Stability and bifurcation in plant–pathogens interactions

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Abstract

We consider a plant–pathogen interaction model and perform a bifurcation analysis at the threshold where the pathogen–free equilibrium loses its hyperbolicity. We show that a stimulatory–inhibitory host response to infection load may be responsible for the occurrence of multiple steady states via backward bifurcations. We also find sufficient conditions for the global stability of the pathogen–present equilibrium in case of null or linear inhibitory host response. The results are discussed in the framework of the recent literature on the subject.

Keywords: plant–pathogen interaction, mathematical model, bifurcation, global stability

1 Introduction

Invasion, persistence and control are among the most relevant aspects of botanical epidemiology and, in particular, of plant–pathogens interaction modelling [15, 20, 26, 27, 28, 43]. A possible and fruitful approach to such issues comes from the qualitative analysis of suitable dynamical systems describing the evolution of botanical epidemics [16, 18, 33, 41]. Such systems are often adaptations of compartmental epidemic models for human/animal diseases [1, 12]. Noteworthy examples are given in the studies by C.A. Gilligan and co-workers (see e. g. [15, 16, 18, 19, 20, 21]). In particular, paper [21] focuses on criteria for invasion in plant–parasite systems. In [21] the authors present a mathematical model based on classical epidemic systems and adapted to take account of the following two relevant special features of plant–parasite interaction:

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(i) plant parasites may infect from primary infection (externally introduced inoculum), and secondary infection (contact between susceptible and infected host) [3, 20]. This is modelled by an infection term of the form:

$$F(S, I, X) = X f_p(S, X) + I f_s(S, I). \tag{1}$$

Here S, I and X are the state variables of the system and represent the density of susceptible host (S), infected host (I) and inoculum (X), respectively. The functions f_p and f_s represent the primary and secondary infection mechanisms, respectively;
(ii) many plants react to infection load with stimulation or inhibition in the production of susceptible tissue in infected plants [19], so that the host reproduction is described by the function:

$$r(S, I) = b(S) - \alpha(I), \tag{2}$$

where $b(S)$ and $\alpha(I)$ represent the host growth in absence of infection and the host response to infection load, respectively.
Considering (1) and (2), the model is described by the following system of nonlinear ordinary differential equations:

$$\begin{aligned} \dot{S} &= b(S) - X f_p(S, X) - I f_s(S, I) - \alpha(I) \\ \dot{I} &= X f_p(S, X) + I f_s(S, I) - mI - hI \\ \dot{X} &= aI - cX, \end{aligned} \tag{3}$$

where h is the natural death rate, m is the recovery rate, a is the parasite reproduction rate and c is the parasite death rate.
In [21] the following three noteworthy cases of host response to infection load are examined:
Case I : no host response, $\alpha(I) = 0$, for all $I \geq 0$,
Case II : linear inhibitory host response, $\alpha(I) = \gamma_0 I$, where γ_0 is a positive constant,
Case III : nonlinear (stimulatory–inhibitory) host response:

$$\alpha(I) = \frac{(\gamma_1 I - \gamma_2)}{\gamma_3 + I^2} I, \tag{4}$$

where γ_i, $i = 1, 2, 3$, are positive constants. Function (4) models the host production of susceptible tissue below a threshold infection density γ_2/γ_1, and the inhibition of host reproduction above such a threshold [19].

Among the relevant results obtained in [21] there is the following:

(a) Assume a logistic growth in absence of infection, with carrying capacity K, and assume a mass-action transmission; that is, function (1) may be written:

$$F(S, I, X) = \beta_p SX + \beta_s SI, \tag{5}$$

where β_p and β_s are two positive constants, representing the transmission rate of primary and secondary infection, respectively. Introduce the basic reproduction number:

$$R_0 = \frac{K}{m+h}\left(\beta_s + \frac{a}{c}\beta_p\right). \tag{6}$$

Then, cases I and II above produce a classical threshold behaviour: if $R_0 < 1$, the parasite is unable to invade and the parasite–free equilibrium is locally stable. If $R_0 > 1$, the parasite–free equilibrium is unstable and system (3) admits a pathogen–present equilibrium (also said *coexistence* equilibrium, i. e. an equilibrium with all positive components).
On the contrary, case III may produce multiple coexistence equilibria: provided that $m+h < \gamma_2/\gamma_3$ (i.e. the infected hosts survive for a sufficiently long time) and $R_c < R_0 < 1$ then there are two coexistence equilibria; R_c being the critical level (sub–threshold) below which invasion is not possible.
Case III may be completed as follows: if $R_0 > 1$, the parasite–free equilibrium is unstable and there exists a unique coexistence equilibrium, so that the parasite is always able to invade. Finally, if $m + h > \gamma_2/\gamma_3$, then the dynamics is analogous to cases I and II.

Note that the paper [21] focuses only on the invasion; the stability of the coexistence equilibria was considered not relevant and therefore not performed. However, such an analysis is crucial to assess the persistence of infection. In a more recent paper [15], persistence for plant pathogens was analysed for model (3) under mass action transmission and specific forms of host reproduction (2). More specifically, the following result has been established (among others):

(b) Assume that the transmission (1) is given by (5) and that in (2) the host response is absent, $\alpha \equiv 0$, and the growth term is given by a *monomolecular* function, i. e.

$$b(S) = b(K - S),$$

where b and K are positive constants representing the birth rate and the carrying capacity, respectively. Then, the above–mentioned classical threshold behaviour occurs, where now a further result is proved: the inequality $R_0 > 1$ ensures also the local asymptotic stability of the (unique) coexistence equilibrium (in such cases the threshold behaviour is sometimes called 'R_0-dogma' [37]).

Note that in *(b)* the stability analysis is local and obtained by means of linearization procedures. Also observe that system (3), completed with an equation for the

removed host (R) and with a monomolecular birth function, is called M–SIRX model [15]. The equation for R is given by:

$$\dot{R} = mI - hR, \tag{7}$$

and can be studied separately from the other three equations of the system.

The aim of this paper is to give a contribution to the analysis of plant–pathogen models. Precisely, in view of the above results *(a)* and *(b)*, we consider the M–SIRX model with mass–action transmission (5) and perform the following investigations:

(a') A bifurcation analysis for all the three above mentioned cases of host response to infection load. We make use of the bifurcation criterion introduced in [13] (summarized in subsection A.1) and based on the use of the *center manifold theory* [22].

(b') A global stability analysis of the M–SIRX model in the case of null or linear inhibition (cases I e II above). The analysis is performed by means of the generalization of the Poincaré-Bendixson criterion for systems of n ordinary differential equations, with $n \geq 3$, introduced by M. Li and J. Muldowney [29, 30, 31] and sometimes called *geometric approach to global stability* (summarized in subsection A.2).

