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Host-parasite population dynamics under combined frequency- and density-dependent transmission

Jonathan J. Ryder, Martin R. Miller, Andy White, Robert J. Knell and Michael Boots

J. J. Ryder (jon.ryder@uea.ac.uk), School of Medicine, Health Policy & Practice, Univ. of East Anglia, Norwich, UK, NR4 7TJ. – M. R. Miller and M. Boots, Dept of Animal & Plant Sciences, Univ. of Sheffield, Sheffield, UK, S10 2TN. – R. J. Knell, School of Biological and Chemical Sciences, Queen Mary, Univ. of London, London, UK, E1 4NS. – A. White, Dept of Mathematics and the Maxwell Inst. for Mathematical Sciences, Heriot-Watt Univ., Edinburgh, Scotland, EH14 4AS.

Many host-parasite models assume that transmission increases linearly with host population density ('density-dependent transmission'), but various alternative transmission functions have been proposed in an effort to capture the complexity of real biological systems. The most common alternative (usually applied to sexually transmitted parasites) assumes instead that the rate at which hosts contact one another is independent of population density, leading to 'frequency-dependent' transmission. This straight-forward distinction generates fundamentally different dynamics (e.g. deterministic, parasite-driven extinction with frequency- but not density-dependence). Here, we consider the situation where transmission occurs through two different types of contact, one of which is density-dependent (e.g. social contacts), the other density-independent (e.g. sexual contacts). Drawing on a range of biological examples, we propose that this type of contact structure may be widespread in natural populations. When our model is characterized mainly by density-dependent transmission, we find that allowing even small amounts of transmission to occur through density-independent contacts leads to the possibility of deterministic, parasite-driven extinction (and lowers the threshold for parasite persistence). Contrastingly, allowing some density-dependent transmission to occur in a model characterized mainly by density-independent contacts (i.e. by frequency-dependent transmission) does not affect the extinction threshold, but does increase the likelihood of parasite persistence.

The idea that directly transmitted parasites exploit different types of host contact is not new, but here we show that the impact on dynamics can be fundamental even in the simplest cases. For example, in systems where density-dependent transmission is normally assumed de facto, we show that parasite-driven extinction can occur if a small amount of transmission occurs through density-independent contacts. Many empirical studies are still guided by the traditional density/frequency dichotomy, but our combined transmission function may provide a better model for systems in which both types of transmission occur.

A useful starting assumption in many host-parasite models is that the rate of transmission increases linearly with population density (Anderson and May 1979, 1981, McCallum et al. 2001). If transmission is direct, this assumption will be valid if contacts between host individuals increase proportionately with population density. However, various refinements to this simple framework have been proposed to capture the complexity of biological systems. The simplest of these assumes that the contact rate is independent of host population density. For example, the rate at which individuals acquire sexual contacts is often thought to remain approximately constant as population density changes. This is assumed to lead to 'frequency-dependent' transmission for sexually transmitted diseases (STDs) (Getz and Pickering 1983, Anderson and May 1991, Thrall et al. 1993, 1995). A similar argument can be applied to vector-transmitted diseases, because relatively large vector populations and vector biting behaviour may compensate for changes in host density (Antonovics et al. 1995).

Although the distinction between density- and frequency-dependence is straight-forward, fundamentally different predictions concerning host-parasite

equilibria apply in each case. For example, there is no threshold density for parasite persistence in models that assume complete frequency-dependence (Anderson and May 1981, Getz and Pickering 1983, Lloyd-Smith et al. 2005). STDs may therefore be able to persist when host population density is too low to allow 'ordinary' infectious diseases (OIDs), with density-dependent transmission, to do so. Deterministic parasite-driven host extinction is also possible with frequency-dependent transmission (Getz and Pickering 1983, Boots and Sasaki 2003). Extinction is only expected to occur with density-dependent transmission if the parasite reduces the size of or otherwise destabilizes its host's population (such that there is a higher risk of stochastic events leading to extinction) or if the parasite is shared by a more common 'reservoir' host (de Castro and Bolker 2005). Finally, parasites cannot regulate their host population independently in models based on a frequency-dependent transmission term (Getz and Pickering 1983).

The frequency-dependent model is generally applied to animal-STD systems, because it is assumed that sexual contact rate is independent of population density. However, further modifications of the transmission function allow for more complex departures from simple density-dependence. For example, Antonovics et al. (1995) modelled frequency- and density-dependence as opposite ends of a continuum using the Holling type II functional response. This assumption can lead to transmission approximating density-dependence at low densities and frequency-dependence as densities increase (see also Thrall et al. 1995, Lockhart et al. 1996, Thrall and Antonovics 1997, Thrall et al. 1998, McCallum et al. 2001). Fenton et al. (2002) have also analysed non-linear deviations from density-dependent transmission using Hochberg's (1991) phenomenological function, which may provide a more realistic basis for approximating the contact structure of a range of biological systems.

