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

27

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

30

31 **1. Introduction**

32

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

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

65 Most attention in plant virus epidemiology has been given to indirect
66 transmission involving arthropod vectors, mostly Homopteran insects, because of
67 their economic impact on crop plants. Based on a classification of transmission types
68 as non-persistent, semi-persistent, persistent-circulative and persistent-propagative we
69 have developed a general model which can be used to explore the epidemiological
70 consequences of transmission parameters appropriate for each class (Jeger *et al.*,
71 1998; Madden *et al.*, 2000). From a phytocentric perspective direct transmission has
72 been modelled in terms of vertical transmission through the use of vegetatively-
73 propagated planting material and its relative contribution to disease development

74 compared with indirect transmission by arthropod vectors (Jeger *et al.*, 2002). From
75 an arthropocentric perspective these models (Jeger *et al.*, 1998; Madden *et al.*, 2000)
76 give some attention to vertical transovarial transmission, known to be an important
77 ecological determinant of fitness in insect-vector vertebrate viruses (Labuda and
78 Nuttall, 2004; White *et al.*, 2005). With plant viruses transmitted in a persistent-
79 propagative manner, transovarial transmission is relatively frequent for some virus
80 genera (Hogehout *et al.*, 2008). However there has been little consideration of the
81 pathways and barriers involved in such transmission. The possibility of direct
82 horizontal transmission between vectors, e.g. during copulation or other contact, has
83 rarely been explored with only one documented case reported (Ghanim and Czosnek,
84 2000). The question can also be asked under what circumstances might direct
85 horizontal transmission between vectors be advantageous for plant virus survival in
86 the absence of the host plant: a question analogous to that posed for venereal
87 transmission of vertebrate viruses in the absence of viremic hosts (Tesh *et al.*, 1992).

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

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

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

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

124 and Tsai, 1998). Insect viruses of relevance in this context are in the Reoviridae.
125 Cypoviruses from various insect hosts are closely related to *Rice ragged stunt virus*
126 (RRSV) (Hagiwara *et al.*, 2002; Li *et al.*, 2006; Zhao *et al.*, 2003) with the implication
127 drawn that plant reoviruses originated from insect reoviruses. RRSV is also closely
128 related to *Nilaparvata lugens reovirus* (NLRV), more so than to other plant (or
129 animal) reoviruses (Upadhyaya *et al.*, 1998) and may have emerged from an insect
130 virus more recently than other plant reoviruses (Ikeda *et al.*, 2001). *N. lugens*, the
131 brown planthopper, is a serious pest of rice and is also the vector of RRSV (Hibino,
132 1996). NLRV does not cause disease in the insect host and does not multiply in rice
133 but can be re-acquired from rice by non-viruliferous *N. lugens* (Noda and Nakashima,
134 1995; Nakashima and Noda, 1995). In *N. lugens* there are several related reoviruses
135 that appear to be exclusively insect viruses and which have not been known to
136 replicate in rice or other plants. The question then arises as to why related reoviruses
137 have or have not evolved the ability to infect plants?

138 Vertical transmission of plant viruses in the vector with persistent-circulative
139 and persistent-propagative transmission is possible where there is transovarial
140 transmission from parent to progeny. With the geminiviruses the circulative pathway
141 has been well-described in whiteflies (Ghanim *et al.*, 2001; Czosnek *et al.*, 2002).
142 Transovarial transmission has been claimed for TYLCV (Goldman and Czosnek,
143 2002) although Bosco *et al.*, (2004) claimed only non-infectious viral DNA was
144 transmitted to the progeny, and thus transovarial transmission was not relevant
145 epidemiologically. In the leaf hopper *Nephotettix cincticeps*, the vector of the
146 reovirus, *Rice dwarf virus* (RDV) (Hibino, 1996), transovarial transmission was
147 confirmed as efficient and enabled the long-term maintenance of RDV in a leaf

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

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

158

159 **2. Model development**

160

161 *2.1 Basic model*

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

166 The plant model with no latent category is

$$167 \quad \frac{dH}{dt} = \beta(K - H) - \phi k_1 T Z \frac{H}{K} \quad (\text{healthy}) \quad (1a)$$

$$168 \quad \frac{dS}{dt} = \phi k_1 T Z \frac{H}{K} - (k_3 + \beta)S \quad (\text{infectious}) \quad (1b)$$

$$169 \quad \frac{dR}{dt} = k_3 S - \beta R \quad (\text{post-infectious}) \quad (1c)$$

