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THE EFFECT OF TIME DELAY IN PLANT–PATHOGEN INTERACTIONS WITH HOST DEMOGRAPHY

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Abstract. Botanical epidemic models are very important tools to study invasion, persistence and control of diseases. It is well known that limitations arise from considering constant infection rates. We replace this hypothesis in the framework of delay differential equations by proposing a delayed epidemic model for plant–pathogen interactions with host demography. Sufficient conditions for the global stability of the pathogen-free equilibrium and the permanence of the system are among the results obtained through qualitative analysis. We also show that the delay can cause stability switches of the coexistence equilibrium. In the undelayed case, we prove that the onset of oscillations may occur through Hopf bifurcation.

1. **Introduction.** In botanical epidemiology, disease invasion, persistence and control represent important aspects in the study of plant–pathogens interaction modelling [15, 21]. Compartmental epidemic models for human and animal diseases [2, 8], whose analytical tractability and ease of simulation is well known, have been adapted and extensively used to model the evolution of botanical epidemics [34].

Models of plant epidemics, either caused by fungal pathogens or by viruses, have received much attention in the last few years [7, 10, 11, 14, 21, 23, 25]. Many aspects of the plant–pathogen interaction have already been considered in the past. For example, Gilligan and coworkers focused on thresholds for invasion, persistence of the disease and effective strategies to control it [10, 11, 14, 15, 16, 17]. Given the importance of the dynamics of the population size, and in particular of the susceptible hosts for the outcome of a successful invasion [1, 2, 35], Cunniffe et al. presented a model including the host demography, in both a linear and nonlinear formulation, to represent how the availability of susceptible tissue changes during the infection [10]. Jeger et al. analysed different models for plant disease epidemics paying particular attention to the plant virus transmission [22, 26], and recently studied the likelihood of successful biocontrol of foliar plant diseases by

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coupling a compartmental SIR model for host–pathogen dynamics with a pathogen-biocontrol agent dynamics [23, 25]. Jeger et al. [22] and van den Bosch et al. [43, 44] incorporated the virus dynamics into a population-based epidemiological model, and explored the evolution of the virus when different types of disease control are introduced. Other epidemiological models do not explicitly consider vectors' or viruses' dynamics, but are based on host plant categories of susceptible, exposed, infectious, and removed [26, 38].

The first model for the spread of plant disease is due to Van der Plank [47], who assumed that the time evolution of the density of infected host tissue (denoted by I) is ruled by the following delay differential equation:

$$\frac{dI(t)}{dt} = \rho\,(I(t-\tau_1) - I(t-\tau_1-\tau_2))\,(1-I(t)). \tag{1}$$

In (1) ρ is a positive constant (corrected infection rate), and τ_1 and τ_2 are constant delays representing the latent and infectious periods, respectively. Despite of many studies that followed Van der Plank's model and that adopted discrete time approximations of it [24, 50], delay differential equations are rarely used in theoretical studies of plant disease [51], perhaps in part because they are difficult to analyse [12, 33]. As a matter of fact, most of the literature for botanical epidemics present models in which either the disease incubation period is considered negligible, which means that once a susceptible individual has been infected it becomes instantly infectious, or the infected susceptible individual goes through a period of latency before becoming infectious. In the former case the dynamics is described by SIR (susceptibles – infectious – removed) compartmental models while in the latter by SEIR-like models, where the class of the exposed is also considered. Only few studies have considered alternative approaches, where these assumptions of constant rates of infection [47] and of exponentially distributed latent and infectious periods [12] are avoided.

Delay differential equations have been widely used to study the spread of human/animal diseases (just to name few contributions, see [5, 20, 27, 31, 32, 36, 40, 41, 42, 52]). Much attention has been given to models with constant delays representing either the infectious period after which the infected individuals are removed, or the constant latency time or both [3, 48, 49]. The global stability properties of equilibria, for SIR [5, 31, 40] and SEIR [3, 29, 38, 49] epidemic models including delay, are among the relevant results that have been obtained through qualitative analysis.

In 2010, Cunniffe and Gilligan [10] presented a particular formulation to model plant epidemics. The aim of their study was to analyse the effect of different host dynamics on invasion, persistence and control of the disease. The model incorporates two sources of infection: a primary infection due to a free-living inoculum (free-living infective stages, fragments of previously infected tissue, spores, other resting structures e. g. sclerotia [17]), denoted by X, and a secondary infection due to the transmission from infected (I) to susceptible hosts (S). Due to the symbols used for denoting the state variables, the model is called "SIRX model" and it has been argued to be appropriate for soil-borne plant diseases caused by fungal pathogens like *Fusarium oxysporum*, *Gaeumannomyces graminis* and *Rhizoctonia solani* [10, 17], or in general for plant parasites such as soil-borne and air-borne fungi, nematodes, and bacteria [17].

The host dynamics plays a crucial role when the generation time of the host is short compared to that of the pathogens. This is the case of diseases in which

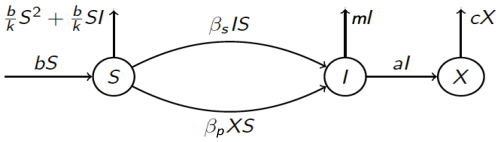

FIGURE 1. Transfer diagram of the plant–pathogen interaction model in terms of Susceptibles, Infectious and free-living inoculum density (System (2) with $\tau = 0$).

the hosts are leaves, roots or other plant organs [14]. In a more recent paper [11], Cunniffe and Gilligan studied the effects of an antagonist population on the interaction between plant host and soil-borne pathogen, where this last interaction is modelled with an SIX model, which is a variant of a class of models analysed by Gubbins et al. [17].

