v = 0.5$, $\xi = 1$, $p = 0.00005$.

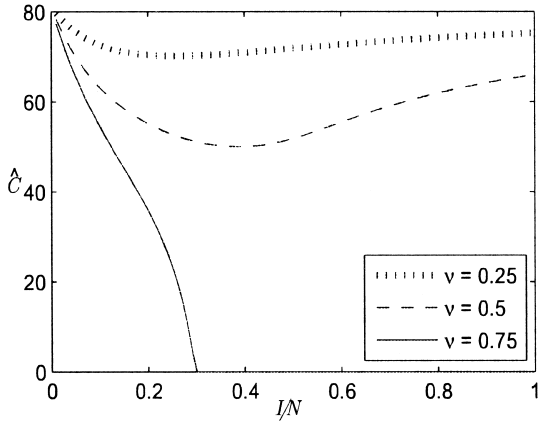

FIG. 3. The optimal contact rate is presented over a range of exogenously determined rates of disease prevalence, with different values of pathogen virulence. At relatively low levels of virulence, the evolutionarily stable contact rate responds nonmonotonically to disease prevalence. At high levels of virulence, the evolutionarily stable contact rate responds negatively to disease prevalence; $p = 0.00005$, $K = 81$, $\rho = gv/(v + \xi)$, $g = 0.1$, $\xi = 1$; $d = 0.15$.

host contact rates. Both high rates of host mortality (Fig. 4a) and low probabilities of disease transmission (Fig. 4b) cause pathogen prevalence to be low (or even zero), selecting for contact rates near the disease-free optimum of K. In essence, we can view being infectious for a short time (due to high mortality) and being poorly transmissive per unit of time (due to low transmission probabilities) as epidemiologically analogous, with both limiting the spread of the disease. At all nonzero rates of pathogen virulence, contact rates fall from their disease-free optimum as disease prevalence rises in response to decreases in background mortality or increases in disease transmission probabilities. Whether contact rates continue to fall as the parameters shift depends on the virulence of the disease. At relatively low rates of pathogen virulence, contact rates eventually begin to rise as prevalence rises. This is expected because we know from Figure 2 that contact rates will rise at high rates of disease prevalence when virulence is low. But when the pathogen threatens a relatively quick death (with high virulence), infection poses a more powerful deterrent to contact (Fig. 2), and the relationship between contact and the parameters d or g become monotonic.

COEVOLUTION OF HOST SOCIALITY AND INFECTIOUS DISEASE

We have shown that ES host contact rates may be nonmonotonic functions of disease parameters for organisms whose survival depends on sociality. However, just as the analysis in the section Optimal Contact Responds to Disease Prevalence is partial in the sense that it treats disease prevalence as exogenous to host contact, the analysis in the section Evolutionarily Stable Contact and Disease Prevalence Respond to Each Other is only partial in that it treats pathogen virulence as exogenous. The question we now pose is: In what way will the host influence the evolution of the pathogen? And if the host is evolving in response to the pathogen, and the pathogen evolves in response to the host, what general properties of this coevolutionary system can we predict? In other words, how does host sociality coevolve with infectious diseases?

Ewald (1994) hypothesized that higher host contact rates select for more virulent strains of pathogens. He argued that this is because there is a positive relationship between pathogen virulence and transmission, and high levels of host contact offer transmission opportunities for the pathogen, increasing the benefits of being transmissive while lowering the costs of virulence. However, because most evolutionary models of simple disease-host systems predict that pathogens maximize their basic reproductive ratio, R_0 (Bremmermann and Thieme 1989; Frank 1996), host behavior that influences birth rates and transmission rates multiplicatively will not have any influence on pathogen evolution at the population equilibrium (Bull 1994; Lipsitch and Nowak 1995; Lipsitch 1997; Day 2001). For example, a typical R_0 for an S-I equation is $R_0 = \beta(C, v)/(d + v)$, where $\beta(C, v) = \rho(v)C$, and $\rho(v) = gv/(v + \xi)$. The evolutionarily stable rate of virulence is, $v^* = \sqrt{\xi d}$, which is a simple positive function of the host's death rate. The reasoning is that, by shortening the time allotment for transmission, high host death rates select for high transmission rates and their corresponding level of virulence. Alternatively, long-lived hosts favor lower pathogen virulence and lower transmission rates. Because the literature regarding evolution of virulence has generally assumed host contact to be independent of host survival, virulence is considered to not influence long-run pathogen evolution.

