The effect of transmission route on plant virus epidemic development and disease control

Michael. J. Jeger, Laurence V. Madden, Frank van den Bosch

www.elsevier.com/locate/yjtbi

The effect of transmission route on plant virus epidemic development and disease control

Michael. J. Jeger, Laurence V. Madden, Frank van den Bosch

PII: S0022-5193(09)00010-1
DOI: doi:10.1016/j.jtbi.2009.01.012
Reference: YJTBI5427

To appear in: *Journal of Theoretical Biology*

Received date: 13 June 2008
Revised date: 6 January 2009
Accepted date: 6 January 2009

Cite this article as: Michael. J. Jeger, Laurence V. Madden and Frank van den Bosch, The effect of transmission route on plant virus epidemic development and disease control, *Journal of Theoretical Biology* (2009), doi:10.1016/j.jtbi.2009.01.012

The effect of transmission route on plant virus epidemic development and disease control

Michael. J. Jeger[1*], Laurence V. Madden[2] and Frank van den Bosch[3]

[1] Division of Biology, Imperial College London, Silwood Park, Ascot, SL5 7PY, UK.

[2] Department of Plant Pathology, Ohio State University, Wooster, OH 44691, USA.

[3] Rothamsted Research, Harpenden, Herts AL5 2JQ, UK.

*Corresponding author: Email: m.jeger@imperial.ac.uk; tel: +44-207 594 2428; fax: +44-207 594 2601.

Abstract

A model for indirect vector transmission and epidemic development of plant viruses is extended to consider direct transmission through vector mating. A basic reproduction number is derived which is the sum of the R_0 values specific for three transmission routes. We analyse the model to determine the effect of direct transmission on plant disease control directed against indirect transmission. Increasing the rate of horizontal sexual transmission means that vector control rate or indirect transmission rate must be increased/decreased substantially to maintain R_0 at a value less than 1. By contrast, proportionately increasing the probability of transovarial transmission has little effect. Expressions are derived for the steady-state values of the viruliferous vector population. There is clear advantage for an insect virus in indirect transmission to plants, especially where the sexual and transovarial

transmission rates are low; however information on virulence-transmissibility relationships is required to explain the evolution of a plant virus from an insect virus.

Keywords: basic reproductive number, transovarial transmission, venereal transmission, plant virus evolution, insect viruses.

1. Introduction

A plant virus is a microparasite, a nucleoprotein that is entirely dependent upon plant cells for its survival and multiplication. It must also have the ability to move between plant cells and ultimately between-plants if it is to persist. In this paper we consider the mechanisms of transmission of viruses between plants, an important and often defining characteristic of plant virus epidemiology determining the rate and extent of disease development in plant populations and which also may constrain the extent of virus variability (Power, 2000; Seal *et al*., 2006). Transmission can be either direct or indirect, involving a vector – predominantly arthropods. Direct transmission can also be horizontal or vertical from parent to progeny. Transmission has been viewed from the perspective of the plant or less frequently from the perspective of the vector. Both perspectives are important in considering the evolutionary origin of plant viruses, *i.e.* from a phytocentric or arthropocentric point of view. The latter perspective may be important for those plant viruses which have established an intimate relationship with the arthropod vector, in which the virus circulates within the vector, may propagate within the vector, and in some cases is passed to progeny in a functional form.

These contrasting perspectives are shown in Table 1 for the different classes of transmission. From the phytocentric perspective, routes of transmission have been well characterised, transmission can be direct and vertical (through seed, pollen or vegetative-propagation), direct and horizontal (through pollen or mechanical damage), as well as indirect and horizontal (through vector transmission). Equally from the arthropocentric perspective, transmission can be direct and vertical (transovarial transmission) as well as indirect and horizontal (through plant transmission). There is little information on direct horizontal transmission, i.e., contact or venereal. Where transmission is indirect, involving a vector (whether the plant or the arthropod), then it can be broken down into the processes of acquisition and inoculation, with an intervening period of variable length determining the survival of the virus (in the plant or in the arthropod) depending on the system being considered. Indirect transmission can involve life history characteristics: for example, in thrips transmission of tospoviruses, only larvae can acquire the virus from an infected plant but do not inoculate healthy plants until adults. Thus the arthropod vector must complete its life cycle for the infection cycle to be completed.

Most attention in plant virus epidemiology has been given to indirect transmission involving arthropod vectors, mostly Homopteran insects, because of their economic impact on crop plants. Based on a classification of transmission types as non-persistent, semi-persistent, persistent-circulative and persistent-propagative we have developed a general model which can be used to explore the epidemiological consequences of transmission parameters appropriate for each class (Jeger *et al*., 1998; Madden *et al*., 2000). From a phytocentric perspective direct transmission has been modelled in terms of vertical transmission through the use of vegetatively-propagated planting material and its relative contribution to disease development

compared with indirect transmission by arthropod vectors (Jeger *et al*., 2002). From an arthropocentric perspective these models (Jeger *et al*., 1998; Madden *et al*., 2000) give some attention to vertical transovarial transmission, known to be an important ecological determinant of fitness in insect-vectored vertebrate viruses (Labuda and Nuttal, 2004; White *et al*., 2005). With plant viruses transmitted in a persistent-propagative manner, transovarial transmission is relatively frequent for some virus genera (Hogenhout *et al*., 2008). However there has been little consideration of the pathways and barriers involved in such transmission. The possibility of direct horizontal transmission between vectors, e.g. during copulation or other contact, has rarely been explored with only one documented case reported (Ghanim and Czosnek, 2000). The question can also be asked under what circumstances might direct horizontal transmission between vectors be advantageous for plant virus survival in the absence of the host plant: a question analogous to that posed for venereal transmission of vertebrate viruses in the absence of viremic hosts (Tesh *et al*., 1992).

Sexually-transmitted diseases of insects, caused by viruses, protists, fungi, nematodes and mites are known (Knell and Webberley, 2004). These diseases can be highly pathogenic, leading to high mortality but often to reduced fecundity of the host. At the same time they can reach high prevalences in the host population. This combination means that sexually-transmitted diseases are likely to be important in the ecology and evolution of the host (Knell and Webberley, 2004). Viruses that are transmitted between insects by mating are often horizontally transmitted viruses of mammalian hosts. Sexual transmission between vectors may be an adaptation that maintains virus in the vector population in the absence of mammalian hosts, or where vertical transmission is inefficient. Venereal and vertical (transovarial) transmission of vertebrate viruses in insect vectors is well known for a wide range of vectors,

notably for mosquitoes (Barreau *et al*., 1997; Rust *et al*., 1999; Schopen *et al*., 1991), sandflies (Tesh *et al*., 1992) and ticks (Labuda and Nuttal, 2004). Venereal transmission can occur from males to females, and vice-versa with or without (Endris and Hess, 1994) transovarial transmission.

The complexity of the role of vertical and/or venereal transmission in the epidemiology of vertebrate viruses was emphasised by Rodhain (1991), who also made the case that similar consideration should be given to other virus-arthropod relationships. The one plant virus that may be analogous to these examples is *Tomato yellow leaf curl virus* (TYLCV) in which horizontal sexual transmission has been demonstrated (Ghanim and Czosnek, 2000). An isolate of TYLCV was shown to be transmitted among whiteflies (*Bemisia tabaci*) in a sex-dependent way in the absence of any other virus source. The virus was transmitted from males to females and *vice versa* but not within the same sex. The virus was considered to follow the same circulative pathway associated with normal acquisition from infected plants but the actual mechanisms involved in sexual transmission remain unknown. The vector-virus system was considered to have features of an insect-pathogen relationship (Rubinstein and Czosnek, 1997; Czosnek *et al*., 2001), although there is no evidence that the virus is propagative in the whitefly.