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

174 The vector model with no latent category is

$$175 \quad \frac{dX}{dt} = \alpha(X + Z) \left[1 - q \frac{Z}{X + Z} \right] + \tau Z - \alpha X - \phi X \lambda T \frac{S}{K} \quad (\text{non-viruliferous}) \quad (1d)$$

$$176 \quad \frac{dZ}{dt} = \phi X \lambda T \frac{S}{K} - \tau Z - \alpha Z + \alpha(X + Z) q \frac{Z}{X + Z} \quad (\text{viruliferous}) \quad (1e)$$

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

179 Introduce the scaling

$$180 \quad \bar{H} = \frac{H}{K}, \bar{S} = \frac{S}{K}, \bar{R} = \frac{R}{K}; \bar{X} = \frac{X}{K}, \bar{Z} = \frac{Z}{K}$$

181 then substituting and dropping overbars gives

$$182 \quad \frac{dH}{dt} = \beta(1 - H) - \phi k_1 T Z H \quad (2a)$$

$$183 \quad \frac{dS}{dt} = \phi k_1 T Z H - (k_3 + \beta)S \quad (2b)$$

$$184 \quad \frac{dZ}{dt} = \phi \left[\frac{P}{K} - Z \right] \lambda T S - [\tau + \alpha(1 - q)]Z \quad (2c)$$

185 with the equations for R and X redundant.

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

$$187 \quad H^* = \frac{\beta}{\beta + k_1 \phi T Z^*}, \quad S^* = \frac{\beta}{k_3 + \beta} \cdot \frac{k_1 \phi T Z^*}{\beta + k_1 \phi T Z^*}$$

188 where

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

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

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

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 $(Z/P)^2$.

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

212

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

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

224 Direct transmission occurs through two processes:

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

227 Assume that a fraction ϕ of the non-viruliferous X individuals in an XZ copulation
 228 gets infected with the virus, then $2\phi\frac{C}{P}$ is the per capita rate of transmission due to

229 copulation, which is simply the sexual transmission rate. This implies that the direct

230 transmission infection rate equals $2\phi\frac{C}{P}\frac{XZ}{P}$.

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

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

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

237 (ii) ZZ : produces both X and Z . The production rate for X is

238 $(1 - q_1)(1 - q_2)(\alpha P)\xi(Z/P)^2$; the production rate for Z is

239 $(q_1 + q_2 - q_1q_2)(\alpha P)\xi(Z/P)^2$.

240 (iii) XZ : produces X and Z . The production rate of X is

241 $((1 - q_1) + (1 - q_2))(\alpha P)\xi(XZ/P^2)$; the production rate of Z is

242 $(q_1 + q_2)(\alpha P)\xi(XZ/P^2)$.

243 Note that adding these three rates gives $\alpha\xi P$, the overall production rate. Thus

244 writing $\alpha' = \alpha\xi$ and $\psi = 2\phi\frac{C}{P}$, the model becomes

$$245 \quad \frac{dX}{dt} = \frac{\alpha'}{P} \left(X^2 + (1 - q_1)(1 - q_2)Z^2 + ((1 - q_1) + (1 - q_2))XZ \right) + \tau Z - \alpha'X - \phi\lambda TX \frac{S}{K} - \psi \frac{XZ}{P}$$

$$246 \quad (4a)$$

$$247 \quad \frac{dZ}{dt} = \frac{\alpha'}{P} \left((q_1 + q_2 - q_1q_2)Z^2 + (q_1 + q_2)XZ \right) - \tau Z - \alpha'Z + \phi\lambda TX \frac{S}{K} + \psi \frac{XZ}{P} \quad (4b)$$

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

249

250 Now for $q_2 = 0$ (no transmission via the sperm cell), and using

251 $X^2 + (1 - q_1)Z^2 + (2 - q_1)XZ = X^2 + Z^2 + 2XZ - q_1(Z^2 + XZ) = P(P - q_1Z)$, we find

252 that the production rate term in 4a is the same as that in equation 1d, with α' replacing

253 α .

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

$$255 \quad \frac{dZ}{dt} = \frac{\alpha'K}{P} \left[(q_1 + q_2 - q_1q_2)Z^2 + (q_1 + q_2) \left(\frac{P}{K} - Z \right) Z \right] - \tau Z - \alpha'Z + \phi\lambda TS \left(\frac{P}{K} - Z \right) + \psi \frac{K}{P} Z \left(\frac{P}{K} - Z \right)$$

$$256 \quad (5)$$

257 where for convenience the overbars have been dropped.