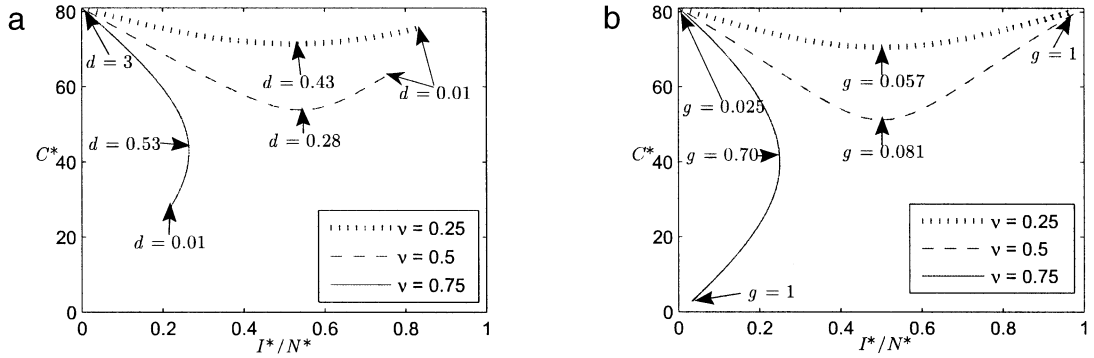

FIG. 4. (a) Evolutionarily stable contact and the corresponding equilibrium disease prevalence are presented for different values of virulence, v, over a range of background mortality rates; $d \in [0.01, 3]$; $K = 81$, $p = 0.00005$, $\rho = gv/(v + \xi)$, $g = 0.1$, $\xi = 1$. (b) Evolutionarily stable contact and the corresponding equilibrium disease prevalence are presented for different values of virulence, v, over a range of transmission probabilities; $g \in [0.025, 1]$; $K = 81$, $p = 0.00005$, $d = 0.15$, $\rho = gv/(v + \xi)$, $\xi = 1$.

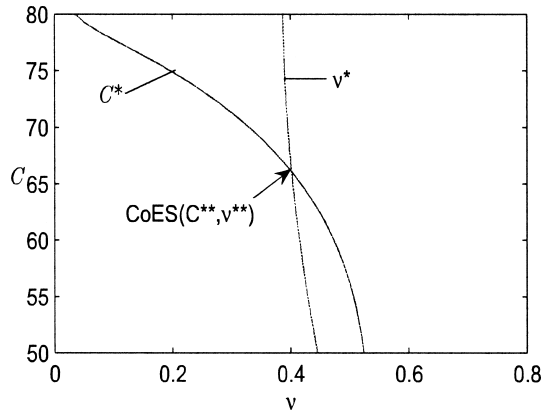

FIG. 5. The coevolutionary equilibrium is where the contact and virulence curves intersect. Notice that the optimal virulence falls as contact rises; $d = 0.15$, $\xi = 1$, $K = 81$, $p = 0.00005$, $g = 0.1$.

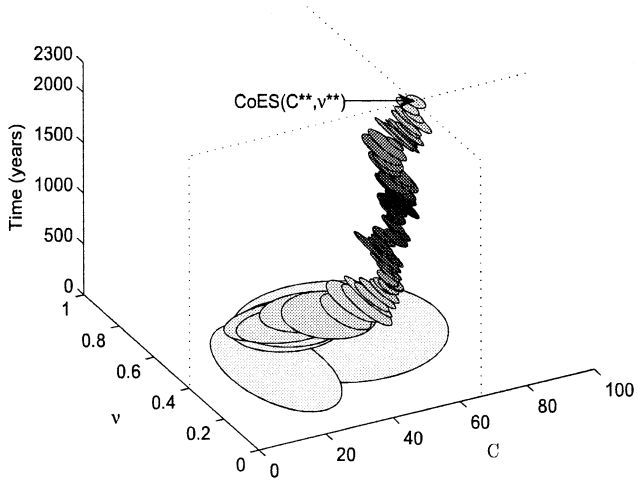

FIG. 6. Contact and virulence coevolve in a stochastic simulation. The coordinates of the center of each disk represent the average contact and virulence at each time period, which is incremented in units of 25 years. The distance between the top and bottom of the disk along the v and C gradients represent the standard deviation of the virulence and contact rates, respectively. The dotted lines represent the analytically predicted values of the coevolutionarily stable equilibrium; $d = 0.15$, $\xi = 1$, $K = 81$, $p = 0.00005$, $g = 0.1$. For a more detailed explanation of the simulation, see Appendix.