It would be of considerable interest to study persistent-propagative plant viruses for direct transmission, both horizontal and vertical, and their relationship with animal viruses. For example, Tospoviruses belong to the family Bunyaviridae, to which many of the mammalian viruses vectored by Dipteran insects belong and where sexual transmission has either been demonstrated or inferred. Tenuiviruses, including *Rice grassy stunt virus* (RGSV), have host ranges including plants and animals, and also characteristics similar to vertebrate-infecting viruses in the Bunyaviridae (Falk

and Tsai, 1998). Insect viruses of relevance in this context are in the Reoviridae. Cypoviruses from various insect hosts are closely related to *Rice ragged stunt virus* (RRSV) (Hagiwara *et al*., 2002; Li *et al.*, 2006; Zhao *et al*., 2003) with the implication drawn that plant reoviruses originated from insect reoviruses. RRSV is also closely related to *Nilaparvata lugens reovirus* (NLRV), more so than to other plant (or animal) reoviruses (Upadhyaya *et al*., 1998) and may have emerged from an insect virus more recently than other plant reoviruses (Ikeda *et al*., 2001). *N. lugens*, the brown planthopper, is a serious pest of rice and is also the vector of RRSV (Hibino, 1996). NLRV does not cause disease in the insect host and does not multiply in rice but can be re-acquired from rice by non-viruliferous *N. lugens* (Noda and Nakashima, 1995; Nakashima and Noda, 1995). In *N. lugens* there are several related reoviruses that appear to be exclusively insect viruses and which have not been known to replicate in rice or other plants. The question then arises as to why related reoviruses have or have not evolved the ability to infect plants?

Vertical transmission of plant viruses in the vector with persistent-circulative and persistent-propagative transmission is possible where there is transovarial transmission from parent to progeny. With the geminiviruses the circulative pathway has been well-described in whiteflies (Ghanim *et al*., 2001; Czosnek *et al.*, 2002). Transovarial transmission has been claimed for TYLCV (Goldman and Czosnek, 2002) although Bosco *et al*., (2004) claimed only non-infectious viral DNA was transmitted to the progeny, and thus transovarial transmission was not relevant epidemiologically. In the leaf hopper *Nephotettix cincticeps*, the vector of the reovirus, *Rice dwarf virus* (RDV) (Hibino, 1996), transovarial transmission was confirmed as efficient and enabled the long-term maintenance of RDV in a leaf

hopper population (Honda *et al*., 2007) although no checks were made on whether there was any contribution of venereal transmission to virus survival.

In this paper we deal with two questions: (1) to what extent is the effectiveness of plant disease control affected where direct transmission in the vector occurs; and (2) does indirect transmission through a plant provide a trajectory for an insect virus towards evolution of a plant virus? We do this by developing further a mathematical model dealing largely with indirect horizontal transmission (Jeger *et al*., 1998, Madden *et al*., 2000) to a form in which the contributions of direct transmission to epidemic development can be evaluated. We then discuss these results in relation to broader questions on plant virus epidemiology.

2. Model development

2.1 Basic model

We first reduce the original model (Jeger *et al*., 1998) by leaving out the latent period in both plant and vector, which for persistently-transmitted viruses could be of approximately the same order. We also take out migration terms and derive new simplified steady-state values and basic reproductive number (R_o) expressions.

The plant model with no latent category is

$$\frac{dH}{dt} = \beta(K - H) - \phi k_1 T Z \frac{H}{K} \qquad \text{(healthy)} \qquad \text{(1a)}$$

$$\frac{dS}{dt} = \phi k_1 T Z \frac{H}{K} - (k_3 + \beta)S \qquad \text{(infectious)} \qquad \text{(1b)}$$

$$\frac{dR}{dt} = k_3 S - \beta R \qquad \text{(post-infectious)} \qquad \text{(1c)}$$

where *H, S,* and *R* are variables representing the density of healthy (disease-free susceptible), infectious, post-infectious (removed) plants. *K* (= *H* + *S* + *R*) is total plant population density, Z is the density of viruliferous vectors, and the model parameters are defined in Table 2.

The vector model with no latent category is

$$\frac{dX}{dt} = \alpha(X+Z)\left[1 - q\frac{Z}{X+Z}\right] + \tau Z - \alpha X - \phi X \lambda T \frac{S}{K} \quad \text{(non-viruliferous)} \quad (1d)$$

$$\frac{dZ}{dt} = \phi X \lambda \mathrm{T}\frac{S}{K} - \tau Z - \alpha Z + \alpha(X+Z)q\frac{Z}{X+Z} \quad \text{(viruliferous)} \quad (1e)$$

where *X* is the density of non-viruliferous (virus-free) vectors. *P (= X + Z)* is the constant vector population density and the model parameters are defined in Table 2.

Introduce the scaling

$$\overline{H} = \frac{H}{K},\ \overline{S} = \frac{S}{K},\ \overline{R} = \frac{R}{K};\ \overline{X} = \frac{X}{K},\ \overline{Z} = \frac{Z}{K}$$

then substituting and dropping overbars gives

$$\frac{dH}{dt} = \beta(1-H) - \phi k_1 T Z H \quad (2a)$$

$$\frac{dS}{dt} = \phi k_1 T Z H - (k_3 + \beta)S \quad (2b)$$

$$\frac{dZ}{dt} = \phi\left[\frac{P}{K} - Z\right]\lambda T S - [\tau + \alpha(1-q)]Z \quad (2c)$$

with the equations for *R* and *X* redundant.

By setting equations 2 to zero, internal equilibria are obtained as

$$H^* = \frac{\beta}{\beta + k_1 \phi T Z^*},\ S^* = \frac{\beta}{k_3 + \beta}\cdot\frac{k_1 \phi T Z^*}{\beta + k_1 \phi T Z^*}$$

where

189 $$Z^* = \frac{\beta\left[\frac{P}{K}(\phi T)^2\,\lambda k_1 - [\tau+\alpha(1-q)](k_3+\beta)\right]}{k_1\,\phi T\{\beta\phi T\,\lambda + [\tau+\alpha(1-q)](k_3+\beta)\}}$$

190 The invasion criterion for the system described by equations 2 is obtained as the
191 inequality:

192 $$\frac{\phi k_1 T \phi \lambda T \frac{P}{K}}{(k_3+\beta)(\tau+\alpha)} + \frac{\alpha}{\tau+\alpha} q > 1 \qquad (3)$$

193 where the first term represents indirect transmission, and the second the contribution
194 of transovarial transmission. Note that $(\tau+\alpha)^{-1}$ gives the average length of time a
195 vector remains viruliferous, the term $\alpha q/(\tau+\alpha)$ has a maximum value of 1 when
196 $\tau = 0,\ q = 1$. The left hand side of the inequality is the basic reproductive number R_0.
197 In this formulation no consideration is given to whether transovarial transmission is
198 parthenogenic or through sexual mating.
199
200 *2.2 Extended model with direct transmission through mating*
201 We now introduce specific terms for vector encounter and mating and,
202 following Jeger *et al*. (1998) and Madden *et al*. (2000), modify birth and death rates
203 so as to maintain a constant population. We then analyse the contribution of
204 transovarial transmission to sexual progeny and venereal transmission, by comparison
205 with indirect transmission, to the development of a plant virus epidemic.
206 The probabilities for the three types of encounter between non-viruliferous *X*
207 and viruliferous *Z* individuals are:
208 (i) probability of an *XX* encounter is $(X/P)^2$.
209 (ii) probability of a *ZZ* encounter is $(Z/P)^2$.

(iii) probability of an *XZ* encounter is *2(X/P)(Z/P)*. Of these encounters *(X/P)(Z/P)* are with *X* as female, and *(X/P)(Z/P)* are with *Z* as female.

We assume that male-female encounters lead to copulation with probability one. The number of such encounters is dependent on the sex ratio of the vector population. Let σ be the proportion of females, $1-\sigma$ the proportion of males in the population, such that $\frac{\sigma}{1-\sigma}$ gives an assumed constant sex ratio. The probabilities of encounter specified above can readily be shown to convert to probability of copulation by multiplying each by $\xi = \sigma(1-\sigma)$. The probabilities then sum to ξ, the proportion of encounters that lead to copulation.

We assume that the total number of copulations in the vector population per time unit, *C*, is proportional to the subsequent birth rate. Define γ as the number of offspring due to one copulation, then with our model this becomes $\frac{C}{P} = (\alpha\xi/\gamma)$, *i.e.* the number of copulations per individual per unit time.

Direct transmission occurs through two processes:

I. Horizontal: copulation between an *X* and a *Z* individual can cause the *X* to become viruliferous.

Assume that a fraction φ of the non-viruliferous *X* individuals in an *XZ* copulation gets infected with the virus, then $2\varphi\frac{C}{P}$ is the per capita rate of transmission due to copulation, which is simply the sexual transmission rate. This implies that the direct transmission infection rate equals $2\varphi\frac{C}{P}\frac{XZ}{P}$.

