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► To cite this version:

Marta Zaffaroni, Loup Rimbaud, Ludovic Mailleret, Nik J Cunniffe, Daniele Bevacqua. The role of interference between vectors in control of plant diseases. 2021. hal-03265550

HAL Id: hal-03265550

<https://hal.inrae.fr/hal-03265550>

Preprint submitted on 21 Jun 2021

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The role of interference between vectors in control of plant diseases

Marta Zaffaroni^{1,2} marta.zaffaroni@inrae.fr

Loup Rimbaud³ loup.rimbaud@inrae.fr

Ludovic Mailleret^{4,5} ludovic.mailleret@inrae.fr

Nik J. Cunniffe^{6*} njc1001@cam.ac.uk

Daniele Bevacqua^{1*} daniele.bevacqua@inrae.fr

Affiliation ¹ INRAE, UR1115 Plantes et Systèmes de culture Horticoles (PSH), Site Agroparc, 84914 Avignon, France. ² Present Address: INRAE, Bordeaux Sciences Agro, ISVV, Santé et Agroécologie du Vignoble, 33140, Villenave d'Ornon, France. ³ INRAE, UR0407 Pathologie Végétale, Domaine St Maurice, 84143 Montfavet, France. ⁴ Université Côte d'Azur, Inria, INRAE, CNRS, Sorbonne Université, Biocore team, Sophia Antipolis, France. ⁵ Université Côte d'Azur, INRAE, CNRS, ISA, 06903, Sophia Antipolis, France. ⁶ Department of Plant Sciences, University of Cambridge, Cambridge CB2 3EA, United Kingdom. * Both authors contributed equally to this work.

Corresponding author: Marta Zaffaroni, marta.zaffaroni@inrae.fr

1

Abstract

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Aphids are the primary vector of plant viruses. Transient aphids, which probe several plants per day, are considered to be the principal vectors of non-persistently transmitted (NPT) viruses. However, resident aphids, which can complete their life cycle on a single host and are affected by agronomic practices, can transmit NPT viruses as well. Moreover, they can interfere both directly and indirectly with transient aphids, eventually shaping plant disease dynamics. By mean of an epidemiological model, originally accounting for ecological principles and agronomic practices, we explore the consequences of fertilization and irrigation, pesticide deployment and roguing of infected plants on the spread of viral disease in crops. Our results indicate that the spread of NPT viruses can be *i*) both reduced or increased by fertilization and irrigation, depending on whether the interference is direct or indirect; *ii*) counter-intuitively increased by pesticide application and *iii*) reduced by roguing infected plants. We show that a better understanding of vectors' interactions would enhance our understanding of disease transmission, supporting the development of disease management strategies.

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Keywords: aphid-borne pathogen; disease ecology; epidemiological model; inter-specific interference; non-persistently transmitted virus; plant–herbivore interactions; plant–pathogen interactions.

23 Introduction

24 Aphids transmit nearly 30% of known plant virus species (Gray and Baner-
25 jee, 1999; Brault et al., 2010). Aphids vector the majority of non-persistent
26 transmitted (NPT) viruses with virus particles (virions) remaining loosely
27 attached to the insect's stylets (Ng and Falk, 2006; Brault et al., 2010). Ac-
28 cording to this transmission mode, virions are rapidly acquired from infected
29 plants, briefly retained by their vector and inoculated to healthy plants dur-
30 ing plant sampling probes (Nault, 1997; Ng and Falk, 2006). NPT viruses
31 are responsible for severe damage to crops (Andret-Link and Fuchs, 2005).
32 For instance, *Plum pox virus* (PPV), which is vectored by more than 20
33 aphid species worldwide, is responsible for sharka, the most devastating dis-
34 ease of stone fruit trees (Rimbaud et al., 2015a). *Potato virus Y* (PVY),
35 which is spread by more than 50 aphid species, threatens the production of
36 a range of solanaceous crops, including potato, tomato, tobacco, and pepper
37 (MacKenzie et al., 2013).

38 The epidemiology of NPT viruses is closely related to the behaviour of
39 aphid vectors, in particular to *i*) aphids' ability to acquire and inoculate
40 the virus during sampling probes and *ii*) their propensity for moving among
41 plants (Ng and Perry, 2004). With respect to a given plant host species, aphid
42 species can be classified as: "residents", which under favourable conditions,
43 spend most of their life on the same host plant individual, or "transients",
44 which land and probe numerous plant individuals in the same day (Van Em-
45 den and Harrington, 2007; Fereres and Moreno, 2009). Although transient
46 aphids are commonly considered the principal vectors of NPT viruses, resi-

47 dent aphids can also efficiently transmit NPT viruses when they are induced
48 to change their host, for example in response to crowding or to changes in
49 plant nutrient contents (Müller et al., 2001). For instance, the green peach
50 aphid *Myzus persicae*, despite being a resident aphid species that colonises
51 peach *Prunus persica* and potato *Solanum tuberosum* plants (Van Emden
52 and Harrington, 2007; Robert et al., 2000), was observed to be an efficient
53 vector of two NPT viruses (PPV and PVY) at laboratory conditions (Robert
54 et al., 2000; Rimbaud et al., 2015a,b). In addition, the presence of resident
55 aphids may affect transient aphids' behaviour (Kaplan and Denno, 2007; Bird
56 et al., 2019; Chisholm et al., 2019). For example, in their experiment with
57 three aphid species, Mehrparvar et al. (2014) showed that aphid presence on
58 a plant discourages other aphid species to visit the same plant.

59 Different mechanisms of interference might characterize interactions be-
60 tween resident and transient aphid species. Resident aphids can interfere
61 *i*) directly through the production of pheromones that can have a repelling
62 effect towards other aphid species (Van Emden and Harrington, 2007); or
63 *ii*) indirectly, by inducing the host plant to produce volatile compounds as
64 a defensive mechanism, which may lower plant attractiveness to other aphid
65 species (Zust and Agrawal, 2016). Such interference mechanisms are likely
66 to reduce the number of plants visited by transient aphids in a given area
67 and increase their propensity for leaving the area (Levins and Culver, 1971;
68 Nee and May, 1992).