In this study, we consider a further refinement of the transmission function that is suggested by the biology of many host-parasite systems. In particular, we note that the transmission of infection often occurs through more than one type of contact, each of which may have a different functional relationship with population density. Various factors at the level of individual behaviour may give rise to such dynamics (see also Thrall and Antonovics 1997, Thrall et al. 1998). For example, STDs may sometimes be transmitted partly through social or other types of non-sexual contact (Vitale et al. 2000, Cattani et al. 2003). We envisage that there may be a component of density-dependence in such cases, in addition to the expected frequency-dependent transmission, because social/non-sexual contacts will often be density-dependent. A similar argument can be applied to OIDs. For example, OIDs may commonly be transmitted during sexual activity, leading to a component of frequency-dependent transmission (Bastos et al. 1999).

Thus, our motivation in this paper is to examine the simple case in which transmission occurs through two different types of contact – one of which is density-dependent, the other density-independent. Our approach is analogous to modelling scenarios along the 'continuum' between density- and frequency-dependence. However, by using a combined linear function, we make it explicit that transmission occurs through two different types of contact. We analyse how the thresholds between the parasite-driven host extinction, endemic and disease-free equilibria vary as: (1) transmission arising through density-dependent contacts (e.g. social transmission) is gradually incorporated into a system otherwise characterized by density-independence (e.g. sexual transmission, frequency-dependence); and (2) transmission arising through density-independent contacts (i.e. frequency-dependence) is gradually incorporated into a system characterized mainly by density-dependent transmission. Thus, our transmission function allows the level of frequency- and density-dependence to be varied independently, enabling us to model outcomes in a range of systems (which we discuss with examples) where elements of density- and frequency-dependent transmission occur simultaneously. We illustrate how our transmission function may be linked to empirical systems.

The model and its biological context

Under the standard density-dependent model (and assuming that the area occupied by the population is fixed), the infection rate is given by $vmNS(I/N)$, where v is the per contact probability of transmission, m is a constant which scales the total population density (N) to give a density-dependent contact rate (mN), and S and I are the densities of susceptible and infected individuals (Begon et al. 2002). The probability that any one contact is with an infected individual is given by I/N. This formulation of the transmission function is equivalent to the more familiar βSI, with vm equating to the transmission coefficient, β. The equivalent frequency-dependent model is $vcS(I/N)$, where c is a density-independent contact rate. This is equivalent to the $\beta' SI/N$ term commonly used for STDs, where vc equates to the corresponding transmission coefficient, β' (Begon et al. 2002).

In these two formulations of the transmission function, $vmNS(I/N)$ and $vcS(I/N)$, the contact rates are given by mN and c, respectively. We now assume that transmission can be described by a function that combines these two types of contacts:

$$\frac{v(c + mN)SI}{N} \quad (1)$$

The rate at which effective contacts are acquired is thus modelled simply as $c+mN$, rather than the traditional mN (density-dependence) or c (frequency-dependence).

A clear demonstration of the biological meaning of this function comes from the familiar dichotomy between STDs and OIDs, to which we referred in the preceding section. STD transmission is generally modelled using a frequency-dependent formulation (i.e. $\beta'SI/N$). From the above argument this is equivalent to setting $m = 0$ in Eq. 1, which reduces to $vcS(I/N)$ with $c > 0$ (i.e. sexual contact rate is positive). If we now allow some transmission to occur through additional density-dependent contacts (e.g. social contacts), such that $m > 0$, then it clear that Eq. 1 will provide a more realistic description of the transmission dynamics than either of the standard linear terms. Similarly, considering again the example we gave in the preceding section concerning the sexual transmission of OIDs, it can be seen that the effect of density-independent (sexual) contacts can be included by setting $c > 0$.

The occurrence of behaviourally distinct types of contact between hosts (e.g. sexual vs non-sexual/social contacts) provides a useful illustration of the biological meaning of Eq. 1. We also note that combined transmission dynamics may arise through heterogeneities in the relationship of one specific type of contact with population density. The relatively simple host behaviour demonstrated in some invertebrate-STD systems provides an example. Male *A. bipunctata* ladybirds mate promiscuously, but females can control their own mating rate by resisting unwanted copulation attempts (Webberley et al. 2002). The transmission of a sexually transmitted mite *Coccipolipus hippodamiae* found in these beetles may contain elements of both frequency- and density-dependence (Ryder et al. 2005, Webberley et al. 2006) because, at any one point in time, only a proportion of the females in a population may demonstrate such resistance. These females may therefore mate at a fixed rate (leading to frequency-dependent transmission), whereas females that continue to mate promiscuously will generate sexual contacts at a density-dependent rate (leading to density-dependent transmission). The combined linear function (Eq. 1) provides a simple means of modelling this type of heterogeneity.

