

Interactions between frequency-dependent and vertical transmission in host–parasite systems

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SUMMARY

We investigate host–pathogen dynamics and conditions for coexistence in two models incorporating frequency-dependent horizontal transmission in conjunction with vertical transmission. The first model combines frequency-dependent and uniparental vertical transmission, while the second addresses parasites transmitted vertically via both parents. For the first model, we ask how the addition of vertical transmission changes the coexistence criteria for parasites transmitted by a frequency-dependent horizontal route, and show that vertical transmission significantly broadens the conditions for parasite invasion. Host–parasite coexistence is further affected by the form of density-dependent host regulation. Numerical analyses demonstrate that within a host population, a parasite strain with horizontal frequency-dependent transmission can be driven to extinction by a parasite strain that is additionally transmitted vertically for a wide range of parameters. Although models of asexual host populations predict that vertical transmission alone cannot maintain a parasite over time, analysis of our second model shows that vertical transmission via both male and female parents can maintain a parasite at a stable equilibrium. These results correspond with the frequent co-occurrence of vertical with sexual transmission in nature and suggest that these transmission modes can lead to host–pathogen coexistence for a wide range of systems involving hosts with high reproductive rates.

1. INTRODUCTION

Theoretical studies of host–parasite dynamics have recognized a fundamental distinction between parasites transmitted as a function of the absolute density of infected hosts (density dependent) versus parasites transmitted as a function of the frequency of infected hosts in the population (frequency dependent). Density-dependent parasites are characterized by a threshold density of hosts below which the parasite cannot persist, and the ability of the parasite to regulate the host population in the absence of any external density-dependent regulation (Anderson & May 1978, 1991). In contrast, parasites characterized by frequency-dependent transmission are predicted to show no critical host density threshold and no regulation of the host population without density-dependent constraints on host birth or death rates (Getz & Pickering 1983). Both vector and sexually transmitted parasites are expected to follow a more frequency-dependent transmission function, whereas parasites shed by the host into the environment are characterized by density-dependent transmission (Anderson & May 1991; Antonovics *et al.* 1995).

A second dichotomy in host–parasite systems occurs between horizontally and vertically transmitted parasites (Fine 1975; Anderson & May 1981).

This difference is based on whether the parasite moves between parent and offspring generations (vertical), or between two members of a population other than a parent–offspring pair (horizontal). A parasite transmitted by a uniparental vertical route cannot persist in a population if infected hosts suffer any fitness costs. Models of vertically transmitted parasites must therefore include some form of horizontal or biparental vertical transmission to achieve host–pathogen coexistence (Fine 1975; Lipsitch *et al.* 1995).

The combination of frequency-dependent and vertical transmission is a feature shared by many diseases. For example, as a consequence of the infection site of sexually transmitted parasites, occasional vertical transmission from mother to offspring occurs. Many human venereal diseases, such as syphilis, gonorrhoea and chlamydia, are often transferred vertically to infants (e.g. Murphy & Patamasucon 1984). The sexual transmission of *Chlamydia psittaci* in koalas may also involve transmission to offspring (Weigler *et al.* 1988). Several mosquito species have been intensively studied due to their role as vectors of vertebrate diseases, and many researchers have documented the combination of vertical and sexual virus transmission within mosquito populations (Nayar *et al.* 1986; Rosen 1987; Tesh & Modi 1987; Rosen *et al.* 1989; Gonzalez *et al.* 1992). These studies sug-

gest that vertical and sexual transmission may be critical to disease maintenance in the vector population in the absence of the vertebrate hosts. In plants, the combination of pollen (sexual) and seed or seedling (vertical) transmission has also been reported for many species (reviewed by Mandehar & Gill 1984; Mink 1993).

Parasite transmission between vectors and primary hosts may be characterized by a frequency-dependent function (Anderson 1981; Antonovics *et al.* 1995) and for many vector species also involves vertical transmission (reviewed by Yuill 1986). While these diseases involve more complex dynamics than the case of a single frequency-dependent transmission function, models incorporating frequency-dependent and vertical transmission may provide a first step toward understanding such systems.

A unique combination of transmission routes involving direct maternal transmission and indirect paternal transmission through pollen or infected sperm has been reported for systems involving both plant and arthropod hosts (Thomson 1958; Seecof 1968; Kellen & Lindegren 1971; Rosen 1987; Mink 1993). In monarch butterflies, *Ophryocystis elektroscirrha*, spores are usually passed maternally, but spores transferred by males during mating can infect the offspring of healthy females (Leong *et al.* 1992; S. Altizer, unpublished data). In such situations, both parents transmit the pathogen vertically, but adults are not directly infected by other adults.