69 As far as we know, the effect of the interference between resident and
70 transient aphids on the spread of NPT viruses has never been explored.
71 Understanding NPT viruses spread is complex because experimentation is

72 costly and difficult: symptoms may be difficult to detect and experimen-
73 tal trials in the vicinity of susceptible commercial crops may be restricted
74 (Cunniffe et al., 2014; Picard et al., 2017). Mathematical models are thus
75 particularly useful to provide complementary insights on virus spread (Jeger,
76 2020), and to design and test management strategies, while circumventing
77 the difficulties associated with experiments [*e.g.* Fabre et al. (2012); Rim-
78 baud et al. (2015b); Cunniffe and Gilligan (2020)]. Numerous models have
79 been developed to study the role of vector population dynamics and vector-
80 host-pathogen interactions on the spread of NPT viruses [*e.g.* Nakazawa
81 et al. (2012); Sisterson and Stenger (2016); Shaw et al. (2017); Jeger et al.
82 (2018); Allen et al. (2019); Donnelly et al. (2019); Crowder et al. (2019)].
83 Shaw et al. (2017) developed a model to assess the contributions of vector
84 life history traits (*e.g.* growth rates, fecundity, and longevity) and behavior
85 (*e.g.* vector preferences for settling and feeding) to pathogen spread. Crow-
86 der et al. (2019) developed a model where the vector life history traits and
87 behaviour were varied to explore the effect of interaction (*e.g.* predation,
88 competition and mutualism) between a vector and a non vector species on
89 the spread of plant pathogens. However, all existing studies consider only
90 a single vector species per virus, which limits the possibility to assess the
91 effects of interference between two or more vector species.

92 In the present work, we develop a general epidemiological model which
93 describes the temporal variation of the number of susceptible and infected
94 plants, and of the number of non viruliferous and viruliferous resident and
95 transient aphids in a single field. We apply the model to explore the role of
96 inter-specific interference upon resident and transient aphid behaviour and

97 the resulting effects on the invasion, persistence and control of NPT viruses
98 in agroecosystems. We use the model to analyze the effects of common agri-
99 cultural practices, such as fertilization and irrigation, pesticide application
100 and roguing, upon the spread of NPT viruses. We apply the model to a
101 general pathosystem composed of a NPT virus vectored by a resident and a
102 transient aphid species.

103 **Methods**

104 **Model outlines and assumptions**

105 The model is schematically represented in Fig.1. The plant population (plant
106 ha^{-1}) is structured into two compartments: susceptible (S) and infected (I).
107 Both resident (subscript "R" hereafter) and transient (subscript "T" here-
108 after) aphid populations (aphid plant^{-1}) are structured into non viruliferous
109 (X_R and X_T) and viruliferous (Z_R and Z_T). The total number of plants
110 per hectare is N_P ($N_P = S + I$), the average number of resident aphids per
111 plant is N_R ($N_R = X_R + Z_R$) and the average number of transient aphids
112 per plant is N_T ($N_T = X_T + Z_T$). A susceptible plant can be infected if it
113 enters in contact with a viruliferous aphid, resident (Z_R) or transient (Z_T).
114 The probability per unit time that a susceptible plant becomes infected (*i.e.*
115 force of infection) depends on *i*) the rate of contact between plant and aphid
116 $\Lambda_i(X_i + Z_i)$ ($i = R, T$) where Λ_i is the number of plants visited by an aphid
117 per unit time and $(X_i + Z_i)$ is the average number of aphids per plant, *ii*)
118 the probability $\frac{Z_i}{X_i + Z_i}$ that the contact is indeed with a viruliferous aphid and

119 *iii*) the probability δ_i that the contact leads to virus inoculation. Therefore
120 the number of infected plants per unit time at the plant population scale is

$$121 \sum_{i=R,T} \Lambda_i \delta_i Z_i S.$$

122 We assumed that infected plants are removed at a *per capita* rate ρ and
123 that both susceptible and infected plants are harvested at a *per capita* rate
124 θ . Furthermore, Γ new susceptible plants are planted per unit time. A non
125 viruliferous aphid can become viruliferous if it enters in contact with an
126 infected plant. The probability per unit time that a non viruliferous aphid
127 acquires the virus from an infected plant depends on *i*) the rate of contact
128 between one aphid and a plant Λ_i , *ii*) the probability $\frac{I}{N_P}$ that the contact is
129 indeed with an infected plant and *iii*) the probability ε_i that the contact leads
130 to virus acquisition. Viruliferous aphids lose viruliferousness at a clearance
131 rate γ . We assume that the population of resident aphids varies following a
132 logistic function with a density dependent *per capita* birth rate $r(1 - \frac{N_r}{h})$,
133 where r is the intrinsic growth rate and h is the plant hosting capacity with
134 respect to aphid population (*i.e.* the resident aphid population size at which
135 birth rate is zero), and a constant aphid mortality rate μ . Resident aphids can
136 leave their host plant and move to another one as a response to unfavourable
137 environmental conditions (Müller et al., 2001): we assumed $\Lambda_T > \Lambda_R >$
138 0 , due to the different moving behaviour of transient and resident aphids.
139 We assume that an average of λ transient aphids per plant immigrate into
140 the system (*i.e.* a patch of 1 ha) per unit time and a fraction π of them
141 are viruliferous. Such a fraction depends on the disease prevalence in the
142 surrounding area. Transient aphids emigrate from the system or die at a
143 rate τ , which is the inverse of their average sojourn time in the system.

144 We assume that resident aphids exert two types of interference towards
 145 transient aphids, "visiting" and "emigration" interference, that will be pre-
 146 sented in the following sections.

147 Model equations

148 The model outlined above can be described by the following set of differential
 149 equations:

$$\left\{ \begin{array}{l} \dot{S} = \Gamma - (\Lambda_R \delta_R Z_R + \Lambda_T \delta_T f(\cdot) Z_T) S - \theta S \\ \dot{I} = (\Lambda_R \delta_R Z_R + \Lambda_T \delta_T f(\cdot) Z_T) S - (\rho + \theta) I \\ \dot{X}_R = r N_R \left(1 - \frac{N_R}{h}\right) - \mu X_R - \Lambda_R \varepsilon_R \frac{I}{N_P} X_R + \gamma Z_R \\ \dot{Z}_R = \Lambda_R \varepsilon_R \frac{I}{N_P} X_R - (\gamma + \mu) Z_R \\ \dot{X}_T = (1 - \pi) \lambda - \Lambda_T \varepsilon_T f(\cdot) \frac{I}{N_P} X_T + \gamma Z_T - \tau g(\cdot) X_T \\ \dot{Z}_T = \pi \lambda + \Lambda_T \varepsilon_T f(\cdot) \frac{I}{N_P} X_T - \gamma Z_T - \tau g(\cdot) Z_T \end{array} \right. \quad (1)$$

150 Where the dot represents the derivative with respect to time t , and param-
 151 eters are as in Table 1. Functions $f(\cdot)$ and $g(\cdot)$ represent, respectively, "vis-
 152 iting interference" and "emigration interference" exerted by resident aphids
 153 towards transient aphids. Details on their functional forms are given in the
 154 following section.

