

Interference and the persistence of vertically transmitted parasites

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Abstract

Given their ubiquity in nature, understanding the factors that allow the persistence of multiple enemies and in particular vertically transmitted parasites (VTPs) is of considerable importance. Here a model that allows a virulent VTP to be maintained in a system containing a host and a horizontally transmitted parasite (HTP) is analysed. The method of persistence relies on the VTP offering the host a level of protection from the HTP. The VTP is assumed to reduce the HTPs ability to transmit to the host through ecological interference. We show that VTPs are more likely to persist with HTPs that prevent host reproduction than with those that allow it. The VTP persists more easily in r -selected hosts and with highly transmittable HTPs. As the level of protection through interference increases the densities of the host also increase. We also show that VTPs when they do persist tend to stabilise the host population cycles produced by free-living HTPs. The study raised questions about persistence of diseases through interactions with others, and also the stabilising effects of VTPs on dynamical systems in a biological control context.

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1. Introduction

Despite the fact that many parasites are transmitted vertically from mother to offspring (Goulson and Cory, 1995; Smits and Vlak, 1988), the persistence of virulent, purely vertically transmitted parasites (VTPs) in nature runs counter to simple ecological theory. This classical theory suggests that a degree of horizontal transmission, in addition to vertical transmission, is also required to enable persistence (Busenberg et al., 1983; Lipsitch et al., 1995; Regniere, 1984). It is clear that vertical transmission alone is not enough to maintain a parasite in a self-limited host population, if infection either lowers birth rate (Regniere, 1984) or increases death rate (Busenberg et al., 1983; Lipsitch et al., 1995). When a single parasite does have vertical transmission in addition to horizontal transmission it increases parasite persistence (Anderson and May, 1981)

and affects the stability (Bonsall et al., 2005; Boots et al., 2003), but theory suggests that purely VTPs will not persist in nature without altering the hosts life history.

These theoretical results have raised the question of what mechanisms may allow the persistence of purely VTPs. VTPs are commonly found in male-killing bacteria, of the genera *Rickettsia* and *Wolbachia* (Hurst et al., 1999; Werren et al., 1994). These distort the sex ratio of the host population by reducing the number of males. The death of the males can enhance the fitness of the surviving brood members due to factors such as reduced intrabrood competition and inbreeding. Models have shown that this fitness compensation allows the parasite to persist despite the death of infected males and female fitness costs (Hurst, 1991, 1993). Another possibility is host metapopulation structure. A model showed that VTPs that reduce host fitness may persist within a metapopulation, if infected offspring are able to invade new patches (Saikkonen et al., 2002).

Here, we examine another mechanism by which a completely VTP may persist through an interaction with another parasite, in a host–parasite–parasite system.

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We consider in detail, the situation where a vertical infection gives the host interference protection against other natural enemies (Lipsitch et al., 1996). This interference can arise in a number of ways. Firstly the infected host's behaviour may be modified so that encounters with other enemies are reduced. One obvious example is where the hosts feeding rates are reduced due to VTP infection (Bellonick, 1996) which results in a reduction in the ingestion of microparasitic infective stages. In addition to behavioural changes, the VTP may offer protection by restricting the uptake or development/replication of a secondary horizontally transmitted parasite (HTP). Despite the relative paucity of multi-enemy studies, interference has been shown to occur in a number of multi-parasite interactions. For example, it has been reported that vertically transmitted bacterial symbionts in aphid hosts interfere with the development of the larvae of parasitic wasps (Oliver et al., 2003). Viruses have also been seen to have an effect on each other's intra-cell replication rate. In *Helicoverpa zea*, a slower killing virus (granulose virus) has been found to inhibit the replication rate of a more pathogenic NPV (nucleopolyhedrovirus) in the host, thereby increasing host fitness (Hackett et al., 2000). Although this occurs in two HTPs, it demonstrates a mechanism of possible viral interference. Rohani et al. (2003) described the interaction of fatal diseases, where infection by one pathogen removed possible susceptibles from the population during a quarantine period, thus preventing infection from another. Modelled in this way, ecological interference was shown to have a profound effect on the dynamics of the system, making multi-annual outbreaks of different infections out of phase. Thus the dynamics can only be explained by the interaction between the two infections and not simply looking at either one in isolation.

A related form of cross-immunity between one horizontally and one VTP has previously been modelled by Allen et al. (2003). In this model, however, the VTP also had a component of horizontal transmission and as such the model did not examine the role of protection in the coexistence and persistence of a purely VTP. Here we present a simpler model that shows how ecological protection can lead to the persistence of an exclusively VTP in a host–parasite system. We assume that hosts infected with the VTP have an increased death rate and lower fecundity, but also gain protection from the HTP in the form of a reduced transmission rate. We consider both directly transmitted and free-living forms of HTPs in order to examine the effect of the persistence of VTPs on host–parasite dynamics. In addition we compare HTPs that castrate their hosts with true parasites that allow reproduction from infected individuals (see Boots, 2004).