While diseases expected to follow a frequency-dependent horizontal transmission function are commonly observed in nature, previous theoretical analyses have shown that the conditions required for such a parasite to invade and persist in a host population are relatively restricted (Getz & Pickering 1983; Thrall *et al.* 1993). In particular, parasite persistence requires that the host's birth rate always be less than the per-contact transmission rate of the disease. Since the latter rate must be less than one, this condition will only be met for hosts with low fecundity, such as large vertebrates (Thrall *et al.* 1993). If density-dependent mortality acts only on juveniles, then for sexually transmitted diseases the infected class is not subject to this density-dependent control. Thrall *et al.* (1993) showed that this can also extend the conditions for coexistence between hosts and frequency-dependent parasites. However, such a result does not apply to parasites with combined vertical and horizontal transmission because both juveniles and adults can acquire the disease.

Given the frequent co-occurrence of vertical and frequency-dependent horizontal transmission, and the restricted conditions for coexistence for either route alone, we address how vertical transmission changes the conditions for the invasion and persistence of parasites with horizontal frequency-dependent transmission. As a natural extension of pre-existing models, we first investigate host-pathogen dynamics for parasites with frequency-dependent horizontal transmission and imperfect vertical transmission. Using computer simulations, we explore competition between parasite strains with

and without vertical transmission to investigate conditions that might favour the evolution of combined vertical and frequency-dependent transmission. Finally, we build upon Fine's (1975) observations concerning biparental vertical transmission by presenting an analytical model that incorporates purely vertical transmission via both parents. By comparing the observed dynamics to previously analysed systems with horizontal transmission alone, we suggest underlying reasons why vertical transmission is often observed in frequency-dependent host-parasite systems.

2. MODEL DEVELOPMENT

The general form for a frequency-dependent host-parasite model was described by Getz & Pickering (1983) as

$$\frac{dS}{dt} = S \left(a - b - \frac{\beta I}{N} \right) + I(a + r), \quad (1)$$

$$\frac{dI}{dt} = I \left(\frac{\beta S}{N} - \alpha - b - r \right), \quad (2)$$

where S is the number of susceptible hosts, I is the number of infected hosts and N is total population size. Host birth and death rates are given by a and b , respectively, α is the disease-induced mortality rate and r is the recovery rate of infected hosts. β is the transmission coefficient which determines the rate at which healthy hosts acquire the disease via the frequency-dependent horizontal route. In the absence of host density-dependent birth or death rates, host-pathogen coexistence is not possible. If density dependence is incorporated as $b = b_0 + b_1 N$, the host population is maintained at carrying capacity $K = (a - b_0)/b_1$ in the absence of disease. The conditions for parasite invasion and coexistence are presented by Thrall *et al.* (1993) and are repeated in table 1a.

(a) *Model 1: frequency-dependent horizontal transmission with vertical transmission*

We extend the Getz & Pickering (1983) model by allowing imperfect uniparental vertical transmission such that

$$\frac{dS}{dt} = S \left(a - b_0 - b_1 N - \frac{\beta I}{N} \right) + I(1 - v)a, \quad (3)$$

$$\frac{dI}{dt} = I \left(\frac{\beta S}{N} - \alpha - b_0 - b_1 N - r \right) + Iva, \quad (4)$$

where v is the probability of vertical transmission and all other parameters are as described above. As in equations (1) and (2), host and pathogen coexistence is not possible without external density-dependent host regulation. The addition of uniparental vertical transmission does not alter the general structure of the equations, and the relative proportions of infected and susceptible hosts at equilibrium are unchanged from the case of sexual trans-

Table 1. *The conditions allowing for parasite invasion of a population and overall host-parasite coexistence for models described by (a) Getz & Pickering (1983) and Thrall et al. (1993); (b)–(d) this study; (e) Fine (1975); and (f) this study*

(Conditions for coexistence are determined by combining the conditions for invasion with the requirement that the total population size at equilibrium is greater than zero and imply local stability. Symbols are as described in the text.)