A similar form of heterogeneity may lead to combined transmission dynamics in vertebrate systems. For example, a number of studies have demonstrated that the rate of extra-pair copulations (EPCs) in birds increases with population density (Hatchwell 1988, Brown and Brown 1996). Females may thus mate at a density-independent rate with their partner, but engage in more extra-pair copulations at higher population densities (i.e. density-dependent extra-pair sexual contacts). This increase in promiscuity will introduce at least a component of density dependence to the transmission dynamics of any STD present.

Our contention is that whether one is interested in either STD or OID transmission dynamics, Eq. 1 allows transmission arising through contacts that might otherwise be considered of negligible importance (and thus be neglected) to be incorporated explicitly. Our interest is in exploring the implications of doing so for host-parasite equilibria. The parameters c and m determine the amount of frequency- and density-dependent transmission, respectively. Assuming $c > 0$, transmission approaches complete frequency-dependence as $m \to 0$, and assuming that $m > 0$, transmission approaches complete density-dependence as $c \to 0$. If we say that $vm = \beta$ and that $vc = \beta'$ then it can be seen that

$$\frac{v(c + mN)SI}{N} = \frac{\beta'SI}{N} + \beta SI \quad (2)$$

This is useful from an empirical perspective, because it is reasonably straight forward to estimate both transmission coefficients by carrying out appropriate experiments (depending, of course, on the particular system), making it possible to estimate the relative amount of each type of transmission in natural systems (Ryder et al. 2005).

Analytically, the question that we are attempting to address is: how do deviations from 'pure' density- or pure frequency-dependence affect dynamics? We therefore consider a situation where the dynamics are described by the following equations:

$$\frac{dS}{dt} = (b - hN)N - \frac{v(c + mN)SI}{N} - uS \quad (3)$$

$$\frac{dI}{dt} = \frac{v(c + mN)SI}{N} - (u + \alpha)I \quad (4)$$

We assume that susceptible and infected individuals reproduce at the same rate (i.e. there are no sterility effects associated with infection). The birth and natural death rates are b and u respectively, α is the rate of disease-induced mortality (i.e. virulence), and h is a coefficient of density-dependent host regulation. Here the carrying capacity is given by $K = r/h$, where $r = b - u$, the intrinsic growth rate of the host population. There is no host recovery, and all parameters are assumed to be positive.

To simplify the analysis, we make the substitutions $N = S + I$ and $p = I/N$. The equations can then be rewritten in terms of the total host density (N) and the prevalence of infection (p):

$$\frac{dN}{dt} = (b - hN)N - uN - \alpha pN \quad (5)$$

$$\frac{dp}{dt} = p[v(c + mN)(1 - p) - (b - hN) - \alpha(1 - p)] \quad (6)$$

As will be shown, this substitution allows a more intuitive explanation of the equilibrium states.

Analysis

The analytical results are derived in Appendix 1. For positive parameters, there are three non-trivial and biologically feasible equilibrium states for Eq. 5–6. In the first equilibrium the parasite is unable to invade the host population, which settles at its uninfected carrying capacity, $N_K = (b - u)/h$. In the second, the dynamics reach a stable endemic equilibrium where the parasite is maintained at a constant prevalence (N^*, p^*). In the third equilibrium state, the parasite drives the host to extinction. These results are illustrated in Fig. 1, in which the (v, α) parameter space is partitioned into regions where we observe the three outcomes. The equilibrium regions are delineated by two thresholds in infection probability, v_t and v_c. The threshold v_t gives the minimum infection probability for persistence of the parasite and delineates the boundary between the

