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Spatial spread of infectious diseases with conditional vector preferences

Frédéric M. Hamelin^{1,*}, Frank M. Hilker², Yves Dumont^{3,4,5}

¹ Institut Agro, Univ Rennes, INRAE, IGEPP, 35000, Rennes, France

² Institute of Mathematics and Institute of Environmental Systems Research, Osnabrück University, D-49069 Osnabrück, Germany

³ CIRAD, UMR AMAP, 97410 St Pierre, Réunion Island, France

⁴ AMAP, Univ Montpellier, CIRAD, CNRS, INRAE, IRD, Montpellier, France

⁵ Department of Mathematics and Applied Mathematics, University of Pretoria, Pretoria, South Africa

* Corresponding author: frederic.hamelin@institut-agro.fr

Abstract We explore the spatial spread of vector-borne infections with conditional vector preferences, meaning that vectors do not visit hosts at random. Vectors may be differentially attracted toward infected and uninfected hosts depending on whether they carry the pathogen or not. The model is expressed as a system of partial differential equations with vector diffusion. We first study the non-spatial model. We show that conditional vector preferences alone (in the absence of any epidemiological feedback on their population dynamics) may result in bistability between the disease-free equilibrium and an endemic equilibrium. A backward bifurcation may allow the disease to persist even though its basic reproductive number is less than one. Bistability can occur only if both infected and uninfected vectors prefer uninfected hosts. Back to the model with diffusion, we show that bistability in the local dynamics may generate travelling waves with either positive or negative spreading speeds, meaning that the disease either invades or retreats into space. In the monostable case, we show that the disease spreading speed depends on the preference of uninfected vectors for infected hosts, but also on the preference of infected vectors for uninfected hosts under some circumstances (when the spreading speed is not linearly determined). We discuss the implications of our results for vector-borne plant diseases, which are the main source of evidence for conditional vector preferences so far.

33 **Keywords:** vector bias, bistability, backward bifurcation, travelling wave, spread-
34 ing speed, front reversal, pushed and pulled waves.

35 **1 Introduction**

36 Vector-borne diseases are a major concern for human, animal and plant health. Since
37 Ross' seminal work (1911), most mathematical models of vector-borne infections
38 consider that vectors visit hosts randomly, independent of their infection status (e.g.,
39 Wonham et al., 2004; Martcheva, 2015). Spatially explicit models are no exception
40 in this regard (e.g., Lewis et al., 2006). However, growing evidence shows that many
41 vectors do not visit hosts randomly (e.g., Gandon, 2018).

42 Vectors may be differentially attracted towards infected and uninfected hosts,
43 independent of whether or not they carry the pathogen (e.g., Lacroix et al., 2005;
44 Mauck et al., 2010; Cornet et al., 2013). This is termed a "vector bias" in the mod-
45 elling literature (Chamchod and Britton, 2011). Kingsolver (1987) was probably the
46 first to include such a vector bias in a model. He showed that vector preferences
47 can induce bistability, meaning that the dynamics converge either to a disease-free
48 state or to an endemic state depending on the initial conditions. However, bistability
49 only occurred in somewhat special cases in which the vector bias was a function of
50 the fraction of infected hosts in the population. Later studies generally assumed a
51 constant vector bias and did not find bistability (Hosack et al., 2008; Chamchod and
52 Britton, 2011; Wang and Zhao, 2017), except when disease-induced host mortality
53 and immigration were included in the model (Buonomo and Vargas-De-León, 2013).

54 Spatially explicit models have also been used to explore the consequences of a
55 vector bias in space. Individual-based models were formulated to investigate the
56 effect of spatial heterogeneity on the spread of vector-borne diseases with a vector
57 bias (McElhany et al., 1995; Sisterson, 2008). Chamchod and Britton (2011) were
58 probably the first to incorporate a vector bias into a partial differential equation (PDE)
59 model. They numerically showed that travelling waves occur, and how their speed
60 can be calculated. Later studies then also considered a vector bias in PDE models
61 (e.g., Xu and Zhao, 2012; Bai et al., 2018). In particular, Xu and Zhang (2015) also
62 showed the existence of travelling wave solutions. In these studies (Chamchod and

63 Britton, 2011; Xu and Zhang, 2015), the models did not exhibit bistability, and the
64 travelling waves had positive speeds, meaning that the disease invades a disease-
65 free spatial domain.

66 Vector preferences, however, may depend on whether or not vectors carry the
67 pathogen (Ingwell et al., 2012; Blanc and Michalakis, 2016; Gandon, 2018; Eigen-
68 brode et al., 2018; Shoemaker et al., 2019; Carr et al., 2020). These are termed
69 “conditional vector preferences”. Roosien et al. (2013) were probably the first to
70 include conditional vector preferences in a model, but they did not fully analyse the
71 model. In particular, whether or not bistability can occur was left implicit. In a more
72 general version of the model (accounting for vector handling times), Gandon (2018)
73 showed that conditional preferences (in particular a preference of uninfected vectors
74 for infected hosts) can lead to bistability, provided vector fecundity depends on host
75 infection status. Similarly, Cunniffe et al. (2021) observed multi-stability in a more
76 complex model accounting for vector population dynamics that depend on the host
77 infection status. However, whether conditional preferences can lead to bistability
78 when the vector population dynamics are independent of the host infection status
79 still remains to be clarified.

80 Bistability may have important implications regarding the spatial spread of the
81 disease in space. In particular, it is well known (Fife and McLeod, 1977; Lewis and
82 Kareiva, 1993; Lewis and van den Driessche, 1993; Owen and Lewis, 2001; Fagan
83 et al., 2002; Hilker et al., 2005, 2007) that bistability can give rise to negative wave
84 speeds, meaning in our context that the disease retreats. This phenomenon is also
85 termed “front reversal”.

86 In this study, we analyse whether and how conditional vector preferences can
87 give rise to bistability and front reversal in vector-borne diseases. The organisation
88 of the paper is as follows. In Section 2, we present a spatio-temporal (reaction-
89 diffusion) model with conditional vector preferences. In Section 3, we provide an
90 analysis of the temporal (non-spatial) model with some numerical simulations. Then,
91 in Section 4, we go back to the spatio-temporal model (with diffusion), showing
92 existence of travelling wave solutions. Numerical simulations illustrate our findings.
93 Lastly, Section 5 concludes the paper with a discussion.

94 **2 Spatio-temporal model**

95 Let $I(x, t)$ be the infected host density at time t and location $x \in \mathbb{R}$. We adopt a uni-
96 dimensional representation of space for simplicity. The total host density is assumed
97 to be a constant N independent of x . The local density of uninfected hosts at time
98 t is therefore $N - I(x, t)$. Let $V(x, t)$ and $U(x, t)$ be the infected (“viruliferous”) and
99 uninfected vector densities, respectively. Let b be the vector “biting” rate. Let p
100 and q be the probabilities of pathogen transmission and acquisition, respectively.
101 Let r be the removal rate of infected hosts. Infected vectors lose the pathogen at
102 rate l (Chapwanya and Dumont, 2018). Let m be the vector mortality rate. For
103 simplicity, we assume that the vector birth rate exactly compensates the mortality
104 rate. In addition, we assume that vectors are born uninfected. There is no vertical
105 transmission in either the vector or the host. Let a be the preference (attraction) of
106 infected vectors for uninfected hosts: $a = 1$ means no preference, $a > 1$ preference
107 and $0 < a < 1$ repulsion. Similarly, let u be the preference of uninfected vectors for
108 infected hosts. As in Chamchod and Britton (2011), only the spatial movement of
109 vectors is considered. Let D be the vector diffusion rate, independent of the vector
110 infection status. The model is:

$$\begin{aligned} I_t &= bpV \frac{a(N-I)}{a(N-I)+I} - rI, \\ V_t &= bqU \frac{uI}{uI+(N-I)} - (m+l)V + DV_{xx}, \\ U_t &= (m+l)V - bqU \frac{uI}{uI+(N-I)} + DU_{xx}, \end{aligned} \quad (1)$$

111 in which the subscripts denote differentiation with respect to t or x , and in which the
112 dependence of the state variables on t and x has been omitted.

113 **2.1 Model simplification**

114 Let $W = U + V$ be the total vector population density. We have $W_t = DW_{xx}$. Assuming
115 $W(x, 0) = K$ (the vector carrying capacity) for all $x \in (-\infty, +\infty)$, $W_t(x, 0) = 0$ for all
116 x , meaning that $W = K$ for all $t \geq 0$ and $x \in (-\infty, +\infty)$. Therefore, we can substitute

117 U with $K - V$ in model (1), which thus simplifies to a two-dimensional system:

$$\begin{aligned} I_t &= bpV \frac{\alpha(N-I)}{\alpha(N-I)+I} - rI, \\ V_t &= bq(K-V) \frac{uI}{uI+(N-I)} - (m+l)V + DV_{xx}. \end{aligned} \quad (2)$$

118 2.2 Non-dimensionalisation

119 We rescale the state variables and parameters by letting

$$\tau = (m+l)t, \quad \xi = x \sqrt{\frac{m+l}{D}}, \quad i = \frac{I}{N}, \quad v = \frac{V}{K}$$

120 and

$$\beta = \frac{bpK}{(m+l)N}, \quad \rho = \frac{r}{m+l}, \quad \theta = \frac{bq}{m+l}.$$

121 A dimensionless version of model (2) is the following:

$$\begin{aligned} i_\tau &= \beta v \frac{\alpha(1-i)}{\alpha(1-i)+i} - \rho i, \\ v_\tau &= \theta(1-v) \frac{ui}{ui+(1-i)} - v + v_{\xi\xi}, \end{aligned} \quad (3)$$

122 in which the subscripts denote differentiation with respect to τ or ξ . Note that the
123 two state variables are both disease prevalences, i.e. they are fractions of the host
124 and the vector being infected, and take values in the unit interval.

125 3 Analysis of the non-spatial system

126 The non-spatial model is:

$$\begin{aligned} i' &= \beta v \frac{\alpha(1-i)}{\alpha(1-i)+i} - \rho i =: f_1(i, v), \\ v' &= \theta(1-v) \frac{ui}{ui+(1-i)} - v =: f_2(i, v). \end{aligned} \quad (4)$$

127 We will also use the following notations: $y = (i, v)^T$ and $f = (f_1, f_2)^T$.

128 **3.1 Basic reproductive number**

129 System (4) was previously explored in Roosien et al. (2013) and Cunniffe et al.
130 (2021). It is known that the disease-free equilibrium $(i, v) = (0, 0)$ is locally asymp-
131 totically stable if and only if

$$\mathcal{R}_0^2 := \frac{b^2 pq K}{rm N} u = \frac{\beta \theta}{\rho} u < 1.$$

132 We refer to \mathcal{R}_0^2 as the basic reproductive number. Note that \mathcal{R}_0 depends on u (the
133 preference of uninfected vectors for infected hosts) but does not depend on α (the
134 preference of infected vectors for uninfected hosts). The results we present next are
135 original. Let u_c be such that $\mathcal{R}_0^2 = 1$, i.e.,

$$u_c = \frac{\rho}{\beta \theta}.$$

136

137 **3.2 The system is cooperative**

138 We have

$$\frac{\partial f_1}{\partial v} \geq 0 \quad \text{and} \quad \frac{\partial f_2}{\partial i} \geq 0,$$

139 since

$$\frac{\partial}{\partial i} \left(\frac{ui}{ui + (1-i)} \right) = \frac{u}{(1 + i(u-1))^2} > 0.$$

140 Therefore, system (4) is cooperative, meaning that the dynamics necessarily con-
141 verge to an equilibrium (convergence to a limit cycle is impossible) (Smith, 2008).