As for goal *(a')*, we remark that the qualitative analysis performed in [21], based on existence of equilibria and local stability of the parasite–free equilibrium, indicates the possibility of two coexistence equilibria for $R_0 < 1$ and hence the potential occurrence of a *backward* bifurcation [4, 23]. This occurrence implies that a stable coexistence equilibria may also exist when R_0 is less than unity. From the epidemiological point of view, this phenomenon has important implications because it might not be sufficient to reduce R_0 below 1 to eliminate the disease, and the basic reproduction number must be further reduced (below a critical threshold R_c) in order to avoid coexistence states and guarantee the eradication of infection. Therefore, we aim to provide a precise indication of the bifurcation thresholds and derive conditions, expressed in terms of the parameters of the system, ensuring that either forward (that is, the classical R_0-dogma) or backward bifurcation occurs.

As far as goal *(b')* is concerned, we first observe that our analysis will enhance the one performed in [15], where the stability of the equilibria was local. Furthermore, we also consider the case of linear inhibition. We also stress that the global stability of the coexistence equilibrium ensures that the infection will persist independently on the initial size of the epidemics (although we will find only sufficient conditions for the global stability) and this completely solve the problem of infection persistence when the system undergoes a forward bifurcation for $R_0 = 1$ (as we prove for cases I and II) since in such scenario the coexistence equilibrium, when exists, is unique.

The rest of the paper is organised as follows: the M–SIRX model is introduced in Section 2 together with the existence and local stability of the pathogen–free equilibrium. The existence of coexistence equilibria is assessed in Section 3 for three different cases of host response to infection load. The bifurcation analysis for $R_0 = 1$ is presented in Section 4 and the global stability properties of coexistence states are provided in Section 5. Conclusions are given in Section 6.

2 M-SIRX model

We consider the following M–SIRX model:

$$\begin{aligned} \dot{S} &= r - \beta_p\, S\, X - \beta_s\, S\, I - hS - h\beta(I) \\ \dot{I} &= \beta_p\, S\, X + \beta_s\, S\, I - mI - hI \\ \dot{R} &= mI - hR \\ \dot{X} &= aI - cX. \end{aligned} \tag{8}$$

This model can be obtained from system (3) with (2), (5), (7) and monomolecular growth rate in absence of infection given by $b(S) = r - hS$. Variables and parameters are specified in Section 1.
Introducing the scaled dimensionless variables,

$$\hat{S} = \frac{h}{r}S, \quad \hat{I} = \frac{h}{r}I, \quad \hat{R} = \frac{h}{r}R, \quad \hat{X} = \frac{h^2}{ar}X, \quad \hat{t} = ht,$$

the dimensionless parameters,

$$\hat{\beta}_p = \frac{ar}{h^3}\beta_p, \quad \hat{\beta}_s = \frac{r}{h^2}\beta_s, \quad \hat{\mu} = \frac{m+h}{h}, \quad \hat{c} = \frac{c}{h}$$

and observing that the equations of S, I, and X are independent of the variable R, system (8) becomes (omitting the hat):

$$\begin{aligned} \dot{S} &= 1 - \beta_p\, S\, X - \beta_s\, S\, I - S - \alpha(I) \\ \dot{I} &= \beta_p\, S\, X + \beta_s\, S\, I - \mu I \\ \dot{X} &= I - cX, \end{aligned} \tag{9}$$

where

$$\alpha(I) = \frac{h}{r}\beta\left(\frac{r}{h}I\right).$$

2.1 Pathogen–free equilibrium

System (9) admits the pathogen–free equilibrium:

$$E_0 = (1, 0, 0).$$

Let us introduce the basic reproduction number R_0, which interestingly can be written as the sum of two independent components corresponding respectively to the primary and the secondary infection [15]:

$$R_0 = \frac{\beta_p}{\mu c} + \frac{\beta_s}{\mu}.$$

We have the following:

Theorem 2.1 *If $R_0 \leq 1$, then the pathogen–free equilibrium E_0 is locally asymptotically stable. If $R_0 > 1$, then E_0 is unstable.*

Proof. The Jacobian matrix of system (9) is given by:

$$J(S, I, X) = \begin{pmatrix} -\beta_p X - \beta_s I - 1 & -\beta_s S - \alpha'(I) & -\beta_p S \\ \beta_p X + \beta_s I & \beta_s S - \mu & \beta_p S \\ 0 & 1 & -c \end{pmatrix}. \tag{10}$$

The Jacobian evaluated at the pathogen–free equilibrium is:

$$J(1, 0, 0) = \begin{pmatrix} -1 & -\beta_s - \alpha'(0) & -\beta_p \\ 0 & \beta_s - \mu & \beta_p \\ 0 & 1 & -c \end{pmatrix}.$$

One eigenvalue is given by $\lambda_1 = -1$. The others can be derived from the submatrix:

$$J_1 = \begin{pmatrix} \beta_s - \mu & \beta_p \\ 1 & -c \end{pmatrix}.$$

Note that

$$tr J_1 = \beta_s - \mu - c = -\frac{\beta_s}{R_0}(1 - R_0) - c - \frac{\beta_p}{cR_0},$$

and

$$\det J_1 = -c(\beta_s - \mu) - \beta_p = \mu c\,(1 - R_0)\,,$$

so that $R_0 < 1$ implies $\det J_1 > 0$ and $\mathrm{tr} J_1 < 0$, that is E_0 is locally asymptotically stable. On the other hand, $R_0 > 1$ implies that E_0 is a saddle. □

3 Coexistence equilibria

Coexistence equilibria $E = (S^*, I^*, X^*)$ can be obtained from the algebraic system:

$$\begin{aligned} &1 - \beta_p\, S^*\, X^* - \beta_s\, S^*\, I^* - S^* - \alpha(I^*) = 0 \\ &\beta_p\, S^*\, X^* + \beta_s\, S^*\, I^* - \mu I^* = 0 \\ &I^* - cX^* = 0, \end{aligned} \tag{11}$$

From the third and the second equation we get

$$X^* = \frac{I^*}{c}; \qquad \frac{\beta_p}{c} S^* + \beta_s S^* = \mu,$$

that is,

$$S^* = \frac{\mu}{\left(\frac{\beta_p}{c} + \beta_s\right)} = \frac{1}{R_0}.$$

From the first equation in (11) we get

$$1 - \frac{\beta_p}{c\, R_0} I^* - \frac{\beta_s}{R_0} I^* - \frac{1}{R_0} - \alpha(I^*) = 0,$$

from which

$$\left(1 - \frac{1}{R_0}\right) - \frac{1}{R_0}\left(\frac{\beta_p}{c} + \beta_s\right) I^* - \alpha(I^*) = 0,$$

so that I^* must solve the equation

$$\left(1 - \frac{1}{R_0}\right) = \mu I^* + \alpha(I^*). \tag{12}$$

We observe that a necessary condition for equation (12) to admit positive solutions is that $R_0 > 1$. The existence of coexistence states (single or multiple) depends on the form of the functional $\alpha(I)$.