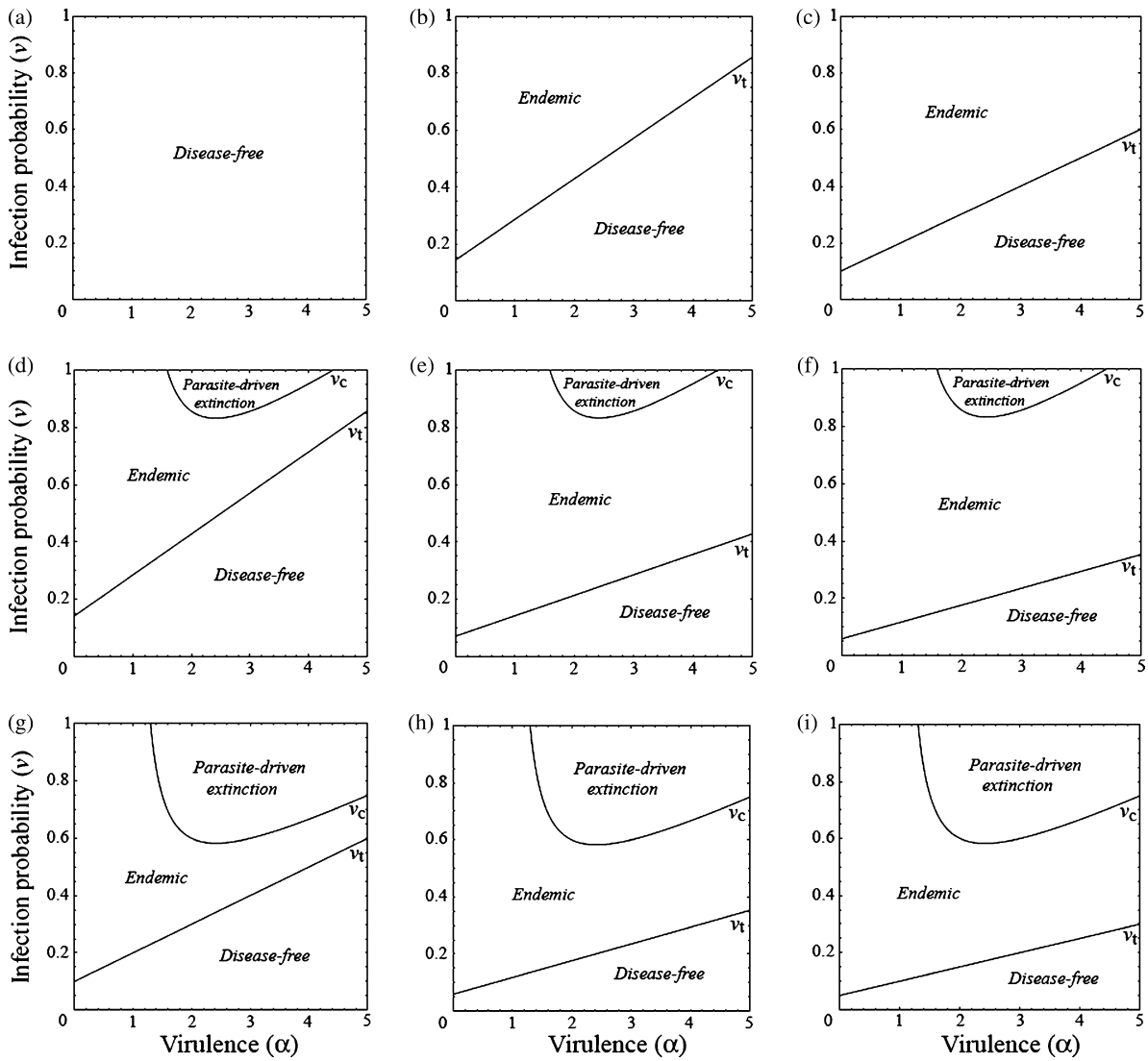

Fig. 1. Outcomes in (v, α) parameter space for different levels of frequency- and density-dependence: (a) m = 0, c = 0; (b) m = 7, c = 0; (c) m = 10, c = 0; (d) m = 0, c = 7; (e) m = 7, c = 7; (f) m = 10, c = 7; (g) m = 0, c = 10; (h) m = 7, c = 10; (i) m = 10, c = 10. The other parameters are: b = 2, u = 1 and h = 1. The carrying capacity is $N_K = 1$.

disease-free and endemic regions:

$$v_t = \frac{u + \alpha}{c + (m(b - u)/h)} \quad (7)$$

A disease-free equilibrium thus requires that $v < v_t$. This threshold increases with virulence (α); a more virulent parasite requires higher infection probability to be able to persist in the population. The upper threshold, v_c, delineates the boundary between the endemic and extinction regions:

$$v_c = \frac{\alpha(u + \alpha)}{c(\alpha - b + u)} \quad (8)$$

This gives the minimum infection probability necessary for the parasite to drive the host to extinction ($v > v_c$). For host-parasite coexistence to occur, we therefore require an intermediate probability, $v_t < v < v_c$. From Eq. 8, below a certain level of virulence ($\alpha < b - u$) there is no possibility of extinction, since births outweigh deaths. Above this level, there is a non-monotonic relationship between virulence (α) and the infection probability (v) required to cause extinction. Thus, at low virulence, reproduction from infecteds provides a mechanism that reduces the cost of parasitism, and extinction can only occur if the infection probability is high. Extremely virulent parasites are also unlikely to cause extinction, because infected hosts die very rapidly, reducing the opportunities for transmission (Fig. 1). It can be shown analytically that if infecteds are unable to reproduce, the Eq. 7 for parasite persistence is unchanged. However, the condition for parasite-driven extinction reduces to the criterion (A4), which merely requires that the frequency-dependent transmission rate, vc, exceed a certain value. This result is analogous to that obtained for a purely frequency-dependent model: allowing reproduction from infected individuals reduces the probability of extinction (Boots and Sasaki 2003).

We now consider the effect of independently varying the amount of frequency- and density-dependence, by allowing the values of c and m to vary. Figure 1 illustrates the effect of varying either m or c whilst the other is held constant.

Probability of parasite persistence

Increasing m from 0 to 10, with c fixed at 10 (i.e. moving from complete frequency-dependence to both frequency- and density-dependence), has the effect of reducing the region of disease-free parameter space (Fig. 1g → 1h → 1i). Thus, by adding an increasing component of density-dependent transmission, parasite persistence becomes possible at lower infection probabilities for a given level of virulence. It is also clear that the relative decrease in the disease-free region with increasing m is more pronounced when c is 7 (Fig. 1d → 1e → 1f) than when c is 10.

Similarly, moving from complete density-dependence to both frequency- and density-dependence (increasing c from 0 to 10, with m fixed at 10, Fig. 1c → 1f → 1i) reduces the size of the disease-free region (i.e. the addition of frequency-dependent transmission allows the parasite to persist at lower infection probabilities). Likewise, the relative decrease in the disease-free region with increasing c is more pronounced when m is 7 (Fig. 1b → 1e → 1h) than when m is 10.

Probability of parasite-driven extinction

The boundary of the parasite-driven host extinction region is unaffected by variation in m: increasing the amount of density-dependence does not affect this equilibrium (e.g. Fig. 1g → 1h → 1i). In contrast, both the existence and size of the parasite-driven extinction region depends on c. When $c = 0$ (pure density-dependence, with $m = 7$ or $m = 10$), parasite-driven extinction cannot occur and the endemic region is unbounded for increasing values of v (Fig. 1b–c). When a component of frequency-dependence is introduced, parasite-driven extinction becomes possible at high infection probabilities (Fig. 1d–f). Further increases in c lower the upper boundary of the endemic region, making extinction possible for a lower infection probability (Fig. 1g–1i).

Probability of endemic persistence

Higher levels of density-dependent transmission always increase the probability of endemic persistence, by lowering the boundary between the endemic and disease-free regions in (v, α) parameter space. In contrast, higher levels of frequency-dependent transmission often reduce the probability of endemic persistence, particularly if the amount of density-dependent transmission is high (Fig. 1f → 1i). In fact, the probability of endemic persistence may actually decrease if c and m increase at the same rate (Fig. 1e → 1i). However, if frequency-dependent transmission increases from low (or zero) to only moderate levels, this may result in an overall increase in the probability of endemic persistence (Fig. 1b → 1e). The ambiguity here is due to the fact that increasing c lowers the threshold for parasite persistence but also the threshold for parasite-driven extinction.

Figure 2 summarizes these results by plotting the disease-free, endemic and parasite-driven host extinction regions in (m, c) parameter space. As the value of m increases, the endemic region expands at the expense

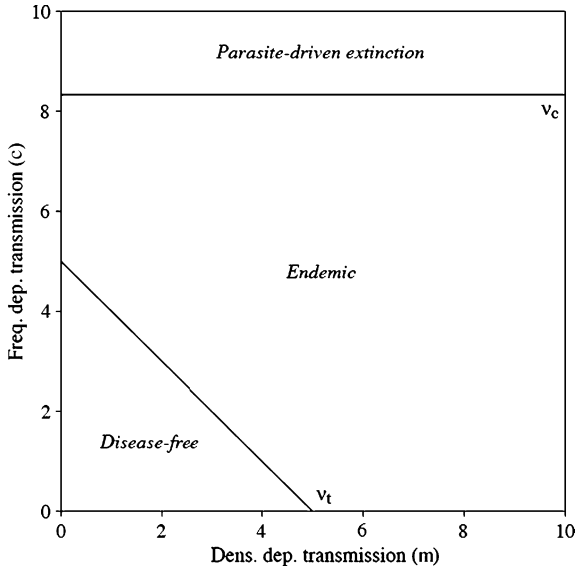

Fig. 2. Outcomes in (c, m) parameter space: $b = 2$, $u = 1$, $h = 1$, $v = 0.7$ and $\alpha = 2.5$. The carrying capacity is $N_K = 1$.