142 **3.3 Endemic equilibrium**

143 Let us solve the system $f_1(i, v) = f_2(i, v) = 0$. An endemic equilibrium (i^*, v^*) , with
144 $i^*, v^* > 0$, satisfies:

$$Q(i^*) = Ai^{*2} + Bi^* + C = 0,$$

145 in which

$$\begin{aligned} A &= (\alpha - 1)(u(1 + \theta) - 1), \\ B &= \left((2 - (1 + \theta)u) - \frac{\beta\theta}{\rho}u \right) \alpha - 1 = -\left(((1 + \theta)u - 1) + (\mathcal{R}_0^2 - 1) \right) \alpha - 1, \\ C &= \alpha \left(\frac{\beta\theta}{\rho}u - 1 \right) = \alpha(\mathcal{R}_0^2 - 1). \end{aligned}$$

146 Let u^* be such that $A = 0$:

$$u^* = \frac{1}{1 + \theta}. \quad (5)$$

147 Note that A has no reason to be zero in general ($A = 0$ only for $\alpha = 1$ or $u = u^*$). The
148 coefficient A can be also expressed as

$$A = (\alpha - 1) \left(\frac{u}{u^*} - 1 \right).$$

149 First, we notice that

$$Q(1) = -u(1 + \theta) < 0. \quad (6)$$

150 Next, we distinguish two cases: $\mathcal{R}_0^2 > 1$ and $\mathcal{R}_0^2 < 1$. (The boundary case $\mathcal{R}_0^2 = 1$ is
151 addressed in Appendix A.1 for the sake of completeness.)

152 **3.4 Case $\mathcal{R}_0^2 > 1$**

153 If $\mathcal{R}_0^2 > 1$, then $Q(0) = C > 0$. Since $Q(1) < 0$ (Eq. 6), there is exactly one root i^* in
154 $[0, 1]$, which is the endemic equilibrium. Appendix A.2 shows that

$$i^* = \begin{cases} -\frac{C}{B} & \text{if } \alpha = 1 \text{ or } u = u^* \text{ (special cases implying } A = 0), \\ \frac{1}{2A}(-B - \sqrt{\Delta}) & \text{otherwise,} \end{cases}$$

155 where $\Delta = B^2 - 4AC$ is the discriminant.

156 **3.5 Case $\mathcal{R}_0^2 < 1$**

157 If $\mathcal{R}_0^2 < 1$, then $C < 0$.

158 Since $Q(0) = C < 0$, and $Q(1) < 0$ (Eq. 6), either there is no root in between
159 0 and 1 or there are two roots (unless the discriminant Δ is zero, in which case

160 there is a single root, of course). The existence of biologically feasible equilibria
 161 requires $A < 0$. Since this implies $AC > 0$, an additional necessary condition for
 162 the existence of endemic equilibria is that the discriminant Δ is non-negative. The
 163 additional conditions are $Q'(0) = B > 0$ and $Q'(1) = 2A + B < 0$.

164 If these conditions ($\mathcal{R}_0^2 < 1$, $A < 0$, $\Delta \geq 0$, $B > 0$, and $2A+B < 0$) are simultaneously
 165 satisfied, there are two positive equilibria with components

$$i_{1,2}^* = \frac{1}{2A}(-B \pm \sqrt{\Delta}), \quad (7)$$

166 since $A < 0$ and $B > 0$. We set $E_1 = (i_1^*, v_1^*)$ and $E_2 = (i_2^*, v_2^*)$ and notice that the
 167 equilibria are ordered, i.e. $E_1 < E_2$, since $i_1^* < i_2^*$ and $v_1^* = g(i_1^*) < v_2^* = g(i_2^*)$, where
 168 $g(i) = \frac{\rho}{\beta} \left(1 + \frac{i}{\alpha(1-i)}\right)$ is an increasing function corresponding to $f_1 = 0$ in (4).

169 3.5.1 Necessary conditions for two equilibria

170 Here, we will derive two necessary conditions on the vector preferences, namely
 171 $\alpha > 1$ and $u < u^* < 1$ (since $u^* = 1/(1 + \theta)$, as defined in Eq. (5)), for two positive
 172 equilibria to coexist.

173 First, B can be expressed as

$$B = -\left(1 + \theta + \frac{\theta\beta}{\rho}\right)au + (2\alpha - 1).$$

174 Therefore, $B > 0$ is equivalent to

$$u < \frac{(2\alpha - 1)}{\left(1 + \theta + \frac{\theta\beta}{\rho}\right)\alpha} =: u^+.$$

175 Second, $B + 2A < 0$ can be expressed as

$$\left((\theta + 1)(\alpha - 2) - \frac{\theta\beta}{\rho}\alpha\right)u + 1 < 0. \quad (8)$$

176 A necessary condition for the inequality (8) to hold is

$$\frac{\theta\beta}{\rho(\theta + 1)} > \frac{\alpha - 2}{\alpha} =: R_1. \quad (9)$$

177 Assuming inequality (9) holds, $B + 2A < 0$ (inequality 8) is equivalent to

$$u > \frac{1}{\frac{\theta\beta}{\rho}a - (\theta + 1)(a - 2)} =: u^-.$$

178 So far, we have shown that conditions $B > 0$ and $B + 2A < 0$ are equivalent to $u < u^+$
 179 and $u > u^-$ (provided inequality 8 holds), respectively. A necessary condition for
 180 these conditions to hold is therefore $u^- < u^+$. The latter inequality can be equiva-
 181 lently expressed as

$$\frac{1}{\frac{\theta\beta}{\rho}a - (\theta + 1)(a - 2)} < \frac{2a - 1}{\left(\left(1 + \frac{\beta}{\rho}\right)\theta + 1\right)a},$$

182 which is equivalent to

$$\left(1 + \frac{\beta}{\rho}\right)\theta + 1 < \left(2 - \frac{1}{a}\right)\left(\frac{\theta\beta}{\rho}a - (\theta + 1)(a - 2)\right).$$

183 After rearrangement, the above inequality can be equivalently expressed as

$$(\theta + 1)(a - 1)^2 < a(a - 1)\frac{\theta\beta}{\rho}.$$

184 A necessary condition for the above inequality to hold is $a > 1$. Assuming $a > 1$, this
 185 inequality can be equivalently expressed as

$$\frac{\theta\beta}{\rho(1 + \theta)} > \frac{a - 1}{a} =: R_2.$$

186 Since $R_2 > R_1$, the above inequality guarantees that inequality (9) is satisfied.

187 We were not able to get more results from this preliminary analysis, but we have
 188 shown that $a > 1$ is a necessary condition for two endemic equilibria to coexist.

189 Assuming $a > 1$, the condition $A < 0$ is equivalent to

$$u < \frac{1}{1 + \theta} = u^*.$$

190 Hence, $a > 1$ and $u < u^* < 1$ are necessary conditions for two positive equilibria to
 191 coexist.

192 **3.5.2 Numerical example of bistability**

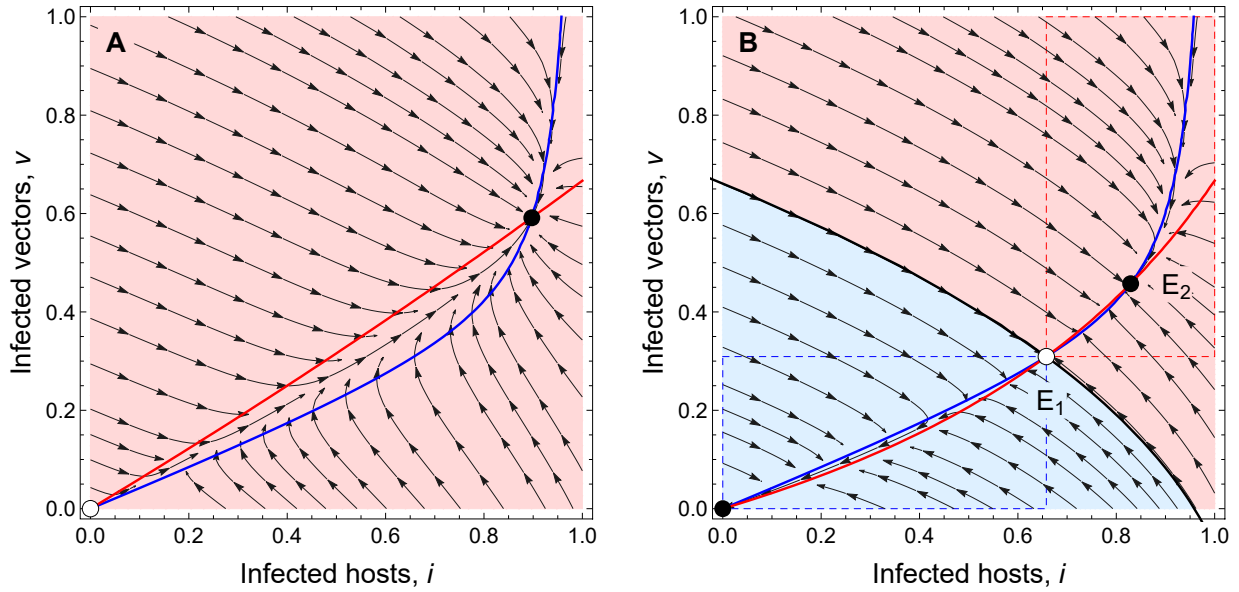


Figure 1: Phase portraits of the non-spatial model (4) with v - and i -nullclines (blue and red curves, respectively). Stable (unstable) equilibria are shown as filled (empty) circles. The basins of attraction to the endemic (disease-free) equilibrium are shown in light red (light blue). **(A)** The endemic equilibrium is the only attractor. Parameter value: $u = 0.3$, so $\mathcal{R}_0^2 = 1.44 > 1$. **(B)** Bistable case. The black line is the separatrix of the two basins of attraction. The dashed rectangles indicate analytically obtained sets of initial conditions that are known to approach the disease-free (blue) or endemic (red) equilibrium. They are part of the actual basins of attraction, see Sect. 3.6 for more details. Parameter value: $u = 0.15$, so $\mathcal{R}_0^2 = 0.72 < 1$. All other parameter values: $a = 15, \beta = 2.4, \rho = 1, \theta = 2$.