2. Models and analysis

The first model considers the density of the susceptible host population, X , the host population infected with the VTP, V , and the host population infected with the HTP, Y .

The HTP is assumed to be directly transmitted. The following model represents the dynamics:

$$\frac{dX}{dt} = (r - q(X + V + Y))X + (1 - p) \times (af - q(X + V + Y))V - \beta XY, \quad (1a)$$

$$\frac{dV}{dt} = p(af - q(X + Y + V))V - (\alpha_V + b)V - \delta\beta VY, \quad (1b)$$

$$\frac{dY}{dt} = (X + \delta V)\beta Y - (\alpha_Y + b)Y. \quad (1c)$$

Here, r (assumed > 0) is the intrinsic rate of growth of the host, equal to the birth rate a , minus the natural death rate b , with density dependence from the total host population acting on the birth rate via the crowding parameter, q . The birth rate of hosts infected with the VTP is reduced by a proportion $(1-f)$, and a proportion, p , of their offspring are born infected with the vertical parasite (therefore $(1-p)$ are born into the susceptible class). Those infected with the HTP are assumed not to reproduce as the parasite acts in a 'predatory' manner (we will relax this assumption later; see below). Both susceptible and vertically infected hosts are susceptible to horizontal infection, at rates βY and $\delta\beta Y$, respectively. We assume a process of super-infection so that there is no co-infection with the two parasites. The parameter $\delta \in [0,1]$ acts to reduce horizontal transmission so that vertically infected hosts are afforded a level of protection from the HTP. Here, $\delta = 0$ equates to total protection while $\delta = 1$ equates to none. The parameters α_V and α_Y are the additional death rates due to infection from the VTP and HTP, respectively.

The model, Eqs. (1a)–(1c), has three biologically relevant equilibria at $(X, V, Y) = (X_1, 0, 0)$, $(X_2, 0, Y_2)$ and (X_3, V_3, Y_3) . (Note: the trivial equilibrium at the origin is always unstable and equilibria of the form $(0, V, 0)$, $(X, V, 0)$ and $(0, V, Y)$ are only attainable when $p = 1$ and always unstable. We therefore do not consider these equilibria). The disease-free equilibrium $(X_1, 0, 0)$ is given by

$$(X_1, 0, 0) = \left(\frac{r}{q}, 0, 0\right). \quad (2)$$

This equilibrium is stable provided the following inequality holds:

$$\frac{r\beta}{q(\alpha_Y + b)} < 1. \quad (3)$$

This is equivalent to $R_0 < 1$, where R_0 is the reproductive ratio of the HTP (Anderson and May, 1981). The equilibrium $(X_2, 0, Y_2)$, which includes the host and the HTP, is given by

$$(X_2, 0, Y_2) = \left(\frac{\alpha_Y + b}{\beta}, 0, \frac{r\beta - q(\alpha_Y + b)}{\beta(\beta + q)}\right). \quad (4)$$

A necessary condition for stability is that the inequality in Eq. (3) is reversed, as this allows the HTP to invade and

coexist with the host. Stability also requires that the VTP is unable to invade (if inequality (5) below is reversed). The equilibrium (X_3, V_3, Y_3) is stable if the following inequality holds:

$$p[af - q(X_2 + Y_2)] > (\alpha_V + b) + \delta\beta Y_2. \tag{5}$$

Inequality (5) can be easily interpreted biologically. It represents the fact that, at the host–HTP equilibrium, net births to the VTP class must be greater than net losses (from natural death, death due to the VTP and from contracting the horizontally transmitted infection). If there is complete protection, $\delta = 0$, the inequality is

$$p[af - q(X_2 + Y_2)] > \alpha_V + b. \tag{6}$$

This is more easily satisfied than that inequality (5); however, it does not mean that V can always invade into the host–HTP equilibrium. Inequality (6) emphasises, rather intuitively, that VTPs with high vertical efficiency (high p) that do not significantly reduce host reproduction (high f) and have low virulence (low α_V) are most likely to coexist.

It is always easier for the VTP to coexist in species with low susceptibility to crowding, (low q) (Fig. 1a). There is a threshold level of the birth rate, below which the VTP cannot coexist (Fig. 1b). Above this threshold, as the birth

rate increases, the VTP can persist for relatively lower levels of protection against the HTP. This effect saturates as the birth rate increases. Productive hosts with high carrying capacities are therefore more likely to support the VTP. The capacity for persistence of the VTP responds to changes in the HTP transmission parameter, β , in a similar way as to changes in birth rate (Fig. 1c). Again there is a threshold level of transmission, below which the VTP does not persist. As the transmission rate of the HTP increases, the VTP can persist at lower levels of protection. Similarly, if the proportion, p , born with the VTP infection is above a threshold, then further increases in p allows the persistence of VTPs that offer lower levels of protection (Fig. 1d).