II. Vertical: a fraction of the offspring can be infected.

Assume that the egg is infected with the virus and give rise to an infected offspring (if the mother is infected) with probability q_1. Assume that the sperm-cell is infected and gives rise to an infected offspring (if the father is infected) with probability q_2. Now the various birth rates from the different copulations are:

(i) *XX*: produces *X* only at rate $(\alpha P)\xi(X/P)^2$

(ii) *ZZ*: produces both *X* and *Z*. The production rate for X is $(1-q_1)(1-q_2)(\alpha P)\xi(Z/P)^2$; the production rate for *Z* is $(q_1+q_2-q_1q_2)(\alpha P)\xi(Z/P)^2$.

(iii) *XZ*: produces *X* and *Z*. The production rate of *X* is $((1-q_1)+(1-q_2))(\alpha P)\xi(XZ/P^2)$; the production rate of *Z* is $(q_1+q_2)(\alpha P)\xi(XZ/P^2)$.

Note that adding these three rates gives $\alpha\xi P$, the overall production rate. Thus writing $\alpha' = \alpha\xi$ and $\psi = 2\varphi\frac{C}{P}$, the model becomes

$$\frac{dX}{dt} = \frac{\alpha'}{P}\left(X^2 + (1-q_1)(1-q_2)Z^2 + ((1-q_1)+(1-q_2))XZ\right) + \tau Z - \alpha' X - \phi\lambda TX\frac{S}{K} - \psi\frac{XZ}{P} \quad (4a)$$

$$\frac{dZ}{dt} = \frac{\alpha'}{P}\left((q_1+q_2-q_1q_2)Z^2 + (q_1+q_2)XZ\right) - \tau Z - \alpha' Z + \phi\lambda TX\frac{S}{K} + \psi\frac{XZ}{P} \quad (4b)$$

We note that the total vector population remains constant under these assumptions.

Now for $q_2 = 0$ (no transmission via the sperm cell), and using $X^2 + (1-q_1)Z^2 + (2-q_1)XZ = X^2 + Z^2 + 2XZ - q_1(Z^2 + XZ) = P(P - q_1 Z)$, we find that the production rate term in 4a is the same as that in equation 1d, with α' replacing α.

Rescaling $\overline{X} = \frac{X}{K}, \overline{Z} = \frac{Z}{K}$, noting that $\overline{X} = \frac{P}{K} - \overline{Z}$, gives

$$\frac{dZ}{dt} = \frac{\alpha' K}{P}\left[(q_1 + q_2 - q_1 q_2)Z^2 + (q_1 + q_2)\left(\frac{P}{K} - Z\right)Z\right] - \tau Z - \alpha' Z + \phi\lambda TS\left(\frac{P}{K} - Z\right) + \psi\frac{K}{P}Z\left(\frac{P}{K} - Z\right) \quad (5)$$

where for convenience the overbars have been dropped.

3. Model analysis

Internal equilibria and the invasion criterion for the system described by equations 2a, 2b and 5 are now derived. By setting these equations to zero, explicit solutions for H^* and S^* are obtained as functions of Z^* as previously, with Z^* obtained as the positive root of the quadratic equation shown in Appendix A.

The invasion criterion is derived in Appendix B as

$$\frac{\phi k_1 T \phi\lambda T \frac{P}{K}}{(k_3+\beta)(\tau+\alpha')} + \frac{\psi}{\tau+\alpha'} + \frac{\alpha'}{\tau+\alpha'}(q_1+q_2) > 1 \quad (6)$$

The first term is simply the R_0-value for indirect transmission (inequality 3), with α' replacing α. The second term is the direct transmission term per introduced vector multiplied by the average length of time a vector remains viruliferous. The term $\alpha'(q_1+q_2)/(\tau+\alpha)$ represents the contribution of transovarial transmission to the R_0-value and has a maximum value of 2 when $\tau = 0$, $q_1 = q_2 = 1$. Thus we conclude that the left hand side of inequality 6 gives an overall R_0-value for the system described by equations 2a, 2b and 5,

R_0 (overall) = R_0(indirect transmission) +

R_0(direct, horizontal transmission) +

R_0(direct, transovarial transmission)

which is valid for the introduction of viruliferous males or viruliferous females.

For the virus to invade in the absence of the host plant, then the sum of the last two (direct) transmission terms must be greater than 1. In the absence of the host plant, by omitting the indirect transmission term from equation 5 and setting to zero, we find

$$\frac{\alpha' K}{P}\left[(q_1+q_2-q_1q_2)Z+(q_1+q_2)\left(\frac{P}{K}-Z\right)\right]-(\tau+\alpha')+\psi\frac{K}{P}\left(\frac{P}{K}-Z\right)=0$$

$$\Rightarrow Z^*=\frac{-[\tau+\alpha'(1-q_1-q_2)]+\psi}{\frac{K}{P}[\psi+\alpha' q_1 q_2]} \qquad (7)$$

which with some re-arranging gives the last two terms for direct transmission in inequality 6 as the condition for Z to be positive; it can also readily be shown that as required $Z^* < \frac{P}{K}$.

4. Numerical results

Typical time plots for H (equation 2a), S (equation 2b) and Z (equation 5) are shown in Figure 1 for parameter values (Table 3) giving a range of R_0 values. In A, C and E the direct transmission parameters are set to zero; high R_0 values give a rapid reduction in healthy plants with a lower eventual steady state size. In B, D and F the direct transmission parameters have non-zero values. The relatively high values of ψ and $q_1 + q_2$ have two effects. Firstly the steady state value for viruliferous vectors (Z) is always higher than when they are zero. Secondly the reduction in the healthy plant population is greater at comparable R_0 values.

We now make direct comparisons of the steady-state values of the viruliferous vector population (Z^*) (Appendix, equation A6) for different values of the compound parameter for indirect horizontal transmission (Fig. 2). In the left-hand column of graphs of Fig. 2, the value of P/K is set at 1 and each graph shows Z^* values for different values of the sexual transmission rate ψ and the joint probability of transovarial transmission ($q_1 + q_2$). The intercept on the ψ axis, where $Z^* = 0$, is the value where $R_0 = 1$. This intercept becomes progressively smaller as $q_1 + q_2$ increases in value. Additionally, where ψ or $q_1 + q_2$ are small then the compound parameter must be sufficiently large for the virus to be endemic in the vector population. At high vector density on plants ($P/K = 10$, right-hand column of Fig. 2), Z^* will be endemic at lower values of the compound parameter. These plots clearly show the advantage to the virus of indirect horizontal transmission especially in cases when direct horizontal and or direct vertical transmission in the vector is low.

The effect of the direct horizontal and vertical transmission terms on R_0 is seen by plotting $R_0 = 1$ isoclines for different values of ψ and $q_1 + q_2$ in relation to the indirect compound transmission parameter ($\phi^2 T^2 k_1 \lambda$) (Fig. 3), the indirect transmission term (inequalities 3, 6). Combinations of $\phi^2 T^2 k_1 \lambda$ and ψ, or $\phi^2 T^2 k_1 \lambda$ and $q_1 + q_2$ above the lines correspond to $R_0 > 1$, and hence virus persistence.Values of ψ and $q + q_2$ greater than 0 reduce the value of indirect transmission necessary for maintaining R_0 above 1, especially when the host infectious period ($1/k_3$) is long. In Fig. 3 the number of vectors per plant (P/K) is set at 1. For higher values of P/K (because the R_0 for indirect transmission depends on P/K) then indirect transmission is sufficient for $R_0 > 1$ irrespective of direct transmission.

5. Modelling disease control options

The objective of plant disease control is to reduce R_0 to below 1. The main plant disease control options to consider are:

1. Roguing, or removal of diseased plants, which means increasing the host mortality rate β.
2. Introducing plant resistance, which means decreasing the compound transmission parameter $\phi^2 T^2 k_1 \lambda$.
3. Vector control, which means increasing vector death rate by an additional term θ.