193 Since necessary and sufficient conditions were hardly expressible with pen and
 194 paper, we used symbolic calculation software (Maple 2022) to disentangle the con-
 195 ditions for two positive equilibria to coexist. To simplify things, we let

$$X = u(1 + \theta) - 1 \quad \text{and} \quad Y = \mathcal{R}_0^2 - 1. \quad (10)$$

196 This way,

$$A = (a - 1)X, \quad B = -(X + Y)a - 1 \quad \text{and} \quad C = aY.$$

197 Since $\mathcal{R}_0^2 < 1$, $Y < 0$. Since $a > 0$, $C < 0$. The previous section showed that $A < 0$,
 198 $a > 1$, and therefore $X < 0$ are necessary conditions for two positive equilibria to
 199 coexist. We thus used the function “solve” in Maple to solve the following system of
 200 inequalities,

$$A < 0, \quad B > 0, \quad (2A + B) < 0, \quad B^2 - 4AC > 0, \quad Y < 0, \quad a > 1, \quad X < 0$$

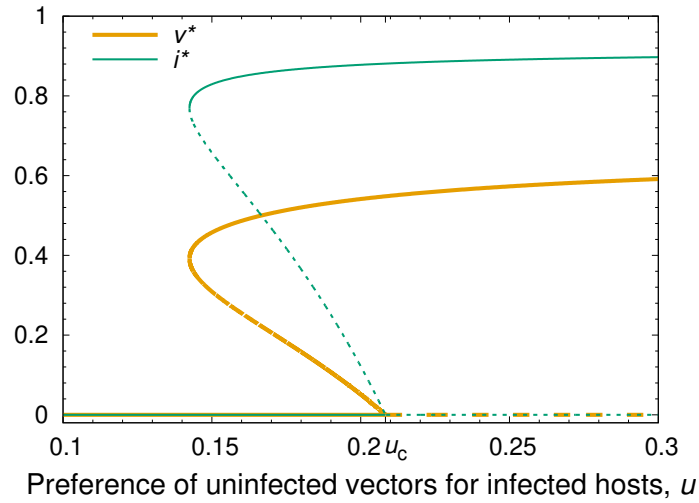


Figure 2: Bifurcation diagram of the non-spatial model (4). Stable (unstable) steady states are shown in solid (dashed) line. There is a backward bifurcation at $u = u_c \approx 0.2083$ and a fold bifurcation at $u \approx 0.1425$. Other parameter values as in Fig. 1.

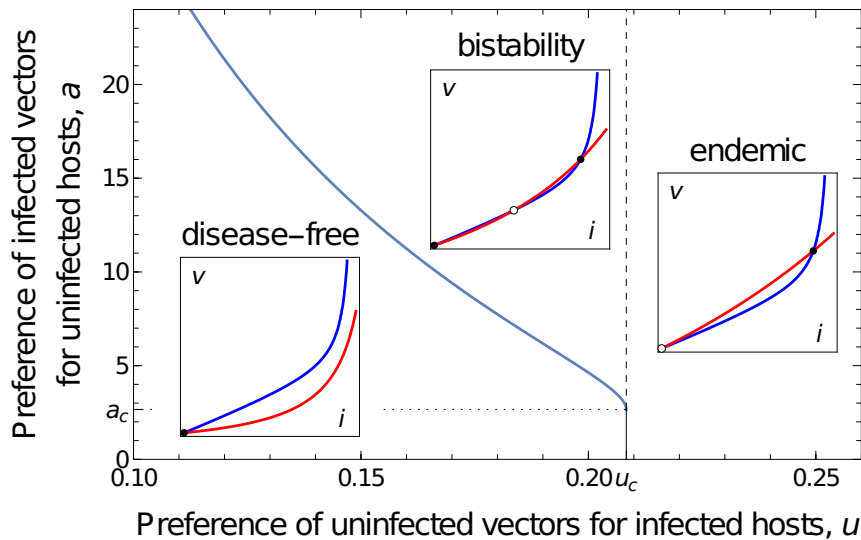


Figure 3: Two-parameter bifurcation diagram of the non-spatial model (4). The fold bifurcation where two endemic equilibria coalesce is shown in blue. The vertical line marks the transcritical bifurcation curve, which occurs at $\mathcal{R}_0^2 = 1$. When the vertical line is solid (dashed), there is a standard transcritical (backward) bifurcation. The fold and transcritical bifurcation curves meet at $(u_c \approx 0.2083, a_c \approx 2.6666)$. The insets are nullcline examples of parameter values leading to different dynamical regimes. Other parameter values as in Fig. 1.

201 with respect to X, Y and a . Letting

$$h(Y, a) := \frac{Ya^2 - (2Y + 1)a - 2\sqrt{-Ya^2(a-1)(Y+1)}}{a^2},$$

202 we obtained the following set of conditions:

203 • If $1 < a < 2$,

$$\frac{Ya + 1}{a - 2} < X < h(Y, a), \quad \text{and} \quad Y > -\frac{a - 1}{a}.$$

204 • If $a = 2$,

$$X < -\frac{1}{2} - \sqrt{-Y(Y-1)} = h(Y, 2), \quad \text{and} \quad Y > -\frac{1}{2}.$$

• If $a > 2$:

$$\begin{cases} X < h(Y, a) & \text{if } Y > -\frac{1}{a}, \\ X < -4\frac{a-1}{a^2} = h\left(\frac{1}{a}, a\right) & \text{if } Y = -\frac{1}{a}, \\ \text{impossible} & \text{if } Y \in \left[-\frac{a-1}{a}, -\frac{1}{a}\right), \\ X < \frac{Ya + 1}{a - 2} & \text{if } Y < -\frac{a-1}{a}. \end{cases}$$

205 This condition set allowed us to find parameter values for which bistability occurs;
206 see Figs. 1–3 for phase portraits, one-parameter, and two-parameter bifurcation di-
207 agrams, respectively.

208 3.6 Local and global asymptotic stability

209 The system being monotone cooperative, proving global asymptotic stability (GAS)
210 relies on local asymptotic stability (LAS) and the use of some appropriate theorems.

The Jacobian of system (4) is

$$J(i, v) = \begin{pmatrix} -\beta v \frac{a}{(a(1-i) + i)^2} - \rho & \beta \frac{a(1-i)}{a(1-i) + i} \\ \theta(1-v) \frac{u}{(ui + (1-i))^2} & -\theta \frac{ui}{ui + (1-i)} - 1 \end{pmatrix}.$$

Notice that $J(i, v)$ is irreducible for all $(i, v) \in [0, 1]^2$. At equilibrium $(0, 0)$, we have

$$J(0, 0) = \begin{pmatrix} -\rho & \beta \\ \theta u & -1 \end{pmatrix},$$

211 from which we deduce that $0 = (0, 0)^T$ is LAS when $\mathcal{R}_0^2 = \beta\theta u/\rho < 1$ and unstable
212 when $\mathcal{R}_0^2 > 1$.

213 When $\mathcal{R}_0^2 > 1$, only one positive endemic equilibrium, E , exists in $[0, 1]^2$. Thus,
214 when $\mathcal{R}_0^2 > 1$, using Theorem 6 in Anguelov et al. (2012) (see also Smith, 2008),
215 with $\mathbf{a} = (0, 0)$ and $\mathbf{b} = (1, 1)$ such that $f(\mathbf{b}) \leq 0 \leq f(\mathbf{a})$, we deduce that the endemic
216 equilibrium E is GAS on $[0, 1]^2$. Similarly, when $\mathcal{R}_0^2 < 1$, in the case when no endemic
217 equilibrium exists, we can show, using the same approach, that 0 is GAS.

218 Assume $\mathcal{R}_0^2 < 1$. In the case where $0, E_1$, and E_2 co-exist such that $0 \ll E_1 \ll E_2$,
219 we already know that 0 is LAS. We can check (at least numerically) that E_1 is unstable
220 and E_2 is LAS. Following Smith (2008, Theorem 2.2.2), it is straightforward to show
221 that the set $\{y \in \mathbb{R}^2 : 0 \leq y < E_1\}$ is in the basin of attraction of 0 , while the set
222 $\{y \in \mathbb{R}^2 : E_1 < y \leq 1\}$, where $1 = (1, 1)^T$, is in the basin of attraction of E_2 (Fig. 1B).

223 4 Back to the system with diffusion

224 In this section, we get back to the system with diffusion, i.e., system (3).

225 4.1 Existence and uniqueness of a solution

System (3), with non-negative initial conditions and appropriate boundary conditions, is a partly dissipative or a partially degenerate system. We consider the following spaces

$$\mathcal{S} = \{(i, v) | v \in L^2(\mathbb{R}); i \in L^\infty(\mathbb{R})\},$$

and

$$\mathcal{S}_{1,1} = \{(i, v) \in \mathcal{S} | 0 \leq v \leq 1; 0 \leq i \leq 1\}.$$

226 Following Rothe (1984, Theorem 1, page 111), or Rauch and Smoller (1978, Theorem 2.1), we can show local existence and uniqueness. Then, using a priori L^∞
227

228 estimates, the fact that the right-hand side of (3) is quasi-positive and the maximum
 229 principle lead to

Theorem 1 (Existence and uniqueness). *For any initial values $(i_0, v_0) \in \mathcal{S}_{1,1}$, system (3) admits a unique non-negative bounded solution such that*

$$i \in C([0, \infty); L^\infty(\mathbb{R})) \cap C^1([0, \infty); L^\infty(\mathbb{R}))$$

and

$$v \in C([0, \infty); L^\infty(\mathbb{R})) \cap C([0, \infty); H^2(\mathbb{R})) \cap C^1([0, \infty); L^2(\mathbb{R})).$$

230 Since the study of the non-spatial system showed us that, depending on parameter
 231 values, it can be monostable or bistable, it seems relevant to study the existence
 232 (or non-existence) of travelling wave solutions.

233 4.2 Monostable case

234 In this section, we assume $\mathcal{R}_0^2 > 1$. We know from the non-spatial system that the
 235 disease-free equilibrium 0 is unstable and the endemic equilibrium E is GAS. Does a
 236 travelling wave solution connecting 0 to E exist?

237 4.2.1 Existence of a travelling wave

238 In the monostable case, the existence of a travelling wave should derive from the
 239 fact that the system is cooperative (Li et al., 2005); the problem is that the system
 240 is partially degenerate (Fang and Zhao, 2009; Li, 2012). However, for this situation
 241 powerful theorems exist (Fang and Zhao, 2014; Li, 2012), see also Doli (2017).

Here, we will use Theorem 4.2 in Li (2012) for the following system:

$$\frac{\partial \mathbf{y}}{\partial t} = D \frac{\partial^2 \mathbf{y}}{\partial x^2} + \mathbf{f}(\mathbf{y}(t, x)),$$

242 with $\mathbf{y} = (y_1(t, x), \dots, y_k(t, x))$, $D = \text{diag}(d_1, \dots, d_k) \geq 0$ and $\mathbf{f} = (f_1, \dots, f_k)$. According
 243 to Li (2012), the following hypotheses have to be checked (Hypotheses 2.1 in Li,
 244 2012):

- 245 1. There is a proper subset Σ_0 of $\{1, \dots, k\}$ such that $d_i = 0$ for $i \in \Sigma_0$ and $d_i > 0$
 246 for $i \notin \Sigma_0$.

- 247 2. $\mathbf{f}(0) = 0$, there is a constant $\gamma \gg 0$ such that $\mathbf{f}(\gamma) = 0$ which is minimal in the
 248 sense that there is no constant ν other than γ such that $\mathbf{f}(\nu) = 0$ and $0 \ll \nu \ll \gamma$,
 249 and the equation $\mathbf{f}(\alpha) = 0$ has a finite number of constant roots.
- 250 3. The system is cooperative.
- 251 4. $\mathbf{f}(\alpha)$ is uniformly Lipschitz in α such that there is $\eta > 0$ such that for any α_i ,
 252 $i = 1, 2$, $\|(\alpha_1) - \mathbf{f}(\alpha_2)\| \leq \eta \|\alpha_1 - \alpha_2\|$.
- 253 5. \mathbf{f} has the Jacobian $\mathbf{f}'(0)$ at 0 with the property that $\mathbf{f}'(0)$ has a positive eigen-
 254 value whose eigenvector has positive components.