The level of protection offered by the VTP also alters the equilibrium values and therefore the proportions of the population in the different classes (Fig. 2). Both types of infected hosts (VTP and HTP) increase as the protection increases up to a threshold at which the protection becomes so large that there are very few individuals infected with the VTP becoming infected by the HTP (and therefore the HTP density decreases). The susceptible host density decreases with increasing protection until they are mostly produced due to the VTPs imperfect vertical transmission ($p < 1$). Due to protection the VTP experiences a lower

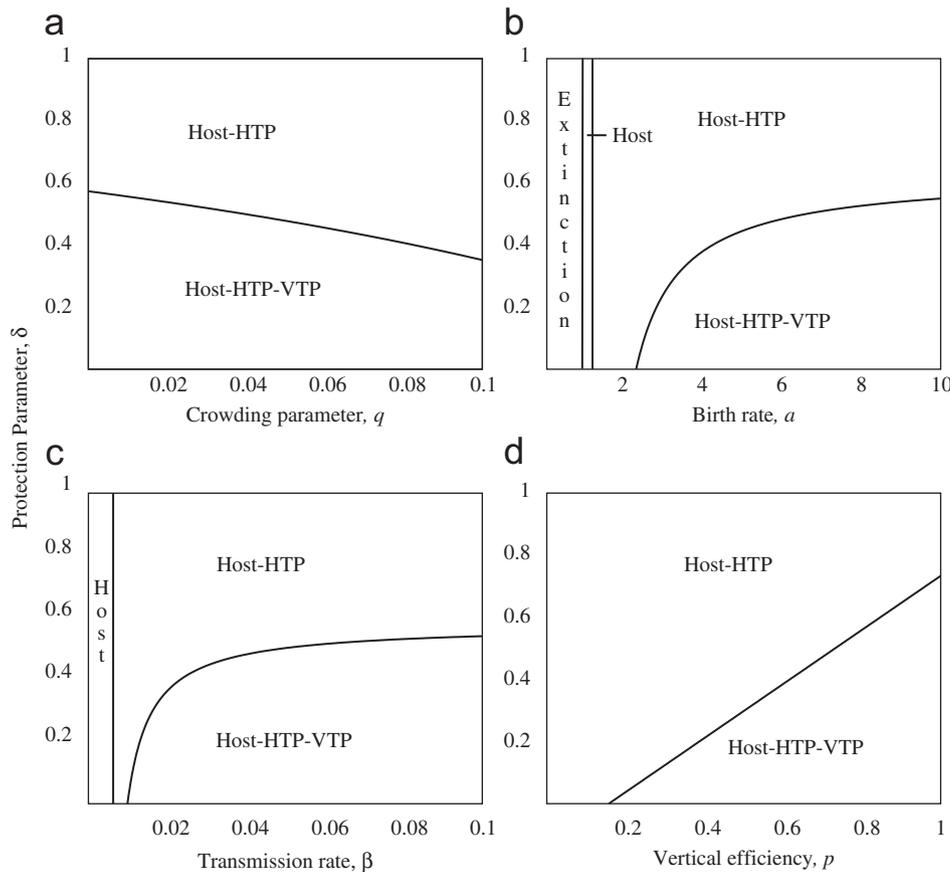


Fig. 1. The role of protection (δ) vs. susceptibility to crowding (q), birth rate (a), transmission rate (β) and vertical transmission efficiency (v) in coexistence of the natural enemies. Parameter space portraits indicating where the different population equilibria are stable for the model defined by Eqs. (1a)–(1c). Parameters used are $\beta = 0.2$; $f = 0.8$; $r = 10$; $q = 0.01$; $b = 1$; $p = 0.8$; $\alpha_v = 0.25$, $\alpha_y = 4$, unless varied in the plots.