transmission routes	characteristics of density-dependent host regulation	conditions for parasite invasion	conditions for host-parasite coexistence
(a) horizontal frequency-dependent	$b = b_0 + b_1N$	$\beta > a + \alpha + r$	$1 < \frac{\beta - b_0 - \alpha - r}{a - b_0} < \frac{\beta}{\alpha}$
(b) horizontal frequency-dependent + vertical	$b = b_0 + b_1N$	$\beta > (1 - v)a + \alpha + r$	$1 < \frac{\beta + av - b_0 - \alpha - r}{a - b_0} < \frac{\beta}{\alpha}$
(c) horizontal frequency-dependent	$a = a_0 - a_1N$	$\beta > b + \alpha + r$	$0 < \frac{\beta - b - \alpha - r}{a_0 - b} < \frac{\beta}{\alpha}$
(d) horizontal frequency-dependent + vertical	$a = a_0 - a_1N$	$\beta > (1 - v)b + \alpha + r$	$v < \frac{\beta + a_0v - b - \alpha - r}{a_0 - b} < \frac{\beta}{\alpha}$
(e) vertical maternal transmission or vertical transmission for asexual host	$b = b_0 + b_1N$ or $a = a_0 - a_1N$		coexistence not possible
(f) frequency-dependent vertical paternal + vertical maternal transmission	$b = b_0 + b_1N$	$\beta \left(av + \frac{av(1 - v)b_1}{\frac{1}{2}a - b_0} \right) > 2\alpha + a(1 - v)$	$1 + \frac{\beta \frac{1}{2}av(1 - v)b_1}{(\frac{1}{2}a - b_0)^2} < \frac{\beta \frac{1}{2}av + \frac{1}{2}av - b_0 - \alpha}{\frac{1}{2}a - b_0} < \frac{\beta}{\alpha} \frac{1}{2}av^2$

mission only (Thrall *et al.* 1993):

$$\frac{I^*}{N^*} = \frac{a - b_0 - b_1N^*}{\alpha}, \tag{5}$$

$$\frac{S^*}{N^*} = \frac{\alpha - a + b_0 + b_1N^*}{\alpha}. \tag{6}$$

However, the introduction of uniparental vertical transmission does alter the internal equilibria of the model and hence the conditions for host-parasite coexistence. We first derive the conditions for parasite invasion of a susceptible host population at equilibrium when $b = b_0 + b_1N$ by setting $N = S = (a - b_0)/b_1$ and solving for $dI/dt > 0$. When vertical transmission is included (equations (3) and (4)), the horizontal transmission coefficient (β) can be reduced by the amount av and parasite invasion is still possible (table 1b).

Several dynamical outcomes of this model are possible (figure 1a–c). For $a < b_0$, the host population size declines uniformly from all starting conditions ($N^* \rightarrow 0$). For $a > b_0$, two equilibria are possible. In the absence of the parasite, or with a non-virulent parasite ($\alpha = 0$), the host population reaches an equilibrium carrying capacity of $N^* = (a - b_0/b_1)$. For $\alpha > 0$, a single internal equilibrium exists where

$$N^* = \frac{\alpha(b_0 + \alpha + r - av) - \beta(\alpha + b_0 - a)}{b_1(\beta - \alpha)}. \tag{7}$$

Stable host-parasite coexistence requires $N^* > 0$ which occurs when

$$b_0 < \frac{\alpha^2 + a\beta - \alpha\beta + \alpha r - \alpha av}{\beta - \alpha}. \tag{8}$$

This inequality, combined with the requirements for parasite invasion, defines the overall conditions for host-parasite coexistence (table 1b). These conditions are identical to the requirement that both eigenvalues of the Jacobian matrix evaluated for the internal equilibrium be negative. Thus, parameter values that allow for the existence of the internal equilibrium also imply local stability.

Previous analyses of frequency-dependent parasite transmission have not considered a host population regulated by density-dependent fecundity. Analysis of equations (1) and (2) when $a = a_0 - a_1N$ shows that the assumption of density-dependence acting on host birth rates alters the conditions for parasite invasion of a host population (table 1c), but does not affect the conditions under which $N^* > 0$ (given in Thrall *et al.* 1993). As a result, the conditions for host-parasite coexistence when only sexual transmission is possible are broader in a host population regulated by density-dependent fecundity (table 1c) compared to a population regulated by density-dependent mortality (table 1a). Inspection of the Jacobian matrix shows that the internal equilibrium for this system is locally stable when it exists.