Assuming $\mathcal{R}_0^2 > 1$, and $k = 2$, it is straightforward to check the first three hypotheses for system (3), where $\gamma = E$. The fourth hypothesis requires long computations for $a \neq 1$ and $u \neq 1$ to be checked. Lastly, we have

$$\mathbf{f}'(0) = \begin{pmatrix} -\rho & \beta \\ \theta u & -1 \end{pmatrix}.$$

255 Since $\mathcal{R}_0^2 > 1$, it is straightforward to show that $\mathbf{f}'(0)$ has a positive eigenvalue,
 256 $\lambda = \frac{1}{2} \left(\sqrt{(1-\rho)^2 + 4\rho\mathcal{R}_0^2} - (1+\rho) \right)$, associated with the positive eigenvector $\left(1, \frac{\lambda + \rho}{\beta} \right)^T$.
 257 Thus, according to Theorem 4.2 in Li (2012), we deduce the existence of a travelling
 258 wave connecting 0 to $\gamma = E$. See, for instance, Fig. 4.

259 4.2.2 Derivation of the linear spreading speed

260 Still assuming $\mathcal{R}_0^2 > 1$, we consider a travelling front connecting the disease-free
 261 equilibrium, 0 to the endemic equilibrium, E . We posit that, in some circumstances,
 262 the front speed is linearly determined by the minimum possible wave speed based
 263 on the linearisation at the leading edge of the wave. We apply the minimum wave
 264 speed approach (Lewis and Schmitz, 1996; Haderler and Lewis, 2002; Bampfyld and
 265 Lewis, 2007; Hilker and Lewis, 2010; Hamelin et al., 2022) to the linearised model to
 266 find the linear spreading speed as a critical point. However, we stress that the linear
 267 spreading speed may be only a lower bound of the actual spreading speed in some
 268 cases (see Fig. 11 in Appendix B.2).

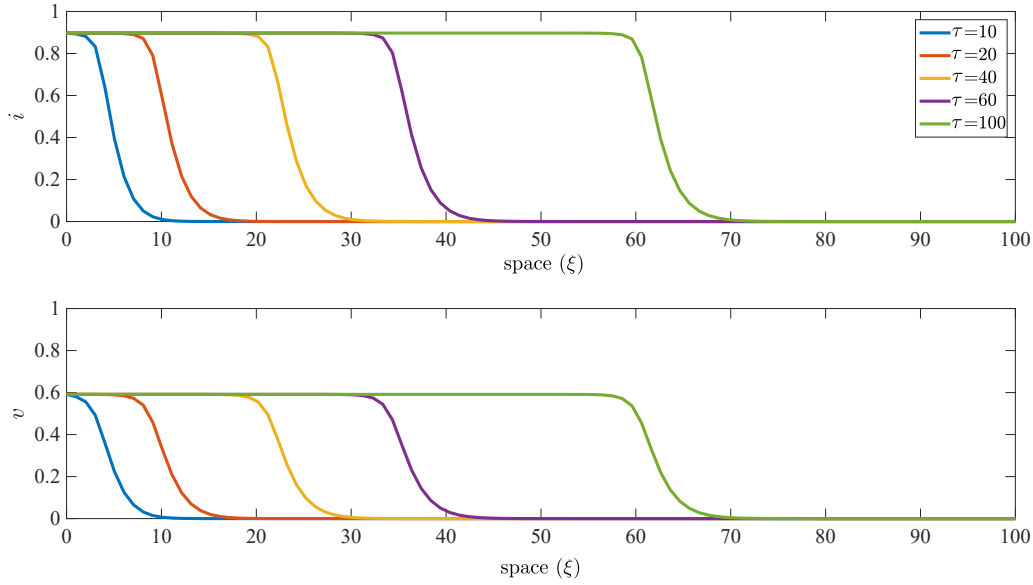


Figure 4: Monostable travelling wave solution of model (3) connecting the disease-free and endemic equilibria 0 and E when $\mathcal{R}_0^2 = 1.44 > 1$. Here $u = 0.3$, other parameter values as in Fig. 1.

269 At the leading edge of the front invading the disease-free equilibrium, i and v
 270 have small positive values. We linearise system (3) at the leading edge:

$$\begin{aligned} i_\tau &= \beta v - \rho i, \\ v_\tau &= \theta u i - v + v_{\xi\xi}. \end{aligned}$$

271 We are interested in travelling wave solutions such that

$$y = \begin{pmatrix} i \\ v \end{pmatrix} = k \exp(-s(\xi - c\tau)),$$

272 in which k is an implicit column vector, c is the linear wave speed, and s is the
 273 exponential decay rate of the wave profile at leading edge.

274 Plugging the previous expression in the system, we obtain

$$s c y = \underbrace{\begin{bmatrix} -\rho & \beta \\ \theta u & -1 + s^2 \end{bmatrix}}_{M_s} y, \quad (11)$$

275 which implies that

$$\det \underbrace{\begin{bmatrix} -\rho - sc & \beta \\ \theta u & -1 + s^2 - sc \end{bmatrix}}_{M_{s-scI}} = 0,$$

276 in which I is the identity matrix. This yields

$$\begin{aligned} 0 &= (-\rho - sc)(-1 + s^2 - sc) - \theta u \beta, \\ &= Fc^2 + Gc + H, \end{aligned} \tag{12}$$

277 with

$$F = s^2, \quad G = s(\rho + 1 - s^2), \quad H = \rho(1 - s^2) - \theta u \beta.$$

278 Next, we follow the approach of using Eq. (12) to calculate the minimum linear wave
279 speed as outlined in Hadeler and Lewis (2002).

280 The discriminant of the quadratic in Eq. (12) is

$$\begin{aligned} \Lambda &= G^2 - 4FH, \\ &= s^2 \left((\rho + 1 - s^2)^2 - 4\rho(1 - s^2) + 4\theta u \beta \right), \\ &= s^2 \left((\rho - 1 + s^2)^2 + 4\theta u \beta \right) > 0. \end{aligned}$$

281 Since $\Lambda > 0$, there are two real roots:

$$z = \frac{-G - \sqrt{\Lambda}}{2F} \quad \text{and} \quad c = \frac{-G + \sqrt{\Lambda}}{2F}.$$

282 First, we show that $z < 0$. If $G > 0$, then $z < 0$ since $F > 0$. Otherwise (if $G < 0$),
283 then $-G - \sqrt{\Lambda} > 0$ is equivalent to $0 > -4FH$, which is impossible since $H < 0$ (this is
284 because $G < 0$ implies $1 - s^2 < 0$).

285 Second, we show that $c > 0$ for all $s > 0$. If $G < 0$, then $c > 0$ since $F > 0$.
286 Otherwise (if $G > 0$), then $-G + \sqrt{\Lambda} > 0 \Leftrightarrow \Lambda > G^2$ is equivalent to $\mathcal{R}_0^2 > 1 - s^2$, which
287 is satisfied since we assume $\mathcal{R}_0^2 > 1$ in this section.

288 The relevant root is therefore c . Since Eq. (12) only depends on three additional

289 parameters, s , ρ and $\beta\theta u$, we express c as a function of these parameters:

$$c(s, \rho, \beta\theta u) = \frac{-(\rho + 1 - s^2) + \sqrt{(\rho - 1 + s^2)^2 + 4\theta u\beta}}{2s}.$$

290 Since c is a convex function of s (Appendix B.1), $\lim_{s \rightarrow 0} c(s, \rho, \beta\theta u) = +\infty$, and
 291 $\lim_{s \rightarrow +\infty} c(s, \rho, \beta\theta u) = +\infty$, there exists a minimum to c with respect to $s > 0$.

292 Equation (12) can also be written to include the dependency of c on s , ρ , and $\beta\theta u$
 293 as

$$P(c(s, \rho, \beta\theta u), s) := (-\rho - sc)(-1 + s^2 - sc) - \theta u\beta.$$

294 Differentiating with respect to s , we have, for all s ,

$$\frac{dP}{ds} = \frac{\partial P}{\partial c} \frac{\partial c}{\partial s} + \frac{\partial P}{\partial s} = 0. \quad (13)$$

295 We are interested in the minimum possible linear wave speed. Let

$$s^*(\rho, \beta\theta u) = \arg \min_s c(s, \rho, \beta\theta u),$$

296 and

$$c^*(\rho, \beta\theta u) = c(s^*(\rho, \beta\theta u), \rho, \beta\theta u).$$

297 Since c^* is such that $\partial c/\partial s = 0$, Eq. (13) yields

$$\frac{\partial P}{\partial s}(c^*(\rho, \beta\theta u), s^*(\rho, \beta\theta u)) = 0. \quad (14)$$

298 Since P is cubic in s , $\partial P/\partial s$ is quadratic in s . We are interested in the conditions
 299 on the coefficients that allow both polynomials to have a common root, s^* . They are
 300 given by cancelling the resultant of the two polynomials. Letting $P = es^3 + fs^2 + gs + h$
 301 yields $\partial P/\partial s = 3es^2 + 2fs + g$. The coefficients are identified as

$$e = -c, \quad f = c^2 - \rho, \quad g = (\rho + 1)c, \quad h = -\theta u\beta + \rho.$$

302 The resultant is $r(e, f, g, h) = -e(f^2g^2 - 4eg^3 - 4f^3h + 18efgh - 27e^2h^2)$, as de-
 303 scribed in Janson (2010, Eq. (4.3)). The equality $r(e, f, g, h) = 0$ can be equivalently

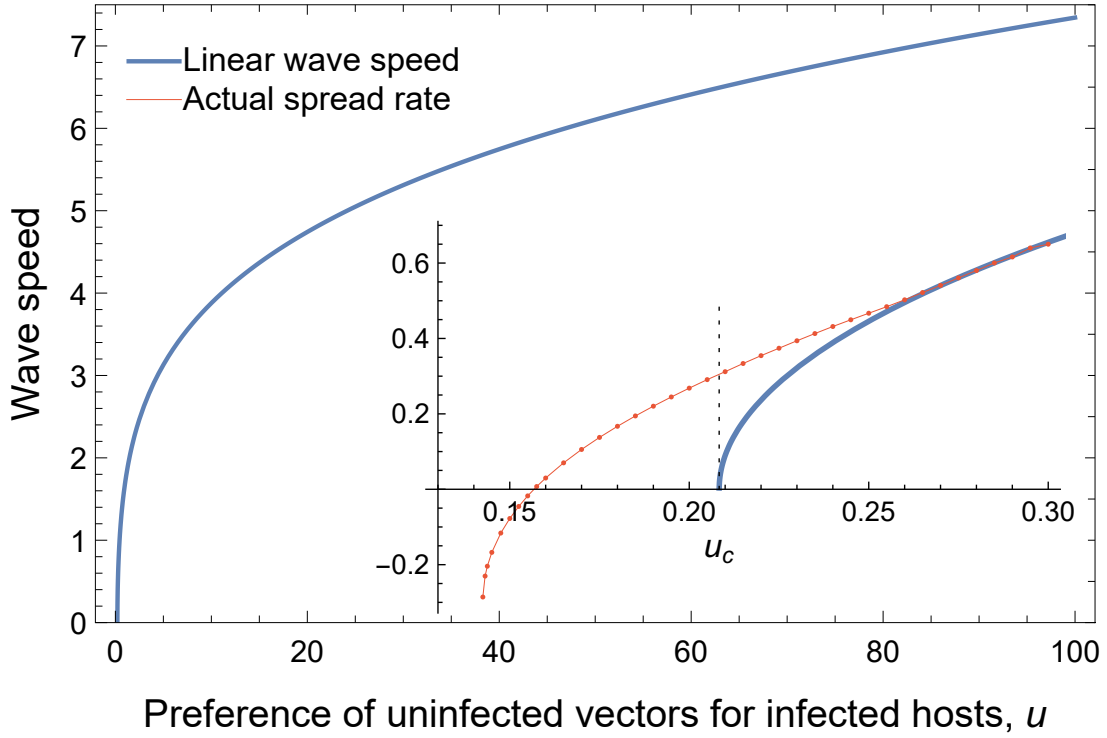


Figure 5: Linear speed of the monostable travelling wave as a function of u (the preference of uninfected vectors for infected hosts). The inset zooms on small values of u and compares the linear and actual (numerically computed) spreading speeds in model (3). The spreading speed is not linearly determined in the bistable case ($u < u_c$) and in the monostable case ($u > u_c$) for u close to u_c . However, the actual speed quickly converges to the linear speed as u increases. Other parameter values as in Fig. 1.