258

259 3. Model analysis

260

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

265 The invasion criterion is derived in Appendix B as

$$266 \quad \frac{\phi k_1 T \phi \lambda T \frac{P}{K}}{(k_3 + \beta)(\tau + \alpha')} + \frac{\psi}{\tau + \alpha'} + \frac{\alpha'}{\tau + \alpha'} (q_1 + q_2) > 1 \quad (6)$$

267 The first term is simply the R_0 -value for indirect transmission (inequality 3), with α'
 268 replacing α . The second term is the direct transmission term per introduced vector
 269 multiplied by the average length of time a vector remains viruliferous. The term
 270 $\alpha'(q_1 + q_2)/(\tau + \alpha')$ represents the contribution of transovarial transmission to the R_0 -
 271 value and has a maximum value of 2 when $\tau = 0$, $q_1 = q_2 = 1$. Thus we conclude that
 272 the left hand side of inequality 6 gives an overall R_0 -value for the system described by
 273 equations 2a, 2b and 5,

$$274 \quad R_0(\text{overall}) = R_0(\text{indirect transmission}) +$$

$$275 \quad R_0(\text{direct, horizontal transmission}) +$$

276 R_0 (direct, transovarial transmission)

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

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

$$282 \quad \frac{\alpha'K}{P} \left[(q_1 + q_2 - q_1q_2)Z + (q_1 + q_2) \left(\frac{P}{K} - Z \right) \right] - (\tau + \alpha') + \psi \frac{K}{P} \left(\frac{P}{K} - Z \right) = 0$$

$$283 \quad \Rightarrow Z^* = \frac{-[\tau + \alpha'(1 - q_1 - q_2)] + \psi}{\frac{K}{P}[\psi + \alpha'q_1q_2]} \quad (7)$$

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

287

288 4. Numerical results

289

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

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

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

321

322 5. Modelling disease control options

323

324 The objective of plant disease control is to reduce R_0 to below 1. The main

325 plant disease control options to consider are:

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

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

334 when

$$335 \frac{\psi}{\tau + \alpha'} + \frac{\alpha'}{\tau + \alpha'} (q_1 + q_2) < 1$$

336 In other words direct sexual transmission, horizontal or vertical, should be sufficiently
337 small that the virus goes extinct if indirect vectored transmission to plants is very
338 small. Note also that vector control through α' affects both the indirect and direct
339 transmission terms in the expression for R_0 (the left hand side of inequality 6).

340 However, the horizontal transmission term $\psi / (\tau + \alpha')$ is unbounded, whereas the
341 transovarial term $\alpha' (q_1 + q_2) / (\tau + \alpha')$ is bounded by the value 2.

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

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

352 A better measure of the effect of changes in parameter values on R_0 and thus
 353 on disease control is obtained by calculating elasticities (Arino *et al.*, 2008) of both
 354 parameters in equation 6; where elasticity in $q_1 + q_2$ is calculated as

355 $\frac{(q_1 + q_2)}{R_0} \frac{\partial R_0}{\partial (q_1 + q_2)}$, and in ψ as $\frac{\psi}{R_0} \frac{\partial R_0}{\partial \psi}$. Clearly these elasticities are equal when

356 $\psi = \alpha'(q_1 + q_2)$. The value of α' used in the simulations (Figs. 1-5) was 0.12
 357 (probably a high value as $\alpha' = \xi\alpha$). This means that $q_1 + q_2$ must be about 8 times
 358 larger than ψ for transovarial transmission to have a greater relative effect than
 359 sexual transmission in reducing R_0 .

360

361 **6. Discussion**

362

363 In this paper we deal with issues arising from two complementary perspectives
 364 in viewing plant virus epidemics. Firstly from the phytocentric perspective. What are
 365 the implications, if any, on disease control options where there is direct horizontal
 366 and/or vertical transmission in the vector population? Secondly from the
 367 arthropocentric perspective what would be the advantage, if any, of indirect
 368 transmission through a plant for an insect virus where there is horizontal and/or
 369 vertical transmission in the vector population? We approached these two issues by
 370 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:

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

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

472

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

473

474 with internal steady-states

$$\hat{H} = \frac{\beta}{\beta + \phi k_1 T \hat{Z}} \quad (\text{A4})$$

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

$$\hat{Z} = \frac{-B \pm \sqrt{B^2 - 4 A C}}{2 A} \quad (\text{A6})$$

476

477 where

$$\begin{aligned} A &= \left(D - \frac{E K}{P} \right) - \psi \frac{K}{P} \\ B &= \frac{\beta}{\phi k_1 T} \left(D - \frac{E K}{P} \right) + (E - (\tau + \alpha')) - \frac{\beta \phi \lambda T}{k_3 + \beta} - \psi \frac{K}{P} \frac{\beta}{\phi k_1 T} + \psi \\ C &= \frac{\beta}{\phi k_1 T} (E - (\tau + \alpha')) + \frac{\beta \phi \lambda T}{k_3 + \beta} \frac{P}{K} + \psi \frac{\beta}{\phi k_1 T} \end{aligned}$$

479

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

480

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

481

482

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

484

485 In the absence of disease and viruliferous vectors

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

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

$$488 \quad \frac{dS}{dt} = \phi k_1 T Z - (k_3 + \beta) S \quad (B1)$$

$$489 \quad \frac{dZ}{dt} = \frac{\alpha' K}{P} \left[(q_1 + q_2 - q_1 q_2) Z^2 + (q_1 + q_2) \frac{ZP}{K} \right] - \tau Z - \alpha' Z + \phi \lambda T S \frac{P}{K} + \psi Z \frac{P}{K}$$

490 which by ignoring quadratic terms

$$491 \quad = \alpha' (q_1 + q_2) Z - \tau Z - \alpha' Z + \phi \lambda T S \frac{P}{K} + \psi Z \quad (B2)$$

492 with the Jacobian

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

494 and determinant

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

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

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

498 i.e.,

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

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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 ($q_1 + q_2$).

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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		- parthenogenic (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	6.4×10^{-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