However, we emphasize here that the reason for contact among social organisms is precisely because it confers greater fitness, which may often be due to lower death rates. Therefore, we predict host contact rates to have a long-term influence on the evolution of virulence. In our model, R_0 is:

$$R_0 = \frac{C\rho(v)}{d + p(K - C)^2 + v}, \tag{4}$$

where $\rho(v) = qv/(v + \xi)$. The ES virulence is then:

$$v^* = \sqrt{\xi[d + p(K - C)^2]}. \tag{5}$$

Our prediction is therefore exactly opposite of the ideas put forth by Ewald (1994). Starting from the disease-free optimum, K, any decrease in C will result in a higher host death rate and greater virulence. Alternatively, any increase in host contact rates from some preexisting equilibrium of C that is necessarily less than K would result in longer life expectancy and lower virulence. Perhaps more importantly, because pathogen evolution is a function of host sociality and host sociality is a function of virulence, the appropriate approach to considering this relationship is by calculating coevolutionarily stable (CoES) strategies (van Baalen 1998; Restif et al. 2001; Gandon et al. 2002; Restif and Koella 2003).

Now we can solve both ES strategies simultaneously to determine the CoES contact rate, $C^{**} = f_4[\rho(v^*), v^*]$, and virulence, $v^{**} = f_5(C^*)$, (note that we use the star notation to denote the ES value and the double-star notation to denote the CoES value). In Figure 5 we can see that a coevolutionary equilibrium is located at the intersection of the host contact curve (or reaction function) and the pathogen virulence curve. Notice that pathogen virulence falls as contact rises, counter to the hypothesis by Ewald (1994). It is also not surprising to see that, as virulence rises, the ES contact rate falls, which can also be seen in Figures 3 and 4. We verified the results in Figure 5 by stochastically simulating coevolution from an initial population of pathogens and hosts with a wide range and even distribution of virulence and contact rates, respectively (Fig. 6). The mean values converge on C^{**} and v^{**} as the distribution of these phenotypes narrows over time. For a more detailed explanation of the simulation, see the Appendix.

Coevolutionarily stable rates of contact are presented with their corresponding pathogen prevalences over a range of parameter values in Figure 7. The important feature is that the relationship between host contact and pathogen prevalence is nonmonotonic, and indeed we see contact rates with maximum values at low and high values of pathogen prevalence. While the ultimate reasons for this are complex, there are partial relationships that play an important role: high levels of disease prevalence negate the value of avoidance, selecting for contact rates near the disease-free optimum, and high contact rates induce lower pathogen virulence, increasing the life expectancy of infected individuals and feeding back to higher disease prevalence.

It is also worth noting that the range of virulence values observed between the two figures varies substantially from $v = 0.1$ to $v = 1.73$ (Fig. 7a), to $v = 0.39$ to $v = 0.42$ (Fig. 7b). This can be understood by considering the strategy of the pathogens (eq. 5), which is a function of background mortality, d, but is not a function of the transmission probability parameter, g. Therefore, when we change the value of g, we alter the strategy of the host but not that of the pathogen. This corresponds to a different contact curve, so that the CoES strategies move along the virulence curve, which, in this case, has a relatively narrow range. However, when we alter the value of d, both strategies change, corresponding to new curves for both players in this coevolutionary game and resulting in a broader suite of values for virulence. Another consequence of d influencing the strategies of both players (instead of only one) is that we find a nonmonotonic relationship between the ES rates of contact and virulence over different d values (Fig. 8). In other words, the coevolutionary outcome suggests a parameter range in which contact and virulence are indeed positively related,

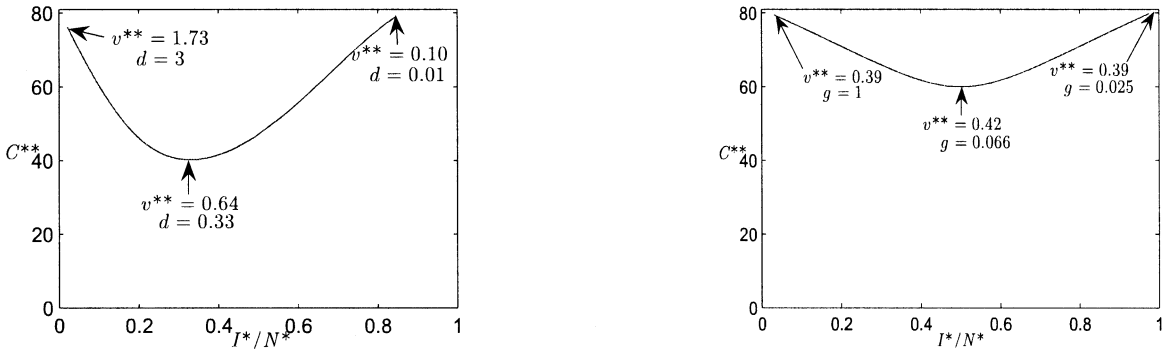

FIG. 7. (left) The coevolutionary relationship between contact and disease prevalence is U-shaped across background mortality rates; $d \in [0.01, 3]$, $\xi = 1$, $K = 81$, $p = 0.00005$, $g = 0.1$. (right) The coevolutionary relationship between contact and disease prevalence is U-shaped across a range of transmission probabilities; $g \in [0.025, 1]$, $d = 0.15$, $\xi = 1$, $K = 81$.