155 The set of differential equations can be used to represent a plant virus
 156 epidemic under very general circumstances. To gain insight on disease trans-
 157 mission, we assume that the total number of plants (N_P) is constant (*i.e.*
 158 every harvested or rogued plant is immediately replaced), which implies that

159 the number Γ of new susceptible plant planted per unit time is given by

$$\Gamma = \theta S + (\rho + \theta)I \quad (2)$$

160 where θ is plant harvesting rate and ρ is infected plant roguing rate. More-
161 over, the total number of transient aphids per plant in absence of resident
162 aphids (T) is constant, which implies that the average number λ of transient
163 aphids immigrating into the system per plant per unit time is given by

$$\lambda = \tau T \quad (3)$$

164 **Modelling interference between resident and transient** 165 **aphids**

166 We assume that interference exerted by resident towards transient aphids
167 can independently induce them to: *i*) visit fewer plants per unit time (visit-
168 ing interference); and/or *ii*) reduce the average sojourn time in the system
169 (emigration interference). Moreover, for both visiting and emigration inter-
170 ference, we consider two interference scenarios:

- 171 • direct [*e.g.* competition for space, (Van Emden and Harrington, 2007)],
172 where interference depends upon the density of resident aphids on the
173 host ($\frac{N_R}{h}$). This implies that, at the same aphid abundance (N_R),
174 the exerted interference is weaker on a bigger plant (*i.e.* higher plant
175 hosting capacity h).
- 176 • Indirect [*e.g.* release of plant volatiles, (Zust and Agrawal, 2016)],

177 where interference depends upon the absolute number of resident aphids
178 and it is independent from the plant hosting capacity.

179 Agricultural practices, such as fertilization and irrigation, that influence plant
180 size, may have different effects on plant virus epidemics according to which in-
181 terference scenario is under consideration. Forms of interference intermediate
182 between these two extreme scenarios are possible and they can be considered
183 in our model, as presented in the *Supplementary Information*.

184 **Visiting interference**

Visiting interference controls the proportionate decrease in the rate at which
transient aphids visit plants, via two functional forms $f(\frac{N_R}{h})$ and $f(N_R)$,
respectively for direct and indirect interference scenarios :

$$f\left(\frac{N_R}{h}\right) = \frac{1}{1 + \left(\nu_1 \frac{N_R}{h}\right)^{\alpha_1}} \quad f(N_R) = \frac{1}{1 + \left(\frac{\nu_1}{h_R} N_R\right)^{\alpha_1}} \quad (4)$$

185 These are a generalisation of the competition function proposed by Bel-
186 lows (1981), extending in continuous time the model of Smith and Slatkin
187 (1972). They are sufficiently flexible to account for a range of possible types
188 of interference (Fig. 2). The “strength” parameter ν_1 controls the magnitude
189 of interference, and so the density of residents that is required to appreciably
190 affect the behaviour of transient aphids. In our model the value of ν_1 is de-
191 fined in reference to the direct interference scenario. To assure that its value
192 is biologically relevant also in the indirect interference scenario, we scale it
193 with the "reference" value of plant hosting capacity (h_R). This implies that,
194 when $h = h_R$ - all other things being equal (*i.e.* the values of α_1 and N_R) - the

195 value of the interference function is the same independently from the under-
196 lying interference scenario. The “curvature” parameter α_1 , controls whether
197 the visiting function presents an inflection point (for $\alpha_1 > 1$) or not (for $0 <$
198 $\alpha_1 \leq 1$).

199 **Emigration interference**

Emigration interference controls the proportionate increase in the rate at
which transient aphids leave the system, via two functional form $g(\frac{N_R}{h})$ and
 $g(N_R)$, respectively for direct and indirect interference scenarios:

$$g\left(\frac{N_R}{h}\right) = 1 + \left(\nu_2 \frac{N_R}{h}\right)^{\alpha_2} \quad g(N_R) = 1 + \left(\frac{\nu_2}{h_R} N_R\right)^{\alpha_2} \quad (5)$$

200 The emigration interference function is characterized by two parameters
201 determining strength and curvature (ν_2 and α_2), and, in case of indirect in-
202 terference scenario, also by the parameter h_R . All three parameters have
203 identical interpretations to those controlling the visiting interference. In the
204 most general form of our model, the analogous parameters for the two func-
205 tions are entirely independent (*e.g.* $\alpha_1 \neq \alpha_2$), which provides sufficient flexi-
206 bility to allow inter-specific interference to affect visiting and/or emigration
207 independently. However in all numerical work in this paper we assumed that
208 $\nu_1 = \nu_2 = \nu$ and $\alpha_1 = \alpha_2 = \alpha$, and so, in turn, that proportionate effects of
209 the density of resident aphids on visiting and emigration rate were similar.

210 **Agricultural practices and disease control**

211 A number of agricultural practices are commonly used to *i*) increase plant
212 growth, *ii*) control aphid populations and *iii*) mitigate the effect of plant
213 diseases. The effect of these practices can be taken into account by modifying
214 the values of some model parameters from their reference value. Practices
215 such as fertilization or irrigation, commonly used to foster crop growth, may
216 increase the abundance of resident aphids dwelling on the plants (Huberty
217 and Denno, 2006; Tamburini et al., 2018) and in our model this translates
218 into an increase of the plant hosting capacity h (while parameter h_R is not
219 varied). Pesticides are commonly used in crops to reduce the number of
220 resident aphids and in our model this translates into an increase of resident
221 aphid mortality μ , while the dynamics of transient aphids is not affected
222 (Perring et al., 1999). In case of spread of plant diseases, frequently producers
223 try to identify as soon as possible the infected plants and replace them with
224 new healthy plants, a practice known as roguing (Sisterson and Stenger,
225 2013). To represent the discrete time process of roguing in our continuous
226 time model, we set the rate of removal of infected host plants (ρ) such that
227 the average period for which plants are symptomatically infected is equal to
228 one half of the interval between successive rounds of roguing (Cunniffe et al.,
229 2014).

230 **Methods of analysis**

231 We first determine the system equilibria and the basic reproduction number
232 of the disease, R_0 . Then, we explore the responses of R_0 to variations of

233 the aforementioned agricultural practices (modeled through parameters h , μ
234 and ρ) under the direct and indirect interference scenarios. In particular,
235 we vary one parameter at a time and we explore the effectiveness of the
236 respective agricultural practices in controlling and eradicating the disease.
237 Finally, we analyse the effectiveness of combinations of agricultural practices
238 in controlling and eradicating the disease by exploring the response of R_0 to
239 simultaneous variation of two parameters at a time. In our analysis, we use
240 biologically plausible parameter values and ranges to reflect a broad range
241 of single host - multi vector system rather than restricting the analysis to a
242 specific system.