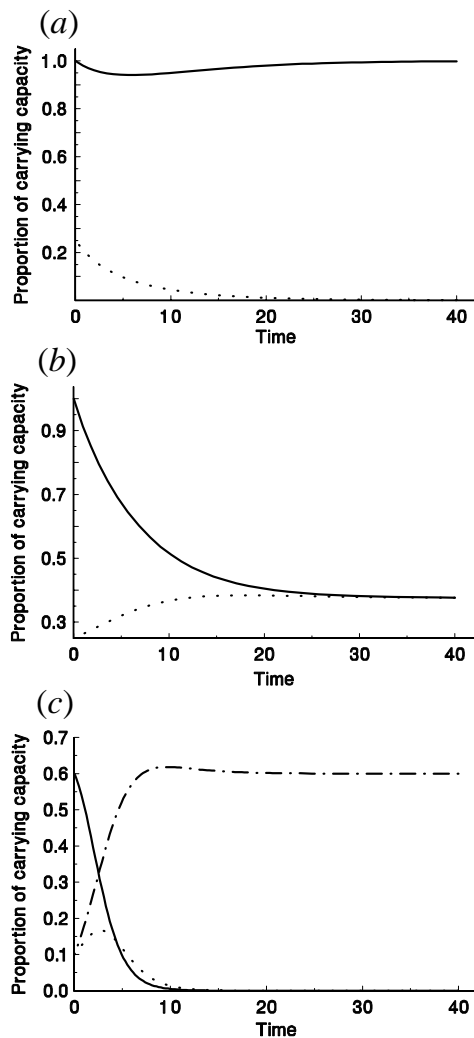


Figure 1. Simulation results of model 1 illustrating different dynamical outcomes of the host–parasite interaction. Host population sizes are shown as proportions relative to the carrying capacity of the host in the absence of disease. Solid lines represent susceptible hosts and dashed lines represent infected hosts. (a) When transmission opportunities are low, the parasite cannot invade the host population ($a = 0.4$, $b_0 = 0.2$, $b_1 = 0.05$, $v = 0.3$, $\beta = 0.2$, $\alpha = 0.1$ and $r = 0$). (b) For moderate transmission rates or moderate-to-low virulence, the parasite can invade and persist at less than 100% prevalence ($a = 0.4$, $b_0 = 0.2$, $b_1 = 0.05$, $v = 0.5$, $\beta = 0.5$, $\alpha = 0.1$ and $r = 0$). (c) When vertical and horizontal transmission rates are high, the parasite can achieve 100% infection at equilibrium. Here we show the contribution of uniparental vertical versus horizontal frequency-dependent transmission to the total number of infected hosts in a population over time. Solid line represents susceptible hosts, dotted line is hosts infected via horizontal route and dot-dash-dot line is hosts infected via vertical route. Parameters used were $a = 0.4$, $b_0 = 0.2$, $b_1 = 0.05$, $v = 1$, $\beta = 0.8$, $\alpha = 0.1$ and $r = 0$.

The form of density-dependent host regulation also influences the conditions for coexistence when vertical transmission is possible. Including uniparental vertical transmission, when a host is regulated by density-dependent fecundity, relaxes the conditions for parasite invasion more than any other case we examined (table 1*a–c* versus 1*d*). However, the con-

ditions for N^* to be positive are still equivalent to equation (8). Combining these two inequalities gives the overall conditions for host–parasite coexistence (table 1*d*), which are identical to the density-dependent mortality case (table 1*b*) when $v = 1$. If these conditions are satisfied, then the eigenvalues of the Jacobian matrix must also be negative and thus the equilibrium is locally stable.

When vertical and frequency-dependent horizontal transmission are combined, 100% disease prevalence is possible (figure 1*c*). This result was also observed by Lipsitch *et al.* (1995) for combinations of vertical and density-dependent horizontal transmission, but cannot be achieved by vertical or horizontal routes alone. The assumption of 100% prevalence for a model with frequency-dependent transmission alone implies $S^* = 0$ and the host’s population dynamics are then described by equation (2). At equilibrium, $dI/dt = 0$, and $N^* = (-b_0 - \alpha - r)/b_1$, thus violating the requirement that the host’s population size be greater than zero. However, assuming 100% prevalence for combined vertical and frequency-dependent horizontal transmission implies $N^* = (va - b_0 - \alpha - r)/b_1$. Thus, 100% infection occurs when the internal equilibrium exists and