As addressed by Cunniffe *et al.* [12], it is sometimes necessary to incorporate the latent period in plant epidemic models. Sporulating plant pathogens such as *Leveillula taurica, Magnaporthe grisea and Ascochyta blight*, that can have different hosts like pepper, rice, chickpea (just to cite some of them), have latent periods that can vary between 5 and 21 days depending on the climatic conditions [37, 39]. Particularly in the cases of polycyclic pathogens, if the inoculum has a short generation time and an abundant spore production, with massive multiplication in susceptible hosts, it can give rise to rapid and devastating epidemics. It is therefore extremely important to understand how the latent time, which is needed for the production of spores, e. g. in the case of fungal pathogens, influences the dynamics of the epidemics.

In this paper we analyse the interaction between plants and (air-) soil-borne pathogens, and consider the primary infection delayed. More precisely, the transmission of primary infection at time t is described by the term $\beta_p X(t-\tau)S(t)e^{-c\tau}$ where c and β_p are positive constants, $S(t)$ is a measure of the susceptible host at time t, $X(t-\tau)$ represents the inoculum produced at the previous time $t-\tau$ and $e^{-c\tau}$ represents the survival probability of inoculum through the latent time $[t-\tau, t]$. We also consider the transmission from infected to susceptible hosts instantaneous, which means that the time needed by the pest to spread from an infected to a susceptible host is negligible compared to the time of latency.

Our goal is to study the effects of the latent time length, described by the delay, on the dynamics of the (air-) soil-borne plant disease. At this aim, methods of qualitative analysis as stability theory, bifurcation theory and uniform persistence theory for infinite dimensional systems are applied.

The organisation of the paper is the following: in Section 2, the model is presented together with its basic properties as positivity and boundedness of solutions. Section 3 is devoted to equilibria existence and local stability analysis. We also give the delay domain of existence of the coexistence equilibrium. In Section 4 the global stability analysis of the disease-free equilibrium is performed. Furthermore, necessary and sufficient conditions for the permanence of the system are given. In Section 5 the undelayed model is studied and some numerical simulations are shown in Section 6. Conclusions are given in Section 7.

2. **The model and its basic properties.** We consider the following system of delay differential equations with discrete delay:

$$\begin{aligned}
\frac{dS(t)}{dt} &= bS(t)\left(1-\tfrac{S(t)+I(t)}{k}\right) - \left[\beta_p X(t-\tau)e^{-c\tau} + \beta_s I(t)\right] S(t) \\
\frac{dI(t)}{dt} &= \left[\beta_p X(t-\tau)e^{-c\tau} + \beta_s I(t)\right] S(t) - \mu I(t) \qquad (2) \\
\frac{dX(t)}{dt} &= aI(t) - cX(t).
\end{aligned}$$

In this model, the state variables are the fractions in which the host is divided: susceptibles (S); infectious (I); and the free-living inoculum, or vector, density (X). The inoculum requires a time τ to become infectious. We assume $\tau \in \mathbb{R}_+$.

The parameters are all positive constants. b is the birth rate of susceptible hosts, k is the carrying capacity of the susceptible host population in absence of pathogens, β_p (resp. β_s) is the transmission rate of the primary (resp. secondary) infection, the term $e^{-c\tau}$ represents the survival probability of inoculum through the latent time $[t-\tau, t]$, μ is the rate of disease induced mortality for the infected hosts, a **is** the rate of production of inoculum by infected hosts, and c is the rate of decay of the inoculum. The transfer diagram of the instantaneous model (i.e. system (2) with $\tau = 0$) is depicted in Figure 1.

When $\tau = 0$, the model reduces to a particular variant of the model introduced by Cunniffe and Gilligan in [11], and therefore a variant of the class of models introduced by Gubbins et al. in [17].

For biological reasons the initial conditions are non-negative continuous functions

$$S(\theta) = \phi_1(\theta); \quad I(\theta) = \phi_2(\theta); \quad X(\theta) = \phi_3(\theta),$$

where $\phi(\theta) = (\phi_1, \phi_2, \phi_3)^T \in C$, are functions such that $\phi_i(\theta) \geq 0$, $(-\tau \leq \theta \leq 0,\ i = 1, 2, 3)$. C denotes the Banach space $C\left([-\tau, 0], \mathbb{R}^3_+\right)$ of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}^3_+$ with the supremum norm

$$\|\phi\| = \sup_{\theta\in[-\tau,0]} |\phi(\theta)|,$$

where $|\cdot|$ is any norm in $\mathbb{R}^3_+$.

Lemma 2.1. *Any solution of (2) with $\phi(\theta) > 0$, $\theta \in [-\tau, 0]$ remains positive whenever it exists.*

Proof. The first equation of (2) can be rewritten as

$$\frac{dS}{dt} - S\,F(S, X, I) = 0,$$

where

$$F(S, X, I) := b\left(1 - \frac{I}{k} - \frac{1}{b}g(X, I) - \frac{1}{k}S\right), \qquad g(X, I) := \beta_p X(t-\tau)e^{-c\tau} + \beta_s I(t).$$

Therefore,

$$\left(\frac{dS}{dt} - SF(S, X, I)\right) e^{-\int_0^t F(r)dr} = 0 \implies \tfrac{d}{dt}\left(S\, e^{-\int_0^t F(r)dr}\right) = 0.$$

Since $S(0) = \phi_1(\theta) > 0$ then $S(t) > 0$, $t \geq 0$.