of the disease-free space, making parasite persistence attainable for a smaller component of frequency-dependence (v_t occurs at decreasing values of c as m increases). However, increasing the amount of density-dependence (m) has no effect on the parasite-driven extinction region (v_c occurs at the same value with respect to c for varying m). Conversely, for any given value of m, the amount of frequency-dependence (c) determines whether the population is in disease-free, endemic or parasite-driven extinction parameter space. The addition of frequency-dependence may therefore allow parasites with principally density-dependent transmission to persist in regions of parameter space where they would otherwise go extinct. On the other hand, a sufficiently high level of frequency-dependence can cause the extinction of both host and parasite deterministically.

Discussion

The view that transmission dynamics will often be too complex to characterize using the traditional density/frequency dichotomy seems widely accepted (Hochberg 1991, Antonovics et al. 1995, Lockhart et al. 1996, Thrall and Antonovics 1997, Thrall et al. 1998, McCallum et al. 2001, Begon et al. 2002, Fenton et al. 2002). Therefore, it is important that the consequences of intermediate forms of transmission for host-parasite equilibria are fully explored (Fenton et al. 2002), because the population outcomes associated with the two traditional models are fundamentally different. In particular, in systems where pure frequency-dependent transmission is assumed, deterministic, parasite-driven extinction can occur (Getz and Pickering 1983, Boots and Sasaki 2003). In addition, host-parasite coexistence is only possible in frequency-dependent models if the host population experiences density-dependent mortality independently of any disease-related factors (Getz and Pickering 1983, Thrall et al. 1993, Lockhart et al. 1996). In contrast, parasites with density-dependent transmission can regulate the host population even if there is no density-dependent mortality, and have threshold densities for persistence (Anderson and May 1981, Lloyd-Smith et al. 2005).

Our model incorporates a combined, linear function, such that transmission can occur through two distinct types of contact, one of which is density-dependent, the other density-independent. When transmission follows simple frequency-dependence (and incorporates a density-dependent host birth rate), host-parasite coexistence is possible for intermediate infection probabilities. Parasite-driven host extinction can also occur if the infection probability rises above the extinction threshold, v_c. If we allow (variable amounts of) frequency- and density-dependent transmission to occur simultaneously, then increasing the amount of density-dependent transmission (i.e. $m > 0$) has no effect on the position of v_c, which depends only on the frequency-dependent parameter, c. However, increasing the amount of density-dependence lowers the boundary between the endemic and disease-free regions (v_t). Thus, if c is fixed, the probability of endemic persistence increases with the strength of density-dependent transmission (m). Conversely, when frequency-dependence is incorporated into a density-dependent model, there is a reduction in the size of the disease-free region, and parasite-driven extinction becomes possible if the infection probability is high enough. As c is increased further (for fixed m), the extinction equilibrium becomes stable for lower values of v, constraining the region within which coexistence is possible.

Our most important conclusions are as follows. First, in host-parasite systems where transmission is normally assumed to be density-dependent, parasite-driven extinction may be possible if even a small amount of transmission occurs through contacts occurring at a density-independent rate (i.e. there is some frequency-dependent transmission). OIDs are generally only implicated in extinctions if they reduce the size of, or otherwise destabilize, their host's population, such that there is a higher risk of stochastic events leading to extinction (Ebert et al. 2000). Alternatively, if OIDs are shared by two hosts, one of which is a reservoir for the parasite while the other is at a low density, there is also a possibility of extinction (de Castro and Bolker 2005). If, however, transmission is in reality only 'mostly' density-dependent in such cases, and also partly

frequency-dependent (e.g. due to additional sexual transmission), then deterministic extinction may also be possible. This finding has broad significance. Many OIDs will have some component of frequency-dependent transmission and even a small degree may be sufficient to place a population above v_c. Parasites have been implicated in the extinction of their host a number of times, including the extinction of the Thylacine, a carnivorous marsupial (McCallum and Dobson 1995), populations of African wild dogs (Burrows et al. 1995), and some amphibian species (Daszak and Cunningham 1999). Our results indicate that a parasite will be even more likely to cause extinction if some transmission takes place through density-independent contacts. From Eq. 8, it is clear that this finding is unaffected by the strength of non-disease density-dependence (h). Intriguingly, recent experimental work on an amphibian-chytrid system detected significant components of both frequency- and density-dependent transmission (T. Garner, pers. comm.). Given that *Batrachochytrium dendrobatidis* has been implicated in the extinction of numerous amphibian species worldwide (Garner et al. 2005, 2006, Pounds et al. 2006), our analysis highlights the potential danger of overlooking frequency-dependence.