304 expressed as a cubic with respect to c^2 :

$$c_3(c^2)^3 + c_2(c^2)^2 + c_1(c^2)^1 + c_0 = 0, \quad (15)$$

305 with

$$\begin{aligned} c_3 &= 4\beta\theta u + (\rho - 1)^2, \\ c_2 &= 2\rho^3 + 2\rho^2 + (6\beta\theta u - 8)\rho + 18\theta u\beta + 4, \\ c_1 &= \rho^4 + 8\rho^3 - (6\beta\theta u + 8)\rho^2 + 36u\rho\beta\theta - 27u^2\beta^2\theta^2, \\ c_0 &= -4\rho^3(\beta\theta u - \rho) = -4\rho^4(\mathcal{R}_0^2 - 1). \end{aligned}$$

306 Since we assume $\mathcal{R}_0^2 > 1$, we have that c_0 is negative and c_3 is positive, which
 307 means that we are in the same configuration as Hadeler and Lewis (2002). This
 308 implies that $c^*(\rho, \beta\theta u)$ is uniquely defined as the square root of the largest root of
 309 the above cubic.

310 Although it is possible to write down the formula for the largest root of a cu-
 311 bic polynomial, we have no simple expression of $c^*(\rho, \beta\theta u)$. Figure 5 shows the
 312 minimum linear speed of the monostable travelling wave solution as a function of u ,
 313 as obtained by solving the cubic equation (15).

314 4.3 Bistable case

315 In this section, we assume $\mathcal{R}_0^2 < 1$.

316 4.3.1 Existence of a travelling wave

317 To show the existence of a bistable travelling wave solution, we will consider The-
 318 orem 4.2 in Fang and Zhao (2009). We have to verify that assumption (L): $f \in$
 319 $C^1(\mathbb{R}^2, \mathbb{R}^2)$ satisfies the following conditions:

- 320 1. $f(0) = f(E_2) = f(E_1) = 0$, with $0 \ll E_1 \ll E_2$. There is no η other than 0 , E_1 and E_2
 321 such that $f(\eta) = 0$, with $0 \leq \eta \leq E_2$.
- 322 2. System (3) is cooperative.
3. $y \equiv 0$ and $y \equiv E_2$ are stable while $y \equiv E_1$ is unstable, that is

$$\lambda_0 := s(f'(0)) < 0, \quad \lambda_{E_2} := s(f'(E_2)) < 0, \quad \lambda_{E_1} = s(f'(E_1)) > 0.$$

- 323 4. $f'(0)$, $f'(E_1)$, and $f'(E_2)$ are irreducible.

324 Assuming that assumption (L) holds, then according to Theorem 4.2 in Fang and
 325 Zhao (2009), system (3) admits a monotone wavefront (U, c) with $U(-\infty) = 0$ and
 326 $U(+\infty) = E_2$.

327 Since $\mathcal{R}_0^2 < 1$, two positive endemic equilibria, E_1 and E_2 , exist. Equilibrium E_1 is
 328 unstable while E_2 is LAS. Thanks to the results obtained in Section 3, it is straight-
 329 forward to check that assumption (L) holds and to conclude that a travelling wave
 330 solution connecting 0 and E_2 exists. See, for instance, Fig. 6.

331 In Fig. 7, we show that for u sufficiently small, the sign of the spreading speed
 332 can change. Thus, in the bistable case, for a given $\alpha \gg 1$, there exist u^\dagger and $u^{*,\dagger}$
 333 such that for $u^\dagger < u < u^*$ the disease travelling wave moves forward, while for

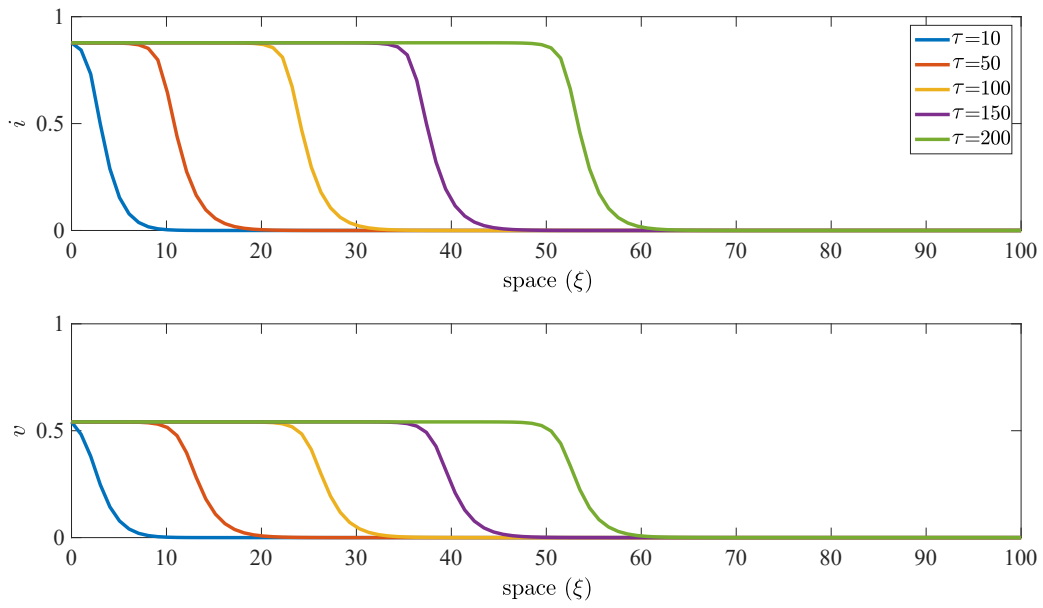


Figure 6: Bistable travelling wave solution of model (3) connecting the disease-free and endemic equilibria 0 and E_2 when $\mathcal{R}_0^2 = 0.96 < 1$. Here $u = 0.2$, other parameter values as in Fig. 1. The disease is invading, $c^* > 0$.

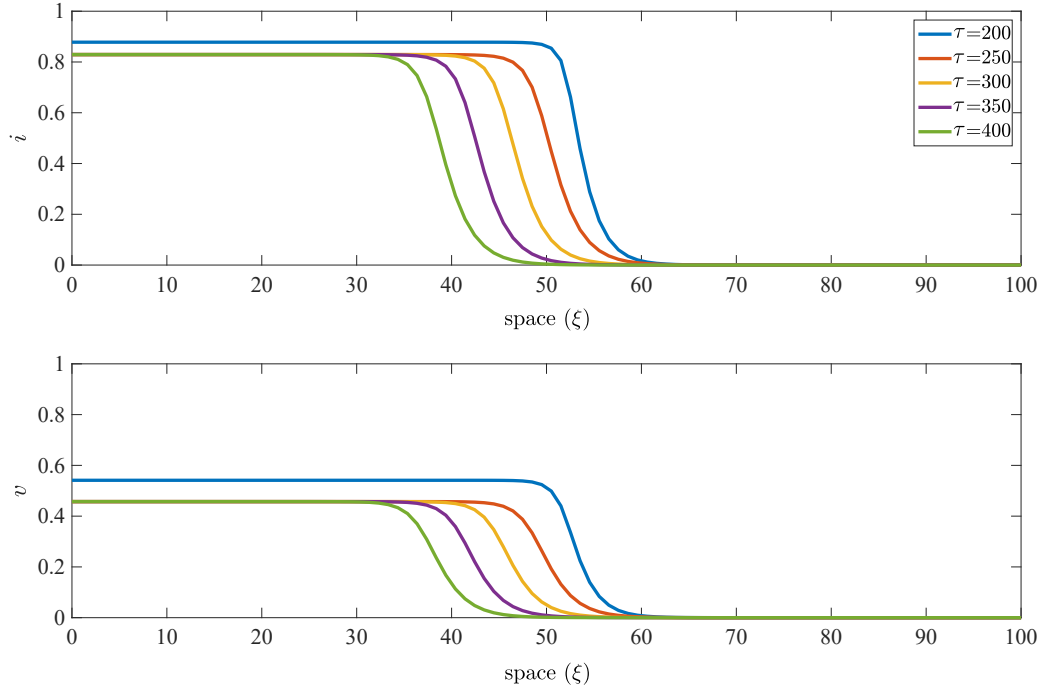


Figure 7: Bistable travelling wave solution of model (3) connecting the disease-free and endemic equilibria 0 and E_2 when $\mathcal{R}_0^2 = 0.72 < 1$. Here $u = 0.15$, other parameter values as in Fig. 1. Starting at $t = 200$ with the solution from Fig. 6 as initial condition, the spread is reversing, $c^* < 0$. This shows that a small variation of the parameter u (switching from $u = 0.2$ in Fig. 6 to $u = 0.15$ in this figure) can make the spreading speed switch from positive to negative. This is why the equilibrium prevalences decrease compared to initial conditions (at $t = 200$).