243 Results

244 Equilibrium analysis

245 The behaviour of the model at equilibrium is summarized in Table 2. When
246 $\mu \geq r$ (*i.e.* the mortality rate of resident aphids is larger than their intrinsic
247 growth rate), resident aphids are not able to survive ($\bar{X}_R = \bar{Z}_R = 0$) and
248 the disease is spread exclusively by transient aphids. When $\mu < r$ both
249 resident and transient aphids are present in the system and may spread
250 the disease. When all the incoming transient aphids are non viruliferous
251 (*i.e.* $\pi = 0$), the disease is able to persist only if the basic reproduction
252 number (R_0 , presented in the following section) is higher than 1, otherwise the
253 disease is eradicated (Diekmann et al., 1990). When a fraction of incoming
254 transient aphids is viruliferous ($\pi > 0$), the disease is always able to persist,

255 regardless of whether the basic reproduction number is smaller or larger than
256 1, because there will always be an influx of some new viruliferous aphids into
257 the system, and infections of plants will result not just from infected plants
258 in the system, but also from viruliferous individuals originating from outside
259 the system [similar to Madden et al. (2000)]. The patterns presented in Table
260 2 can be explained analytically, with the mathematical details derived in the
261 *Supplementary Information*.

262 **The basic reproduction number**

263 In our system, when there is no immigration of viruliferous aphids ($\pi = 0$),
264 the basic reproduction number R_0 is expressed as

$$R_0 = \frac{1}{\rho + \theta} \left(\frac{\Lambda_R^2 \delta_R \varepsilon_R \bar{N}_R}{\gamma + \mu} + \frac{\Lambda_T^2 \delta_T \varepsilon_T f(\cdot)^2 \bar{N}_T}{\gamma + \tau g(\cdot)} \right) \quad (6)$$

265 and it can be written as the sum of two components, $R_0 = R_0^R + R_0^T$, where
266 $R_0^R = \frac{\Lambda_R^2 \delta_R \varepsilon_R \bar{N}_R}{(\rho + \theta)(\gamma + \mu)}$ and $R_0^T = \frac{\Lambda_T^2 \delta_T \varepsilon_T f(\cdot)^2 \bar{N}_T}{(\rho + \theta)(\gamma + \tau g(\cdot))}$ (see the *Supplementary Information*
267 for further details). The first component accounts for disease transmission by
268 resident aphids and the second by transient aphids. Such a representation
269 of the basic reproduction number is typical for plant disease models with
270 multiple routes of transmission (Jeger et al., 2009; Cunniffe and Gilligan,
271 2010; Cunniffe et al., 2012).

272 In the equation of the basic reproduction number, the term $(\rho + \theta)^{-1}$
273 indicates the average time spent by a plant as infected, before it is rogued
274 or harvested; the terms $(\gamma + \mu)^{-1}$ and $(\gamma + \tau g(\cdot))^{-1}$ indicate the average
275 time spent by a viruliferous aphid, respectively resident or transient, in the

276 system. The terms $\Lambda_R \delta_R \bar{N}_R$ and $\Lambda_T \delta_T f(\cdot) \bar{N}_T$ represent the rates at which a
277 susceptible plant is infected by resident or transient aphids, respectively. The
278 terms \bar{N}_R and \bar{N}_T indicate the number of, respectively, resident and transient
279 aphids per plant at the steady state. The terms $\Lambda_R \varepsilon_R$ and $\Lambda_T \varepsilon_T f(\cdot)$ are
280 the rates at which a resident or a transient aphid acquires the virus from
281 an infected plant, respectively. The terms $f(\cdot)$ and $g(\cdot)$ are, respectively,
282 the function for visiting and emigration interference evaluated at the steady
283 state, for the direct interference scenario [$f(\cdot) = f(\frac{\bar{N}_R}{h})$ and $g(\cdot) = g(\frac{\bar{N}_R}{h})$]
284 and for the indirect interference scenarios ($f(\cdot) = f(\bar{N}_R)$ and $g(\cdot) = g(\bar{N}_R)$].

285 The size of the resident aphid population (\bar{N}_R) appears both at the nu-
286 merator of the R_0^R component and as an argument of the interference func-
287 tions $f(\cdot)$ and $g(\cdot)$ which respectively decrease and increase with \bar{N}_R . This
288 implies that the resident component, R_0^R , of the basic reproduction number
289 increases with \bar{N}_R while the transient component, R_0^T , decreases with it. Ac-
290 cording to the assumed population dynamics of resident and transient aphids,
291 $\bar{N}_R = h(1 - \mu/r)$ if $\mu < r$, $\bar{N}_R = 0$ if $\mu \geq r$ and $\bar{N}_T = \lambda/(\tau g(\cdot))$ (see the *Sup-*
292 *plementary Information* for further details). This suggests that the response
293 of the basic reproduction number to h , μ and r might be non-monotonic.

294 Note that, whenever the basic reproduction number is higher than 1, the
295 disease is not eradicated and the incidence of virus infection in plant can be
296 computed as $\frac{\bar{I}}{N_P}$, where \bar{I} is the size of infected plant population at equilib-
297 rium and N_P is the total number of plants in the system (assumed constant).
298 The response of the incidence of virus infection in plants to variation of pa-
299 rameters h , μ and ρ correlates strongly with that of R_0 (see Fig. 2 in the
300 *Supplementary Information*).

301 **The role of plant hosting capacity**

302 The response of the basic reproduction number to aphid hosting capacity is
303 summarized in Fig. 3A. When the system conditions can sustain a popula-
304 tion of resident aphids (*i.e.* $r > \mu$), its equilibrium value increases with the
305 plant hosting capacity h . This always translates into an increase of the term
306 R_0^R . On the other hand, the effect of increasing h on the term R_0^T is mediated
307 by the interference functions, $f(\cdot)$ and $g(\cdot)$. Such an effect is null when the
308 interference is direct (see equations 4 and 5, for $\bar{N}_R = h(1 - \mu/r)$) and it is
309 negative when the interference between aphids is indirect. Increasing plant
310 size, and consequently its hosting capacity, is followed by an increase of the
311 population of resident aphids dwelling on a plant. This has no effect on the
312 transient aphid population when the interference is direct, because the den-
313 sity of resident aphids is kept constant, but it negatively affects the transient
314 aphid population when the interference is indirect. When the interference is
315 direct, increasing the value of h increases the basic reproduction number but,
316 in the presence of indirect interference the response of R_0 is non-monotone,
317 with a minimum value obtained for intermediate values of hosting capacity.