We note first that introducing roguing and plant resistance will affect only the indirect transmission term and can only ever be effective (i.e. reduce R_0 to less than 1) when

$$\frac{\psi}{\tau + \alpha'} + \frac{\alpha'}{\tau + \alpha'}(q_1 + q_2) < 1$$

In other words direct sexual transmission, horizontal or vertical, should be sufficiently small that the virus goes extinct if indirect vectored transmission to plants is very small. Note also that vector control through α' affects both the indirect and direct transmission terms in the expression for R_0 (the left hand side of inequality 6). However, the horizontal transmission term $\psi/(\tau + \alpha')$ is unbounded, whereas the transovarial term $\alpha'(q_1 + q_2)/(\tau + \alpha')$ is bounded by the value 2.

The R_0 isoclines ($R_0 = 1$) in bi-plots of vector control rate θ and roguing rate β (with other parameter values held constant, Table 3) are plotted in Fig. 4 for three values of: (a) the sexual transmission rate parameter ψ and (b) the joint probability of transovarial transmission ($q_1 + q_2$). As ψ increases then both θ and β must increase substantially to keep the R_0 value below 1. The same proportionate increase in

transovarial transmission appears to have less effect on the R_0 isoclines. The same isoclines are plotted in relation to the compound transmission parameter (Fig. 5(a) and (b)). As ψ increases, the indirect transmission rate must reduce considerably for any value of β to keep $R_0 < 1$; again there appears to be little impact of the same proportionate increase in transovarial transmission on R_0 isoclines.

A better measure of the effect of changes in parameter values on R_0 and thus on disease control is obtained by calculating elasticities (Arino *et al*., 2008) of both parameters in equation 6; where elasticity in $q_1 + q_2$ is calculated as $\frac{(q_1+q_2)}{R_0}\frac{\partial R_0}{\partial(q_1+q_2)}$, and in ψ as $\frac{\psi}{R_0}\frac{\partial R_0}{\partial \psi}$. Clearly these elasticities are equal when $\psi = \alpha'(q_1+q_2)$. The value of α' used in the simulations (Figs. 1-5) was 0.12 (probably a high value as $\alpha' = \xi\alpha$). This means that $q_1 + q_2$ must be about 8 times larger than ψ for transovarial transmission to have a greater relative effect than sexual transmission in reducing R_0.

6. Discussion

In this paper we deal with issues arising from two complementary perspectives in viewing plant virus epidemics. Firstly from the phytocentric perspective. What are the implications, if any, on disease control options where there is direct horizontal and/or vertical transmission in the vector population? Secondly from the arthropocentric perspective what would be the advantage, if any, of indirect transmission through a plant for an insect virus where there is horizontal and/or vertical transmission in the vector population? We approached these two issues by developing and analysing an epidemiological model of plant virus dynamics which

371 includes indirect horizontal transmission from plant to plant through a vector and both
372 horizontal and vertical transmission in the vector population. Modelling is
373 increasingly a valuable tool for unravelling the complexities of plant-virus-vector
374 interactions and epidemic development (Jeger *et al*., 2004) and as far as we are aware
375 this is the first model to encompass each of these possible transmission mechanisms
376 and thus analyse their relative importance. We derived the basic reproductive number
377 for the model system and internal steady state values for the host plant and vector
378 categories defined in the model, using R_0 isoclines ($R_0 = 1$) to determine the effect of
379 including sexual transmission, both horizontal through mating and vertical through
380 transovarial transmission, and the steady-state values for the viruliferous vector
381 population as a measure of virus fitness defined in terms of the model parameters.
382 From the phytocentric perspective there would be clear advantage for a plant
383 virus with indirect horizontal transmission to be transmitted, either horizontally or
384 vertically, within the vector population. Such transmission would ensure survival of
385 the virus in cases where the host is absent, especially in crops where spatially and
386 temporally varying rotations are being practised or the environment becomes less
387 favourable for the virus. Seed transmission is, especially that through the tissues of
388 the embryo, frequently found for many plant viruses. However it is generally
389 unknown (Nault 1997; Hogenhout *et al*. 2008) for persistently transmitted viruses,
390 although there appear to be a small number of counter examples (Hull, 2002; Table
391 12.1). Viruses with wide host ranges such as the tospovirus (Whitfield et al., 2005)
392 have an effective means of survival in the absence of any given host. Similarly the
393 geminivirus *Beet curly top virus* has a host range of 300 plant species in 44 families.
394 On the other hand transovarial transmission of *Rice stripe virus* and *Rice dwarf virus*
395 seems to act to ensure survival (Hibino, 1996). In such cases depending on the

396 population dynamics and mobility of the vector the virus could persist locally, and/or
397 migrate to regions where the host plant is present. In the simplest case with
398 parthogenic reproduction only, direct vertical (transovarial) transmission by itself
399 cannot maintain the virus without there also being indirect transmission, as can be
400 seen from inequality 3. Where there is both direct horizontal (sexual/venereal) and
401 vertical transmission then the virus can persist in the absence of the plant host
402 (inequality 6). If we then impose a disease control strategy that is aimed at reducing
403 the overall R_0, then sexual transmission, horizontal or vertical, must be sufficiently
404 small that even if indirect horizontal transmission was reduced considerably, in the
405 limit to zero, the overall R_0 would still be less than 1. As the sexual transmission rate
406 ψ (largely determining direct horizontal transmission) increases, then the achieved
407 level of disease control, through some combination of roguing of diseased plants,
408 introduction of host resistance, and vector control, must increase substantially to keep
409 R_0 less than 1 (Figs 4a, 5a). By contrast, the level of disease control required to keep
410 R_0 less than 1 is less affected by the same proportionate increase in the probability of
411 transovarial transmission (Figs 4b, 5b), at least for the range of parameter values
412 investigated (Table 3). In addition to exploring the parameter space, we derived
413 elasticity expressions for each parameter to determine regions where changes in
414 transovarial transmission have a greater impact than horizontal transmission. From
415 these expressions it is clear that vector turnover rate, adjusted by the population sex
416 ratio, effectively constrains the effect of transovarial compared with horizontal
417 transmission. Thus we are confident that, although the results shown in Figs 4 and 5
418 are specific for the parameters used, the general result can be justified by the R_0 and
419 the elasticity expressions derived. In this analysis we have lumped together the
420 various parameters determining vector transmission, and hence the level of host

421 resistance, in an indirect transmission compound parameter ($\phi^2 T^2 k_1 \lambda$). In a more
422 detailed analysis, van den Bosch *et al*. (2006) examine the extent to which different
423 forms of resistance may cause the virus to evolve to more virulent forms that would
424 largely make the resistance obsolete. Subsequently it was shown (van den Bosch *et*
425 *al*., 2007) that methods of cultural control did not lead to the same evolutionary
426 pressure on the virus as the introduction of host resistance. In relation to the
427 propagative viruses considered in this paper, it is highly relevant that within vector
428 populations there can be a high level of genetic variability in the ability to transmit to
429 plants than can be selected for, but how this relates to genetic variability in direct
430 transmission is not known. The other aspect to be considered is that where
431 monogenetic resistance to a plant pest that is also a virus vector (e.g. *Nilaparvata*
432 *lugens*) breaks down (Hibino, 1996), then the plant can be seriously affected by the
433 viruses transmitted (e.g. RRSV and RGSV).

434 In relation to the second issue – what, from an arthropocentric perspective, is
435 the advantage to an insect virus to be capable of indirect transmission through a plant
436 host – we have some preliminary insight from the model analysis. If we accept the
437 size of the steady state viruliferous vector population as a measure of virus fitness,
438 then there is a clear advantage for the virus in indirect transmission through a plant
439 host, especially when direct transmission in the vector population, either horizontal or
440 vertical, or population density, is low (Figs 2). It is also the case that some insect
441 viruses, such as the rice reoviruses, can be relatively benign in the insect host, causing
442 little or no disease, and are only partially adapted to plant hosts (Nakashima and
443 Noda, 1995). What is unclear is the relationship between direct transmission and
444 virulence in the insect host. If these insect viruses are relatively benign then it might
445 be assumed that direct transmission is relatively efficient (according to the postulated

446 inverse relationship between virulence and transmissibility). Conversely, with more
447 virulent insect virus forms such as *Rice dwarf* and the rice tenuiviruses (Hibino,
448 1996), direct transmission may be more circumscribed with sexual and transovarial
449 transmission much reduced. In those cases there would be clear advantage in the
450 insect virus being transmissible through a plant where again virulence characteristics
451 would be subject to further selection different in character from that occurring in the
452 insect host. What the model cannot deal with at present is the relationship between
453 virulence and transmissibility in the vector and the effect on vector performance.
454 Using the approach outlined in van den Bosch *et al*. (2006, 2007) it may be possible
455 to determine trades-off in these characteristics and whether or not the outcome in such
456 a virus-insect-plant evolutionary game is one in which a plant virus emerges from an
457 insect virus.