3.1 Case I: no inhibition, $\alpha(I) = 0$, for all $I > 0$

If $\alpha \equiv 0$ we get the unique coexistence equilibrium: $E_{noi} = (S^*, I^*, X^*)$, where

$$S^* = \frac{1}{R_0}; \quad I^* = \frac{R_0 - 1}{\mu R_0}; \quad X^* = \frac{R_0 - 1}{c\mu R_0};$$

3.2 Case II: linear inhibition, $\alpha(I) = \gamma_0 I$

If $\alpha(I) = \gamma_0 I$ we get the unique coexistence equilibrium: $E_{lin} = (S^*, I^*, X^*)$, where:

$$S^* = \frac{1}{R_0}; \quad I^* = \frac{R_0 - 1}{(\mu + \gamma_0) R_0}; \quad X^* = \frac{R_0 - 1}{c(\mu + \gamma_0) R_0}.$$

3.3 Case III: nonlinear inhibition

As in [21], we consider the nonlinear representation (4). We prove the following:

Theorem 3.1 *If $R_0 > 1$, then system (9) admits an unique coexistence equilibrium. If $R_0 \leq 1$, then system (9) may admit two coexistence equilibria provided that $\mu\gamma_3 - \gamma_2 < 0$. In all other cases there are no coexistence equilibria.*

Proof. From equation (12) we obtain:

$$\left(1 - \frac{1}{R_0}\right) = \eta(I^*), \tag{13}$$

where

$$\eta(I^*) = \mu I^* + \frac{\left(\gamma_1 \left(I^*\right)^2 - \gamma_2 I^*\right)}{\gamma_3 + \left(I^*\right)^2}.$$

Observe that: $\eta(0) = 0$, $\eta(+\infty) = +\infty$ and

$$\eta'(I^*) = \frac{\gamma_3 \left(\mu\gamma_3 - \gamma_2\right) + \mu \left(I^*\right)^4 + \left(2\mu\gamma_3 + \gamma_2\right) \left(I^*\right)^2 + 2\gamma_1\gamma_3 I^*}{\left(\gamma_3 + \left(I^*\right)^2\right)^2}.$$

Now,
(a) if $\mu\gamma_3 - \gamma_2 > 0$, then $\eta'(I^*) > 0$, for all $I^* > 0$. As a consequence, equation (13) admits an unique solution for $R_0 > 1$ and no solutions for $R_0 < 1$. That is to say, system (9) admits an unique coexistence equilibrium.
(b) if $\mu\gamma_3 - \gamma_2 < 0$, then observe that equation $\eta'(I^*) = 0$ can be written:

$$\xi(I^*) := \mu \left(I^*\right)^4 + \left(2\mu\gamma_3 + \gamma_2\right) \left(I^*\right)^2 + 2\gamma_1\gamma_3 I^* = \gamma_3 \left(\gamma_2 - \mu\gamma_3\right)$$

which admits an unique solution I^{**} being $\xi(0) = 0$, $\xi(+\infty) = +\infty$, and $\xi'(I^*) > 0$ for all $I^* > 0$. Moreover, $\eta' > 0$ for $I^* > I^{**}$, and $\eta' < 0$ for $I^* < I^{**}$. Hence I^{**} is a minimum for η. This means that $\eta(I^*)$ is negative for all $I^* \in [0, I^{**}]$. Consequently, there exists a range of values for $R_0 < 1$, say $R_0 \in (R_c, 1)$, such that equation (13) admits two coexistence equilibria. □

We remark that Theorem 3.1 indicates the possibility of two coexistence equilibria for $R_0 < 1$ and hence the potential occurrence of a backward bifurcation.

4 Bifurcation analysis

In this section we will make use of Theorem A.1, summarized in the appendix, which has been obtained in [13] and is based on the use of the *center manifold theory* [22].

Theorem A.1 gives a practical tool to detect the occurrence of forward (supercritical) and backward (subcritical) bifurcations when the pathogen–free equilibrium loses its hyperbolicity at $R_0 = 1$. More precisely, two coefficients of the normal form representing the system dynamics on the central manifold must be evaluated (namely, a and b, given by (27) and (28)). Then, their sign will give indications on which kind of bifurcation occurs. More precisely, if $a < 0$ and $b > 0$, then the bifurcation is *forward*, if $a > 0$ and $b > 0$ then the bifurcation is *backward*.

Theorem 4.1 *If $R_0 < 1$, then system (9) exhibits a forward bifurcation at $R_0 = 1$ in both the cases of no inhibition and linear inhibition. When the inhibition α has the nonlinear representation (4), then system (9) exhibits a backward bifurcation at $R_0 = 1$ provided that $\mu\gamma_3 - \gamma_2 < 0$, and a forward bifurcation when the reverse inequality holds.*

Proof. We choose β_p as bifurcation parameter. The critical value (corresponding to $R_0 = 1$) is:

$$\beta_p^* = (\mu - \beta_s)\, c. \tag{14}$$

Note that the eigenvalues of the matrix

$$J(E_0, \beta_p^*) = \begin{pmatrix} -1 & -\beta_s - \alpha'(0) & -(\mu - \beta_s)\, c \\ 0 & \beta_s - \mu & (\mu - \beta_s)\, c \\ 0 & 1 & -c \end{pmatrix}, \tag{15}$$

are given by $\lambda_1 = -1$ and by the solutions of

$$(\beta_s - \mu - \lambda)\,(-c - \lambda) + c(\beta_s - \mu) = 0,$$

that is: $\lambda_2 = \beta_s - \mu - c$ (which is negative when $R_0 = 1$, see (14)) and $\lambda_3 = 0$. Hence, when $R_0 = 1$, the pathogen–free equilibrium E_0 is a nonhyperbolic equilibrium: the assumption (A1) of Theorem A.1 is then verified.
The right eigenvectors $\mathbf{w} = (w_1, w_2, w_3)^T$ of (15) are given by: $J(E_0, \beta_p^*)\mathbf{w} = 0$. We obtain:

$$\begin{aligned} &-w_1 + [-\beta_s - \alpha'(0)]\, w_2 + (\beta_s - \mu) c w_3 = 0 \\ &(\beta_s - \mu) w_2 - (\beta_s - \mu) c w_3 = 0 \\ &w_2 - c w_3 = 0, \end{aligned}$$

so that:

$$w_1 = c\left[-\alpha'(0) - \mu\right] w_3; \qquad w_2 = c w_3.$$

The left eigenvectors $\mathbf{v} = (v_1, v_2, v_3)^T$ of (15) are given by: $J(E_0, \beta_p^*)^T \mathbf{v} = 0$. We obtain:

$$\begin{aligned} & v_1 = 0 \\ & [-\beta_s - \alpha'(0)]\, v_1 + (\beta_s - \mu)\, v_2 + v_3 = 0 \\ & (\beta_s - \mu)\, c v_1 - (\beta_s - \mu)\, c v_2 - c v_3 = 0, \end{aligned}$$

so that

$$v_1 = 0; \qquad v_2 = \frac{v_3}{\mu - \beta_s}.$$

The coefficients a and b given in (27) and (28) may be now explicitly computed. Taking into account of system (9) and considering only the nonzero components of the left eigenvector $\mathbf{v}$, it follows that:

$$a = \quad 2 v_2 w_1 w_2 \frac{\partial^2 f_2}{\partial S \partial I}(E_0, \beta_p^*) + 2 v_2 w_1 w_3 \frac{\partial^2 f_2}{\partial S \partial X}(E_0, \beta_p^*),$$

and

$$b = v_2 w_3 \frac{\partial^2 f_2}{\partial X \partial \beta_p}(E_0, \beta_p^*),$$

where f_2 is the right hand side of second equation of system (9), $f_2 = \beta_p S X + \beta_s S I - \mu I$. It can be checked that:

$$\frac{\partial^2 f_2}{\partial S \partial I}(E_0, \beta_p^*) = \beta_s, \qquad \frac{\partial^2 f_2}{\partial S \partial X}(E_0, \beta_p^*) = \beta_p^*, \qquad \frac{\partial^2 f_2}{\partial X \partial \beta_p}(E_0, \beta_p^*) = 1.$$