318 **The role of resident aphid mortality**

319 The effect of aphid mortality (μ) on the basic reproduction number is summa-
320 rized in Fig. 3B. Increasing μ has a negative effect on the R_0^R term reducing
321 both the resident population density and the average time spent by a vir-
322 uliferous resident aphid in the system. On the other hand, it has a positive
323 effect on the R_0^T term by releasing interference forces exerted by resident

324 aphids and consequently increasing transient aphids movement and sojourn
325 time in the system. In this case, the interference scenario (whether direct or
326 indirect) has no influence because the plant hosting capacity is considered at
327 its reference value $h = h_R$ (see equations 4 and 5, for $h = h_R$). Also in this
328 case, a minimum value of R_0 is obtained for intermediate values of resident
329 aphid mortality.

330 **The role of roguing**

331 An increase to the roguing rate ρ decreases the basic reproduction number
332 as it reduces the time that an infected plant spends in the system before it
333 is rogued (see equation 6 and Fig. 3C). As before, the interference scenario
334 has no influence on the value of R_0 because the plant hosting capacity is
335 considered at its reference value $h = h_R$.

336 **Agricultural practices and disease control**

337 The response of R_0 to the simultaneous variation of two control parameters
338 is summarize in Fig. 4. It is always possible to eradicate the disease for some
339 combination of two of the considered parameters. Eradication is possible
340 without the addition of pesticides ($\mu = 0.04 \text{ day}^{-1}$, *i.e.* resident aphid natu-
341 ral mortality) for relatively small plant hosting capacity (h , which increases
342 with fertilization and irrigation) (Fig. 4A-B). Eradication is not possible for
343 relative high values of resident aphid mortality (μ), independently from the
344 value of h , in the direct interference scenario (Fig. 4B), while it is possible in
345 the indirect interference scenario, where the value of μ that leads to disease

346 eradication, increases with the value of h (Fig. 4A). Increasing roguing rate
347 (ρ) increases the range of h and μ values that lead to disease eradication.
348 Both for direct and indirect interference scenarios, if infected plant are iden-
349 tified and eliminated every periods of a maximum of 50 days (*i.e.* $\rho \geq 0.04$
350 day^{-1}), disease eradication is possible for nearly all the considered h values
351 (Fig. 4 C-D) and for nearly all the considered μ values (Fig. 4E).

352 Discussion

353 Meta-analytical evaluations (Bird et al., 2019; Kaplan and Denno, 2007) sug-
354 gests that both direct (*i.e.* aphid mediated) and indirect (*i.e.* plant mediated)
355 interferences between herbivorous insects shape their behaviour and perfor-
356 mance. For example, in an experimental work, Mehrparvar et al. (2014)
357 showed that interference between different aphids species affect host selec-
358 tion behaviour, with alate aphid individuals rarely choosing a plant occupied
359 by individuals of another aphids' species. However, these interference mecha-
360 nisms have been ignored in epidemiological analyses despite a few exceptions
361 [*e.g.* Crowder et al. (2019); Chisholm et al. (2019); Thaler et al. (2010)].
362 Yet, to our knowledge, the existing experimental and theoretical works only
363 consider interferences between vector and non-vector insects. For example,
364 Chisholm et al. (2019) observed higher rates of *Pea enation mosaic virus*
365 spread when the vector *Acythosiphon pisum* individuals shared hosts with a
366 non vector herbivore *Sitona lineatus*.

367 In the present work, we modeled the interaction between two aphid vec-
368 tors via two interference functions, which account for the reduction of the

369 number of plants visited per unit time and the average resident time in the
370 system of transient aphids. Both direct and indirect interference scenario
371 can be represented depending on the considered pathosystem. By means of
372 mathematical and numerical analyses of our model, we have demonstrated
373 that interference can have profound effects on the invasion, persistence and
374 control of plant NPT viruses. In theoretical work, ecological interactions
375 that alter vector movement limiting the number of plants visited by a vector,
376 have already been shown to be more effective in controlling the spread of
377 NPT viruses than those targeting vector abundance (Crowder et al., 2019).
378 Indeed, when the length of time an infectious vector spends moving, search-
379 ing for a new host to visit, is long in comparison to pathogen retention time,
380 the pathogen may be cleared in transit before visiting a new susceptible host
381 (Ng and Falk, 2006; Killiny and Almeida, 2014). The theoretical analysis
382 of our model confirm these findings: visiting interference $f(\cdot)$, affecting the
383 number of plants visited by transient aphids, reduces both the probability
384 of acquiring and inoculating the virus. That is why, no matter whether
385 the underlying mechanism is direct or indirect, visiting interference appears
386 squared in the basic reproduction number, which determines a greater effect
387 of the visiting interference in diminishing the invasion and persistence of the
388 disease respect to emigration interference.

389 Our results suggest that commonly used agricultural practices, such as
390 fertilization and irrigation, pesticide application and roguing of diseased
391 plants, can have unexpected results upon the spread of NPT viruses. Fertil-
392 ization and irrigation are commonly used in agriculture to meet plants' nutri-
393 ent and water needs and increase plant growth and production (Gliessman,

394 2015). Yet they can impact disease development and spread, possibly affect-
395 ing plant physiology, pathogens and/or vector population dynamics (Dordas,
396 2008; Miller et al., 2015). On the one hand nitrogen is involved in the resis-
397 tance mechanisms of the host plant, *i.e.* its ability to limit the development
398 and reproduction of the invading pathogen, possibly decreasing the incidence
399 of disease in crop plants (Dordas, 2008). On the other hand, fertilization and
400 irrigation can increase vector populations via changes in plant nutrient and
401 irrigation status, potentially impacting the spread of plant diseases. For ex-
402 ample, the growth rate of wheat curl mite, vector of the *Wheat streak mosaic*
403 *virus*, was observed to increase with fertilization on winter wheat (Miller
404 et al., 2015). Populations of bird cherry-oat aphid (*Rhopalosiphum padi* L.),
405 vector of the *Barley yellow dwarf virus*, have been observed to increase with
406 irrigation (Hale et al., 2003) and fertilization (Liang et al., 2019) on different
407 grass species. Our results show that an intermediate plant size, which sus-
408 tains a population of resident aphids large enough to appreciably reduce the
409 spread of the virus by transient aphids, but not too large to prevent disease
410 spread by resident vectors, may lead to disease eradication.