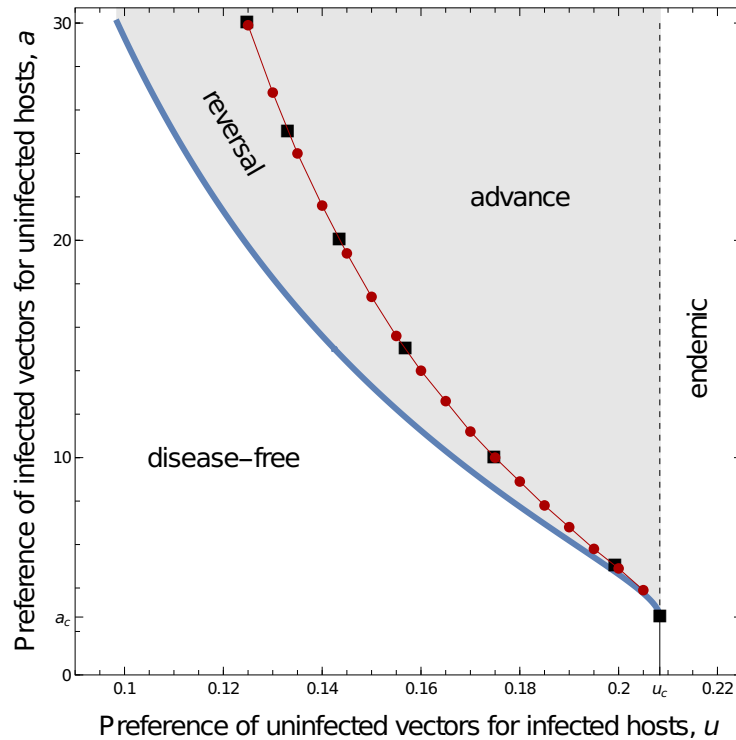


Figure 8: Within the bistable parameter domain (light grey), travelling waves connecting the disease-free and endemic equilibria can reverse or advance. The parameter domains of reversal and advance are separated by a curve corresponding to stalled waves with zero wave speed. Here, the zero-wave speed curve is obtained, on the one hand, by numerical integration of the PDE system (3) with $\rho = 1$, $\beta = 2.4$, $\theta = 2$ and identifying parameter values to result in zero wave speed, accurate to at least the third decimal place (grey squares). On the other hand, the zero wave speed curve was indicated by Eq. (25) in Appendix B.2 using a quasi-steady-state assumption (red points). Other curves as in Fig. 3.

334 $u^{*,\dagger} < u < u^\dagger$ the disease travelling wave moves backward. When $u < u^{*,\dagger}$ then the
 335 system converges to 0.

336 4.3.2 Quasi-steady-state approximation

337 For the case of a quasi-steady-state approximation (QSSA) (see Appendix B.2 for
 338 details), we can gain more information on the parameter domain for which the trav-
 339 elling wave moves forward or backward. The direction is given by the expression (25)
 340 in Appendix B.2.

341 Figure 8 marks the boundary between wave advancement and reversal by red
 342 dots. This boundary corresponds to stalled traveling waves with speed zero. The
 343 QSSA results match very well with the zero wave speeds in the original system with-
 344 out a quasi-steady-state approximation (dark grey squares). This match may be
 345 particularly surprising because we have chosen a time scale parameter of $\rho = 1$ for
 346 the simulations, while the QSSA is based on the assumption $\rho \gg 1$. However, Fig. 9

347 in Appendix B.2 suggests that the wave speed approximations do not deviate much
348 from the exact solutions for small spreading speeds and $\rho \geq 1$. This behaviour might
349 explain why the QSSA correctly locates the $c = 0$ curve in Fig. 8.

350 In the monostable case, the QSSA allows us to derive an explicit expression for
351 the linear wave speed (see Eq. (23) in Appendix B.2), which is simply $c^* = 2\sqrt{\mathcal{R}_0^2 - 1}$.
352 However, the linear spreading speed is only a lower bound of the actual spreading
353 speed in some cases (Fig. 11).

354 **5 Discussion**

355 We have shown that conditional vector preferences may result in bistability between
356 the disease-free equilibrium and an endemic equilibrium. The novelty compared
357 to Gandon (2018) and Cunniffe et al. (2021) is that bistability here occurs in the
358 absence of any epidemiological feedback on vector population dynamics.

359 More specifically, we have shown that conditional vector preferences can cause a
360 “backward bifurcation” (Fig. 2), meaning that $\mathcal{R}_0^2 < 1$ is not a sufficient condition for
361 the disease to go extinct (e.g., Haderer and van den Driessche, 1997).

362 **5.1 Bistability conditions**

363 We have shown that for bistability to occur, the following necessary conditions must
364 be satisfied: $\mathcal{R}_0^2 < 1$, $a > 1$ and $u < u^* < 1$. The first condition ($\mathcal{R}_0^2 < 1$) means that
365 the basic reproductive number of the pathogen is not large enough for the pathogen
366 to invade a disease-free population. Hence, the disease-free equilibrium is locally
367 stable. However, if the prevalence of the infection is initially high, and if infected
368 vectors have a sufficiently strong preference for uninfected hosts ($a > 1$), we have
369 shown that the pathogen may persist in the population (an endemic equilibrium
370 is locally stable as well) even though $\mathcal{R}_0^2 < 1$. These two conditions ($a > 1$ and
371 $\mathcal{R}_0^2 < 1$) are not too surprising. The third condition (implying $u < 1$) is less intuitive.
372 To interpret it, we recall that \mathcal{R}_0^2 is proportional to u . If $\mathcal{R}_0^2 < 1$ in spite of $u > 1$
373 (uninfected vectors prefer infected hosts, which is advantageous for the pathogen),
374 this means that the pathogen has poor reproductive abilities. Therefore, even if
375 the prevalence of the infection is initially high, the pathogen still goes extinct. By

376 contrast, if $\mathcal{R}_0^2 < 1$ while $u < 1$ (uninfected vectors prefer uninfected hosts), it may
377 be that the pathogen has strong enough reproductive abilities not to go extinct when
378 its prevalence is initially high, and infected vectors prefer uninfected hosts ($a > 1$).

379 **5.2 Travelling waves**

380 **5.2.1 Monostable case**

381 In the monostable case ($\mathcal{R}_0^2 > 1$), the disease invades the spatial domain. We have
382 shown that the linear spreading speed depends only on ρ and $\beta\theta u$, meaning that it
383 does not depend on a , the preference of infected vectors for uninfected hosts. The
384 interpretation is the same as for the basic reproductive number, $\mathcal{R}_0^2 = \beta\theta u/\rho$, which
385 does not depend on a either (Roosien et al., 2013; Gandon, 2018; Cunniffe et al.,
386 2021). In a situation close to the disease-free equilibrium, like at the leading edge
387 of the front, there are so few infected hosts that the preference of infected vectors
388 for uninfected hosts has a negligible effect on the dynamics. However, even in the
389 monostable case, the spreading speed may not be linearly determined (Fig. 5), im-
390 plying that it may depend on a (Fig. 12). This is due to the fact that disease spread
391 is not driven by the leading edge of the invasion front (“pulled wave”). Instead, the
392 disease invasion is driven by the whole of the front (“pushed wave”) (Stokes, 1976;
393 Lewis and Kareiva, 1993). In particular, the disease spread may be maximum for
394 intermediate prevalences because of the conditional preferences (similar to weak
395 and strong Allee effects where population growth is strongest at intermediate densi-
396 ties). By contrast, dynamics of pulled waves are independent from the nonlinearities
397 behind the leading edge of the front.

398 **5.2.2 Bistable case**

399 In the bistable case (requiring $\mathcal{R}_0^2 < 1$), the disease either invades or retreats, de-
400 pending on parameter values. More specifically, travelling waves may have negative
401 speeds, meaning that the disease retreats.

402 In an epidemiological context, such a “front reversal” has been shown to occur
403 when host population dynamics in the absence of disease are bistable, due, for
404 instance, to a strong Allee effect in the host (Hilker et al., 2005, 2007). However,

405 to our knowledge, such a phenomenon has seldom (Bocharov et al., 2016) been
406 shown to occur when bistability is due solely to the epidemiological dynamics.

407 **5.3 Biological implications**

408 Although conditional vector preferences might occur in human and animal diseases,
409 they have so far been shown mainly in plant diseases (Gandon, 2018). Therefore,
410 we now discuss plant diseases more specifically.

411 Plant diseases are a main threat to global food security (Ristaino et al., 2021).
412 Many plant diseases are caused by pathogens (viruses, bacteria and others) that
413 are transmitted by insect vectors such as aphids, whiteflies, and others (Eigenbrode
414 et al., 2018). Infected vectors can be attracted to uninfected plants. This is for
415 instance the case for aphids, *Rhopalosiphum padi*, infected by the Barley yellow
416 dwarf virus (BYDV) (Ingwell et al., 2012). In his review of evidence for conditional
417 vector preferences, Gandon (2018) identified the volatile compounds emitted by
418 infected plants as an attraction mechanism for (uninfected) vectors. For instance,
419 plants infected by the *Cucumber mosaic virus* (CMV) or the *Tomato chlorosis virus*
420 (ToCV) produce volatiles that attract aphids or whiteflies (Ferreles et al., 2016). Note
421 also that vectors can be attracted to infected plants by visual cues, such as, for
422 instance, yellow leaves.

423 Since the basic reproductive number of the pathogen (\mathcal{R}_0^2) is proportional to u
424 (the preference of uninfected vectors for infected hosts), a plant variety that emits
425 fewer volatiles could be considered resistant to disease. When visual cues are re-
426 sponsible for vector preferences, a plant variety that expresses fewer symptoms,
427 and is therefore less attractive to uninfected vectors, could also be considered resis-
428 tant. Deployment of such resistant hosts might make it possible to obtain $\mathcal{R}_0^2 < 1$.
429 We have shown that $u \geq 1$ (a preference of uninfected vectors for infected hosts)
430 ensures disease extinction in this case ($\mathcal{R}_0^2 < 1$), since bistability requires $u < 1$.
431 This means that breeding for varieties that emit, when infected, a concentration
432 of volatiles that is lower than that of standard varieties is a possible strategy for
433 the control of vector-borne diseases (in combination with other strategies such as
434 roguing - i.e., removing - infected plants, for instance).

435 **5.4 Mathematical prospects**

436 An alternative for modelling vector preference could be density-dependent advec-
437 tion (in analogy to preytaxis, this could perhaps be called “hosttaxis”). It has been
438 shown that preytaxis in the presence of disease, where predators are attracted to or
439 repelled by infected prey, can speed up or even lead to irregularly fluctuating travel-
440 ling waves (Bate and Hilker, 2019). While Chamchod and Britton (2011) considered
441 a “hosttaxis” term in their model, it was only a vector bias towards infected hosts,
442 regardless of whether the vector carries the pathogen or not. Modelling conditional
443 vector preferences with a hosttaxis term is beyond the scope of this paper and is left
444 for future research.

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456

457 **A Side results on the non-spatial model**

458 **A.1 Case $\mathcal{R}_0^2 = 1$ (boundary case)**

459 If $\mathcal{R}_0^2 = 1$, or equivalently

$$u = \frac{\rho}{\beta\theta},$$

then $C = 0$ and $i^* = -B/A$. Using the above expression of u yields

$$i^* = 1 - \frac{1}{(a-1)\left(\frac{\beta\theta}{(1+\theta)^\rho} - 1\right)}.$$

460 If $a = 1$, the endemic equilibrium does not exist. In what follows, we assume $a \neq 1$.

461 Let the fraction of susceptible hosts at equilibrium be

$$s^* = \frac{1}{(a-1)\left(\frac{\beta\theta}{(1+\theta)^\rho} - 1\right)}.$$

462 We have:

$$s^* > 0 \Leftrightarrow \begin{cases} \frac{\beta\theta}{(1+\theta)^\rho} > 1 & \text{if } a > 1, \\ \frac{\beta\theta}{(1+\theta)^\rho} < 1 & \text{if } a < 1. \end{cases}$$

463 Assuming $s^* > 0$, $s^* < 1$ is equivalent to

$$(a-1)\left(\frac{\beta\theta}{(1+\theta)^\rho} - 1\right) > 1.$$

464 Two cases can then be distinguished:

465 • If $a > 1$, $s^* < 1$ is equivalent to

$$\frac{\beta\theta}{(1+\theta)^\rho} - 1 > \frac{1}{a-1} \Leftrightarrow \frac{\beta\theta}{(1+\theta)^\rho} > \frac{a}{a-1}.$$

466 • If $a < 1$, $s^* < 1$ is equivalent to

$$\left(1 - \frac{\beta\theta}{(1+\theta)^\rho}\right)(1-a) > 1 \Leftrightarrow 1 - \frac{\beta\theta}{(1+\theta)^\rho} > \frac{1}{1-a} \Leftrightarrow -\frac{\beta\theta}{(1+\theta)^\rho} > \frac{a}{1-a},$$

467 which is impossible.