It follows:

$$b = \frac{v_3 w_3}{\mu - \beta_s},$$

so that b is positive, and

$$a = 2 v_2 w_1 w_2 \beta_s + 2 v_2 w_1 w_3 \beta_p^* = \frac{2c\,[\alpha'(0) - \mu]\, v_3 w_3^2}{\mu - \beta_s} \left(c\beta_s + \beta_p^* \right),$$

that is, taking into account of (14),

$$a = \frac{2\mu c^2\,[-\alpha'(0) - \mu]\, v_3 w_3^2}{\mu - \beta_s}.$$

We can now distinguish between the three cases in Section 3:
Case I and Case II: if $\alpha \equiv 0$, or $\alpha = \gamma_0 I$, then a is always negative and the transcritical bifurcation is forward.
Case III: if α is given by (4), then

$$\alpha'(I) = \frac{2\gamma_1 \gamma_3 I - \gamma_2 \gamma_3 + \gamma_2 I^2}{\left(\gamma_3 + I^2\right)^2},$$

and hence

$$\alpha'(0) = -\frac{\gamma_2}{\gamma_3},$$

so that if $\mu\gamma_3 - \gamma_2 < 0$, then a is positive and the transcritical bifurcation is backward. If the reverse inequality holds, then the bifurcation is forward. □

In conclusion, a backward bifurcation scenario occurs when the condition $\mu\gamma_3 - \gamma_2 > 0$ is verified. A bifurcation diagram for system (9) with nonlinear inhibition (4) was obtained with parameter values from [21], and is shown in Figure 1. We note from the picture that bistability occurs in the range $[R_c, 1]$, where R_c is the subthreshold $R_c = 0.3776$ (corresponding to $\beta_p^* = 0.1776$). Also two-dimensional phase plots for system (9) in Case III are shown in Figure 2 to better understand how the bifurcation results in Theorem 4.1 affect the dynamics of (9).
A general expression for the subthreshold R_c may be obtained by observing that the equality (13) may be written as a third order equation,

$$x^3 + Ax^2 + Bx + C = 0,$$

where:

$$A = \frac{\gamma_1}{\mu} + \frac{1}{\mu}\left(\frac{1}{R_0} - 1\right), \quad B = \frac{\mu\gamma_3 - \gamma_2}{\mu}, \quad C = \frac{\gamma_3}{\mu}\left(\frac{1}{R_0} - 1\right).$$

It is well known from basic algebra that the nature of its roots may be obtained by the analysis of the discriminant:

$$\Delta = \frac{Q^2}{4} + \frac{P^3}{27},$$

where:

$$P = \frac{-A^2 + 3B}{3}; \quad Q = \frac{2A^3 - 9AB + 27C}{27}.$$

It can be checked that solving $\Delta = 0$ for R_0 will give the desired subthreshold R_c.

5 Global stability

In this section we prove the global stability of the coexistence equilibria in the case of linear inhibition (Case II). The case of no inhibition (Case I) will follow as an obvious consequence. First of all, we have the following result:

Proposition 5.1 *In case of linear or no inhibition, the set:*

$$\Omega = \left\{(S, I, X) \in R_+^3 : \ 0 \le S + I \le 1, \ 0 \le X \le c^{-1}\right\},$$

is positively invariant and absorbing and, as a consequence, the orbits of (9) are bounded, provided that $(S(0), I(0), X(0)) \ge (0, 0, 0)$.

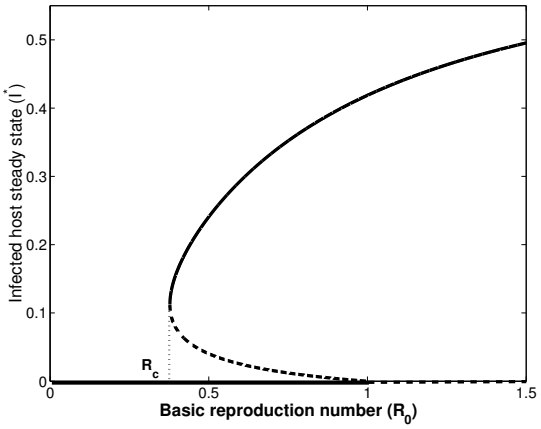

Figure 1: Bifurcation diagram for system (9) with nonlinear inhibition (4), obtained by using the following parameters (from [21]): $\gamma_1 = 0.5$, $\gamma_2 = 0.6$, $\gamma_3 = 0.6$, $\mu = 2$, $\beta_s = 0.4$, $c = 0.5$. The steady states of infected host, I^*, are plotted versus the basic reproduction number R_0, which is the bifurcation parameter. The solid lines (-) denote stability; the dashed lines (- -) denote instability. Condition $\mu\gamma_3 - \gamma_2 > 0$ is verified and backward bifurcation scenario occurs. In particular, bistability occurs in the range $[R_c, 1]$.

Proof. Set $N = S+I$, from (9) it follows $\dot{N} < (1 - N)$. Hence, $\limsup_{t\to+\infty} N(t) \leq 1$. On the other hand, from the inequality: $\dot{X} \leq c\left(c^{-1} - X\right)$, it follows that: $\limsup_{t\to+\infty} X(t) \leq c^{-1}$. □

When $R_0 > 1$, the pathogen–free equilibrium, which is located on the boundary $\partial\Omega$, is unstable and this implies that system (9) is uniformly persistent [17], i.e. there exists a constant $\epsilon_0 > 0$ such that any solution $(S(t), I(t), X(t))$ with $(S(0), I(0), X(0))$ in the interior of Ω, satisfies:

$$\min\{\liminf_{t\to\infty} S(t), \liminf_{t\to\infty} I(t), \liminf_{t\to\infty} X(t)\} > \epsilon_0.$$

The uniform persistence together with the boundedness of Ω is equivalent to the existence of a compact set in the interior of Ω which is absorbing for (9), see [24]. This condition is required by the Li- Muldowney approach, together with a specific Bendixson criterion (inequality (30) in the Appendix) which will be the goal of the next theorem.