411 Pesticide application is the most common aphid control method, but it is
412 well known that its ability to prevent the spread of NPT viruses by transient
413 aphids is limited because inoculation occurs rapidly and before a pesticide
414 can take effect on the transient vector (Perring et al., 1999). Furthermore
415 pesticide may affect local pest community structure as differential suscepti-
416 bility to pesticide may result in species dominance shift favoring secondary
417 pest outbreaks (Mohammed et al., 2019; Zhao et al., 2017). In their exper-
418 iments with two aphids species, *Rhopalosiphum padi* and *Sitobion avenae*,

419 Mohammed et al. (2019) showed that pesticide exposure led to a shift in the
420 outcome of interspecific competition between the two aphid species, compro-
421 mising the dominance of *R. padi* in pesticide-free plants, while favouring the
422 prevalence of the *S. avenae* under pesticides exposure. Our results show that
423 small pesticide application has the potential to slightly reduce the spread of
424 NPT viruses. However, large pesticide application, reducing the interference
425 exerted by resident towards transient aphids, could be counter productive in
426 reducing NPT viruses, because it favours the prevalence of transient aphids,
427 increasing the spread of the virus by the more mobile vector.

428 Roguing infected plants has often been implemented to control the spread
429 of plant pathogens (Sisterson and Stenger, 2013; Rimbaud et al., 2015a).
430 The success of roguing in slowing disease spread depends on how rapidly in-
431 fected plants are identified and removed (Cunniffe et al., 2015; Fabre et al.,
432 2021). Yet, there are various logistical issues associated with identification
433 and removal of infected plants in large-scale agriculture. Firstly, the identi-
434 fication of diseased plants may be hampered by a lack of appropriate and/or
435 cost-effective diagnostic tests. Further, growers can be reluctant to remove
436 diseased plants as soon as symptoms are identified, since infected plants may
437 continue to produce a marketable yield (Sisterson and Stenger, 2013). Fi-
438 nally, the degree of coordination among farmers concerning the decision of
439 roguing is likely to affect the success in slowing disease spread (Laranjeira
440 et al., 2020). Our results unsurprisingly suggest that the incidence of the
441 disease decreases with the effort put into roguing. Yet, they would benefit
442 from further economic evaluations, given the cost of roguing and replanting
443 operations.

444 Despite our efforts to provide a realistic representation of the complex
445 epidemiological and ecological components of an agroecosystem, we had to
446 introduce a number of simplifying assumptions. We have assumed the res-
447 ident aphid mortality rate to be constant, in accordance to several authors
448 (Crowder et al., 2019; Donnelly et al., 2019; Allen et al., 2019). In reality,
449 the effects of chemical control on pest mortality do not remain constant,
450 but vary with the repeated application and subsequent decay of pesticides'
451 concentration. We have assumed that NPT viruses do not manipulate host-
452 vector behavior in order to enhance their own transmission, for example by
453 making infected plant more attractive to aphids but inhibiting aphid set-
454 tling on infected plants (Mauck et al., 2012; Donnelly et al., 2019). This
455 may not always hold, for example, it was shown that squash plants (*Cu-*
456 *curbita pepo*) infected with *Cucumber mosaic virus* firstly emit a blend of
457 volatile organic compounds that attracts aphids, and secondly produce anti-
458 feedant compound, which deter aphids from prolonged feeding (Pickett and
459 Khan, 2016). Yet, a non negligible number of pathosystems involving viruses
460 considered to be of the NPT transmission type do not follow this "attract
461 and deter" trend (Mauck et al., 2012). Finally, it is possible that plants
462 put in place other types of defensive mechanisms which may impair resident
463 aphid fecundity (Zust and Agrawal, 2016) and which can be fostered by fer-
464 tilization (Zaffaroni et al., 2020). Although all these mechanisms could be
465 included in our model, we have chosen to avoid the proliferation of parame-
466 ters which would have been associated with more complex models, possibly
467 hiding the underlying message of this work. Yet, despite the simplifying as-
468 sumptions outlined above and noting that further experimental works are

469 clearly required to confirm our findings, our work suggests that the impacts
470 of inter-specific interference should be incorporated more broadly into the
471 planning of disease management strategies for the control and eradication of
472 aphid vectored NPT viruses.

473 **Statement of authorship:** MZ, LR, NJC and DB conceived the ideas.
474 MZ, NJC and DB designed the methodology and produced the results. All
475 authors discussed the results and contributed to the writing and editing of
476 the manuscript.

477 **Data accessibility statement:** Data sharing is not applicable to this
478 article as no new data were created or analyzed in this study.

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685 **Figures and tables**

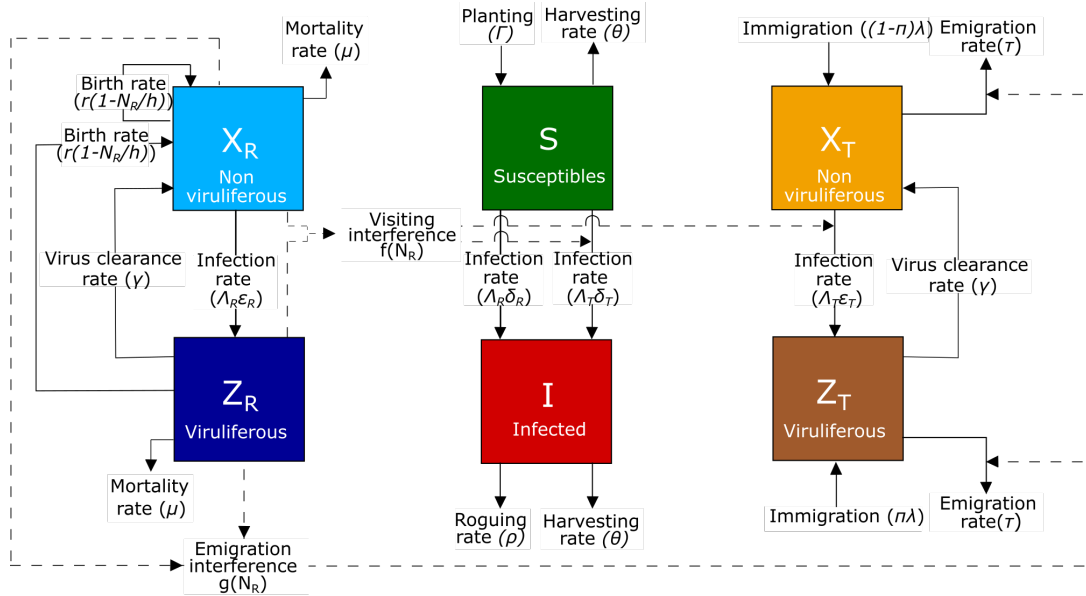


Figure 1: Schematic representation of the single host-multi vector model, where the the total number of host plants is partitioned into susceptible (S) and infected (I) individuals. Aphids are partitioned into non viruliferous (X_i) and viruliferous (Z_i), and are classified as resident ($i = R$) or transient ($i = T$). Dashed arrows identify the contacts between viruliferous aphids and susceptible plants, and between infected plants and non viruliferous aphids, which affect the infection rates. Circles identify the processes affected by interferences exerted by resident towards transient aphids (visiting interference in white and emigration interference in black). The total number of plants per hectare is $N_P = S + I$, the average number of resident aphids per plant is $N_R = X_R + Z_R$ and the average number of transient aphids visiting a plant per unit time is $N_T = X_T + Z_T$. Details on the processes involved are given in the main text.