$$va > b_0 + \alpha + r. \quad (9)$$

(b) Competition between parasite strains with and without vertical transmission

We used numerical simulations to examine competition between two parasite strains within a single host population. Both strains were capable of frequency-dependent horizontal transmission, but the second strain was also vertically transmitted. We assumed that susceptible hosts could be infected with one or both strains simultaneously and that superinfected hosts transmitted strain 1 at the horizontal rate β_1 and strain 2 at the horizontal rate β_2 plus the vertical rate v . The mathematical expressions and all parameters follow from equations (1)–(4) above:

$$\begin{aligned} \frac{dS}{dt} = & S(a - b_0 - b_1N) \\ & - \frac{S}{N}(\beta_1 I_1 + \beta_2 I_2 + (\beta_1 + \beta_2)\frac{1}{2}I_3) \\ & + a(I_1 + (I_2 + I_3)(1 - v)), \end{aligned} \quad (10)$$

$$\begin{aligned} \frac{dI_1}{dt} = & \frac{\beta_1 S}{N}(I_1 + \frac{1}{2}I_3) - I_1(\alpha + b_0 + b_1N) \\ & - \frac{I_1 I_2 \beta_2}{N}, \end{aligned} \quad (11)$$

$$\begin{aligned} \frac{dI_2}{dt} = & \frac{\beta_2 S}{N}(I_2 + \frac{1}{2}I_3) - I_2(\alpha + b_0 + b_1N) \\ & - \frac{I_1 I_2 \beta_1}{N} + va(I_2 + I_3), \end{aligned} \quad (12)$$

$$\frac{dI_3}{dt} = (\beta_1 + \beta_2)\frac{I_1 I_2}{N} - I_3(\alpha + b_0 + b_1N). \quad (13)$$

We simulated these dynamics for all possible combinations of β_1 , β_2 and v at 0.05 intervals ranging from 0 to 1. Each simulation was iterated for 2000 generations. The following sets of additional parameters were used: $b_0 = 0.2$; $b_1 = 0.05$; $\alpha = \{0, 0.1, 0.5\}$;

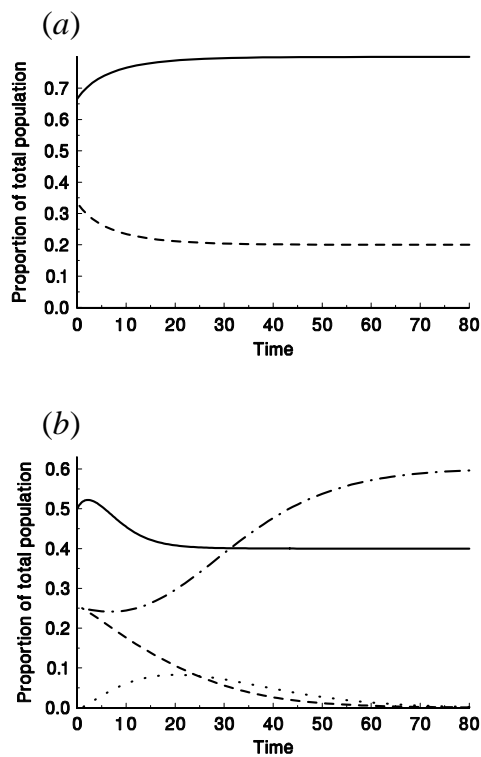


Figure 2. (a) Equilibrium infected and susceptible host densities for a parasite that is transmitted by a frequency-dependent horizontal route only; and (b) competition between two disease strains with and without vertical transmission. Proportions are shown relative to total population density, with lines representing susceptible hosts (solid line), hosts infected with the frequency-dependent strain (strain 1; dashed line), hosts infected with the frequency-dependent/vertical strain (strain 2; dot-dash-dot line) and hosts with both strains (dotted line). Parameter values for both simulations were $a = 0.4$, $b_0 = 0.2$, $b_1 = 0.05$, $\beta_1 = \beta_2 = 0.6$, $\alpha = 0.1$, $v = 0.5$ and $r = 0$. Parameters were chosen such that strain 1 could persist in the population at equilibrium.

and $a = \{0.4, 0.8, 1.2\}$. Values of $\alpha < 0.5$ were chosen based on virulence characters of sexually transmitted diseases presented in Lockhart *et al.* (1996). Given parameter ranges for which strain 1 alone can persist in a host population, the addition of strain 2 can drive the parasite lacking vertical transmission to extinction (figure 2). This was always the case for equal horizontal transmission rates ($\beta_1 = \beta_2$ for any $v > 0$) and occurred for all chosen values of disease-induced mortality.