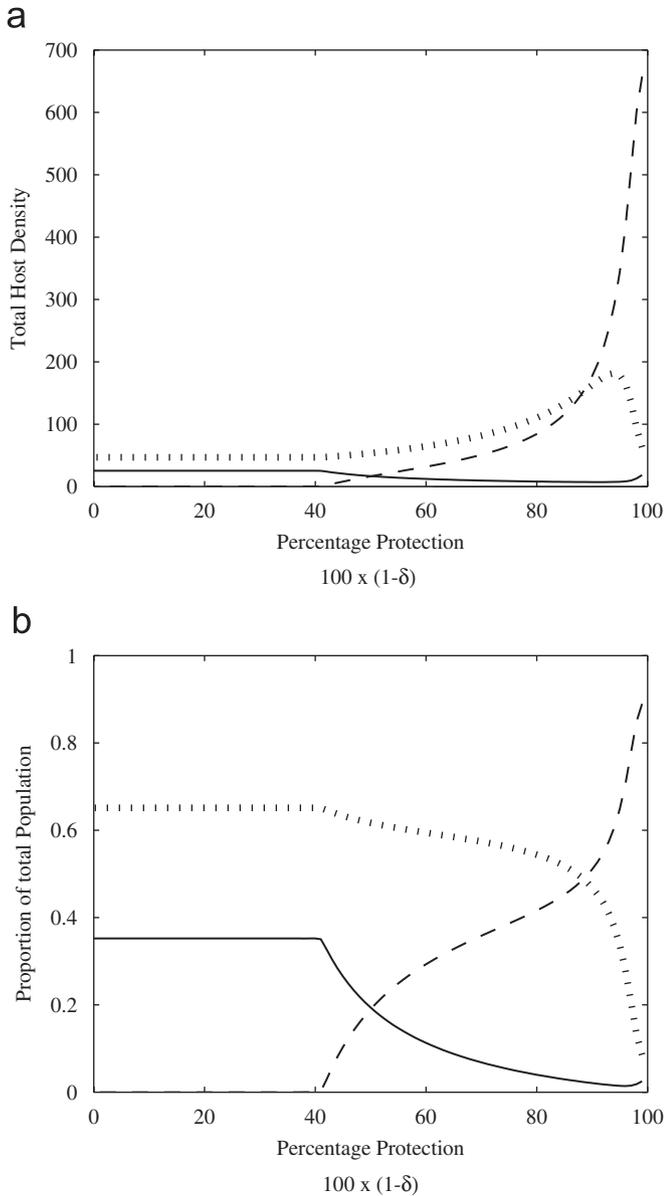


Fig. 2. (a) The equilibrium densities of susceptibles X (solid line), horizontally transmitted infecteds Y (dotted line), vertically transmitted infecteds V (dashed line) to increasing percentage of protection ($100 \times (1-\delta)$) against the HTP for the model defined by Eqs. (1a)–(1c). (b) The proportional densities are shown. Parameters used are $\beta = 0.2$; $p = 0.8$; $f = 0.8$; $b = 1$; $r = 10$; $q = 0.01$; $\alpha_v = 0.25$; $\alpha_y = 4$.

transmission rate from the HTP and therefore supports a higher density VTP infected host. This in turn supports a higher density of HTP infected hosts that acts to depress the susceptible host density further. As protection tends towards 100% ($\delta \rightarrow 0$), the density of hosts infected with the HTP decreases. This decreases the infection pressure on susceptible hosts, thus, as well as the increased input from the imperfect efficiency of the VTPs, allows their density to increase.

The above results correspond to a HTP that castrates the host. If we modify the model (Eqs. (1a)–(1c)) to allow reproduction from individuals infected with the HTP in a

more traditional ‘parasitic’ sense, then Eq. (1a) becomes

$$\frac{dX}{dt} = (r - q(X + V + Y))(X + Y) + (1 - p) \times (af - q(X + V + Y))V - \beta XY. \quad (7)$$

This modification does not change the type of equilibria that can be obtained or the conditions on their stability (Eqs. (3) and (5)). It does however change the population values at the equilibria ($X_2, 0, Y_2$) and (X_3, V_3, Y_3) and, therefore, affects the parameter regions over which the VTP can persist (Fig. 3). Here it is assumed the HTP is a super-infection, however simulations have shown the same qualitative results occur if co-infection is allowed and the HTP can contribute to the VTP growth rate.

The distinction between infectious organisms that allow reproduction and those that castrate their host is important, since in the absence of recovery, it distinguishes ecologically predatory and parasitic life-histories (Godfray, 1994; Boots, 2004). Fig. 3 shows the difference between a ‘parasitic’ and a ‘predatory’ HTP. In all the plots it is clear that the VTP is supported for a greater range of parameters if the HTP is predatory. For a predatory HTP, persistence of the VTP decreases as the crowding parameter increases. For the parasitic HTP, the VTP shows maximum persistence at intermediate values of crowding (Fig. 3a). For a parasitic HTP the VTP can only persist at intermediate levels of the birth rate, whereas it shows an increasing saturating response to birth rate for a predatory HTP (Fig. 3b). VTP persistence shows an increasing saturating response to HTP transmission for a predatory HTP and a similar response to a parasitic HTP, although here VTP persistence gradually declines as HTP transmission increases, rather than saturates (Fig. 3c). Both predatory and parasitic HTP allow for similar persistence of the VTP for high levels of the death rate due to the HTP, α_y . However the VTP cannot be supported at low levels of α_y when the HTP is parasitic (Fig. 3d).