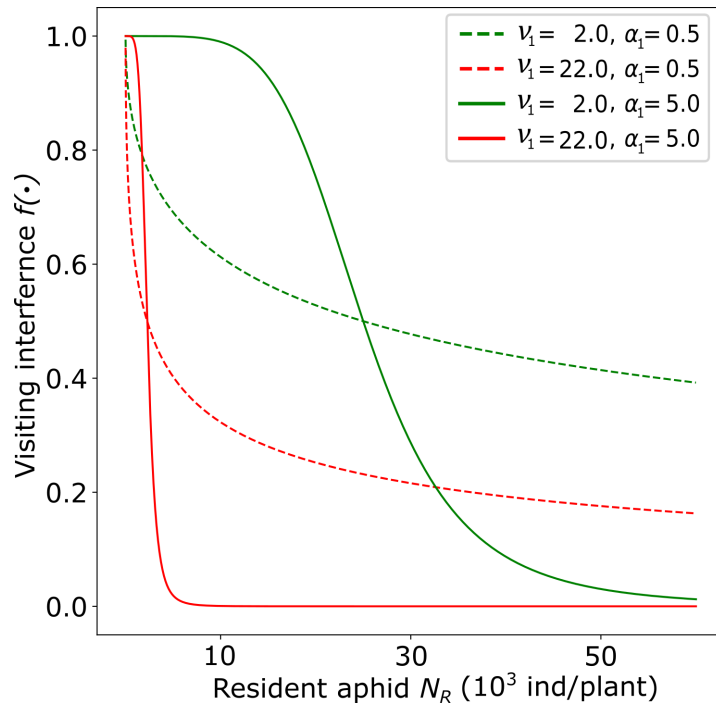


Figure 2: Proportionate decrease of the number of plants visited by a transient aphid as a function of the abundance of resident aphids, for two values of parameter ν_1 and α_1 , in a plant displaying a reference plant hosting capacity (*i.e.* $h = h_R$, thus the value of $f(\cdot)$ is independent of the interference scenario).

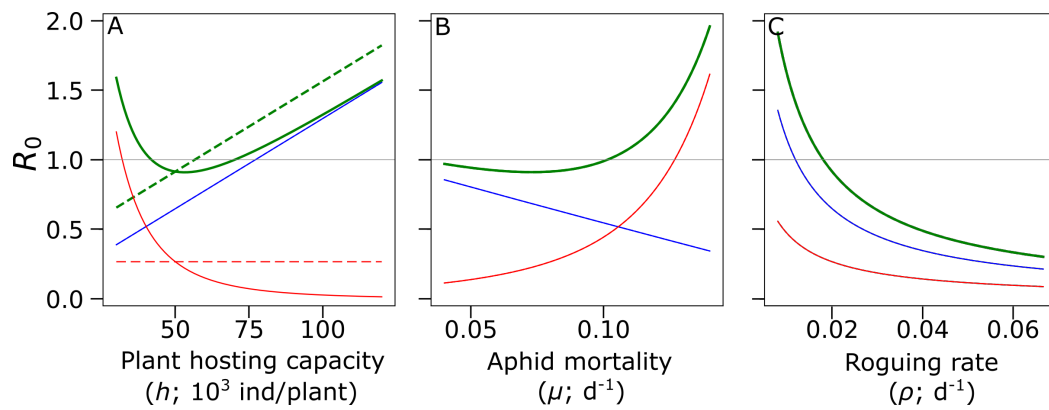


Figure 3: Response of the basic reproduction number R_0 (in bold and green) and its components R_0^R (in blue) and R_0^T (in red) to changes in (A) plant hosting capacity (h) under indirect (continuous line) and direct (dashed line) interference scenarios, (B) resident aphids mortality (μ), (C) roguing rate (ρ). Note that in (A) blue continuous and dashed lines overlap.