To prove the lemma for $I(t)$ let $(S(t), I(t), X(t))$ be a solution of (2) associated with positive initial conditions. First, let us assume that $\bar{t} > 0$ is the first time such that $X(\bar{t}) = 0$, $X(t) < 0$ for $t \in (\bar{t}, \bar{t} + \varepsilon)$, with $\varepsilon > 0$ and sufficiently small ($\varepsilon < \tau$), and for $-\tau \le t < \bar{t}$, $X(t) > 0$. From the third equation of system (2) we get

$$\left.\frac{dX}{dt}\right|_{t=\bar{t}} = aI(\bar{t})$$

Solving the second equation of system (2) we obtain

$$I(\bar{t}) = \left[I_0 + \int_0^{\bar{t}} \beta_p X(t-\tau) S(t) e^{-c\tau} e^{\int_0^t (\mu - \beta_s S(\theta)) d\theta} dt\right] e^{\int_0^{\bar{t}} (\beta_s S(\theta) - \mu) d\theta} > 0$$

Consequently, on the interval $(\bar{t}, \bar{t} + \varepsilon)$, $X(t)$ is negative and increasing, which contradicts $X(\bar{t}) = 0$, so we conclude that $X(t)$ is positive for $t \ge 0$. The positivity of I follows from X and S by observing that

$$\frac{dI}{dt} = \left[I_0 + \int_0^t \beta_p X(\xi - \tau) S(\xi) e^{-c\tau} e^{\int_0^\xi (\mu - \beta_s S(\theta)) d\theta} d\xi\right] e^{\int_0^t (\beta_s S(\theta) - \mu) d\theta} > 0$$

□

Lemma 2.2. *The compact set*

$$\Omega = \left\{(S, I, X) \in \mathbb{R}^3_{+0} : S + I \le LM,\ X \le \frac{a}{c} LM\right\}, \tag{3}$$

where $M = \max\{S(0), k\}$, $L = \frac{b+1}{\mu_m}$, $\mu_m = \min\{1, \mu\}$ *is globally attractive and invariant for the solutions of (2).*

Proof. From the first equation of (2) we obtain

$$\frac{dS}{dt} < bS\left(1 - \frac{S}{k}\right) \implies \limsup_{t \to +\infty} S(t) \le M.$$

By introducing

$$z(t) = S(t) + I(t),$$

we get

$$\frac{dz}{dt} \le (b+1)M - \mu_m z.$$

Therefore, by applying the theorem of differential inequalities [6] we have

$$z(t) < z(0)e^{-\mu_m t} + \frac{(b+1)M}{\mu_m}(1 - e^{-\mu_m t}) \implies \lim_{t \to +\infty} z(t) \le \frac{b+1}{\mu_m} M.$$

With the same technique it is possible to prove the result for X. □

3. **Equilibria and local stability properties.** By introducing the scaled dimensionless variables

$$\hat{S} = \frac{S}{k},\ \ \hat{I} = \frac{I}{k},\ \ \hat{X} = \frac{bX}{ak},\ \ \hat{t} = bt,\ \ \hat{\tau} = b\tau,$$

and the dimensionless parameters

$$\hat{\beta}_p = \frac{\beta_p ak}{b^2},\ \ \hat{\beta}_s = \frac{\beta_s k}{b},\ \ \hat{\mu} = \frac{\mu}{b},\ \ \hat{c} = \frac{c}{b},$$

and by dropping the hats, we get the dimensionless system

$$\begin{aligned}\frac{dS}{dt} &= S(t)\left(1 - S(t) - I(t)\right) - \left[\beta_p X(t-\tau)e^{-c\tau} + \beta_s I(t)\right] S(t)\\ \frac{dI}{dt} &= \left[\beta_p X(t-\tau)e^{-c\tau} + \beta_s I(t)\right] S(t) - \mu I(t)\\ \frac{dX}{dt} &= I(t) - cX(t),\end{aligned} \tag{4}$$

with the following initial conditions

$$(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in C\left([-\tau, 0], \mathbb{R}^3_+\right),\ \phi_i(0) \geq 0,\ i = 1, 2, 3. \tag{5}$$

The basic reproductive number of the pathogen is given by

$$R_0 = \frac{\beta_p e^{-c\tau} + \beta_s c}{\mu c}. \tag{6}$$

We recall that, generally speaking, R_0 is defined as the total number of infections arising from one infected individual that is introduced into a healthy population. In the context of plant diseases, Van de Bosh *et al.* emphasise how particularly indicative is the equivalent definition of generation–to–generation multiplication factor of the pathogen population at low pathogen density [45]. The correspondence between this definition and the mathematical expression (6) is particularly evident when R_0 is written in terms of the dimensional parameters. Furthermore, it is interesting to notice that R_0 can be written as partitioned into two independent components corresponding to the primary and the secondary infection respectively, $R_0 = R_0^p + R_0^s$.

In terms of the dimensionless parameter we can see that $R_0^p = \frac{\beta_p e^{-c\tau}}{\mu c}$, therefore it represents the average number of new infections produced with a rate β_p by the free-living inoculum, which survived the latent period of length τ over the mean lifetime of the inoculum $1/c$, over the mean lifetime of the infection $1/\mu$. Analogously, $R_0^s = \frac{\beta_s}{\mu}$, so that it represents the average number of new infections produced with a rate β_s by the infected host (e. g. through release of spores) over $1/\mu$.

Theorem 3.1. *For all values of the parameters, system (4) admits two steady state solutions*

(i) The trivial *equilibrium* $E_0 = (0, 0, 0)$*;*
(ii) The pathogen–free *equilibrium* $E_1 = (1, 0, 0)$.