The ability of the VTP to better persist in a ‘predatory’ rather than a ‘parasitic’ system is due to the uninfected hosts having no contribution to the growth rate from the HTP. This reduces the ability of the uninfected host to out-compete the VTP for resources and therefore means the VTP is required to provide less protection from the HTP to persist in the system. Simulations have shown that these effects are also apparent when the HTP reduces the fecundity of infected individuals rather than completely castrating them and therefore falls between our ‘predatory’ and ‘parasitic’ limiting cases.

The model (Eqs. (1a)–(1c)) was also modified to consider the persistence of a VTP when the HTP takes the form of a free-living stage. The model is described as follows:

$$\frac{dX}{dt} = (r - q(X + V + Y))X + (1 - p) \times (af - q(X + V + Y))V - \beta XW, \quad (8a)$$

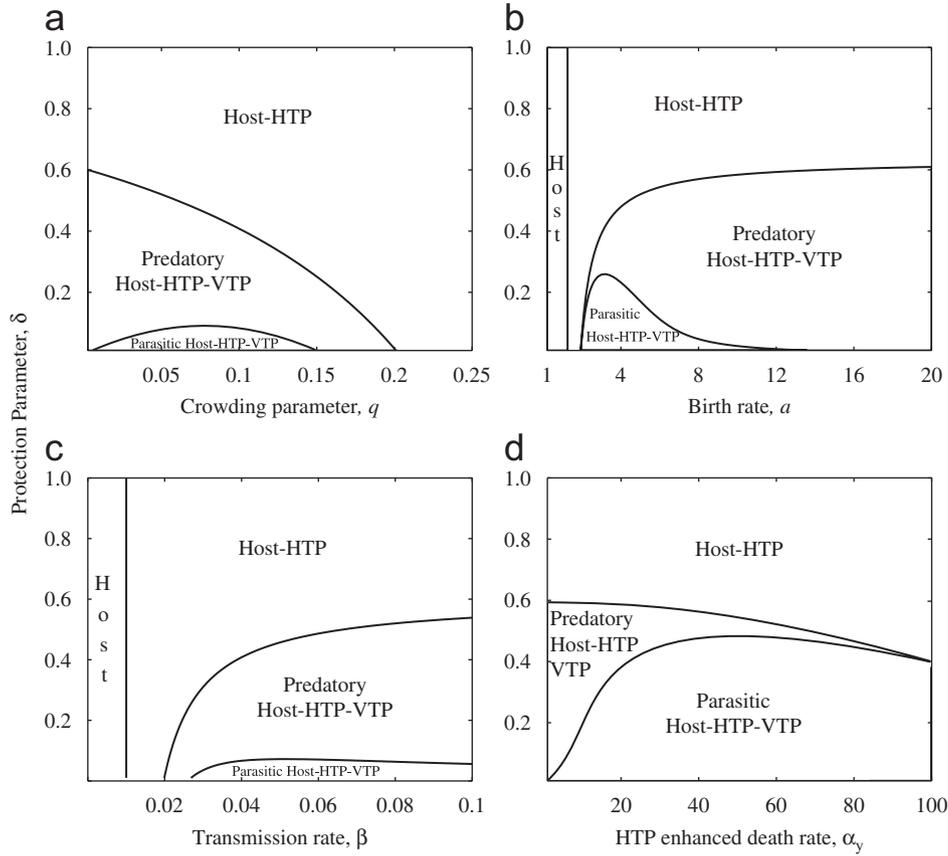


Fig. 3. A comparison of coexistence of parasitic and predatory natural enemies. Parameter space portraits indicating where the different population equilibria are stable for the model defined by Eqs. (1a)–(1c) (predatory HTP) and the modified model where Eq. (1a) is replaced by Eq. (8), (parasitic HTP). Parameter are $r = 10$; $b = 1$; $\alpha_y = 0.25$; $q = 0.01$; $\beta = 0.1$; $f = 0.8$; $p = 0.8$, unless varied in the plots.