Second, we find that the threshold for parasite persistence reduces as frequency-dependent transmission becomes more important – even where the amount of density-dependent transmission is fixed. This result is also surprising, because frequency-dependence is not usually associated with an increased likelihood of parasite persistence (Getz and Pickering 1983, Thrall et al. 1993, Lockhart et al. 1996).

Third, in a host-parasite system otherwise characterized by frequency-dependence, increasing the amount of density-dependent transmission does not affect the threshold for parasite-driven extinction, although it does lower the threshold for persistence. It is not surprising that density-dependence is associated with an increased probability of host-parasite coexistence. However, it is striking that the extinction threshold is governed only by the amount of transmission that takes place through density-independent contacts (i.e. frequency-dependent transmission).

There is currently very little suitable data with which our model can be tested, but as was shown above, the combined transmission function can be converted to a form that should facilitate in estimating the two transmission coefficients, β and β' (Eq. 2). We recently used this approach (Ryder et al. 2005) to estimate the relative contribution of density- and frequency-dependent transmission in an animal-STD system in which previous studies had assumed frequency-dependent dynamics (Webberley et al. 2004, 2006). The coefficients were estimated experimentally, using a range of host densities, by fitting the model to data on the proportion of individuals that became infected within a set period. Surprisingly, the transmission data were best explained by the standard density-dependent term alone (i.e. βSI), although we anticipate that a larger sample size would have detected a component of frequency-dependence (Ryder et al. 2005). The dynamics of this system in the field are complex (Webberley et al. 2006), but our model may help to explain why coexistence is possible despite regular epidemics of infection, because density-dependent transmission should increase the probability of endemic persistence, particularly at high density (Eq. 7).

We know of no data on the relationship between STD transmission and population density in other animal-STD systems, but numerous studies have reported relationships between mating rate and population density (reviewed by Ryder et al. 2005). Where STDs are present in such cases, it seems probable that at least some transmission will take place through contacts that vary with population density (e.g. EPCs in birds; and density-dependent variation in mating rate in insects: Harshman et al. 1988, Gage 1995). Density-dependent changes in mating rate are also likely in higher vertebrate groups, such as red deer (Clutton-Brock et al. 1997). We emphasize that what is currently lacking in all of these taxa is empirical data on the relationship between STD transmission and population density. The frequency-dependent model has proven particularly useful in a plant-STD system, *Silene alba* infected with anther-smut fungus (Thrall and Jarosz 1994a, 1994b, Thrall et al. 1995). However, animal-STD systems are extraordinarily diverse (Lockhart et al. 1996, Knell and Webberley 2004) and the various studies we cite above suggest that many systems will exhibit components of both density- and frequency-dependence.

When considering human diseases, social factors such as fixed class sizes in childhood may lead to a substantial frequency-dependent component to transmission dynamics (Bjørnstad et al. 2002). Although this may mean that some childhood diseases, for example, have elements of both types of transmission, further analysis of our model indicates that the effectiveness of vaccination programmes should be unaffected. In short, although vaccination reduces the proportion of the population that is susceptible, this will affect the probability of transmission in the same way whether contacts are density-dependent or density-independent. Similarly, if we allow for recovery in our model, the threshold for disease persistence is reduced, but this affects density- and frequency-dependent transmission equally.

Our results may also be relevant in light of recent work on the evolutionary dynamics of sexual (frequency-dependent) versus nonsexual (density-dependent) transmission modes (Thrall and Antonovics 1997, Thrall

et al. 1998). Generally speaking, sexual transmission is predicted to evolve in response to low population densities and non-sexual transmission in response to high densities. Indeed, frequency-dependent (i.e. sexual) transmission modes are often thought to have evolved as a mechanism enabling pathogens to survive in smaller populations, an idea which has considerable empirical support (Lockhart et al. 1996). This is corroborated by our results, which indicate that highly frequency-dependent pathogens are more likely to persist at lower carrying capacities. However, as frequency-dependent transmission may lead to extinction of both host and pathogen, a significant component of density-dependent transmission may often be preserved in smaller populations.

In summary, our combined transmission function provides a basis for modelling dynamics in the simple, but important case where transmission occurs through two distinct types of contact that each have a different functional relationship with population density – one density-dependent, the other density-independent. As the number of reported cases of animal STDs continues to increase (Lockhart et al. 1996, Knell and Webberley 2004), we believe that this approach will help to encourage the integration of theoretical and empirical approaches to STD ecology. Similarly, we hope that our model will stimulate further research on the ecological dynamics of diseases normally assumed to have purely density-dependent transmission. In particular, our results show that the likelihood of host extinction may be increased if even a small amount of transmission occurs through density-independent contacts.