Assuming a cost to vertical transmission in strain 2 (so that β_2 decreases as v increases) increases the range over which both strains coexist (figure 3). In order for strain 2 to invade and persist, v must be at least as large as the difference between the two horizontal rates ($v \geq \beta_1 - \beta_2$). The equilibrium abundance of strain 1 then decreased as v increased above this condition, leading to a loss of strain 1 for high rates of vertical transmission (figure 3).

As birth rates increased, invasion and persistence of both strains were restricted to a smaller parameter space. Substantial vertical transmission allowed strain 2 to persist when birth rates were high ($a > 1$),

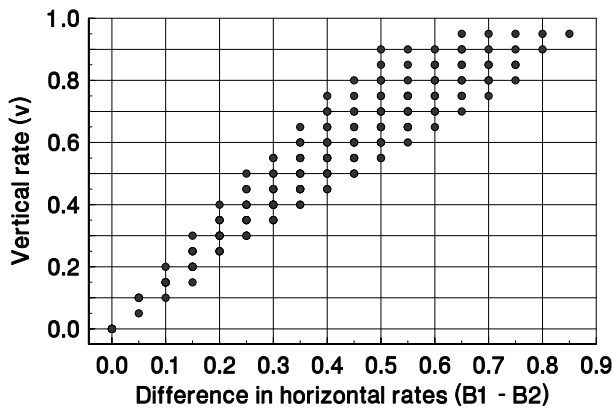


Figure 3. Range of transmission parameters that allow for coexistence between strains 1 and 2 when vertical transmission imposes a cost to horizontal transmission in strain 2. Below the range of the dotted area (where $v < \beta_1 - \beta_2$) strain 2 cannot persist at equilibrium. Above the dotted range, only strain 1 is present at equilibrium. For each of the simulations, parameter values were $a = 0.4$, $b_0 = 0.2$, $b_1 = 0.05$, $\alpha = 0.1$ and $r = 0$. For strain 1 alone to invade, $\beta_1 > 0.5$.

a result not possible for strain 1. When we increased disease-induced mortality from 0.1 to 0.5, the upper range of transmission parameters (β_1 , β_2 and v high) led to total extinction of host and parasite, thus further restricting the conditions for which both strains coexist.

(c) *Model 2: vertical maternal transmission and indirect vertical paternal transmission*

Sexually reproducing host species that transmit a parasite biparentally may experience dynamics similar to those described above as a consequence of transmission from the male parent via a combined sexual/vertical route. Fine (1975) demonstrated that host-parasite coexistence is possible given biparental vertical transmission, but did not present a generalized model. We consider biparental vertical transmission as a special case of sexual and vertical transmission in which females that mate with infected males can pass paternally acquired parasites to offspring without becoming infected themselves.

We use a frequency-dependent function to describe the probability of matings between healthy and infected adults and include a combined sexual/vertical transmission probability for the paternal contribution. Susceptible hosts are augmented by births from healthy females that avoid paternal transmission to their offspring and by infected females that avoid both maternal and paternal transmission. Infected hosts are augmented by births from infected females that pass the disease either maternally or paternally and by susceptible females whose offspring acquire the disease paternally. Assuming a 50:50 sex ratio at birth, this system can be described by

$$\frac{dS}{dt} = \frac{1}{2}a \left[S \left(1 - \frac{\beta I v}{N} \right) + I(1 - v) \left(1 - \frac{\beta I v}{N} \right) \right] - S(b_0 + b_1 N), \tag{14}$$

$$\frac{dI}{dt} = \frac{1}{2}a \left[Iv + I(1-v) \left(\frac{\beta Iv}{N} \right) + S \left(\frac{\beta Iv}{N} \right) \right] - I(b_0 + b_1N + \alpha). \tag{15}$$

Parameters a , b_0 and b_1 are as previously defined. Infected females transmit the parasite to offspring at probability v , while males transmit parasites to offspring at probability βv , or the product of sexual and maternal transmission. The sum $v + (1-v)\beta v$ describes the transmission probability for two infected parents. For simplicity, we assume that host recovery does not occur (i.e. $r = 0$). Because we are describing a sexually reproducing population, the host birth rate equals $\frac{1}{2}a$ (i.e. only females reproduce).