$$\frac{dV}{dt} = p(af - q(X + V + Y))V - (\alpha_v + b)V - \delta\beta VW, \quad (8b)$$

$$\frac{dY}{dt} = (X + \delta V)\beta W - (\alpha_Y + b)Y, \quad (8c)$$

$$\frac{dW}{dt} = \lambda Y - \mu W. \quad (8d)$$

The model is based on the free-living infective stage model G of Anderson and May (1981). The classes and parameters are the same as in Eqs. (1a)–(1c) except we additionally include a new class, W , which represents the density of the external free-living infective stage of the parasite. These external stages decay at a rate μ in the environment and are produced at a rate λ per individual ($\lambda = \Lambda(\alpha_Y + b)$), where Λ is the number of viral particles released upon death of an infected host. Transmission of the HTP therefore takes place when a susceptible host comes into contact with free-living infective stage, W . The uptake of free-living particles by the hosts has been shown to be negligible relative to typical biological rates of production and decay, and thus has little or no effect on the dynamics of host and parasite (Boots, 1999; Dwyer, 1994) and is thus passed over here to maintain a simple

model. In the free-living model, the VTP is able to invade the host–HTP equilibrium if it is stable when inequality (9) is satisfied

$$p[af - q(X^* + Y^*)] > \delta\beta W^* + (\alpha_v + b), \quad (9)$$

where the host–HTP equilibrium is given by

$$(X^*, 0, Y^*, W^*) = \left(\frac{\mu}{\Lambda\beta}, 0, \frac{\Lambda\beta\Psi - 2q\mu \pm \sqrt{\Lambda\beta\Psi^2 + 4\Lambda\beta\alpha_y q\mu}}{2q\Lambda\beta}, \frac{\Lambda(\alpha_y + b)Y^*}{\mu} \right) \quad (10)$$

where $\Psi = a - b - \alpha_y$.

Eq. (9) is analogous to Eq. (5). However, it is possible for the host–HTP populations to coexist in the form of population cycles (Anderson and May, 1981; White et al., 1996). When this is the case, inequality (9) cannot be used to determine whether the VTP can invade. Instead the invasion success of the VTP is determined by numerical calculation of the Floquet multiplier (Ferriere and Gatto, 1993; Iooss and Joseph, 1980). This equates to calculating whether the growth rate of invading VTP population is positive or negative over time in a varying host–HTP

population cycle. The analysis showed the invasion of the VTP stabilised the dynamics, either by decreasing the amplitude of the systems oscillations or stabilising cycles to point equilibrium (Fig. 4). Thus, persistence of VTPs may aid the stability of host–parasite systems. The presence of the VTP reduces the likelihood of cycles, by affording the host a level of protection from the HTP that reduces its

overall transmission, which has been seen to cause cycles when high (Anderson and May, 1981).

Fig. 5 shows the invasion ability of the VTP into the coexisting host–free-living HTP population. Parameters in Fig. 5 are chosen such that the HTP–host population exhibits oscillatory dynamics. The parameter region in which the VTP can invade is similar for parasites with

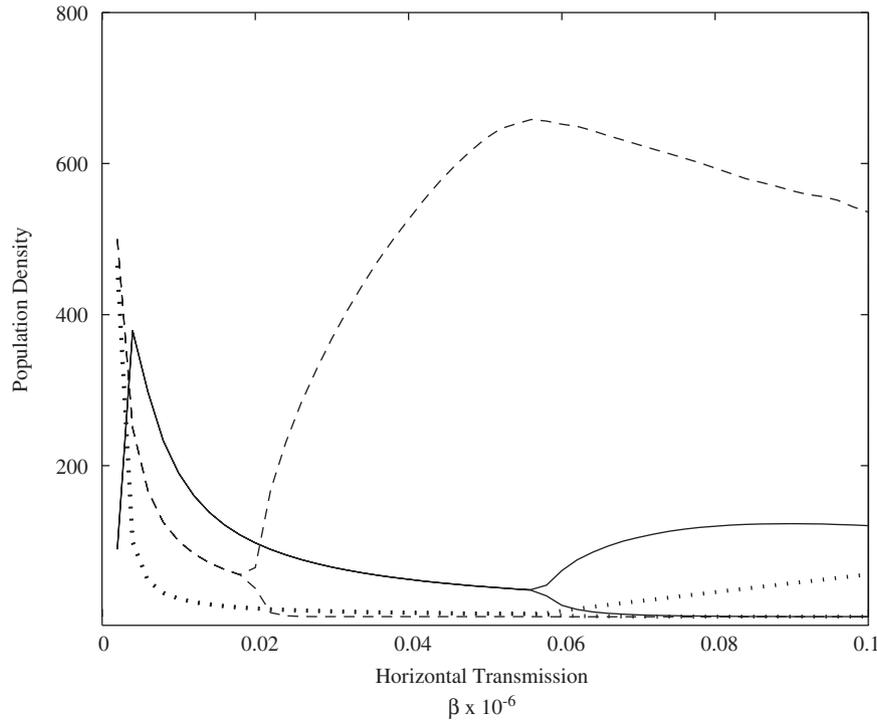


Fig. 4. Bifurcation diagram that plots of maximum and minimum values produced in the dynamics of host–HTP system and the host–HTP–VTP system with free-living parasites. The dashed line denotes the dynamics of the uninfected host in the presence of the HTP without a VTP present in the system (where the line splits it indicates that the dynamics change from point equilibrium to population cycles). The solid and dotted lines are the VTP and uninfected host dynamics, respectively. Parameters are $f = 0.8$; $r = 10$; $q = 0.01$; $\alpha_y = 4$; $b = 1$; $p = 0.8$; $A = 1 \times 10^6$; $\delta = 0.4$; $\alpha_v = 0.25$.