Theorem 5.1 *If $R_0 > 1$ and*

$$\mu > 2\beta_s + \gamma_0 + 1, \tag{16}$$

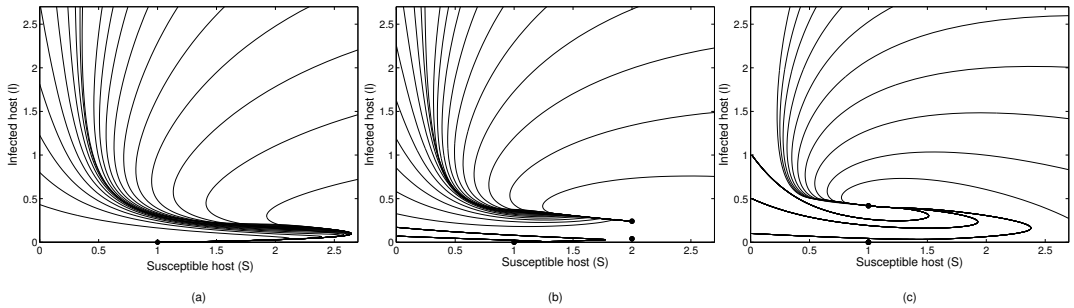

Figure 2: Two-dimensional phase is plotted for system (9) with nonlinear inhibition (4), obtained by using the following parameters (from [21]): $\gamma_1 = 0.5$, $\gamma_2 = 0.6$, $\gamma_3 = 0.6$, $\mu = 2$, $\beta_s = 0.4$, $c = 0.5$. In (a) $R_0 < R_c$, E_0 is the only feasible equilibrium and it is locally asymptotically stable. In (b) $R_0 \in [R_c, 1]$, system (9) has three steady state solutions E_0, E_1 and E_2, the condition $\mu\gamma_3 - \gamma_2 > 0$ is verified and bistability occurs. Finally, in (c) $R_0 > 1$, there exists only one coexistence equilibrium which is stable and E_0 is unstable.

then the equilibrium E_{lin} of system (9) with linear inhibition $\alpha(I) = \gamma_0 I$ exists and is globally asymptotically stable with respect to solutions of (9) initiating in the interior of Ω.

Proof. From the Jacobian matrix $J(S, I, X)$ corresponding to (9), which is given in (10), we can deduce the second additive compound matrix $J^{[2]}(S, I, X)$:

$$J^{[2]} = \begin{pmatrix} -\beta_p X - \beta_s I - 1 + \beta_s S - \mu & \beta_p S & \beta_p S \\ 1 & -\beta_p X - \beta_s I - c - 1 & -\beta_p S - \gamma_0 I \\ 0 & \beta_p X + \beta_s I & \beta_s S - \mu - c \end{pmatrix}.$$

Now we consider the function

$$P = P(S, I, X) = diag\left\{1, \frac{I}{X}, \frac{I}{X}\right\}. \tag{17}$$

It follows:

$$P_f P^{-1} = \text{diag}\left\{0, \frac{\dot{I}}{I} - \frac{\dot{X}}{X}, \frac{\dot{I}}{I} - \frac{\dot{X}}{X}\right\},$$

and

$$PJ^{[2]}P^{-1} = \begin{pmatrix} -\beta_p X - \beta_s I - 1 + \beta_s S - \mu & \beta_p \frac{SX}{I} & \beta_p \frac{SX}{I} \\ \frac{I}{X} & -\beta_p X - \beta_s I - c - 1 & -\beta_p S - \gamma_0 I \\ 0 & \beta_p X + \beta_s I & \beta_s S - \mu - c \end{pmatrix}.$$

so that

$$B = P_f P^{-1} + PJ^{[2]}P^{-1} = \begin{bmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{bmatrix},$$

where
$B_{11} = -\beta_p X - \beta_s I - 1 + \beta_s S - \mu, \quad B_{12} = \left[\begin{array}{cc} \beta_p \frac{SX}{I}, & \beta_p \frac{SX}{I} \end{array} \right], \quad B_{21} = \left[\begin{array}{cc} \frac{I}{X}, & 0 \end{array} \right]^T,$
and

$$B_{22} = \left[\begin{array}{cc} \frac{\dot{I}}{I} - \frac{\dot{X}}{X} - \beta_p X - \beta_s I - c - 1 & -\beta_p S - \gamma_0 I \\ \\ \beta_p X + \beta_s I & \frac{\dot{I}}{I} - \frac{\dot{X}}{X} + \beta_s S - \mu - c \end{array} \right].$$

Consider now the norm in $\mathbf{R}^3$ as

$$|(u, v, w)| = \max\{|u|, |v| + |w|\}, \tag{18}$$

where (u, v, w) denotes the vector in $\mathbf{R}^3$ and denote by $\mathcal{L}$ the Lozinskiĭ measure with respect to this norm. It follows, [34]:

$$\mathcal{L}(B) \leq \sup\{g_1, g_2\} \equiv \sup\{\mathcal{L}_1(B_{11}) + |B_{12}|,\ \mathcal{L}_1(B_{22}) + |B_{21}|\}, \tag{19}$$

where $|B_{21}|$, $|B_{12}|$ are matrix norms with respect to the L^1 vector norm and $\mathcal{L}_1$ denotes the Lozinskiĭ measure with respect to the L^1 norm[3]

$$\mathcal{L}_1(B_{11}) = -\beta_p X - \beta_s I - 1 + \beta_s S - \mu, \tag{20}$$

$$|B_{12}| = \beta_p \frac{SX}{I}, \quad |B_{21}| = \frac{I}{X}, \tag{21}$$

$$\mathcal{L}_1(B_{22}) = \frac{\dot{I}}{I} - \frac{\dot{X}}{X} - c + \max\{-1,\ 2\beta_s S - \mu + \gamma_0 I\}. \tag{22}$$

Taking into account (19) and (20)-(22), the general expressions of g_1 and g_2 for system(9) are thus

$$g_1 = -\beta_p X - \beta_s I - 1 + \beta_s S - \mu + \beta_p \frac{SX}{I}, \tag{23}$$

and

$$g_2 = \frac{\dot{I}}{I} - \frac{\dot{X}}{X} - c + \frac{I}{X} + \max\{-1,\ 2\beta_s S - \mu + \gamma_0 I\}. \tag{24}$$

Observe that system (9) provides the following equalities:

$$\frac{\dot{I}}{I} = \beta_p \frac{SX}{I} + \beta_s S - \mu, \quad \frac{\dot{X}}{X} = \frac{I}{X} - c.$$

[3] i.e., for the generic matrix $A = (a_{ij})$, $|A| = \max_{1 \leq k \leq n} \sum_{j=1}^{n} |a_{jk}|$ and $\mathcal{L}(A) = \max_{1 \leq k \leq n}(a_{kk} + \sum_{j=1(j \neq k)}^{n} |a_{jk}|)$.