Conditions for parasite invasion are presented in table 1*f*. Host density has a single internal equilibrium where

$$N^* = \frac{\alpha(b_0 + \alpha - \frac{1}{2}av) + \frac{1}{2}av\beta(\frac{1}{2}av - b_0v - \alpha)}{b_1(\frac{1}{2}a\beta v^2 - \alpha)}. \tag{16}$$

The relative equilibrium density of infected and susceptible individuals in the population is the same as for model 1, with the exception that the host birth rate a is replaced by $\frac{1}{2}a$. Host-parasite coexistence again requires $N^* > 0$, which occurs when

$$b_0 < \frac{(\frac{1}{2}a\beta v - \alpha)(\frac{1}{2}av - \alpha)}{\frac{1}{2}a\beta v^2 - \alpha}. \tag{17}$$

Eigenvalues of the Jacobian matrix show that local stability also requires that $\frac{1}{2}a(\beta v^2) > \alpha$. Finally, 100% disease prevalence can result from model 2; here we assume $S^* = 0$ and $N^* = I^*$ and solve equation (15) for N^* . In this case, $N^* > 0$ when

$$\frac{1}{2}a(v + (1-v)\beta v) > \alpha + b_0. \tag{18}$$

3. DISCUSSION

(a) *Model 1: effects of vertical transmission on host-parasite coexistence*

Previous theoretical studies of frequency-dependent horizontal transmission demonstrate that host-parasite coexistence is limited to a relatively restricted set of conditions precluding hosts with high reproductive rates (i.e. greater than one (Getz & Pickering 1983)). Further theoretical and empirical work shows that conditions for coexistence may be extended when density dependence does not act on the infected class (Thrall *et al.* 1993), but such conditions exclude the possibility of vertical transmission by assuming that only adults acquire the disease. We showed here that the ability to be transmitted vertically enables a parasite to successfully invade a host population for a broader range of conditions than when vertical transmission is not possible and allows coexistence with host species with reproductive rates greater than one.

Previous analyses of frequency-dependent parasite transmission have not examined the case in which the host population is regulated by density-dependent fecundity. We showed that a host population regulated by density-dependent fecundity rather than

mortality can be more easily invaded by a frequency-dependent transmission route. This results from the fact that as a host population regulated by mortality approaches carrying capacity, individuals continue to produce offspring at a constant rate. The population at carrying capacity therefore has a shorter relative generation time and healthy offspring enter the population at a faster rate than a host regulated by fecundity. This result, combined with the observation that the form of density dependence does not alter the equilibrium population densities, allows host-parasite coexistence to occur for a broader range of conditions when a host is regulated by density-dependent fecundity (table 1*a* versus 1*c*).

The form of density-dependent host regulation also interacts with the influence of vertical transmission on the conditions for host-parasite transmission. The addition of vertical transmission to a system regulated by density-dependent fecundity allows a parasite to invade more easily than any other case considered. However, the overall conditions for coexistence are more restricted when vertical transmission is possible than when the parasite is transmitted by a frequency-dependent route alone (table 1*c* versus 1*d*). For combined sexual and vertical transmission, overall coexistence conditions are broader for hosts regulated by density-dependent fecundity versus mortality (table 1*b* versus 1*d*). The magnitude of this difference depends on the efficiency of vertical transfer, such that in the extreme case of perfect vertical transmission, the conditions for coexistence are identical for both types of density-dependent host regulation.

In all situations, the addition of vertical transmission reduces the total equilibrium population size as the vertical transmission rate increases from zero to one. However, for any given equilibrium population size, the ratio of infected to susceptible hosts in the population remains unchanged for systems with and without vertical transmission. While vertical transmission does not directly increase the ratio of infected to susceptible hosts for a given N^* , selection for vertical transmission may result from the increased range of conditions for which the parasite can invade the population (table 1*a* versus 1*b* and table 1*c* versus 1*d*). This is especially important for hosts with high birth rates (greater than 1) and may also be important in highly dynamic systems where local extinctions and re-introductions of the parasite are occurring within a host meta-population.

We found that the entire host population can be infected in a system with vertical and frequency-dependent horizontal transmission. This occurs when host birth rates and vertical transmission rates are relatively high and host mortality and parasite-induced mortality are relatively low (equation (9)). Such a combination of high birth rates into the infected class and low mortality rates for diseased hosts leading to 100% infection is also observed when vertical and density-dependent horizontal transmission are combined (Lipsitch *et al.* 1995). It is important to

note that for cases where equilibrium disease prevalence is extremely high, most new infections will result from vertical transmission, although the contribution of horizontal transmission is critical to disease establishment (figure 1c).