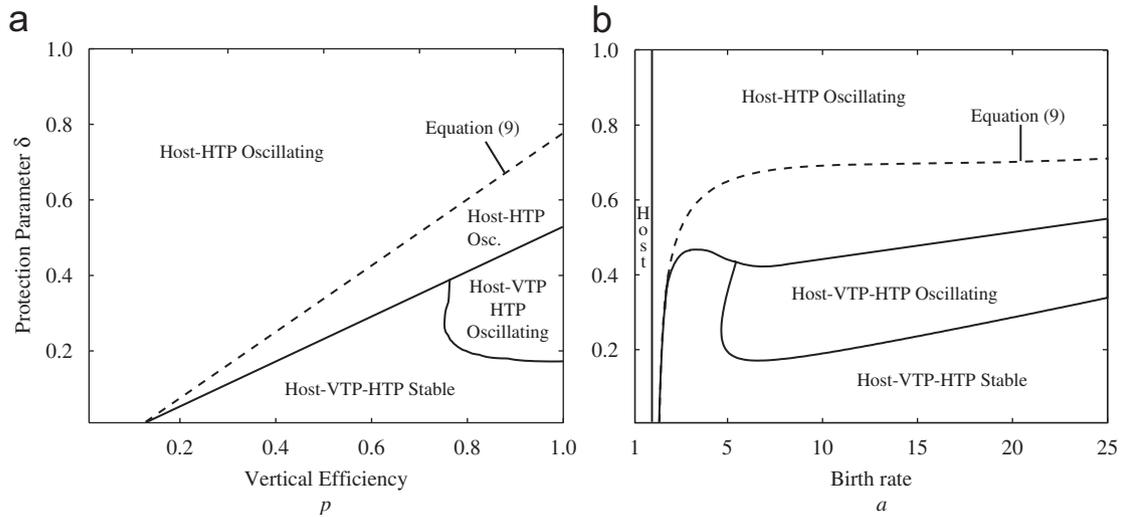


Fig. 5. The role of protection (δ) vs. vertical efficiency and birth rate in coexistence and stability. The parameter space portraits indicate where the different population equilibria are stable for the model with free-living HTP defined by Eqs. (8a)–(8d). The dashed line represents the invasion boundary for the VTP determined by Eq. (9) and is plotted for comparison with the true invasion lines. Parameters are $f = 0.8$; $p = 0.8$; $r = 10$; $q = 0.01$; $\alpha_y = 4$; $\alpha_v = 0.25$; $b = 1$; $p = 0.8$; $A = 1 \times 10^6$, unless varied in the plots.

free-living stages as for directly transmitted ones (compare Figs. 1 and 5). Again there is a threshold level in the vertical efficiency of the VTP, above which the VTP can persist. As vertical efficiency increases, persistence of the VTP can be achieved at lower levels of protection (Fig. 5a). Persistence of the VTP shows an increasing saturating response for birth rate against protection (Fig. 5b). Importantly, both figures show that for a large region of parameter space, VTP persistence acts to remove the oscillations and stabilise the population dynamics. This occurs most readily at high levels of protection (Fig. 5). For the purpose of comparison the region of VTP invasion as determined from Eq. (9) is also plotted in Fig. 5. If the HTP–host dynamics were in equilibrium then the VTP could invade below this line. The actual region of VTP invasion requires higher levels of protection, indicating that it is more difficult for the VTP to invade oscillatory populations.

3. Discussion

A VTP's reduction of host fitness (whether by decreasing fecundity and/or increasing host mortality) is generally thought to lead its exclusion from a population (Lipsitch et al., 1995; Regniere, 1984). Here we have shown when interference can allow a VTP, without any horizontal transmission, to be maintained in a host population. This occurs when the VTP confers protection against a secondary, horizontally transmitted, parasite in the system. Persistence depends critically on the pathogenicity of the HTP and the transmission efficiency of the VTP. In addition the mode of action of the HTP is important. If the HTP is a functional predator (Boots, 2004) it is much more likely to maintain the VTP in the population. In addition we have shown that the persistence of VTP through interference may stabilise host population dynamics. Interference may therefore be an important factor in the maintenance of complex multi-enemy systems in the wild, allowing persistence of VTPs and stabilisation. It is, however, more difficult for the VTP to persist if the system it invades is oscillating rather than stable.