Therefore, from (23) one gets

$$g_1 = \frac{\dot{I}}{I} - \beta_p X - \beta_s I - 1 \leq -1,$$

and, from (24)

$$g_2 = \frac{\dot{I}}{I} + \max\left\{-1,\ 2\beta_s S - \mu + \gamma_0\right\}.$$

Taking into account that $S, I \leq 1$, it follows:

$$g_2 \leq \frac{\dot{I}}{I} + \max\left\{-1,\ 2\beta_s - \mu + \gamma_0\right\}.$$

Now because of assumption (16) we obtain

$$g_2 \leq \frac{\dot{I}}{I} - 1.$$

Hence, from (19)

$$\mathcal{L}(B) \leq \sup\left\{g_1, g_2\right\} = \frac{\dot{I}}{I} - 1,$$

and

$$\frac{1}{t}\int_0^t \mathcal{L}(B)ds \leq \frac{1}{t}\log\frac{I(t)}{I(0)} - 1,$$

which implies

$$\limsup_{t\to\infty} \sup_{x_0\in\Gamma} \frac{1}{t}\int_0^t \mathcal{L}(B(x(s,x_0)))ds < 0,$$

so that the Bendixson criterion given in [31] is verified. □

6 Conclusions

We considered the plant–pathogen interaction model (9) which includes a monomolecular birth rate of susceptibles in absence of infection and a mass–action transmission. We studied two noteworthy analytical aspects. The first is the bifurcation at the threshold $R_0 = 1$, where the pathogen–free equilibrium loses its hyperbolicity. At this purpose we used a bifurcation criterion based on the use of the center manifold theory [13]. The second analysis concerned with the global stability of the coexistence equilibria and has been performed with the geometric approach to global stability [31].

Both the used methods are nowadays extensively applied in the analysis of epidemic models for human/animal disease (see for example [2, 7, 8, 9, 10, 36, 38, 40]

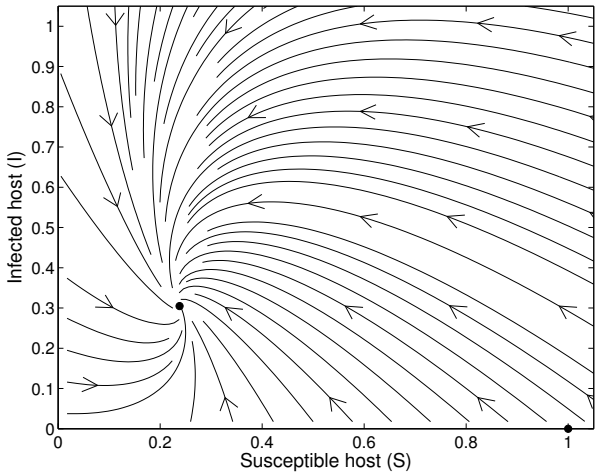

Figure 3: Two-dimensional phase is plotted for system (9) with linear inhibition (Case II), obtained by using the following parameters (from [21]): $\gamma_0 = 0.5$, $\mu = 2$, $\beta_s = 0.4$, $c = 0.1$, $\beta_p = 0.8$. Here $R_0 > 1$ and system (9) has two steady state solutions E_0 and E_1. Condition (16) is not verified but the stability of the coexistence equilibrium E_1 is preserved.

and [5, 6, 11, 14, 25, 32, 39, 42, 44] respectively). However this is the first time, as far as we know, that they are applied in botanical epidemiology.

We found the following main results:

(i) The dynamics of the M–SIRX model without host response to infection load ($\alpha \equiv 0$) follows the classical R_0-dogma: if $R_0 < 1$, then the parasite is unable to invade and the parasite–free equilibrium is stable. If $R_0 > 1$, the parasite–free equilibrium is unstable and there is a stable coexistence equilibrium. In terms of bifurcation analysis, a forward bifurcation occurs at $R_0 = 1$.

(ii) A linear host response (with rate γ_0) does not alter the qualitative behaviour of the system (Theorem 4.1). However the infection level of the coexistence equilibria depends on γ_0 (it decreases as γ_0 increases).

(iii) A nonlinear host response to infection load may be responsible for the occurrence of multiple steady states via backward bifurcations. In particular, we have shown (Theorem 4.1) that a backward bifurcation may occur when the host response is represented by the stimulatory–inhibitory function (4). This happens if the infected host survive for a sufficiently-long time, that is $\mu < \gamma_2/\gamma_3$. A forward bifurcation occurs if the reversed inequality holds. These results enhance the one obtained in [21], where the occurrence of mul-

tiple coexistence equilibria have been detected solely by analysis of existence. As matter of fact, the bifurcation analysis gives also indications on the stability of the coexistence states. We stress that the occurrence of a backward bifurcation is an aspect of relevant interest in the perspective of the disease control. Indeed, in such a case condition $R_0 < 1$ is no longer sufficient for disease eradication and the system could stabilize at an endemic level of infection in the range $[R_c, 1]$, where R_c is a critical threshold corresponding to the saddle–node bifurcation which causes the appearing of the two (respectively stable and unstable) coexistence equilibria.

(iv) In case of forward bifurcation, we enhanced the local result obtained in [15] by providing sufficient conditions for the global stability of the coexistence equilibrium (Theorem 5.1). Under such conditions the infection will persist independently of the initial size of the infection. We recall that forward bifurcation does not allow for multiple coexistence states, so that the global stability of the unique coexistence equilibrium completely solve the problem of infection persistence. Note that the inequalities (16) and $R_0 > 1$ may be combined to give

$$2\beta_s + \gamma_0 + 1 < \mu < \beta_s + \frac{\beta_p}{c}, \tag{25}$$

which in turn implies

$$\beta_s + \gamma_0 + 1 < \frac{\beta_p}{c}.$$

According to this last inequality, the primary infection plays a key role in ensuring that the infection will persist. Indeed it is required a large value of transmission rate of primary infection, β_p, and/or small parasite death rate, c. In such cases, global stability is guaranteed if the infected host mortality μ is in the range given by (25). We finally underline that the bounds on μ may appear to be quite restrictive. As a matter of fact it is possible to find parameter values that do not satisfy condition (25) but still ensure the stability of the coexistence equilibrium, as shown in Figure 3. However, the geometric approach to stability is based on two crucial choices: the entries of the matrix P and the vector norm; in our case (17) and (18). Therefore the sufficient conditions we found here might be improved in principle by choosing in a different way the matrix and the vector norm. This could lead to better conditions in the sense that the restrictions on the parameters may be weakened.

Acknowledgements. The present work has been performed under the auspices of the Italian National Group for Mathematical Physics (GNFM-Indam) and the granted scientific

project GNFM 2012 entitled "Analisi e controllo di processi di diffusione". Thanks are given to the anonymous referees for the careful reading and valuable comments, which have led to the improvement of our original manuscript.

A Appendix

A.1 Bifurcation theorem

Let us consider a general system of ODEs with a parameter ϕ:

$$\dot{x} = f(x, \phi); \qquad f : \mathbf{R}^n \times \mathbf{R} \to \mathbf{R}^n, \qquad f \in C^2(\mathbf{R}^n \times \mathbf{R}). \tag{26}$$

Without loss of generality, we assume that $x = 0$ is an equilibrium for (26).