458 We suggest that rice viruses referred to in this paper make a suitable model
459 system to investigate such an evolutionary trajectory. It seems that the whole
460 spectrum of viruses from strictly an insect virus to a completely adapted plant virus,
461 with in both cases, variations in virulence (in plant and vector) and transmission
462 (direct and indirect), present. Rice has a long continuous history as a cultivated crop
463 in Asia which would have enabled a co-evolutionary dynamic between plant, viruses
464 and vectors to occur. It is only in the last half century that major changes in rice
465 production systems have occurred which may have affected this dynamic and led to
466 the different patterns of virus epidemics that have occurred during this period (Thresh,
467 1988, 1991).

468

469 **Appendix A: Solution of the steady-state equations for transmission to offspring**

470

471 The system is:

$$\frac{dH}{dt} = \beta(1-H) - \phi k_1 T\ Z H \tag{A1}$$

$$\frac{dS}{dt} = \phi k_1 T\ Z H - (k_3+\beta)S \tag{A2}$$

472

$$\frac{dZ}{dt} = \frac{\alpha' K}{P}\left[(q_1+q_2-q_1q_2)Z^2 + (q_1+q_2)\left(\frac{P}{K}-Z\right)Z\right] - \tau Z - \alpha' Z + \\ + \phi\lambda T\, S\left(\frac{P}{K}-Z\right) + \psi\frac{K}{P}Z\left(\frac{P}{K}-Z\right) \tag{A3}$$

473

474 with internal steady-states

$$\hat{H} = \frac{\beta}{\beta+\phi k_1 T\hat{Z}} \tag{A4}$$

475

$$\hat{S} = \frac{\beta}{k_3+\beta}\frac{\hat{Z}}{\left(\beta/\phi k_1 T\right)+\hat{Z}} \tag{A5}$$

$$\hat{Z} = \frac{-B \pm \sqrt{B^2 - 4\ A\ C}}{2\ A} \tag{A6}$$

476

477 where

$$A = \left(D - \frac{E\,K}{P}\right) - \psi\frac{K}{P}$$

478

$$B = \frac{\beta}{\phi k_1 T}\left(D - \frac{E\,K}{P}\right) + \left(E - (\tau+\alpha')\right) - \frac{\beta\phi\lambda T}{k_3+\beta} - \psi\frac{K}{P}\frac{\beta}{\phi k_1 T} + \psi$$

$$C = \frac{\beta}{\phi k_1 T}\left(E - (\tau+\alpha')\right) + \frac{\beta\phi\lambda T}{k_3+\beta}\frac{P}{K} + \psi\frac{\beta}{\phi k_1 T}$$

479

480

$$D = \frac{\alpha' K}{P}(q_1 - q_2 - q_1 q_2)$$

$$E = \alpha'(q_1+q_2)$$

481

482

483 **Appendix B: Derivation of the invasion criterion**

484

485 In the absence of disease and viruliferous vectors

486 $$\hat{S} = \hat{Z} = 0, \hat{H} = 1, \hat{X} = \frac{P}{K}.$$

487 Introduce an infinitesimally small amount of *S* and Z. Then

488 $$\frac{dS}{dt} = \phi k_1 TZ - (k_3 + \beta)S \qquad \text{(B1)}$$

489 $$\frac{dZ}{dt} = \frac{\alpha' K}{P}\left[(q_1 + q_2 - q_1 q_2)Z^2 + (q_1 + q_2)\frac{ZP}{K}\right] - \tau Z - \alpha' Z + \phi\lambda TS\frac{P}{K} + \psi Z\frac{P}{K}$$

490 which by ignoring quadratic terms

491 $$= \alpha'(q_1 + q_2)Z - \tau Z - \alpha' Z + \phi\lambda TS\frac{P}{K} + \psi Z \qquad \text{(B2)}$$

492 with the Jacobian

493 $$J = \begin{bmatrix} -(k_3 + \beta) & \phi k_1 T \\ \phi\lambda T\dfrac{P}{K} & -[\tau + \alpha'(1 - q_1 - q_2)] + \psi \end{bmatrix}$$

494 and determinant

495 $$\det(J) = (k_3 + \beta)([\tau + \alpha'(1 - q_1 - q_2)] - \psi) - \phi k_1 T\phi\lambda T\frac{P}{K}$$

496 Invasion will occur if $\det(J) < 0$

497 $$(k_3 + \beta)[\tau + \alpha'(1 - q_1 - q_2)] - (k_3 + \beta)\psi - \phi k_1 T\phi\lambda T\frac{P}{K} < 0$$

498 i.e.,

499 $$\frac{\phi k_1 T\phi\lambda T\dfrac{P}{K}}{(k_3+\beta)(\tau + \alpha')} + \frac{\psi}{\tau + \alpha'} + \frac{\alpha'}{\tau + \alpha'}(q_1 + q_2) > 1 \qquad \text{(B3)}$$

References

Arino, J., Brauer, F., van den Driessche, P., Watmough, J., Wu, J., 2008. A model for influenza with vaccination and antiviral treatment. J. Theor. Biol. 253, 118-130. doi:10.1016/j.jtbi.2008.02.026

Barreau, C., Jousset, F.X., Bergoin, M., 1997. Venereal and vertical transmission of the *Aedes albopictus* parvovirus in *Aedes aegypti* mosquitoes. Am. J. Trop. Med. Hyg. 57, 126-131.

Bosco, D., Mason, G., Accotto, G.P., 2004. TYLCSV DNA, but not infectivity, can be transovarially inherited by the progeny of the whitefly vector *Bemisia tabaci* (Gennadius). Virology 323, 276-283. doi:10.1016/j.virol.2004.03.010

Czosnek, H., Ghanim, M., Ghanim, M., 2002. The circulative pathway of begomoviruses in the whitefly vector *Bemisia tabaci* – insights from studies with *Tomato yellow leaf curl virus*. Ann. Appl. Biol. 140, 215-231. doi:10.1111/j.1744-7348.2002.tb00175.x

Czosnek, H., Morin, S., Rubinstein, G., Fridman, U., Zeidan, M., Ghanim, M., 2001. Tomato yellow leaf curl virus: A disease sexually transmitted by whiteflies. In: Harris, K., Smith, O. P. & Duffus, J. E. (Eds.), Virus-Insect-Plant Interactions, San Diego: Academic Press, pp. 1-27.

Endris, R.G., Hess W.R., 1994. Attempted transovarial and venereal transmission of African swine fever virus by the Iberian soft tick Ornithodoros (Pavlovskyella) marocanus (Acari, Ixodoidea, Argasidae). J. Med. Entomol. 31, 373-381.

Falk, B.W., Tsai, J.H., 1998. Biology and molecular biology of viruses in the genus Tenuivirus. Annu. Rev. Phytopathol. 36, 139-163. doi:10.1146/annurev.phyto.36.1.139

Ghanim, M., Czosnek, H., 2000. Tomato yellow leaf curl geminivirus (TYLCV-Is) is transmitted among whiteflies (*Bemisia tabaci*) in a sex-related manner. J. Virol. 74, 4738-4745.

Ghanim, M., Morin, S., Czosnek, H., 2001. Rate of *Tomato yellow leaf curl virus* translocation in the circulative transmission pathway of its vector, the whitefly *Bemisia tabaci*. Phytopathology 91, 188-196. doi:10.1094/PHYTO.2001.91.2.188

Goldman, V., Czosnek, H., 2002. Whiteflies (*Bemisia tabaci*) issued from eggs bombarded with infectious DNA clones of *Tomato yellow leaf curl virus* from Israel (TYLCV) are able to infect tomato plants. Arch. Virol. 147, 787-801. doi:10.1007/s007050200026

Hagiwara, K., Rao, S.J., Scott, S.W., Carner, G.R., 2002. Nucleotide sequences of segments 1, 3 and 4 of the genome of *Bombyx mori* cypovirus 1 encoding putative capsid proteins VP1, VP3 and VP4, respectively. J. Gen. Virol. 83, 1477-1482.