which is not the case for our model in which only the pathogen evolves. Alternatively, plotting changes in g values would simply reproduce the monotonic virulence curve in Figure 5 because the pathogen strategy is not a direct function of g.

DISCUSSION

There are two separate literatures on the relationship between host contact or sociality and disease prevalence. On the one hand, it is suggested that because higher contact will induce greater disease transmission we can expect a positive correlation between contact and prevalence (Anderson and May 1979; Møller et al. 1993; Arneberg et al. 1998; Ezenwa 2003). On the other hand, Freeland (1976, 1979), Hart (1988a, 1990), Loehle (1995), Moore (2002), and others have argued that the host may respond to higher prevalence with lower contact. These seemingly conflicting positions need to be addressed by noting, first, that the two variables—contact and prevalence—are determined simultaneously for systems in which diseases represent a cost to sociality. Moreover, systematic evolutionary responses from the pathogen must also be accounted for. Because of the complexity of this system, it is helpful to first consider the different processes in isolation to better understand their interactions.

We present a model that differs from the standard S-I framework in two ways: host contact rates are assumed to determine both the rates of disease transmission and host mortality, as is expected to be the case for many social species. Specifically, we assume that in the absence of the pathogen the host would evolve to some finite optimal value of contact and that the impacts on host survival that result from deviations from this optimum can be represented by a quadratic function (though an alternative functional form produces similar results; see Supplementary Material available online only).

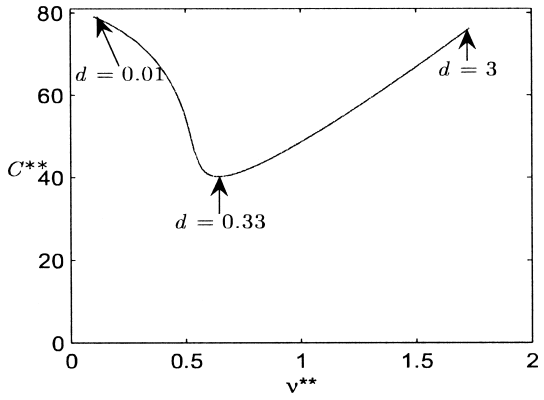

FIG. 8. The coevolutionarily stable values of contact and prevalence are nonmonotonic over a range of background mortality rates; $d \in [0.01, 3]$, $\xi = 1$, $K = 81$, $p = 0.00005$, $g = 0.1$.

We show that, as generally hypothesized, exogenously increasing disease prevalence from initial low values results in a decrease in the evolutionarily stable rate of contact (Fig. 2). However, for a large range of the parameter space, the optimal contact rate increases as prevalence rises past a threshold level. In other words, though the disease never causes sociality to rise above the disease-free optimum, higher disease prevalence can actually induce greater sociality. This result is similar to that of van Baalen (1998), who found that the optimal investment in immunity rises and then falls as infection probabilities rise. The reason for both of these results is that the costs of the host's response to the disease, in terms of higher mortality, eventually overwhelm the benefits of those responses when infection is sufficiently difficult to evade. In our model, this means that the host would then evolve greater sociality at high levels of disease prevalence. An exception to this relationship is when the pathogen is sufficiently virulent to eliminate the value of contact after infection (Fig. 3). In this case, the optimal contact will fall monotonically as prevalence rises.

These partial phenomena explain the more complex interplay between equilibrium levels of disease prevalence and host contact, which is depicted in Figure 4. We alter the equilibrium disease prevalence through changes in background mortality (Fig. 4a) and the transmission probability (Fig. 4b). In both cases, we observe a U-shaped relationship between contact rates and prevalence for relatively low levels of pathogen virulence. At high virulence the relationship is backward-bending—that is, contact rates continue to fall as the parameters shift, but the equilibrium disease prevalence also falls with it. In all cases, the relationship is nonmonotonic.