Furthermore,

(iii) If $R_0 > 1$*, system (4) admits the* coexistence *equilibrium* $E_2 = (S^*, I^*, X^*)$*, where*

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

Proof. Points *(i)* and *(ii)* can be easily checked. As for *(iii)*, the coexistence equilibrium $E_2 = (S^*, I^*, X^*)$ can be obtained as solution of the algebraic system

$$1 - S^* - I^* - \left(\beta_p X^* e^{-c\tau} + \beta_s I^*\right) = 0 \tag{7}$$

$$\left(\beta_p X^* e^{-c\tau} + \beta_s I^*\right) S^* - \mu I^* = 0 \tag{8}$$

$$I^* - cX^* = 0. \tag{9}$$

□

Remark 1. The delay domain Γ of existence of the coexistence equilibrium E_2 requires $R_0 > 1$. We observe that

(i) If $\mu < \beta_s$ then $\Gamma = \mathbb{R}^+$;
(ii) If $\mu > \beta_s$ then $\Gamma = \{\tau \in \mathbb{R}^+ : \tau < \tau_{\max}\}$, where

$$\tau_{\max} := \frac{1}{c} \ln\left(\frac{\beta_p}{c(\mu - \beta_s)}\right).$$

In order to determine the stability properties of the equilibria we write the characteristic equation corresponding to E_0, E_1 and E_2, respectively. The characteristic equation of system (4) at a generic equilibrium $\bar{E} = (\bar{S}, \bar{I}, \bar{X})$ is given by

$$\begin{bmatrix} 1 - 2\bar{S} - \bar{I} - \beta_s\bar{I} - \beta_p e^{-c\tau}\bar{X} - \lambda & -(\beta_s + 1)\bar{S} & -\beta_p e^{-(\lambda+c)\tau}\bar{S} \\ \beta_s\bar{I} + \beta_p e^{-c\tau}\bar{X} & \beta_s\bar{S} - \mu - \lambda & \beta_p e^{-(\lambda+c)\tau}\bar{S} \\ 0 & 1 & -c - \lambda \end{bmatrix} = 0. \quad (10)$$

At $E_0 = (0, 0, 0)$ equation (10) becomes

$$(1 - \lambda)(-\mu - \lambda)(-c - \lambda) = 0. \quad (11)$$

Since equation (11) has a positive root, we can conclude that E_0 is an unstable equilibrium (saddle point).

At E_i, $i = 1, 2$ equation (7) is satisfied, and the characteristic equation (10) can be written as a third order transcendental equation

$$P_0(\lambda) + P_1(\lambda)e^{-\lambda\tau} = 0, \quad (12)$$

where the polynomials $P_0(\lambda)$ and $P_1(\lambda)$ can be written as follows

$$P_0(\lambda) = (c + \lambda)\left[(\bar{S} + \lambda)(\beta_s\bar{S} - \mu - \lambda) - (\beta_s + 1)\mu\bar{I}\right],$$
$$P_1(\lambda) = \beta_p e^{-c\tau}\left[\bar{S}(\bar{S} + \lambda) - \mu\bar{I}\right].$$

The stability analysis for E_1 follows.

Theorem 3.2. *The pathogen free equilibrium $E_1 = (1, 0, 0)$ of system (4) is*

(i) unstable if $R_0 > 1$;
(ii) linearly neutrally stable if $R_0 = 1$;
(iii) asymptotically stable if $R_0 < 1$.

Proof. At E_1 the characteristic equation (12) becomes

$$(\lambda + 1)\left((\beta_s - \mu - \lambda)(-c - \lambda) - \beta_p e^{-(\lambda+c)\tau}\right) = 0. \quad (13)$$

Equation (13) has a real negative root $\lambda = -1$ and the other roots are solutions of

$$\Delta(\lambda) := (\beta_s - \mu - \lambda)(-c - \lambda) - \beta_p e^{-(\lambda+c)\tau} = 0. \quad (14)$$

(i) Assuming $R_0 > 1$, then

$$\Delta(0) = -c(\beta_s - \mu) - \beta_p e^{-c\tau} = \mu c(1 - R_0).$$

Therefore, $\Delta(0) < 0$. Since $\lim_{\lambda\to+\infty} \Delta(\lambda) = +\infty$, there exists at least one positive root of (14).

(ii) If $R_0 = 1$, then $\lambda = 0$ is a simple characteristic root of (14). Let $\lambda = \alpha + i\omega$ any of the other solutions, then (14) turns into:

$$(\alpha + i\omega)^2 + (\alpha + i\omega)(c + \mu - \beta_s) + c(\mu - \beta_s) = e^{-(\alpha+i\omega)\tau}\beta_p e^{-c\tau}, \quad (15)$$

By using Euler's formula and by separating real and imaginary parts we can write

$$\begin{aligned} -\omega^2 + \alpha^2 + \alpha(c+\mu-\beta_s) + c(\mu-\beta_s) &= e^{-(\alpha+c)\tau}\beta_p \cos\omega\tau, \\ 2\alpha\omega + \omega(c+\mu-\beta_s) &= -e^{-(\alpha+c)\tau}\beta_p \sin\omega\tau. \end{aligned} \tag{16}$$

Observing that $R_0 = 1$ implies $c(\mu-\beta_s) = \beta_p e^{-c\tau}$. Moreover, if there exist roots satisfying both equations (16), then they also satisfy the equation obtained by squaring and adding them member to member, we obtain

$$((c+\alpha)^2+\omega^2)((\alpha+\mu-\beta_s)^2+\omega^2) = c^2 e^{-2\alpha\tau}(\mu-\beta_s)^2. \tag{17}$$

For equation (17) to be verified we must have $\alpha \le 0$. Therefore E_1 is linearly neutrally stable.

(iii) Let be $R_0 < 1$ (which implies $(\mu-\beta_s > 0)$. Observe that all roots of (14) have negative real part for $\tau = 0$. Our goal is to prove that for any values of the parameters the characteristics roots cannot reach the imaginary axis. This means that for any values of the parameters and for all delays τ it happens that $Re(\lambda) < 0$. Let $\lambda = i\omega$, $\omega \in \mathbb{R}_+$ be a root of (14). Then, it must satisfy

$$\omega^4 + \omega^2(c^2 + (\mu-\beta_s)^2) + c^2(\mu-\beta_s)^2 - \beta_p^2 e^{-2c\tau} = 0.$$

Since $R_0 < 1$ then there are no positive real roots ω. Therefore, all roots of (14) must have negative real part and E_1 is asymptotically stable.