Hibino, H., 1996. Biology and epidemiology of rice viruses. Annu. Rev. Phytopathol. 34, 249-274. doi:10.1146/annurev.phyto.34.1.249.

Hogenhout, S.A., Ammar, E.-D., Whitfield, A.E., Redinbaugh, M.G., 2008. Insect vector interactions with persistently transmitted viruses. Annu. Rev. Phytopathol. 46, 327-359. doi:10.1146/annurev.phyto.022508.092135

Honda, K., Wei, T., Hagiwara, K., Higashi, T., Kimura, I., Akutsu, K., Omura T., 2007. Retention of *Rice dwarf virus* by descendants of pairs of viruliferous vector insects after rearing for 6 years. Phytopathology 97, 712-716. doi:10.1094/PHYTO-97-6-0712

Hull, R., 2002. Matthew's Plant Virology, 4th edition. Academic Press, San Diego, 1001 pages.

Ikeda, K., Nagaoka, S., Winkler, S., Kotani, K., Yagi, H., Nakanishi, K., Miyajima, S. Kobayashi, J., Mori, H., 2001. Molecular characterization of *Bombyx mori* cytoplasmic polyhedrosis virus genome segment 4. J. Virol. 75, 988-995. doi:10.1128/JVI.75.2.988-995.2001

Jeger, M.J., Holt, J., van den Bosch, F., Madden, L.V., 2004. Epidemiology of insect-transmitted plant viruses: modelling disease dynamics and control interventions. Physiol. Entomol. 29, 291-304. doi:10.1111/j.0307-6962.2004.00394.x

Jeger, M.J., van den Bosch, F., Dutmer, M.Y., 2002. Modelling plant virus epidemics in a plantation-nursery system. IMA J. Math. Appl. Med. Biol. 19, 75-94. doi:10.1093/imammb/19.2.75

Jeger, M.J., van den Bosch, F., Madden, L.V., Holt, J., 1998. A model for analysing plant-virus transmission characteristics and epidemic development. IMA J. Math. Appl. Med. Biol. 15, 1-18. doi:10.1093/imammb/15.1.1

Knell, R.J., Webberley, K.M., 2004. Sexually transmitted diseases of insects: distribution, evolution, ecology and host behaviour. Biol. Rev. 79, 557-581. doi:10.1017/S1464793103006365

Labuda, M., Nuttal, P.A., 2004. Tick-borne viruses. *Parasitology* **129**, S221-S245.

Li, Y., Tan, L., Li, Y.Q., Chen, W.G., Zhang, J.M., Hu, Y.Y., 2006. Identification and genome characterization of *Heliothis armigera* cypovirus types 5 and 14 and *Heliothis assulta* cypovirus type 14. J. Gen. Virol. 87, 387-394. doi:10.1099/vir.0.81435-0

Madden, L.V., Jeger, M.J., van den Bosch, F., 2000. A theoretical assessment of the effects of vector-virus transmission mechanism on plant virus disease epidemics. Phytopathology 90, 576-594. doi:10.1094/PHYTO.2000.90.6.576

Madden, L.V., Hughes, G., van den Bosch, F., 2007. The Study of Plant Disease Epidemics. APS Press, 432 pages. ISBN 978-0890543542

Nakashima, N., Noda, H., 1995. Non pathogenic *Nilaparvata lugens* reovirus is transmitted to the brown plant hopper through rice plant. Virology 207, 303-307.

Nault, L.R., 1997. Arthropod transmission of plant viruses: a new synthesis. Ann. Entomol. 90, 521-541.

Noda, H., Nakashima, N., 1995. Nonpathogenic reoviruses of leafhoppers and planthoppers. Sem. Virol. 6, 109-116.

Power, A.G., 2000. Insect transmission of plant viruses: a constrain on virus variability. Curr. Op. Pl. Biol. 3, 336-340. doi:10.1016/S1369-5266(00)00090-X

Rodhain, F. 1991 Function of virus-vector systems. Ann. Soc. Belg. Méd. Trop. 71, 189-199.

Rubinstein, G., Czosnek, H., 1997. Long-term association of tomato yellow leaf curl virus (TYLCV) with its whitefly vector *Bemisia tabaci*: effect on the insect transmission capacity, longevity and fecundity. J. Gen. Virol. 78, 2683-2689.

Rust, R.S., Thompson, W.H., Matthews, C.G., Beaty, B.J., Chun, R.W.M., 1999. La Crosse and other forms of California encephalitis. J. Child Neurol. 14, 1-14. doi:10.1177/088307389901400101

Schopen, S., Labuda, M., Beaty, B., 1991. Vertical and venereal transmission of California group viruses by *Aedes-triseriatus* and *Culiseta-inornata* mosquitos. Acta Virol. 35, 373-382.

Seal, S.E., van den Bosch, F., Jeger, M.J., 2006. Factors influencing begomovirus evolution and their increasing global significance: Implications for sustainable control. Crit. Rev. Pl. Sci. 25, 23-46. doi:10.1080/07352680500365257

Tesh, R.B., Lubroth, J., Guzman, H., 1992. Simulation of Arbovirus overwintering – survival of Toscana virus (Bunyaviridae, Phlebovirus) in its natural sand fly vector *Phlebotomos-perniciosus*. Am. J. Trop. Med. Hyg. 47, 574-581.

Thresh, J.M., 1988. Rice viruses and 'The Green Revolution'. Asp. Appl. Biol. 17, 187-194.

Thresh, M.J., 1991. The ecology of tropical plant viruses. Pl. Pathol. 40, 324-329. doi:10.1111/j.1365-3059.1991.tb02386.x

Upadhyaya, N.M., Ramm, K. Gellatly, J.A., Li, Z. Kositratana, W., Waterhouse, P.M., 1998. Rice ragged stunt oryzavirus genome segment S4 could encode an RNA dependent RNA polymerase and a second protein of unknown function. Arch. Virol. 143, 1815-1822. doi:10.1007/s007050050419

van den Bosch, F., Akudibilah, G., Seal, S.E., Jeger, M.J., 2006. Host resistance and the evolutionary response of plant viruses. J. Appl. Ecol. 43, 506-516. doi:10.1111/j.1365-2664.2006.01159.x

van den Bosch, F., Jeger, M.J., Gilligan, C.A., 2007. Disease control and its selection for damaging plant virus strains in vegetatively propagated staple food crops. Proc. R. Soc. B 274, 11-18. doi:10.1098/rspb.2006.3715

White, D.M., Wilson, W.C., Blair, C.D., Beaty, B.J., 2005. Studies on overwintering of bluetongue viruses in insects. J. Gen. Virol. 86, 453-462.

Whitfield, A.E., Ullman, D.E., German, T.L., 2005. Tospovirus-thrips interactions. Annu. Rev. Phytopathol. 43, 459-489

Zhao, S.L., Liang, C.Y., Hong, J.J., Peng, H.Y., 2003. Genomic sequence analyses of segments 1 to 6 of *Dendrolimus punctatus* cytoplasmic polyhedrosis virus. Arch. Virol. 148, 1357-1368. doi:10.1007/s00705-003-0103-zGenomic

Figure legends

Fig. 1. Numerical output of model for parameter values giving a range of R_0 values. In A, C and E parameters ψ, q_1 and q_2 are set to zero. In B, D and F they take on non-zero values.

Fig. 2. Effects of ψ, $q_1 + q_2$, and the indirect compound transmission parameter, $\phi^2 T^2 k_1 \lambda$, on the steady-state–viruliferous vector population density (Z^*). Lines correspond to different values of $\phi^2 T^2 k_1 \lambda$ (from top to bottom: 0.52, 0.13, 8.1×10^{-3}, and 5.6×10^{-5}), and graphs correspond to different values of $q_1 + q_2$. Left-hand graphs: *P/K* is set to 1 vector/plant; right-hand graphs: *P/K* is set to 10 vectors/plant.