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Appendix 1.

There are four equilibrium solutions of Eq. 5–6. Taking the variables in the order (N, p), the equilibria are (0,0), (N_K, 0), (0,ρ_E) and (N*,p*). The relevance and stability of the equilibrium values are determined below.

(1) The trivial equilibrium, (0, 0) has Jacobian matrix:

$$\begin{pmatrix} b-u & 0 \\ 0 & -b-\alpha+cv \end{pmatrix} \tag{A1}$$

Assuming the birth rate (b) exceeds the natural death rate (u), the eigenvalue $\lambda_1 = b-u$ will be positive and the equilibrium is unstable.

(2) At the disease-free equilibrium (N_K, 0), the population reaches its carrying capacity, $N_K = (b-u)/h$. The associated Jacobian matrix is:

$$\begin{pmatrix} -b+u & \dfrac{(-b+u)\alpha}{h} \\ 0 & -u-\alpha+\left(c+\dfrac{m(b-u)}{h}\right)v \end{pmatrix} \tag{A2}$$

The first eigenvalue, $\lambda_1 = -b+u$, is negative. Stability therefore depends upon the other eigenvalue, $\lambda_2 = -u-\alpha+(c+m(b-u)/h)v$, having negative sign. This condition can be expressed as:

$$v < \frac{u+\alpha}{c+(m(b-u)/h)} = v_t \tag{A3}$$

Thus, if $v > v_t$ then the parasite is able to invade the uninfected host population. Note that increasing either c or m will reduce v_t.

(3) The parasite-driven extinction equilibrium (0,ρ_E) is defined by $N = 0$ and $p_E = 1-b/(vc-\alpha)$. The equilibrium is feasible provided:

$$vc > b+\alpha \tag{A4}$$

The associated Jacobian matrix is:

$$\begin{pmatrix} -u-\alpha+\dfrac{bcv}{-\alpha+cv} & 0 \\ \left(1+\dfrac{b}{\alpha-cv}\right)\left(h+\dfrac{bmv}{-\alpha+cv}\right) & b+\alpha-cv \end{pmatrix} \tag{A5}$$

The second eigenvalue is given by $\lambda_2 = (b+\alpha)-cv$, which is negative provided the relevance criterion A4 is satisfied. The other eigenvalue is $\lambda_1 = -u-\alpha+bcv/(-\alpha+cv)$. The condition for a stable equilibrium can therefore be expressed as:

$$v > \frac{\alpha(u+\alpha)}{c(\alpha-b+u)} = v_c \tag{A6}$$

Note that Eq. A6 requires that $\alpha > b-u = r$.

(4) The endemic equilibrium (N*,p*) is defined by

$$N^* = \frac{-\Phi_c + \sqrt{\Phi_c^2 + 4vmh\theta_c}}{2vmh},$$
$$p^* = \frac{\Phi_t - \sqrt{\Phi_t^2 - 4vm\alpha\theta_t}}{2vm\alpha} \tag{A7}$$

Here $\Phi_c = v(ch+m\alpha-m(b-u))$, $\Phi_t = v(ch+m\alpha+m(b-u))$, $\theta_c = vcb-vcu+\alpha(\alpha+u)-vc\alpha$ and $\theta_t = vch-h\alpha+vmb-vmu-uh$. Assuming $r = b-u > 0$ we know that $\Phi_t > 0$. For $p^* > 0$ we therefore require

$\theta_t > 0$, which is the same as $v > v_t$ (Eq. A3). To obtain $N^* > 0$ there are two possibilities. If $\alpha > r$ then $\Phi_c > 0$ and we require $\theta_c > 0$, which is the same as $v < v_c$ (Eq. A6). If $\alpha < r$ then $\theta_c > 0$ is always true and so $N^* > 0$. Therefore $(N,p) = (N^*,p^*)$ is feasible if:

$$v_t < v < v_c \tag{A8}$$

The associated Jacobian matrix is

$$\begin{pmatrix} -hN^* & -\alpha N^* \\ p^*(vm(1-p^*)+h & -p^*v(c+mN^*)+\alpha p^* \end{pmatrix} \tag{A9}$$

It can be shown that this matrix has negative trace and positive determinant, provided $0 < N^* < N_k$ and $0 < p^* < 1$. Thus, whenever the equilibrium (N^*,p^*) is feasible it is also stable. This requires that $v_t < v < v_c$.