□

Now let us focus on the coexistence equilibrium E_2. As we have seen, E_2 exists when $R_0 > 1$. According to Remark 1 the stability properties of E_2 have to be investigated for each τ if $\mu < \beta_s$ and for $\tau < \tau_{\max}$ if $\mu > \beta_s$. At E_2 the characteristic equation (12) is

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 + (b_0 + b_1\lambda)e^{-\lambda\tau} = 0, \tag{18}$$

where

$$a_2 = c + \mu + S^* - \beta_s S^*; \quad a_1 = cS^* + c\mu + S^*\mu + I^*(\beta_s+1)\mu - cS^*\beta_s - S^{*2}\beta_s$$
$$a_0 = c\mu S^* + c(\beta_s+1)\mu I^* - c\beta_s S^{*2}; \quad b_1 = -S^*\beta_p e^{-c\tau}; \quad b_0 = (\mu I^* - S^{*2})\beta_p e^{-c\tau}.$$

The characteristic equation (18) has delay dependent coefficients and it is quite involved. Therefore, it is difficult to obtain information on the nature of the eigenvalues and the occurrence of stability switches. However, in the next section we will show that system (4) is permanent when $R_0 > 1$. That is, every solution of (4) will eventually enter and remain in a compact region in the interior of the feasible set (3). This means that for $R_0 > 1$ the disease will maintain itself in the environment. Furthermore, in Section 5 we will show that some stability properties for E_2 can be inferred from the analogous properties of the coexistence equilibrium for $\tau = 0$.

4. **Permanence and global stability.**

First, we remark that using the same technique as in Lemma (2.2) it is possible to prove the following:

Lemma 4.1. *All feasible solutions of system (4) are bounded and enter the region*

$$\Omega_\epsilon = \left\{ (S,I,X) \in \mathbb{R}^3_{+0} : S + I \le \frac{2M}{\mu_m} + \epsilon,\ X \le \frac{2M}{c\mu_m} + \epsilon \text{ for all } \epsilon > 0 \right\}$$

where $\mu_m = \min\{\mu, 1\}$ *and* $\limsup_{t\to\infty} S(t) \le M$ *with* $M = \max\{1, S(0)\}$.

Theorem 4.2. *If* $R_0 \le 1$, *then the solutions of (4) initiating in the interior of* Ω_ϵ *for any positive* ϵ, *satisfy* $(S(t), I(t), X(t)) \to (1,0,0)$ *as* $t \to \infty$.

Proof. Let us consider the first case $R_0 < 1$. We introduce the following non-negative Lyapunov functional [3, 52]:

$$V(t) = X(t) + \omega_1 I(t) + \omega_2 \int_{t-\tau}^{t} X(u)du + \omega_3 \left(S(t) - 1\right)^2,$$

with $\omega_i > 0$, $(i = 1, 2, 3)$. Along the solutions of (4) we obtain (from now on the upper dot denotes the time derivative)

$$\begin{aligned} \dot{V}|_{(4)} = \; & -\Big\{2\omega_3 \left(1 - S(t)\right)^2 S(t) + (c - \omega_2)X(t) \\ & + \left[2\omega_3\beta_p e^{-c\tau} S(t)^2 - \beta_p e^{-c\tau}(\omega_1 + 2\omega_3)S(t) + \omega_2\right] X(t-\tau) \\ & + \left[2\omega_3(\beta_s + 1)S(t)^2 - (2\omega_3(\beta_s + 1) + \omega_1\beta_s)S(t) + \mu\omega_1 - 1\right] I(t)\Big\}. \end{aligned}$$

We choose ω_i $(i = 1, 2, 3)$ satisfying the following conditions (see Remark 2):

$$(\omega_1 + 2\omega_3)^2 \beta_p e^{-c\tau} < 8\omega_2\omega_3, \tag{19}$$

$$\left[(\omega_1 + 2\omega_3)\beta_s + 2\omega_3\right]^2 < 8\omega_3(\beta_s + 1)(\mu\omega_1 - 1). \tag{20}$$

In this way we have: $\dot{V}(t)|_{(4)} \leq 0$. The maximum invariant set in $\{(S, I, X) \in \Omega_\epsilon : \dot{V}(t)|_{(4)} = 0\}$ is E_1, since E_0 is always an unstable saddle point. Therefore, by applying the Lyapunov–LaSalle type theorem [28], we get $(S(t), I(t), X(t)) \to (1, 0, 0)$ as $t \to \infty$ in Ω_ϵ when $R_0 < 1$.

Let us now consider the case $R_0 = 1$ or, equivalently, $\beta_p e^{-\tau} + \beta_s c = \mu c$. From the first equation of (4) we have $\dot{S} \leq S(t)(1 - S(t))$. Therefore $S(t)$ will be always decreasing when above 1, and if there exist a time such that $S(t)$ goes below 1 then it must stay below 1 for all following times. Two cases are possible:
(a) $S(t) \to 1$ from above as $t \to \infty$;
(b) there exists a T such that $S(t) < 1$ for all $t > T$.

In case *(a)* we only need to show that $X(t) \to 0$. Integrate the first equation of system (4) from τ to $t + \tau$. We have,

$$\begin{aligned} S(t+\tau) - S(\tau) = \; & \int_\tau^{t+\tau} (1 - S(u))S(u)du - \Big(\int_\tau^{t+\tau} (1 + \beta_s)I(u)S(u)du \\ & + \int_\tau^{t+\tau} \beta_p e^{-c\tau} X(u-\tau)S(u)du\Big) \\ & \leq \int_\tau^{t+\tau} (1 - S(u))S(u)du - \Big(\int_\tau^{t+\tau} (1 + \beta_s)I(u)du \\ & + \int_\tau^{t+\tau} \beta_p e^{-c\tau} X(u-\tau)du\Big) \\ & \leq -\int_0^t \beta_p e^{-c\tau} X(u)du. \end{aligned}$$

Hence,

$$\int_0^t \beta_p e^{-c\tau} X(u)du \leq -S(t+\tau) + S(\tau) \leq S(0).$$

Letting $t \to \infty$, we can conclude that $X(t) \in L^1(0, \infty)$ and therefore, $X(t) \to 0$. From the second equation of (4) and by observing that $\beta_s - \mu < 0$ we get that $\lim_{t\to\infty} X(t) = 0$ implies $\lim_{t\to\infty} I(t) = 0$.