Fig. 3. $R_0 = 1$ isoclines for different values of the direct transmission terms (either ψ or $q_1 + q_2$) in relation to the indirect compound transmission parameter, $\phi^2 T^2 k_1 \lambda$. In the left hand column, either ψ or $q_1 + q_2$ is set at 0; in the right hand column, either ψ is set to 0.2 or $q_1 + q_2$ is set to 0.4. The three lines represent k_3 values of 0.05, 0.1 and 0.2 (left to right). The vector density *P/K* = 1. For *P/K* = 10, for example, the lower right figure would be blank (i.e., all transmission rates give $R_0 > 1$).

Fig. 4. $R_0 = 1$ isoclines for different values of roguing rate (β) and vector control rate (θ), for three different values of either: (a) direct horizontal transmission during mating (ψ); or (b) direct vertical transmission following mating ($q_1 + q_2$).

Fig. 5. R_0 = 1 isoclines for different values of roguing rate (β) and indirect compound transmission rate ($\phi^2 T^2 k_1 \lambda$),for three different values of either: (a) direct horizontal transmission during mating (ψ); or (b) direct vertical transmission following mating ($q_1 + q_2$).

Table 1. Different classes of plant virus transmission from (a) phytocentric and (b) arthropocentric perspectives

(a) Phytocentric view of plant-virus transmission		(b) Arthropocentric view of plant virus transmission	
Direct transmission		Direct transmission	
Horizontal	Mechanical	Horizontal	Contact
	Soil		Venereal
	Pollen		
Vertical	Seed	Vertical	Transovarial
	Pollen		- sexual (diploidy)
	Vegetative propagation		- parthogenic (haploidy)
			- haplo-diploid
Indirect transmission		Indirect transmission	
Horizontal	Vector transmission from plant to plant*	Horizontal	Plant transmission from vector to vector**
Vertical	Vector transmission from parent plant to progeny	Vertical	Plant transmission from adult vector to progeny

* acquisition/inoculation may depend on life stage of vector e.g. larvae acquire (the plant inoculates)/adults inoculate (the plant acquires)

** acquisition/inoculation may depend on plant development e.g. seedlings acquire (the vector inoculates)/mature plants inoculate (the vector acquires)

Table 2. Parameters of basic model (equations 2a, b, c) together with typical values (Jeger *et al*., 1998) for the persistent classes of transmission

Parameter	**Transmission class**	
	Circulative	Propagative
Inoculation rate per day, λ	96	48
Acquisition rate per day, k_1	48	12
Host infectious period, $1/k_3$ (days)	10-25	10-25
Vector turnover rate per day, α	0-0.25	0-0.25
Host mortality rate per day, β	0.01	0.01
Fraction of viruliferous offspring, q	0	0.5
Feeding time per vector per day, ϕT	0-0.02	0-0.02
Vector infectious period, $1/\tau$ (days)	0.1	life

Table 3. Compound parameter values used in numerical solutions to extended model (equations 2a, b and 5) (Jeger *et al*., 2004; Madden *et al*., 2007)

Parameter	**Interpretation**	**Value**
$\phi^2 T^2 k_1 \lambda$	Compound transmission parameter	6.4 x 10^{-5}
P/K	Vector density per plant	1 – 10
$\alpha' = \alpha'_0 + \theta$	Vector turnover rate per day	0.12 + additional mortality due to control
k_3	Plant harvest rate per day (assumed equal to 1/infectious period)	0.003
β	Roguing (or mortality) rate per day	0 – 0.02
$1/\tau$	Vector infectious period (days)	$\tau = 0.1$
ψ	Sexual transmission rate per day	0 – 0.2
$q_1 + q_2$	Joint probability of transovarial transmission following mating	0-1