Theorem A.1 [13] *Assume:*
(A1) $A = D_x f(0,0)$ is the linearization matrix of system (26) around the equilibrium $x = 0$ with ϕ evaluated at 0. Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts;
(A2) Matrix A has a (nonnegative) right eigenvector $\mathbf{w}$ and a left eigenvector $\mathbf{v}$ corresponding to the zero eigenvalue.
Let f_k denotes the k-th component of f and,

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \tag{27}$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0). \tag{28}$$

Then the local dynamics of system (26) around $x = 0$ are totally determined by a and b.
i) $a > 0$, $b > 0$. When $\phi < 0$, with $|\phi| << 1$, $x = 0$ is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 < \phi << 1$, $x = 0$ is unstable and there exists a negative and locally asymptotically stable equilibrium;
ii) $a < 0$, $b < 0$. When $\phi < 0$, with $|\phi| << 1$, $x = 0$ is unstable and there exists a positive stable equilibrium; when $0 < \phi << 1$, $x = 0$ is locally asymptotically stable and there exists a negative unstable equilibrium;
iii) $a > 0$, $b < 0$. When $\phi < 0$, with $|\phi| << 1$, $x = 0$ is unstable and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi << 1$, $x = 0$ is stable and a positive unstable equilibrium appears;
iv) $a < 0$, $b > 0$. When ϕ changes from negative to positive, $x = 0$ changes its stability from stable to unstable. Correspondently, a negative unstable equilibrium becomes positive and locally asymptotically stable.

Remark A.1 *Taking into account of Remark 1 in [13], we observe that if the equilibrium of interest in Theorem A.1 is a non negative equilibrium x_0, then the requirement that w is non negative is not necessary. When some components in w are negative, one can still apply the theorem provided that $w(j) > 0$ whenever $x_0(j) = 0$; instead, if $x_0(j) > 0$, then $w(j)$ need not to be positive. Here $w(j)$ and $x_0(j)$ denote the j-th component of w and x_0 respectively.*

A.2 Geometric approach to global stability

Here we will shortly describe the general method developed in Li and Muldowney, [31]. Consider the autonomous dynamical system:

$$\dot{x} = f(x), \tag{29}$$

where $f : D \to \mathbf{R}^n$, $D \subset \mathbf{R}^n$ open set and simply connected and $f \in C^1(D)$. Let x^* be an equilibrium of (29), i.e. $f(x^*) = 0$. We recall that x^* is said to be *globally stable* in D if it is locally stable and all trajectories in D converge to x^*.
Assume that the following hypotheses hold:
$(H1)$ there exists a compact absorbing set $K \subset D$;
$(H2)$ the equation (29) has a unique equilibrium x^* in D.
The basic idea of this method is that if the equilibrium x^* is (locally) stable, then the global stability is assured provided that $(H1)$-$(H2)$ hold and no non-constant periodic solution of (29) exists. Therefore, sufficient conditions on f capable to preclude the existence of such solutions have to be detected.
Li and Muldowney showed that if $(H1)$-$(H2)$ hold and (29) satisfies a Bendixson criterion that is robust under C^1 local ϵ-perturbations[4] of f at all non-equilibrium non-wandering[5] points for (29), then x^* is globally stable in D provided it is stable. Then, a new Bendixson criterion robust under C^1 local ϵ-perturbation and based on the use of the Lozinskiĭ measure is introduced.
Let $P(x)$ be a $\binom{n}{2} \times \binom{n}{2}$ matrix-valued function that is C^1 on D and consider

$$B = P_f P^{-1} + P J^{[2]} P^{-1},$$

where the matrix P_f is

$$(p_{ij}(x))_f = (\partial p_{ij}(x)/\partial x)^T \cdot f(x) = \nabla p_{ij} \cdot f(x),$$

[4] A function $g \in C^1(D \to \mathbf{R}^n)$ is called a C^1 local ϵ-perturbation of f at $x_0 \in D$ if there exists an open neighbourhood U of x_0 in D such that the support $\mathrm{supp}(f-g) \subset U$ and $|f-g|_{C^1} < \epsilon$, where $|f-g|_{C^1} = \sup\{|f(x) - g(x)| + |f_x(x) - g_x(x)| : x \in D\}$.

[5] A point $x_0 \in D$ is said to be non-wandering for (29) if for any neighbourhood U of x_0 in D and there exists arbitrarily large t such that $U \cap x(t, U) \neq \emptyset$. For example, any equilibrium, alpha limit point, or omega limit point is nonwandering.

and the matrix $J^{[2]}$ is the second additive compound matrix of the Jacobian matrix J, i.e. $J(x) = Df(x)$. Generally speaking, for a $n \times n$ matrix $J = (J_{ij})$, $J^{[2]}$ is a $\binom{n}{2} \times \binom{n}{2}$ matrix (for a survey on compound matrices and their relations to differential equations see [35]) and in the special case $n = 3$, one has

$$J^{[2]} = \begin{bmatrix} J_{11} + J_{22} & J_{23} & -J_{13} \\ J_{32} & J_{11} + J_{33} & J_{12} \\ -J_{31} & J_{21} & J_{22} + J_{33} \end{bmatrix}.$$

Consider the Lozinskiĭ measure $\mathcal{L}$ of B with respect to a vector norm $|\cdot|$ in $\mathbf{R}^N$, $N = \binom{n}{2}$ (see [34])

$$\mathcal{L}(B) = \lim_{h \to 0^+} \frac{|I + hB| - 1}{h}.$$

It is proved in [31] that if (H1) and (H2) hold, condition

$$\limsup_{t \to \infty} \sup_{x_0 \in \Gamma} \frac{1}{t} \int_0^t \mathcal{L}(B(x(s, x_0)))ds < 0, \tag{30}$$

guarantees that there are no orbits giving rise to a simple closed rectifiable curve in D which is invariant for (29), i.e. periodic orbits, homoclinic orbits, heteroclinic cycles. In particular, condition (30) is proved to be a robust Bendixson criterion for (29). Besides, it is remarked that under the assumptions (H1)-(H2), condition (30) also implies the local stability of x^*.
As a consequence, the following theorem holds:

Theorem A.2 [31] *Assume that conditions* (H1)-(H2) *hold. Then* x^* *is globally asymptotically stable in* D *provided that a function* $P(x)$ *and a Lozinskiĭ measure* $\mathcal{L}$ *exist such that condition* (30) *is satisfied.*

References

[1] Anderson R. M., May R. M., Infectious Diseases of Humans. Dynamics and Control, Oxford University Press, Oxford, 1991

[2] Blayneh K. W., Gumel A. B., Lenhart S., Clayton T., Backward bifurcation and optimal control in transmission dynamics of West Nile Virus, Bull. Math. Biol., 72, 1006–1028 (2010)

[3] Brasset P. R., Gilligan C. A., A model for primary and secondary infection in botanical epidemics, Z. Pflanzenk. Pflanzen, 95, 325–360 (1988)