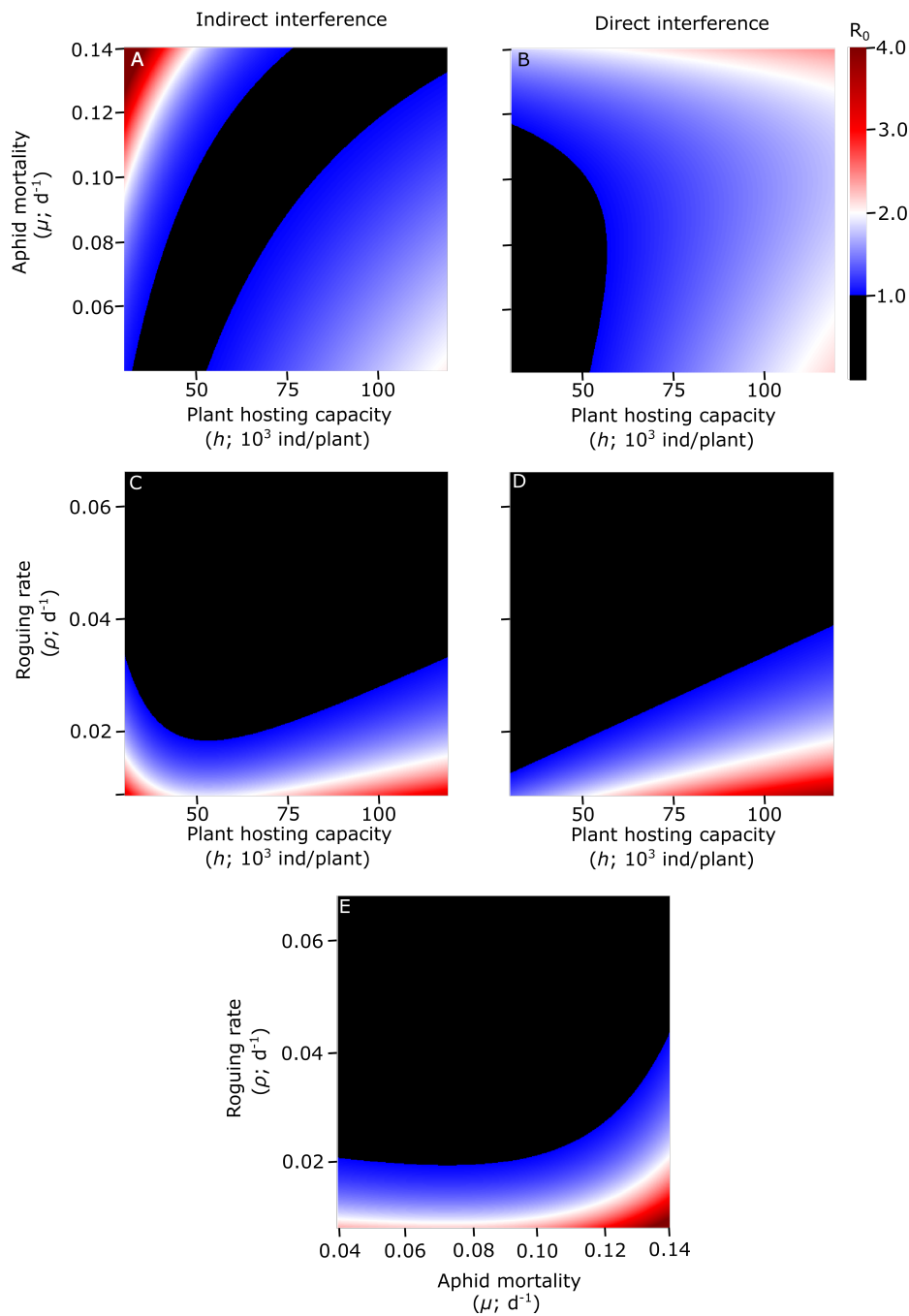


Figure 4: Response of R_0 to changes in: plant hosting capacity (h) and resident aphid mortality (μ) (A-B); plant hosting capacity (h) and roguing rate (ρ) (C-D); resident aphid mortality (μ) and roguing rate (ρ) (E), under different interference scenarios (indirect and direct). Note that the interference scenario has no effect on R_0 when μ and ρ are simultaneously varied (E). Black areas identify values of $R_0 < 1$, corresponding to disease eradication. Other model parameters are set to default values (Table 1).

Table 1: Model state variables and parameters

Sources: (1) Jeger et al. (1998); (2) Jeger et al. (2004); (3) Jeger et al. (2008); (4) Holt et al. (1997); (5) Madden et al. (2000); (6) Rimbaud et al. (2019); (7) Rousselin et al. (2016); (8) Fixed to an arbitrary, biologically-plausible reference value

Variable	Description	Dimensions		
S	Susceptible plants	plant ha ⁻¹		
I	Infected plants	plant ha ⁻¹		
X_R	Non viruliferous resident aphids	aphid plant ⁻¹		
Z_R	Viruliferous resident aphids	aphid plant ⁻¹		
X_T	Non viruliferous transient aphids	aphid plant ⁻¹		
Z_T	Viruliferous transient aphids	aphid plant ⁻¹		
Parameter	Description	Dimensions	Values	Source
Γ	Planting rate	plant day ⁻¹	$\theta S + (\rho + \theta)I$	
Λ_R	Number of plants visited by a resident aphid	plant aphid ⁻¹ day ⁻¹	0.05	(8)
Λ_T	Number of plants visited by a transient aphid	plant aphid ⁻¹ day ⁻¹	8.5	(8)
δ_R	Probability of virus transmission from the resident aphid to the plant	dimensionless	0.04	(1)
δ_T	Probability of virus transmission from the transient aphid to the plant	dimensionless	0.04	(1)
ε_R	Probability of virus transmission from the plant to the resident aphid	dimensionless	0.02	(1)
ε_T	Probability of virus transmission from the plant to the transient aphid	dimensionless	0.02	(1)
α_1	Visiting interference curvature	dimensionless	1.00	(8)
ν_1	Visiting interference strength (for direct interference)	dimensionless	12.0	(8)
α_2	Emigration interference curvature	dimensionless	1.00	(8)
ν_2	Emigration interference strength (for direct interference)	dimensionless	12.0	(8)
ρ	Infected plant roguing rate	day ⁻¹	0.02	(2,3)
θ	Plant harvesting rate	day ⁻¹	0.003	(4)
r	Intrinsic growth rate of resident aphids	day ⁻¹	0.21	(4)
h	Plant hosting capacity	aphid plant ⁻¹	50,000	(7)
h_R	Reference plant hosting capacity	aphid plant ⁻¹	50,000	(8)
μ	Mortality rate of resident aphids	day ⁻¹	0.08	(8)
γ	Virus clearance rate in aphid vectors	day ⁻¹	4	(1)
λ	Average number of transient aphids immigrating per plant	aphid plant ⁻¹ day ⁻¹	$\tau T = 250$	derived
π	Fraction of viruliferous transient aphids entering the system	dimensionless	0	(8)
τ	Transient aphids emigration rate in absence of resident aphids	day ⁻¹	0.5	(2,5)
N_P	Total number of plants	plant ha ⁻¹	720	(6)
T	Average number of transient aphids per plant in absence of resident aphids	aphid plant ⁻¹	500	(8)

Table 2: Summary of equilibrium behaviour. The value of state variables at the equilibrium are presented in the *Supplementary Information*.

Viruliferous aphids enter the system ($\pi > 0$)	Resident aphids are present ($\mu < r$)	Basic reproduction number (eq. 6)	$(\bar{S}, \bar{I}, \bar{X}_R, \bar{Z}_R, \bar{X}_T, \bar{Z}_T)$	Explanation
no	no	$R_0 < 1$	$(+, 0, 0, 0, +, 0)$	Transient aphids do not bear the disease from outside the system. Resident aphids are absent, the disease is spread by transient aphids but it does not persist in the system.
no	no	$R_0 > 1$	$(+, +, 0, 0, +, +)$	Transient aphids do not bear the disease from outside the system. Resident aphids are absent, the disease is spread by transient aphids.
no	yes	$R_0 < 1$	$(+, 0, +, 0, +, 0)$	Transient aphids do not bear the disease from outside the system. Resident and transient aphids spread the disease, but it does not persist in the system.
no	yes	$R_0 > 1$	$(+, +, +, +, +, +)$	Transient aphids do not bear the disease from outside the system. Resident and transient aphids spread the disease.
yes	no	- *	$(+, +, 0, 0, +, +)$	Transient aphids bear the disease from outside the system. Resident aphids are absent, transient aphids spread the disease.
yes	no	- *	$(+, +, +, +, +, +)$	Transient aphids bear the disease from outside the system. Resident and transient aphids spread the disease.

* The disease is always able to persist, regardless of whether the basic reproduction number is smaller or larger than 1.