468 Therefore, $0 < s^* < 1$ if and only if

$$a > 1 \quad \text{and} \quad \frac{\beta\theta}{(1+\theta)^\rho} > \frac{a}{a-1} > 1. \quad (16)$$

469 **A.2 Case $\mathcal{R}_0^2 > 1$**

470 To derive the expression of the endemic equilibrium, we consider three cases: $a = 1$,

471 $0 < a < 1$ and $a > 1$.

472 **Case $a = 1$.** If $a = 1$, then $A = 0$ and

$$i^* = -\frac{C}{B}.$$

473 Since $\mathcal{R}_0^2 > 1$,

$$B = -\left((1 + \theta)u + \mathcal{R}_0^2 - 1\right) < 0.$$

474 Therefore,

$$i^* = \frac{\mathcal{R}_0^2 - 1}{(1 + \theta)u + \mathcal{R}_0^2 - 1}.$$

475 We have $0 < i^* < 1$.

476 **Case $0 \leq a < 1$.** Since

$$B = -\left(\left((1 + \theta)u + \mathcal{R}_0^2 - 1\right)a + (1 - a)\right),$$

477 we deduce that $B < 0$. Then, we have three sub-cases to consider:

478 • If $u > u^*$, then $A < 0$. The relevant root is therefore the largest:

$$\frac{1}{2A}(-B - \sqrt{\Delta}),$$

479 since the other root is negative.

480 • If $u = u^*$, $A = 0$. We obtain

$$i^* = \frac{\mathcal{R}_0^2 - 1}{\mathcal{R}_0^2 - 1 + \frac{1}{a}}. \tag{17}$$

481 We have $0 < i^* < 1$.

482 • If $u < u^*$, then $A > 0$. The relevant root is therefore the smallest:

$$\frac{1}{2A}(-B - \sqrt{\Delta}),$$

483 since both roots are positive.

484 **Case $a > 1$.** We again distinguish three sub-cases:

Table 1: The component i^* of the endemic equilibrium in the specific case $\mathcal{R}_0^2 > 1$, expressed as a function of A , B , C and $\Delta = B^2 - 4AC$.

	$0 < a < 1$	$a = 1$	$a > 1$
$u < u^*$	$\frac{1}{2A}(-B - \sqrt{\Delta})$	$-\frac{C}{B}$	$\frac{1}{2A}(-B - \sqrt{\Delta})$
$u = u^*$	$-\frac{C}{B}$	$-\frac{C}{B}$	$-\frac{C}{B}$
$u > u^*$	$\frac{1}{2A}(-B - \sqrt{\Delta})$	$-\frac{C}{B}$	$\frac{1}{2A}(-B - \sqrt{\Delta})$

- 485 • If $u > u^*$, then $A > 0$ and $B < 0$. The relevant root is therefore the smallest:

$$\frac{1}{2A}(-B - \sqrt{\Delta}),$$

486 since both roots are positive.

- 487 • If $u = u^*$, $A = 0$. We again find expression (17).

- 488 • If $u < u^*$, then $A < 0$. The relevant root is therefore the largest:

$$\frac{1}{2A}(-B - \sqrt{\Delta}),$$

489 since the other root is negative.

490 These results are summarized in Tab. 1.

491

492 **A.3 Case $\mathcal{R}_0^2 < 1$**

493 We here focus on the necessary condition $\Delta = B^2 - AC > 0$ for the existence of
 494 two endemic equilibria in the case $\mathcal{R}_0^2 < 1$. Using the notations $X = u(1 + \theta) - 1$ and
 495 $Y = \mathcal{R}_0^2 - 1$ (Eq. (10)) yields the following expression of Δ as a quadratic function of
 496 a :

$$\Delta = (X - Y)^2 a^2 + 2(X(1 + Y) + Y(1 + X))a + 1.$$

497 Since $(X - Y)^2 > 0$, this parabola has a U-shape. We also have $\Delta(0) = 1 > 0$. There-
 498 fore, either there are two positive roots, a_c^- and a_c^+ , or there are none. In the latter
 499 case, $\Delta > 0$ regardless of the value of a . In case there are two roots, the largest one,

$$a_c^+ = \frac{-((X(1 + Y) + Y(1 + X)) + 2\sqrt{XY(1 + X)(1 + Y)})}{(X - Y)^2},$$

500 can also be expressed as

$$a_c^+ = \left(\frac{\sqrt{-X(1+Y)} + \sqrt{-Y(1+X)}}{X-Y} \right)^2.$$

501 Similarly, a_c^- can be expressed as

$$a_c^- = \left(\frac{\sqrt{-X(1+Y)} - \sqrt{-Y(1+X)}}{X-Y} \right)^2.$$

502 Since $1+X = u(1+\theta) > 0$, $1+Y = \mathcal{R}_0^2 > 0$, and $Y = \mathcal{R}_0^2 - 1 < 0$, the existence of two
 503 conjugate roots requires $X = u(1+\theta) - 1 < 0$, or equivalently $u < u^*$.

504 We now focus on the condition $a > a_c^+$ (implying $\Delta > 0$) since it happens to co-
 505 incide with the separatrix we numerically obtained in the parameter space (Fig. 3).

506 Let us express a_c^+ as a function of the original parameters:

$$a_c^+ = \left(\frac{\sqrt{-(u(1+\theta)-1)\mathcal{R}_0^2} + \sqrt{-(\mathcal{R}_0^2-1)u(1+\theta)}}{u(1+\theta) - \mathcal{R}_0^2} \right)^2, \quad (18)$$

507 or equivalently:

$$a_c^+(u) := \left(\frac{\sqrt{(1-\frac{u}{u^*})\mathcal{R}_0^2} + \sqrt{(1-\mathcal{R}_0^2)\frac{u}{u^*}}}{\frac{\mathcal{R}_0^2}{u^*}(u_c - u^*)} \right)^2.$$

508 Assuming $a_c^+(u)$ is defined for all $u \in [0, u_c]$ implies $u_c < u^*$.

509 In particular, since u_c is such that $\mathcal{R}_0^2 = 1$, we have

$$a_c^+(u_c) = \frac{1}{1 - \frac{u_c}{u^*}} =: a_c. \quad (19)$$

510 The condition $a > a_c$ (implying $a > 1$) is equivalent to the condition we obtained for
 511 the existence of an endemic equilibrium in the boundary case $\mathcal{R}_0^2 = 1$, see Eq. (16).

512 This means that in Fig. 3, the line $\mathcal{R}_0^2 = 1$ and the separatrix between the “disease-
 513 free” and “bistability” regions meet at the point (u_c, a_c) .

514

515 **B Side results on the spatial model**

516

517 **B.1 Existence of a minimum linear spreading speed**

518 The function of the form $\xi \mapsto \exp(-\xi s)y$ is a solution of system (3) linearised
 519 around the disease-free equilibrium if and only if $scy = M_s y$, in which

$$M_s = \begin{pmatrix} -\rho & \beta \\ u\theta & -1 + s^2 \end{pmatrix},$$

520 see Eq. (11). Since M_s is irreducible and, for all $s > 0$, essentially non-negative,
 521 the Perron-Frobenius theorem provides the existence of a unique eigenvalue κ_s of
 522 M_s associated to a positive eigenvector (Crooks, 1996)[Theorem 1.4]. Therefore,
 523 $sc = \kappa_s$. Since $c = \kappa_s/s$ is the dominant eigenvalue of $\frac{1}{s}M_s$, c is a convex function of s
 524 (Cohen, 1981).

525 **B.2 Quasi-steady-state approximation**

526 In this section, we make a quasi-steady-state approximation to reduce our model to
 527 a single dimension (similarly to Hamelin et al., 2016).

528 Model (3) can be equivalently expressed as:

$$\begin{aligned} \frac{1}{\rho}i_\tau &= \frac{\beta}{\rho}v \frac{a(1-i)}{a(1-i)+i} - i, \\ v_\tau &= \theta(1-v) \frac{ui}{ui+1-i} - v + v_{\xi\xi}. \end{aligned} \tag{20}$$

529 We consider the case where the infected vector removal rate ($m + l$) is much lower
 530 than the removal rate of infected hosts r , so $\rho = r/(m + l) \gg 1$. This might happen in
 531 plant viruses if roguing occurs frequently relative to the vector lifespan ($r \gg m$), and
 532 the virus is persistent in the vector ($l = 0$).

533 We apply the quasi-steady-state approximation to the first equation of (20) to
 534 yield the fraction of infected hosts i directly in terms of the fraction of infected vec-
 535 tors v as

$$0 < i^\#(v) := \frac{\left(\frac{\beta}{\rho}v + 1\right)a - \sqrt{\left(\left(\frac{\beta}{\rho}v - 1\right)^2 a + 4\frac{\beta}{\rho}v\right)a}}{2(a-1)} < 1.$$

536 (It can be easily shown that the other root is greater than unity.)

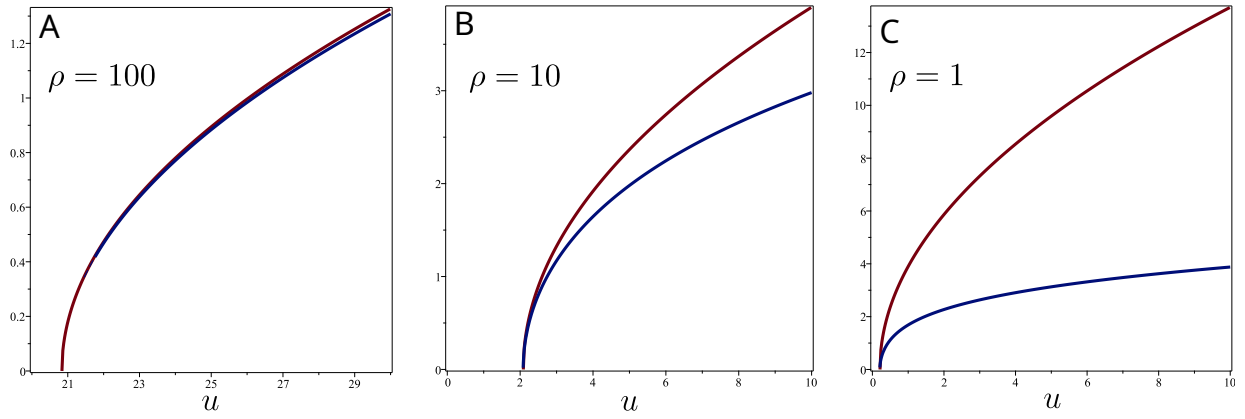


Figure 9: Exact linear spreading speed (c^* , in red), as given by numerically solving Eq. (15), and approximated linear spreading speed (c^* , in blue), as given by Eq. (23) in the monostable case. Our quasi-steady-state-approximation (QSSA) assumes $\rho \gg 1$. It is therefore unsurprising that the QSSA performs badly when $\rho \leq 10$. However, the approximation does not seem to deviate much from the exact solution for small spreading speeds and $\rho \geq 1$. This might explain why the QSSA correctly locates the $c = 0$ curve in Fig. 8. Here, $\beta = 2.4\rho$, other parameter values as in Fig. 1.