The vertical efficiency of the VTP, p , is crucial to persistence. If the efficiency decreases, then it not only reduces the VTP infected host's competitive ability but also mechanistically increases the uninfected host's birth rate and, therefore, its competitive ability. VTPs benefit linearly from increased vertical transmission efficiency, with correspondingly less protection required for persistence. Therefore, providing that the parasite has a high vertical transmission, only a relatively small level of protection is required to allow persistence. The microsporidia *Dictyocoela* sp. (roeselum) possess a moderate vertical transmission efficiency (55%) (Haine et al., 2004), which would suggest that a substantial reduction in virulence of a HTP is needed to enable its persistence. Vertical transmission efficiencies of over 70% have been seen in some granuloviruses and cytoplasmic polyhedral viruses (CPV) (Burden et al., 2002; Sikorowski et al., 1973). This high transmission efficiency leads to the requirement of much less interference

with a HTP virus to enable persistence. CPVs are also known to be passed into the environment via larval faeces (Bong and Sikorowski, 1991; Sikorowski et al., 1973). However there is little known of the scale at which this possible horizontal transmission occurs and therefore it may be less important in the persistence of these VTPs than an interference interaction with other parasites.

The host–HTP system displays a dramatic increase in overall population density when the VTP also persists (Fig. 2). VTPs may therefore help to allow the persistence of host–parasite interactions in nature. Increasing the protection by the VTP also somewhat counter-intuitively may benefit the HTP since more HTP infected hosts can be supported. Therefore whilst the two parasites may be viewed as competing for the host in some sense, both the HTP and VTP may benefit from the interaction. The free-living model showed that the presence of a protecting VTP acts to reduce the amplitude of population oscillations (or replace them with a stable equilibrium). Therefore VTPs may be of value as biological controls in systems that have a tendency to exhibit large population oscillations since they reduce the tendency to cycles and hence limit population outbreaks.

It is increasingly recognised that there are important differences between parasites that act as functional predators (obligate killers from which the host cannot recover) compared with those that act as classical parasites (Boots, 2004). Functional predators are common insect natural enemies, including the baculoviruses, and of course many, but not all parasitoids (Godfray, 1994). We have shown that such predatory HTPs always provide a greater opportunity for a VTP to persist compared to when the HTP acts in a parasitic manner. Furthermore, when the HTP has low virulence the VTP is only likely to persist with a predatory HTP, whereas it can persist with both predatory and parasitic HTPs that have high virulence. These differences reflect the contrasting effects of virulence on the prevalence of infection of the two types of parasite.

Productive hosts that have high carrying capacities through low susceptibility to crowding are more likely to support the VTP if the HTP is predatory. In contrast, when the HTP is parasitic the VTP is supported over a greatly reduced region of parameter space, which is maximised at an intermediate susceptibility to crowding. We may therefore expect that VTPs would be seen in r -selected species, such as pest species that are also vulnerable to other HTPs, particularly if these are predatory. For example the winter moth, *Operophtera brumata*, is affected by viral predatory pathogens and parasitoids that infect the host's larval stage (Graham et al., 2004; Kerslake and Hartley, 1997; Kerslake et al., 1996). It is therefore a prime host for a VTP that provides some interference protection. Further investigation into the vertically transmitted cytoplasmic viruses that occur in winter moth populations (Graham et al., 2006) should examine whether they protect their hosts from the HTPs.

Here we have assumed that the interaction is between a VTP that reduces the chance of parasitism from another HTP. This is closely related to the findings of Oliver

et al. (2003) who showed that a vertically transmitted bacterial endosymbiont could persist if it provides a level of protection to an aphid host that is attacked by a *Braconidae* parasitic wasp. Host infected with the vertically transmitted bacteria exhibited a reduction in parasitism. This is analogous to the protection parameter that reduces the HTP transmission ability in our study. Oliver et al. (2003) do not consider the classical host parasitoid system with discrete generations. In their study the aphids can reproduce when parasitised and so it is equivalent to the parasitic HTP in our study. This is one example where protection may allow the persistence of a VTP. Our model suggests that persistence is even more likely with the predatory infections that are common in insect pathogens. Many vertically transmitted diseases seem to persist asymptotically or have very little pathogenic effect, but any deleterious effect on the host may be masked by the fact that they are protecting the host from a more pathogenic HTP in the environment.

Our models show it is important to determine if the VTPs commonly found in many different insects occur due to ecological interference with more pathogenic, HTPs. We have shown here that there are important implications to the host population dynamics from the coexistence of these different parasites. Given the widespread presence of virulent VTPs in nature, understanding the mechanisms by which they persist remains a key theoretical and empirical challenge.

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