Competitive interactions between two parasite strains showed that a wide range of conditions favour vertically transmitted parasites over strains with only frequency-dependent horizontal transmission. When both strains were transmitted at the same horizontal rate, any degree of vertical transmission in strain 2 led to the eventual extinction of strain 1, even in the absence of disease-induced mortality. If vertical transmission imposes a cost on the rate of horizontal transmission, both strains can coexist over a wider range of parameters; this range declines with increasing host birth rates. High birth rates (near to or in excess of one) always resulted in a loss of the strain without vertical transmission, whereas the vertically transmitted strain could still persist. It is therefore likely that the degree of vertical transmission observed in host–parasite systems may result from competitive exclusion between parasite strains in addition to the invasion advantages of vertical transmission.

Sexual versus vertical transmission modes may at times exert conflicting selective pressures on pathogen characters such as disease-induced sterility. Sexually transmitted diseases are predicted to increase host sterility when this leads to increased mating attempts between hosts (Lockhart *et al.* 1996). However, suppression of host fecundity lowers the transmission rates of vertically acquired pathogens. Such a situation is illustrated by koala populations infected with *Chlamydia psittaci*. Females that do not mate successfully may undergo two or three ovulation cycles during the breeding season (Handasyde *et al.* 1990). Since koalas typically bear only one offspring per breeding season (Eberhard 1978), *Chlamydia* strains that induce sterility in females may create more transmission opportunities than strains that do not decrease host fecundity. Selection against disease-induced sterility would be more likely in hosts with higher birth rates and thus there would be greater potential for vertical transmission.

(b) Model 2: biparental vertical transmission in a sexually reproducing host

The observation of indirect transmission of *O. elektroscirra* spores from male monarch butterflies to offspring via mating (S. Altizer, unpublished data) prompted our consideration of biparental vertical transmission. For sexually reproducing hosts, transmission from the male parent to offspring allows for host–parasite coexistence in the absence of any direct horizontal transmission between adults in the population (table 1f). As demonstrated conceptually by Fine (1975), this differs from the uniparental vertical transmission case in which coexistence is not possible given any degree of parasite virulence.

Model 2 predicts host–parasite coexistence for high host reproductive rates, low parasite-induced mortal-

ity and a large degree of vertical transmission (table 1f). These conditions are consistent with current data regarding virulence and transmission rates of *O. elektroscirra*. Although severely infected monarch butterflies die shortly after emergence, mean life-spans for infected and healthy adults differ by only a few days (Leong *et al.* 1992; S. Altizer, unpublished data). Maternal transmission rates by heavily infected females measured in the laboratory approach 100% and females that mate with infected males can result in up to 75% progeny with heavy parasite loads (S. Altizer, unpublished data).

As in systems combining horizontal and uniparental vertical transmission (model 1, Lipsitch *et al.* 1995), a model incorporating both maternal and paternal vertical transmission (equations (14) and (15)) predicts that 100% disease prevalence is possible. This outcome requires high host birth rates and vertical transmission rates relative to host mortality and virulence. Interestingly, extremely high infection frequencies (approaching 100%) have been recorded for non-migratory monarch populations infected by *O. elektroscirra* in southern Florida (S. Altizer, unpublished data) and Hawaii (Leong *et al.* 1997).

This overall analysis indicates that the evolution of vertical transmission in a sexually or vector transmitted disease is most likely related to (i) the increased ability of a vertically transmitted parasite to invade a host population; and (ii) the ability of parasites with vertical transmission to outcompete parasite strains capable of only horizontal transmission. The magnitude of this effect depends on the host's birth rate, as parasite invisibility is increased by a factor that depends on vertical transmission through new births. Thus, vertical parasite transmission is not expected to be as common among host organisms with low reproductive rates. Examples of this transmission combination in larger vertebrates come from hosts such as humans and koalas where vertical transmission appears to occur primarily as a side effect of infected genital tissues, while a much larger number of examples involve host species with high reproductive rates (e.g. Rosen *et al.* 1989; Leong *et al.* 1992; Mink 1993). While the important transmission routes of many parasites remain undocumented, these analyses suggest that vertical transmission will occur in combination with horizontal frequency-dependent transmission for a wide range host–parasite systems involving hosts with high reproductive rates.

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