537 This yields

$$v_t \approx \theta(1-v) \frac{ui^\#(v)}{ui^\#(v) + 1 - i^\#(v)} - v + v_{\xi\xi} =: W(v) + v_{\xi\xi}. \quad (21)$$

538

539 B.2.1 Monostable case ($\mathcal{R}_0^2 > 1$)

540 It is useful to notice that in the monostable case ($\mathcal{R}_0^2 > 1$), $W(0) = 0$, $W(v^*) = 0$,
 541 and $W(v) > 0$ for all $v \in (0, v^*)$. It is well known that if

$$\frac{W(v)}{v} < W'(0) \quad \text{for all } v \in (0, v^*), \quad (22)$$

542 the spreading speed of the wave is linearly determined (Stokes, 1976; Lewis and
 543 Kareiva, 1993):

$$c^* = 2\sqrt{W'(0)} = 2\sqrt{\frac{\beta}{\rho}\theta u - 1} = 2\sqrt{\mathcal{R}_0^2 - 1}. \quad (23)$$

544

545 Fig. 9 compares the linear speed under the QSSA (Eq. (21)) with the exact linear
 546 spreading speed given by Eq. (15). The QSSA performs well for large values of ρ , but
 547 performs increasingly badly for smaller values of ρ that do not meet the assumption
 548 $\rho \gg 1$ behind the QSSA.

549 Note, however, that if condition (22) is not satisfied, the spreading speed may

550 not be linearly determined. A sufficient condition for condition (22) not to hold is
 551 $W''(0) > 0$. We have

$$W''(0) = -\frac{2\frac{\beta}{\rho}u\theta\left((1+(u-1)a)\frac{\beta}{\rho}+a\right)}{a},$$

552 and so $W''(0) > 0$ is equivalent to

$$u < \frac{\frac{\beta}{\rho}(a-1)-a}{a\frac{\beta}{\rho}}.$$

553 Or equivalently,

$$(u-1)\frac{\beta}{\rho}+1 < 0 \quad \text{and} \quad a > \frac{\frac{\beta}{\rho}}{-((u-1)\frac{\beta}{\rho}+1)} =: \tilde{a}(u). \quad (24)$$

554 We also have

$$\tilde{a}(u_c) = \frac{\frac{\beta}{\rho}}{\frac{\beta}{\rho}-\frac{1}{\theta}-1} = \frac{1}{1-\frac{u_c}{u^*}} = a_c,$$

555 see Eq. (19). This means that the curve separating pulled waves (linear speed)
 556 with pushed waves (nonlinear speed) in the parameter plane “originates” at (u_c, a_c)
 557 (Fig. 10).

558

559 **B.2.2 Bistable case ($\mathcal{R}_0^2 < 1$)**

560 In the bistable case ($\mathcal{R}_0^2 < 1$), the wave speed is not linearly determined. How-
 561 ever, it is well known (Fife and McLeod, 1977) that

$$\text{sign}(c^*) = \text{sign}\left(\int_0^{v_2^*} W(v)dv\right), \quad (25)$$

562 where v_2^* is the stable nontrivial equilibrium of (21). Hence, the travelling wave has
 563 positive (negative) speed when the net area between the growth dynamics $W(v)$
 564 of the approximated system (21) and the horizontal axis in the range between the
 565 disease-free state and the stable endemic state is positive (negative, respectively).

566 Figure 11 compares the linear spreading speed with the actual (numerically com-
 567 puted) spreading speed under the QSSA. It shows that in the bistable case ($u < u_c$),
 568 the spreading speed can be either negative or positive. In the monostable case

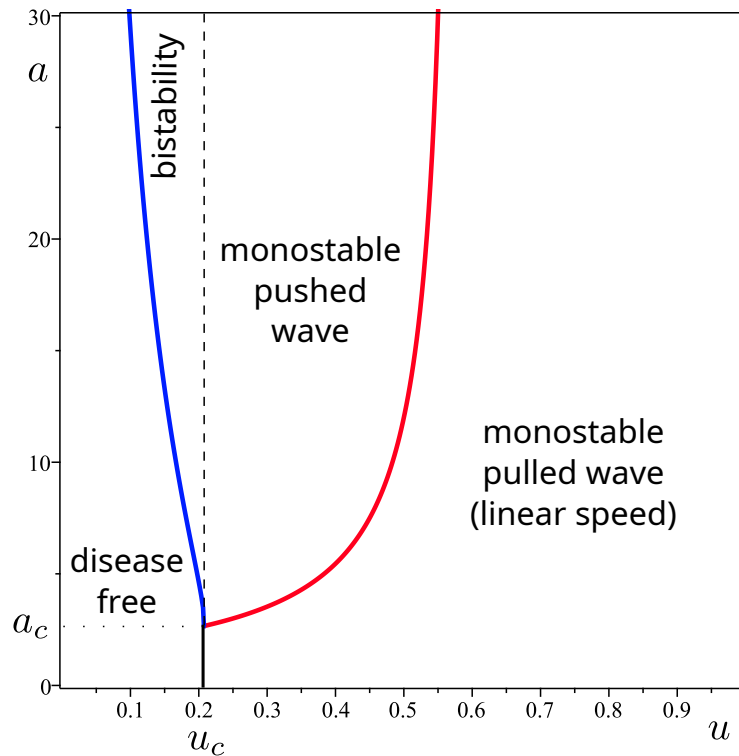


Figure 10: Two-parameter bifurcation analysis under the quasi-steady-state approximation (QSSA), i.e., model (21). The black line connecting $(u_c, 0)$ to (u_c, a_c) is the boundary between the monostable case with linear speed and the disease-free region. The blue line separates the disease-free region from the bistability region, as given by Eq. (18). The red line separates the monostable/pushed wave from the monostable/pulled wave (linear speed) region, as given by Eq. (24). Note that Eq. (24) only depends on β/ρ , while Eq. (18) only depends on θ and β/ρ through $\mathcal{R}_0^2 = \beta\theta u/\rho$. Parameter values: $\theta = 2$ and $\beta/\rho = 2.4$. Note, however, that ρ must be much greater than 1 for the QSSA to hold.

569 $(u > u_c)$, the actual spreading speed significantly deviates from the linear speed for
 570 u close to u_c , but the actual speed converges to the linear speed as u increases.

571 Figure 12 shows that the actual spreading does not depend on a when it is well
 572 approximated by the linear speed (for $u > 0.3$), while it increasingly depends on a as
 573 u decreases from $u = 0.3$. The dependency is greater in the bistable case ($u = 0.15$)
 574 than in the monostable pushed case ($u = 0.2$). As expected, the spreading speed is
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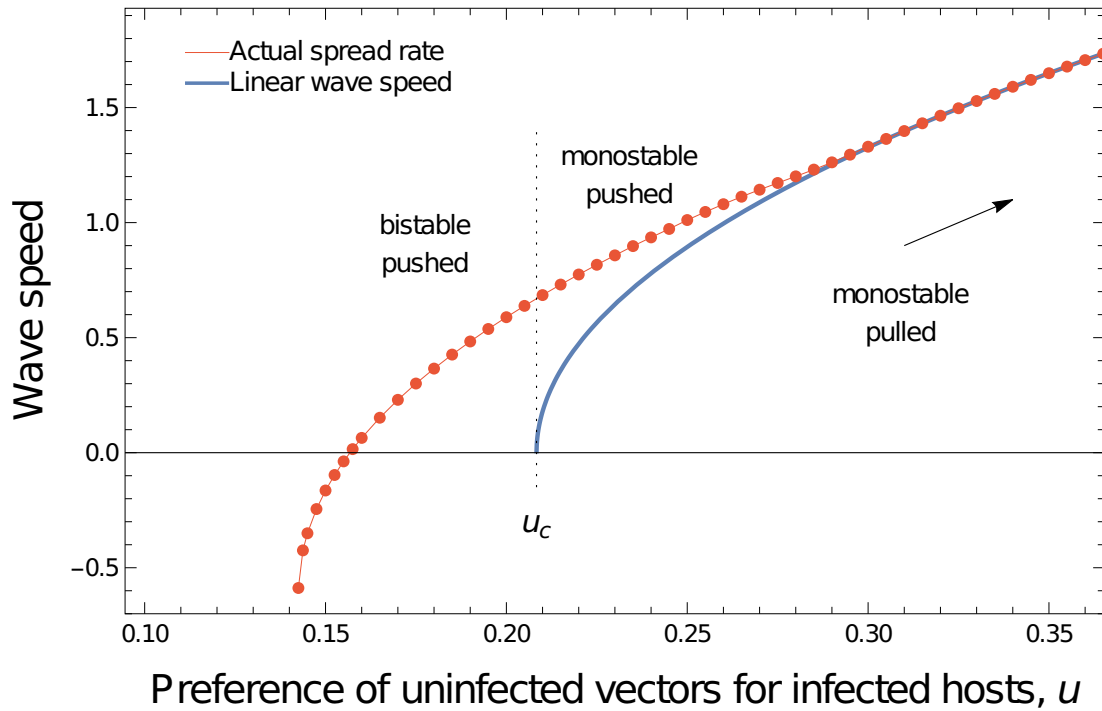


Figure 11: Comparing the linear wave speed, given by equation (23), with the actual (numerically computed) wave speed under the QSSA, i.e., model (21). Here, $\rho = 100$, $\beta = 2.4\rho$, other parameter values as in Fig. 1.

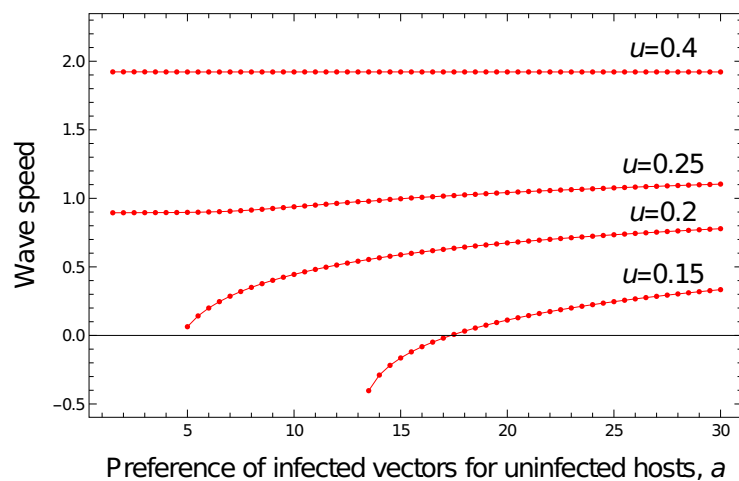


Figure 12: Shows whether and how the actual (numerically computed) spreading speed depends on a . This is for the QSSA, i.e., model (21), but the results are very similar in the original model (3), for $\rho = 1$. Here, $\rho = 100$, $\beta = 2.4\rho$, other parameter values as in Fig. 1.

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