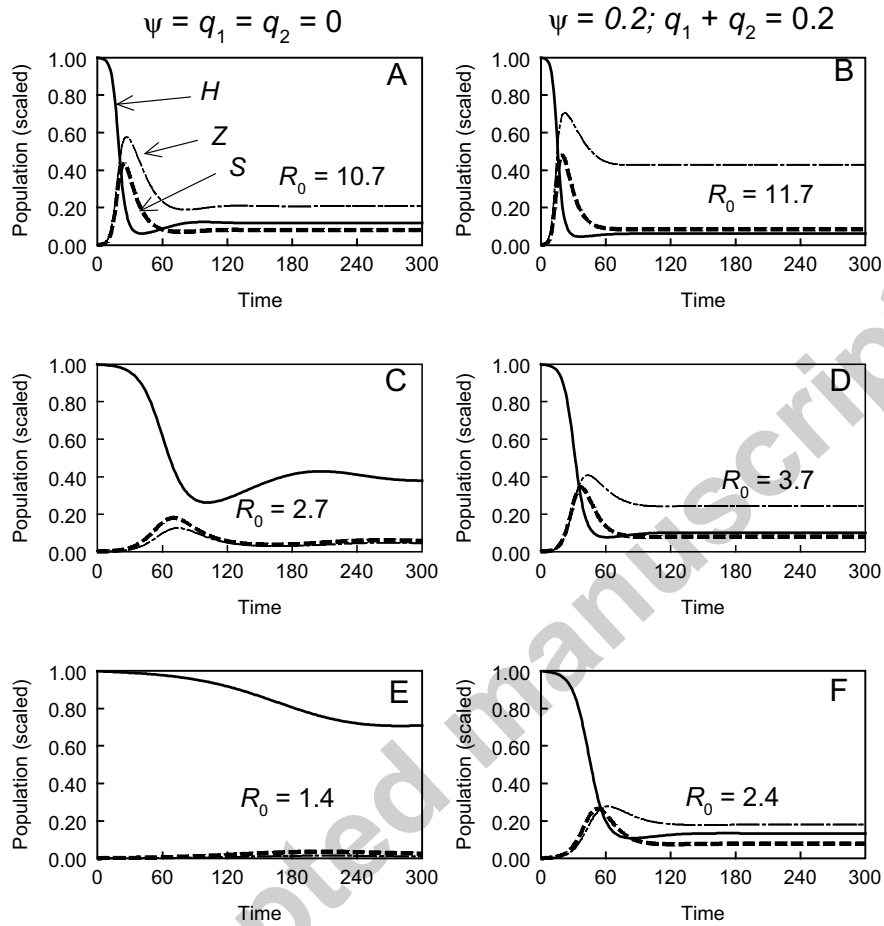


Fig. 2

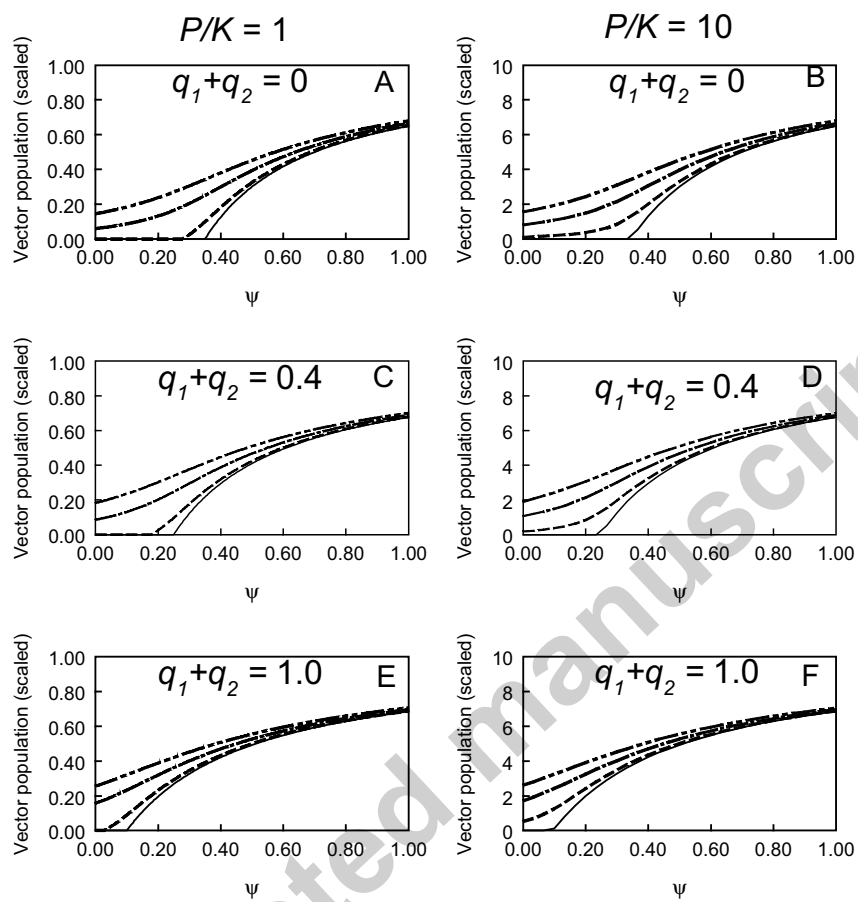


Fig. 3