Finally, we consider pathogen evolution. We show that

lower contact increases pathogen virulence, counter to Ewald's (1994) hypothesis. This is because, as contact rates fall from the disease-free optimum, so does host survival, thus lowering the benefits for the pathogen of preserving the host and increasing the advantages of being transmissive. As we alter various parameter values, we continue to find a U-shaped relationship between the CoES level of host sociality and the equilibrium disease prevalence (Fig. 7). We also find a U-shaped relationship between the CoES level of host sociality and pathogen virulence (Fig. 8).

How well does this model explain the evidence? While we are unaware of any studies that have tracked all three variables—contact rates, disease prevalence, and virulence—simultaneously, several studies have considered both disease prevalence and host group size (which is assumed to be highly correlated with contact rates), with a positive relationship often being observed (Davies et al. 1991; Côté and Poulin 1995; Dobson and Meagher 1996; Arneberg et al. 1998; Ezenwa 2003). Such a positive relationship would always be predicted in cases where evolutionary forces were expected to be irrelevant. This would be the case if variations in group size merely represented stochastic variations around an evolutionary equilibrium and not genetic heterogeneity due to localized differences in selection pressures such as background mortality.

However, if the variation in group size can be attributed to evolutionary forces operating under different ecological conditions, then there are some statistical issues that would complicate the analysis. A necessary condition for making statistical inference from simple linear regression analyses is that the regressor (or independent variable) is independent of the regressand (or dependent variable). However, in cases where host demographics can be attributed to genetic variation responding to selection pressure from diseases, both group size and disease prevalence are dependent on each other. In other words, such analyses suffer from an endogeneity bias. This is in addition to an omitted variable bias, with the relevant omitted variable being pathogen virulence. Unfortunately, the system we are describing is nonlinear and our expected relationship between these variables is not even monotonic. Therefore, we have no a priori expectation of the direction (up or down) of such biases; it depends on the parameter values that generate the different equilibrium outcomes for these subpopulations. Thus, it is not surprising that Ezenwa (2003), for example, found no significant correlation between group size and disease prevalence for four of six populations of African bovids she studied. One might also wonder how many unpublished studies found no significant correlations.

In contrast to the studies that found a positive relationship between group size and disease prevalence, the absence of studies that found a negative general correlation is conspicuous. This is especially surprising given the mounting evidence that animals change their behavior in response to risks of infection. Such behaviors include avoidance of infected conspecifics—observed, for example, experimentally in mice (Edwards 1988) and guppies (Kennedy et al. 1987)—as well as individual preference for parasite-free habitats, such as nesting sites (Emlen 1986; Christe et al. 1994) and grazing space (Hart 1988b). If diseases constitute strong enough selection pressure to alter animal behavior temporarily, why have we not seen evidence of more substantial evolutionary influences on social structure such as grouping? One possible answer is simply that we would expect both positive and negative relationships, something that cannot be distilled from simple linear regression analyses. Those that have found positive relationships may be capturing population ecological forces that are expected to be positive in the absence of evolutionary responses.

Because of all of the important interacting factors between host contact rates, pathogen prevalence, and virulence, it would appear that the proper first step to confirming a theory such as this is in the laboratory, where the relevant parameter values can be systematically altered and the analysis can be conducted on data with high resolution.

In their classic paper, Brown and Brown (1986, p. 1217) suggested that, ''without compensating benefits of coloniality, the cost of ectoparasitism would quickly select for solitary nesting in Cliff Swallows.'' Similarly, Møller et al. (2001, p. 142) argued that ''if the cost of parasitism is greater in colonial species than in solitary ones, there should be selection for early fledging within species since nestlings thereby could evade their parasites.'' This logic is common. Counter-intuitively, our analysis indicates that increased prevalence of infectious diseases can actually induce greater sociality.

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Appendix

Stochastic Simulation of Coevolving Contact and Virulence

The system is simulated using the Gillespie algorithm (Gillespie 1977), with birth, death, and transmission events occurring stochastically in accordance with the probabilities that correspond to the deterministic equations (1) and (2). Initially, we assign 600 host phenotypes with contact efforts that are evenly distributed between zero and 10. We also assign 400 pathogen phenotypes with rates of virulence that are evenly distributed between zero and one. For 99% of birth events, the offspring inherits the contact effort of its parent. For 1% of the birth events, the contact effort mutates within -1 and 1 units from its parent's contact effort, with a uniform probability for all values within that range. For 99% of transmission events, the newly infected individual contracts a pathogen that is equally virulent to the individual that infected it. For 1% of the transmission events, the pathogen virulence mutates within -0.2 and 0.2 units, with a uniform probability for all values within that range. For a detailed account of the conversion of a deterministic model into a stochastic simulation using the Gillespie algorithm, see Wearing et al. (2004).