In case *(b)* we can consider the functional

$$V(t) = (\mu - \beta_s)X(t) + I(t) + \beta_p e^{-c\tau} \int_{t-\tau}^{t} X(u)du.$$

Then, for all $t > T + \tau$, we obtain,

$$\dot{V}|_{(4)} = -\beta_p e^{-c\tau}(1 - S(t))X(t-\tau) - \beta_s(1 - S(t))I(t) < 0.$$

Applying the Lyapunov–LaSalle theorem we obtain that $(I(t), X(t)) \to 0$ as $t \to \infty$ and therefore $\lim_{t\to\infty}(S(t), I(t), X(t)) = (1, 0, 0)$. □

Remark 2. As an example of $\omega_1, \omega_2, \omega_3$ satisfying (19) and (20), take $\omega_2 = c$. Then, (20) is equivalent to $(1+\beta_s)^2\omega_3^2 + 2(1+\beta_s)(\beta_s\omega_1 + 2 - 2\mu\omega_1)\omega_3 + \beta_s^2\omega_1^2 < 0$ which is true for some $\omega_3 > 0$ if $\beta_s\omega_1 + 2 - 2\mu\omega_1 < 0$ and $(\beta_s\omega_1 + 2 - 2\mu\omega_1)^2 - \beta_s^2\omega_1^2 > 0$. The latter two conditions are satisfied if $\omega_1 > \frac{1}{\mu-\beta_s}$. By observing that $R_0 < 1$ is equivalent to $\beta_p e^{-\tau} < c(\mu - \beta_s)$ and with the previous choices of $\omega_i, (i = 1, 2)$ we obtain that also (19) is satisfied for any ω_3.

From Theorems 3.2 and 4.2 we obtain the following corollary:

Corollary 1. *The pathogen free equilibrium $E_1 = (1, 0, 0)$ of system (4) is globally asymptotically stable in Ω_ϵ for any $\epsilon > 0$ if $R_0 < 1$.*

In the following we will prove that the instability of E_1 implies that system (4) is permanent. Some definitions and a lemma are necessary to establish the main result.

Definition 4.3. System (4) is said to be permanent if there exists a compact region, say $U \in \mathbb{R}^3_+$, such that every solution of (4) with positive initial condition will eventually enter and remain in the region U.

We begin by observing that for a dissipative system uniform persistence is equivalent to permanence. To prove the permanence of system (4) we will use the uniform persistence theory for infinite dimensional systems [19]. Let U be a complete metric space. Suppose that U^0 is an open set dense in U, and U_0 is a set such that $U^0 \cup U_0 = U$, $U^0 \cap U_0 = \emptyset$. Assume that $T(U)$ is a C^0 semigroup on U satisfying

$$T(t) : U^0 \to U^0, \qquad T(t) : U_0 \to U_0. \tag{21}$$

Let $T_b(t) = T(t)|_{U_0}$ and let A_b be the global attractor for $T_b(t)$.

Definition 4.4. The semigroup $T(t)$ is said to be point dissipative in U if there is a bounded non empty set B in U such that, for any $u \in U$, there is a $t_0 = t_0(u, B)$ such that $T(t)u \in B$ for $t \geq t_0$.

Definition 4.5. The semigroup $T(t)$ is said to be uniformly persistent if there is an $\eta > 0$ such that for any $u \in U^0$, $\liminf_{t\to\infty} d(T(t)u, U_0) \geq \eta$.

Lemma 4.6. *Suppose that $T(t)$ satisfies (21) and we have the following:*

- *(i) there is a $t_0 \geq 0$ such that $T(t)$ is compact for $t > t_0$;*
- *(ii) $T(t)$ is a point dissipative in U;*
- *(iii) $\tilde{A}_b = \bigcup_{u\in A_b} \omega(u)$ is isolated and has an acyclic covering $\hat{M}$, where*

$$\hat{M} = \{M_1, M_2, \ldots, M_n\}.$$

Then $T(t)$ is uniformly persistent if and only if for each $M_i \in \hat{M}$

$$W^s(M_i) \cap U^0 = \emptyset \quad \text{for} \quad i = 1, 2, \ldots, n$$

The proof of this lemma can be found in [19]. A consequence of this result is that U_0 is a uniform repeller with respect to U^0, i.e., there is an $\varepsilon > 0$ such that for any $u \in U^0$, $\liminf_{t\to+\infty} d(T(t)u, U_0) \geq \varepsilon$, where d is the distance of $T(t)u$ from U_0.

We are now able to prove the following result:

Theorem 4.7. *System (4) is permanent provided that $\mu < \beta_s$.*

Proof. We begin by showing that the boundary planes of $\mathbb{R}^3_+$ repel the positive solutions of system (4) uniformly. Let $U = C\left([-\tau, 0], \mathbb{R}^3_+\right)$, $U^0 = \text{int}U$ and $U_0 = \partial U$. We will now verify that the conditions of Lemma 4.6 are satisfied. By the definitions of U^0 and system (4) it is easy to see that U^0 is positively invariant. Moreover, conditions (i) and (ii) of Lemma 4.6 are trivially satisfied. We therefore only need to verify (iii) and that $W^s(M_i) \cap U^0 = \emptyset$. Since $\mu < \beta_s$ implies $R_0 > 1$ then (4) admits two constant solutions E_0 and E_1 on U_0 corresponding to $S(t) = I(t) = X(t) = 0$ and $S(t) = 1$, $I(t) = X(t) = 0$. If $(S(t), I(t), X(t))$ is a solution of (4) with initial conditions in $C_1 = \{(\phi_1, \phi_2, \phi_3) \in C([-\tau, 0], \mathbb{R}^3_+) : \phi_1 = 0, \theta \in [-\tau, 0]\}$ then we have $\dot{S}(t)|_{((\phi_1,\phi_2,\phi_3)\in C_1)} = 0$, from which we obtain $S(t)|_{((\phi_1,\phi_2,\phi_3)\in C_1)} = 0$ and respectively from the second and the third equation of (4) we get $\dot{I}(t) \leq -\mu I(t)$ and $\dot{X}(t) \leq -cX(t)$. Hence $I(t) \to 0$ and $X(t) \to 0$ as $t \to \infty$, that is all solutions starting in C_1 approach E_0, i.e. $C_1 = W^s(E_0)$. It is also easy to prove that all the solutions of (4) starting in $C_2 = \{(\phi_1, \phi_2, \phi_3) \in C([-\tau, 0], \mathbb{R}^3_+) : \phi_2 = 0, \phi_3 = 0, \phi_1 \neq 0, \theta \in [-\tau, 0]\}$ approach E_1, that is $C_2 = W^s(E_1)$. Since if $R_0 > 1$ then E_0 and E_1 are unstable, $\tilde{A}_b$ is just the union of the constant solutions, and we can take the M_i to be these constant solutions, which are also isolated invariant sets. Therefore $\{E_0, E_1\}$ is our isolated and acyclic covering of $\tilde{A}_b$, satisfying condition (iii) of Lemma 4.6.

We now want to show that $W^s(M_i) \cap U^0 = \emptyset$ for $i = 1, 2, \ldots, n$. We will only prove that $W^s(E_1) \cap U^0 = \emptyset$ since the proof of $W^s(E_0) \cap U^0 = \emptyset$ is simple. Let us assume the contrary, that is $W^s(E_1) \cap U^0 \neq \emptyset$ then there exists a positive solution $(S(t), I(t), X(t))$ of system (4) such that $\lim_{t\to\infty}(S(t), I(t), X(t)) = (1, 0, 0)$. Since $\mu < \beta_s$, then $\mu < \beta_s(1 - \epsilon)$ for a sufficiently small ε, and there exists a positive constant $T = T(\varepsilon)$ such that

$$S(t) > 1 - \varepsilon > 0, \quad 0 < I(t) < \varepsilon, \quad 0 < X(t) < \varepsilon, \quad \forall t \geq T,$$

from the second equation we obtain

$$\begin{aligned} \dot{I}(t) &= (\beta_p X(t-\tau)e^{-c\tau} + \beta_s I(t))\, S(t) - \mu I(t) \geq \\ &\geq (\beta_s(1-\varepsilon) - \mu) I(t), \quad \forall t \geq T + \tau. \end{aligned}$$

According to the comparison principle, if $\mu < \beta_s$ then $\lim_{t\to\infty} I(t) = +\infty$, contradicting $I(t) < \varepsilon$. Therefore we have $W^s(E_1) \cap U^0 = \emptyset$. Finally, from Lemma 4.6 we are able to conclude that U_0 repels the positive solutions of (4) uniformly, and therefore that system (4) is permanent. □

5. **Undelayed system.** This section is devoted to the study of the classical approach to plant–pathogen interaction. That is, we consider the case of instantaneous transmission of primary infection. We perform a qualitative analysis of model (4) without delay, i.e., we set $\tau = 0$. This analysis has interest in itself and will also allow to get some information on the stability of the coexistence equilibrium in the case with delay.

As shown in the previous sections, the delayed model undergoes **a** bifurcation when the basic reproduction number crosses unity. Therefore, it is useful to investigate the stability properties of system (4), without delay, near the criticality (that is at E_1 and $R_0 = 1$). To this aim, we use the bifurcation theory approach developed in [9, 13, 46], which is based on the centre manifold theory [18]. In particular, we are interested to assess if there is a stable coexistence equilibrium bifurcating from

E_1, and E_1 changes from being stable to unstable. This behaviour is called *forward* bifurcation [9, 13, 46].

In short, it can be shown that the normal form representing the dynamics of the system on the central manifold is given by

$$\dot{u} = au^2 + b\mu u,$$

where,

$$a = \frac{\mathbf{v}}{2} \cdot D_{\mathbf{xx}}\mathbf{f}(\mathbf{x}_0, 0)\mathbf{w}^2 \equiv \frac{1}{2} \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\mathbf{x}_0, 0), \tag{22}$$

and

$$b = \mathbf{v} \cdot D_{\mathbf{x}\varphi}\mathbf{f}(\mathbf{x}_0, 0)\mathbf{w} \equiv \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \varphi}(\mathbf{x}_0, 0). \tag{23}$$

In the (22) and (23) φ denotes a bifurcation parameter to be chosen, f_k's denote the right hand side of system (4) with $\tau = 0$, $\mathbf{x}$ denotes the state vector, $\mathbf{x}_0$ the disease–free equilibrium E_1 and $\mathbf{v}$ and $\mathbf{w}$ denote, respectively, the left and right eigenvectors corresponding to the null eigenvalue of the Jacobian matrix of system (4) with $\tau = 0$, evaluated at criticality.