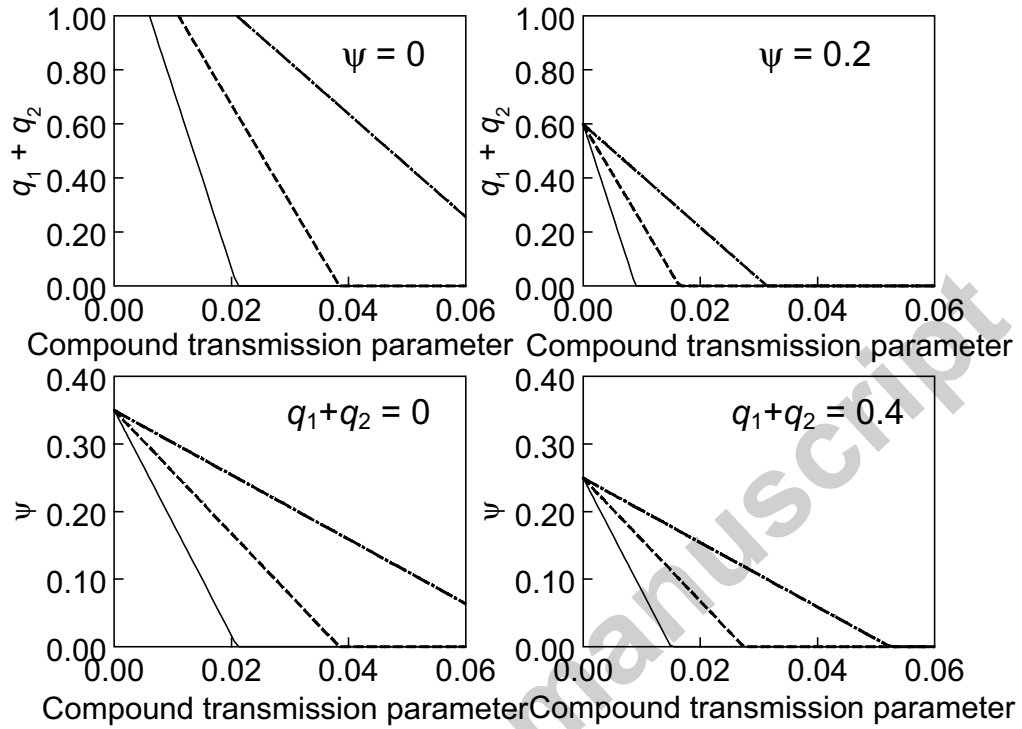


Fig. 4

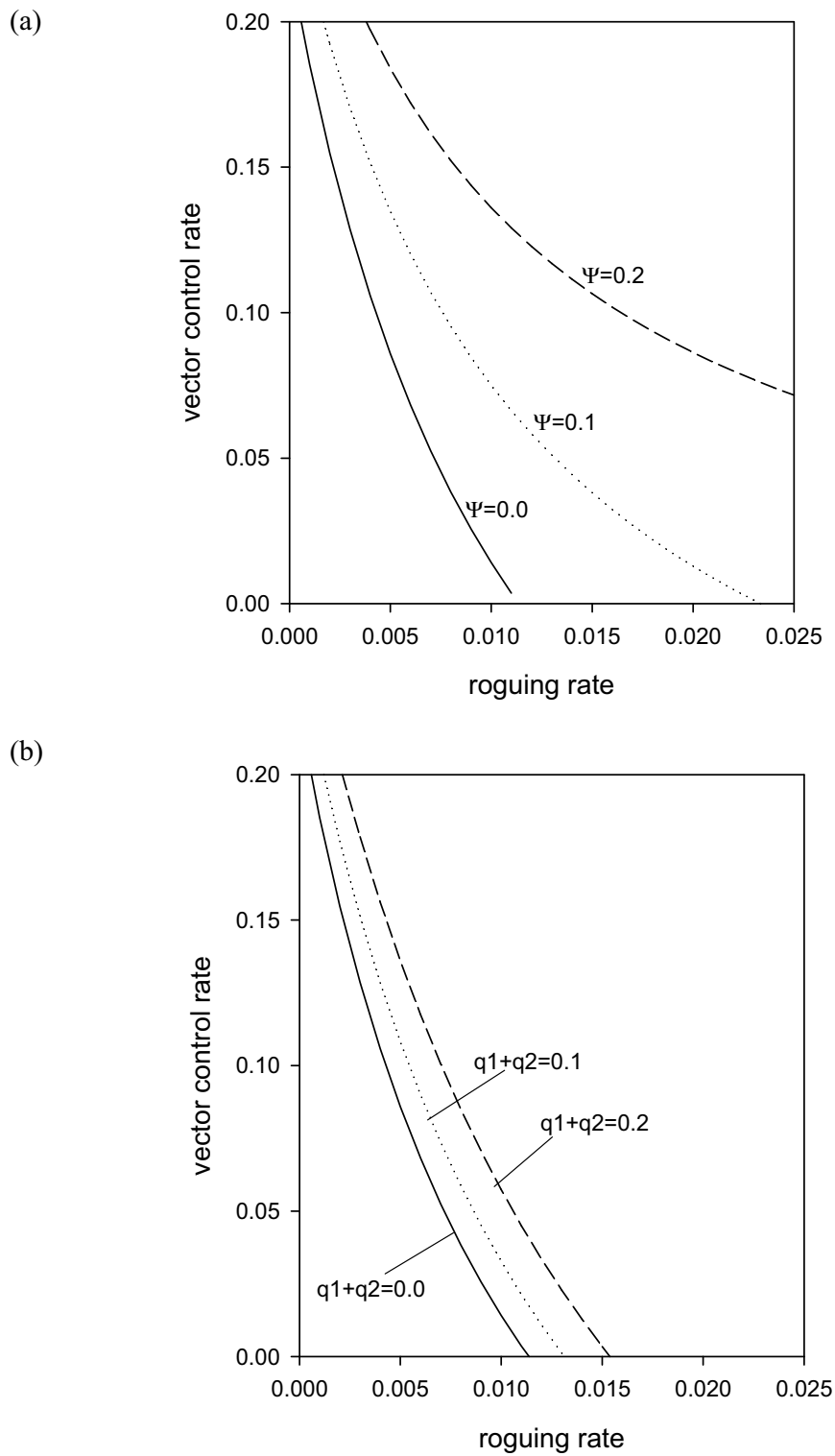


Fig. 5