Fig. 1

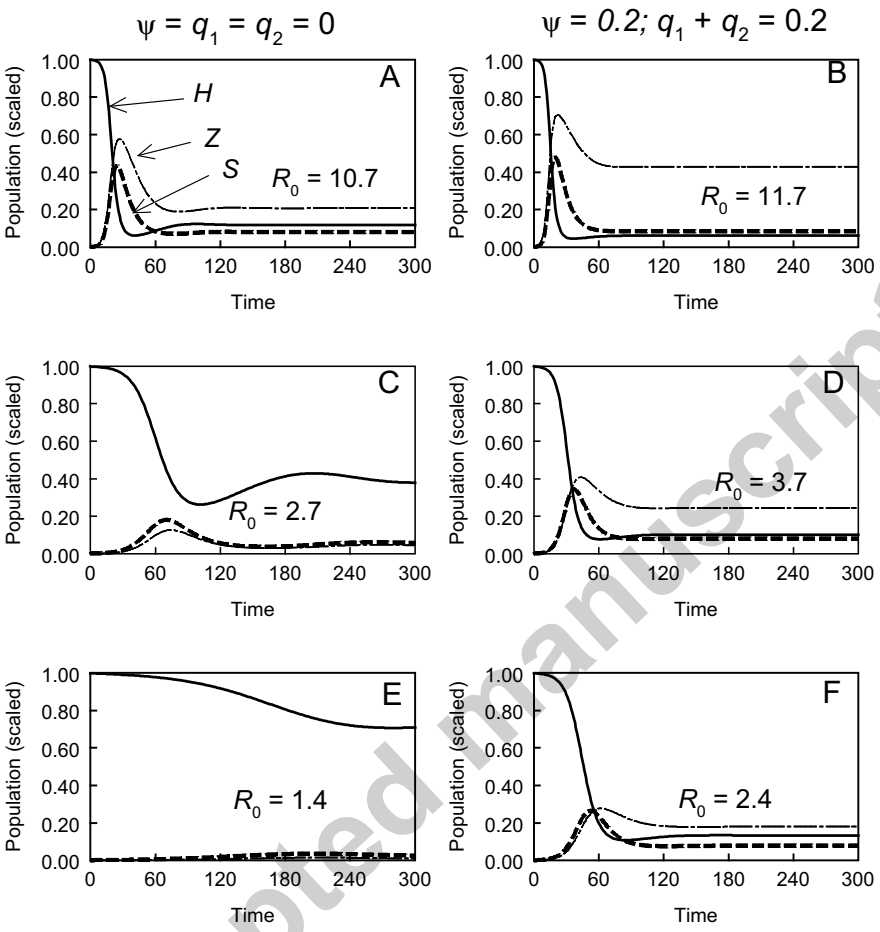


Fig. 2

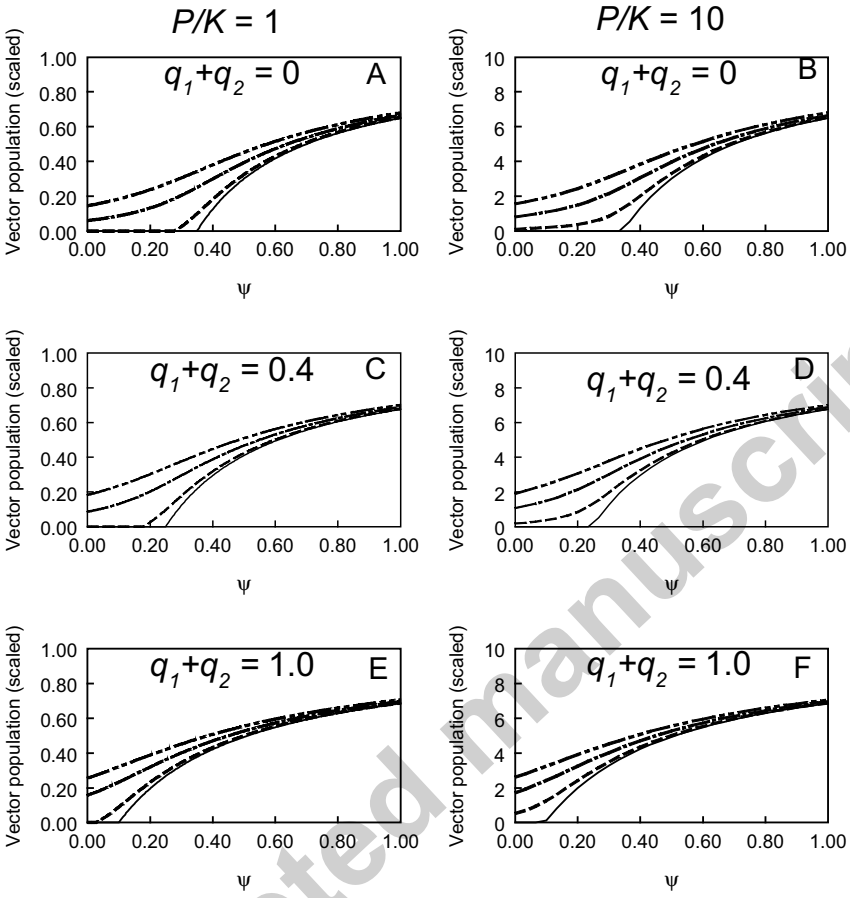


Fig. 3

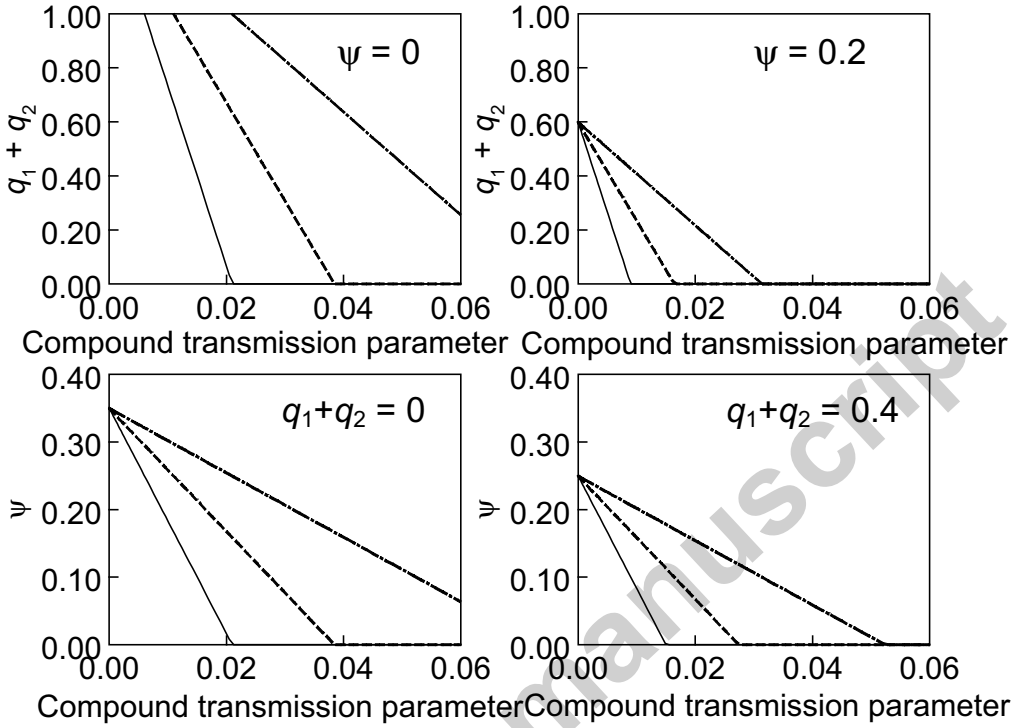
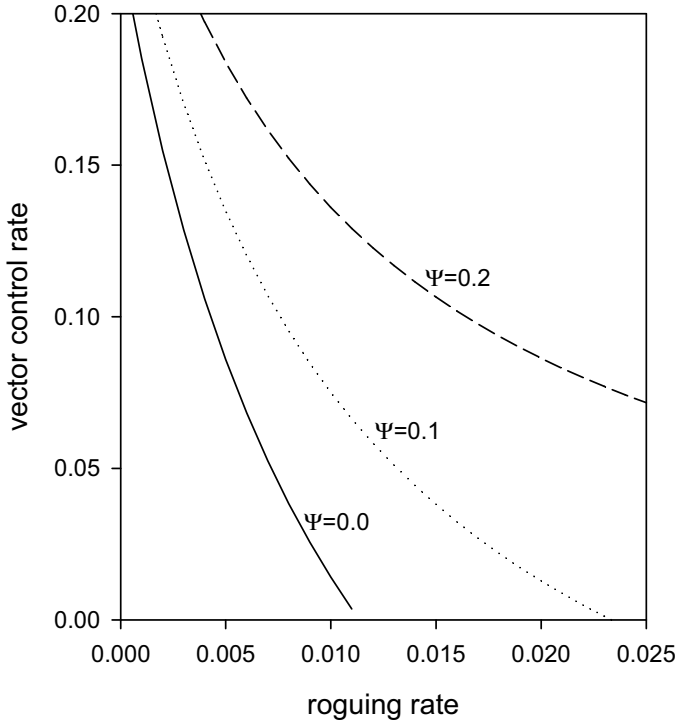


Fig. 4

(a)



(b)

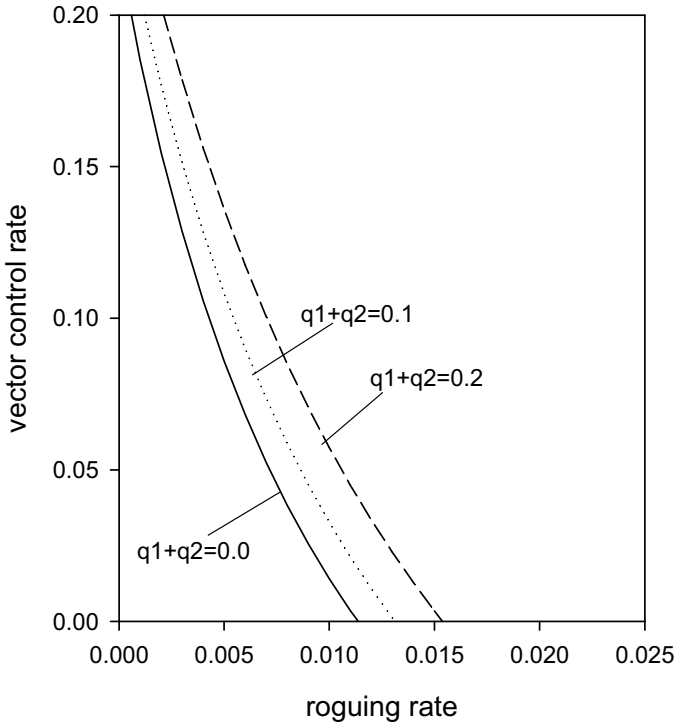
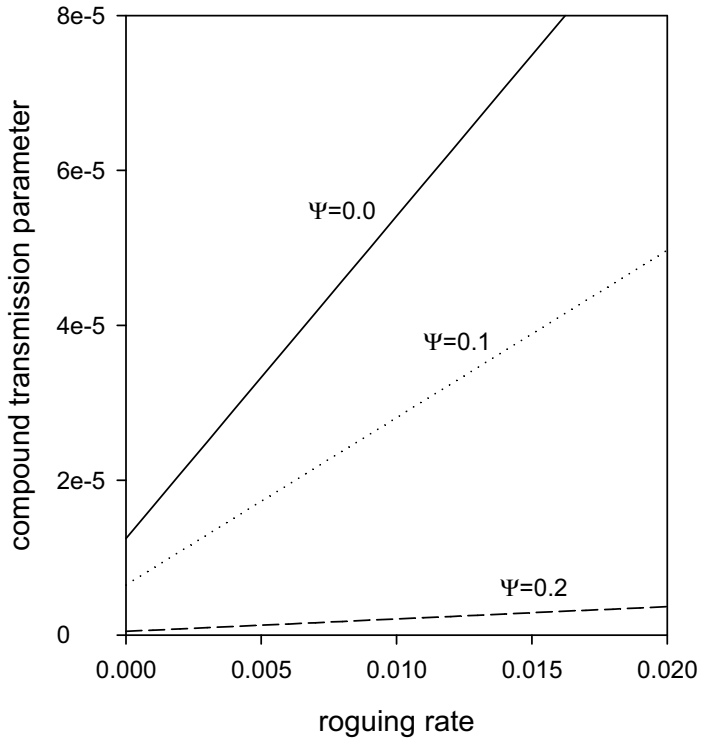


Fig. 5

(a)



(b)