[4] Brauer, F., Backward bifurcations in simple vaccination models, J. Math. Anal. Appl., 298, 418–431 (2004)

[5] Buonomo B., d'Onofrio A., Lacitignola D., Global stability of an SIR epidemic model with information dependent vaccination, Math. Biosci., 216, 9–16 (2008)

[6] Buonomo B., d'Onofrio A., Lacitignola D., The geometric approach to global stability in behavioral epidemiology. In: Modeling the Interplay Between Human Behavior and the Spread of Infectious Diseases (P. Manfredi and A. d'Onofrio eds), 289–308, Springer, 2013

[7] Buonomo B., Lacitignola D., On the dynamics of an SEIR epidemic model with a convex incidence rate, Ric. Mat., 57, 261–281 (2008)

[8] Buonomo B., Lacitignola D., Analysis of a tuberculosis model with a case study in Uganda, J. Biol. Dyn., 4, 571–593 (2010)

[9] Buonomo B., Lacitignola D., On the backward bifurcation of a vaccination model with nonlinear incidence, Nonlinear Anal. Model. Control, 16, 30–46 (2011)

[10] Buonomo B., Lacitignola D., Vargas-De-León C., Qualitative analysis and optimal control of an epidemic model with vaccination and treatment, Submitted.

[11] Buonomo B., Vargas-De-León C., Global stability for an HIV-1 infection model including an eclipse stage of infected cells, J. Math. Anal. Appl., 385, 709–720 (2012)

[12] Capasso V., Mathematical Structures of Epidemic Systems. Lecture Notes in Biomath., 97. Springer-Verlag, Berlin, 1993

[13] Castillo-Chavez C., Song B., Dynamical models of tuberculosis and their applications, Math. Biosci. Eng., 1, 361–404 (2004)

[14] Cunniffe N. J., Gilligan C. A., Invasion, persistence and control in epidemic models for plant pathogens: the effect of host demography, J. R. Soc. Interface, 7, 439–451 (2010)

[15] Cunniffe N. J., Stutt R. O. J. H., van den Bosch F., Gilligan C. A., Time-dependent infectivity and flexible latent and infectious periods in compartmental models of plant disease, Phytopathology, 102, 365–380 (2012)

[16] Freedman H. I., Ruan S., Tang M., Uniform persistence and flows near a closed positively invariant set, J. Diff. Eq., 6, 583–600 (1994)

[17] Gilligan C. A., Sustainable agriculture and plant disease: An epidemiological perspective, Phil. Trans. R. Soc. London B, 363, 741–759 (2008)

[18] Gilligan C. A., Gubbins S., Simons S. A., Analysis and fitting of an SIR model with host response to infection load for a plant disease, Phil. Trans. R. Soc. London B, 352, 353–364 (1997)

[19] Gilligan C. A., Kleczkowski A., Population dynamics of botanical epidemics involving primary and secondary infection, Phil. Trans. R. Soc. London B, 352, 591–608 (1997)

[20] Gubbins S., Gilligan C. A., Kleczkowski A., Population dynamics of plant–parasite interactions: threshold for invasion, Theor. Pop. Biol., 57, 219–233 (2000)

[21] Guckenheimer J., Holmes P., Nonlinear Oscillations, Dynamical Systems and Bifurcations of Vector Fields. Springer-Verlag, Berlin, 1983

[22] Hadeler, K. P., van den Driessche P., Backward bifurcation in epidemic control, Math. Biosci., 146, 15–35 (1997)

[23] Hutson V., Schmitt K., Permanence and the dynamics of biological systems, Math. Biosci., 111, 1–71 (1992)

[24] Iwami S., Takeuchi Y., Liu X., Avian-human influenza epidemic model, Math. Biosci., 207, 125 (2007)

[25] Jeger M. J., Asymptotic behaviour and threshold criteria for model plant disease epidemics, Plant Pathol, 35, 355–361 (1986)

[26] Jeger M. J., van den Bosch F., Threshold criteria for model plant disease epidemics. I. Asymptotic results, Phytopathology, 84, 24–27 (1994)

[27] Jeger M. J., van den Bosch F., Threshold criteria for model plant disease epidemics. II. Persistence and endemicity, Phytopathology, 84, 28–30 (1994)

[28] Li M. Y., Muldowney J. S., On Bendixson's criterion, J. Diff. Equat., 106, 27–39 (1993)

[29] Li M. Y., Muldowney J. S., Global stability for the SEIR model in epidemiology, Math. Biosci., 125, 155–164 (1995)

[30] Li M. Y., Muldowney J. S., A geometric approach to global-stability problems, SIAM J. Math. Anal., 27, 1070–1083 (1996)

[31] Li G., Wang W., Jin Z., Global stability of an SEIR epidemic model with constant immigration, Chaos Solitons Fractals, 30, 1012–1019 (2006)

[32] Madden L. V., Botanical epidemiology: Some key advances and its continuing role in disease management, Eur. J. Plant Pathol., 115, 3–23 (2006)

[33] Martin R. H. Jr., Logarithmic norms and projections applied to linear differential systems, J. Math. Anal. Appl., 45, 432–454 (1974)

[34] Muldowney J. S., Compound matrices and ordinary differential equations, Rocky Mount. J. Math., 20, 857–872 (1990)

[35] Qesmi R., Wu J., Wu J., Heffernan J. M., Influence of backward bifurcation in a model of hepatitis B and C viruses, Math. Biosci., 224, 118–125 (2010)

[36] Reluga T. C., Medlock J., Perelson A. S., Backward bifurcations and multiple equilibria in epidemic models with structured immunity. J. Theor. Biol., 252, 155–165 (2008)

[37] Safan M., Rihan F. A., Mathematical analysis of an SIS model with imperfect vaccination and backward bifurcation, Math. Comput. Simulation, 96, 195-206 (2014)

[38] Sharomi O., Gumel A. B., Curtailing smoking dynamics: A mathematical modeling approach, Appl. Math. Comput., 195, 475-499 (2012)

[39] Sharomi O., Podder C. N., Gumel A. B., Mahmud S. M., Rubinstein E., Modelling the transmission dynamics and control of the novel 2009 swine influenza (H1N1) pandemic, Bull. Math. Biol., 73, 515–548 (2011)

[40] Sherm H., Ngugi H. K., Ojiambo P. S., Trends in theoretical plant epidemiology, Eur. J. Plant. Pathol., 115, 61–73 (2006)

[41] Shu H., Wang L., Role of $CD4^+$T–cell proliferation in HIV infection under antiretroviral therapy, J. Math. Anal. Appl., 394, 529-544 (2012)

[42] Swinton J., Anderson R. M., Model frameworks for plant–pathogen interaction, In "Ecology of Infectious Diseases in Natural Populations" (B. T. Grenfell and A. P. Dobson, Eds), 280–294, Cambridge University Press, Cambridge, UK (1995)

[43] Wang X., Song X., Global stability and periodic solution of a model for HIV infection of $CD4^+$T cells, Appl. Math. Comput., 189, 1331-1340 (2007)