We have the following result:

Theorem 5.1. *When $\tau = 0$, system (4) exhibits a forward bifurcation at E_1 and $R_0 = 1$.*

Proof. We assume the parameter values such that $R_0 = 1$ that is $\beta_p + \beta_s c = \mu c$. By choosing β_p as bifurcation parameter, the critical value is:

$$\beta_p^* = (\mu - \beta_s)\, c. \tag{24}$$

Note that the eigenvalues of the matrix,

$$J(E_1, \beta_p^*) = \begin{pmatrix} -1 & -\beta_s - 1 & -(\mu - \beta_s)\, c \\ 0 & \beta_s - \mu & (\mu - \beta_s)\, c \\ 0 & 1 & -c \end{pmatrix}, \tag{25}$$

are given by $\lambda_1 = -1$ and 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 (24)) and $\lambda_3 = 0$. Hence, when $R_0 = 1$, the disease–free equilibrium E_1 is a non-hyperbolic equilibrium.

The right eigenvectors $\mathbf{w} = (w_1, w_2, w_3)^T$ of (25) are given by: $J(E_1, \beta_p^*)\mathbf{w} = 0$. We obtain:

$$\begin{aligned} &-w_1 + [-\beta_s - 1]\, w_2 + (\beta_s - \mu) c w_3 = 0 \\ &(\beta_s - \mu) w_2 - (\beta_s - \mu) c w_3 = 0 \\ &w_2 - c w_3 = 0, \end{aligned}$$

so that:

$$w_1 = c\,[-1 - \mu]\, w_3; \qquad w_2 = c w_3.$$

The left eigenvectors $\mathbf{v} = (v_1, v_2, v_3)^T$ of (25) are given by: $J(E_1, \beta_p^*)^T\mathbf{v} = 0$. We obtain:

$$\begin{aligned} &v_1 = 0 \\ &[-\beta_s - 1]\, v_1 + (\beta_s - \mu)\, v_2 + v_3 = 0 \\ &(\beta_s - \mu)\, c v_1 - (\beta_s - \mu)\, c v_2 - c v_3 = 0, \end{aligned}$$

so that

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

The coefficients a and b given in (22) and (23) may be now explicitly computed. Taking into account system (4) with $\tau = 0$ and considering only the nonzero components of the left eigenvector $\mathbf{v}$, it follows that:

$$a = \quad 2v_2w_1w_2\frac{\partial^2 f_2}{\partial S\partial I}(E_1, \beta_p^*) + 2v_2w_1w_3\frac{\partial^2 f_2}{\partial S\partial X}(E_1, \beta_p^*),$$

and

$$b = v_2w_3\frac{\partial^2 f_2}{\partial X\partial \beta_p}(E_1, \beta_p^*),$$

where f_2 is the right hand side of second equation of system (4) with $\tau = 0$, $f_2 = \beta_p SX + \beta_s SI - \mu I$. It can be checked that:

$$\frac{\partial^2 f_2}{\partial S\partial I}(E_1, \beta_p^*) = \beta_s, \qquad \frac{\partial^2 f_2}{\partial S\partial X}(E_1, \beta_p^*) = \beta_p^*, \qquad \frac{\partial^2 f_2}{\partial X\partial \beta_p}(E_1, \beta_p^*) = 1.$$

It follows:

$$b = \frac{v_3w_3}{\mu - \beta_s},$$

so that b is positive, and:

$$a = 2v_2w_1w_2\beta_s + 2v_2w_1w_3\beta_p^* = \frac{2c\,[1-\mu]\,v_3w_3^2}{\mu - \beta_s}\left(c\beta_s + \beta_p^*\right),$$

which can be written, taking into account of (24),

$$a = \frac{2\mu c^2\,[-1-\mu]\,v_3w_3^2}{\mu - \beta_s}.$$

Therefore, a is negative and the bifurcation is forward. □

The theorem above states that for values of R_0 greater than 1 but close to 1, the model admits a unique infected equilibrium, which is locally asymptotically stable. In the next theorem we find a sufficient condition ensuring that this property holds true for R_0 greater than 1.

Theorem 5.2. *If $R_0 > 1$ and $\tau = 0$ then the infected equilibrium equilibrium $E_2 = (S^*, I^*, X^*)$ is locally asymptotically stable if $\mu \geq \beta_p$.*

Proof. When $\tau = 0$ equation (18) becomes

$$\lambda^3 + a_2\lambda^2 + (a_1 + b_1)\lambda + a_0 + b_0 = 0. \tag{26}$$

Since

$$a_2 = c + \frac{1}{R_0} + \frac{\beta_p}{cR_0} > 0 \qquad \text{and} \qquad a_0 + b_0 = \mu I^*(\beta_p + c\beta_s + c) > 0,$$

from the Routh–Hurwitz criterion we know that all roots of (26) have negative real parts if

$$a_2(a_1 + b_1) - (a_0 + b_0) > 0,$$

that is

$$F(R_0) := (c^4 + cI^*(\beta_p + c\beta_s)(\mu - \beta_p) + cI^*\mu(\beta_p\beta_s + c))R_0^2 + c^2(2\beta_p + c)R_0 + \beta_p(\beta_p + c) > 0,$$

which is verified if the hypothesis holds. □

As corollary of Theorem 5.2, it follows that for $\mu < \beta_p$ the onset of oscillations may take place. In particular from a direct application of the criterion in [30], we have the following result:

Corollary 2. *If* $\tau = 0$, $R_0 > 1$ *and* $c^4 + cI^*(\beta_p + c\beta_s)(\mu - \beta_p) + cI^*\mu(\beta_p\beta_s + c) < 0$ *then there exist a* R_c *such that if* $\frac{dF(R_0)}{dR_0}\Big|_{R_0=R_c} \neq 0$ *at* $R_0 = R_c$ *system (4) undergoes* **a** *Hopf bifurcation.*

6. **Numerical results and further developments.** We can observe that once the stability properties of the equilibrium E_2 are known for the case without delay (τ=0), stability switches may happen for certain values of the delay τ when the characteristic roots reach the imaginary axis. Let $\lambda = i\omega$, $\omega > 0$, be a root of (18). Then, by separating real and imaginary parts and using Euler's formula, we get

$$\begin{array}{ll} -a_2\omega^2 + a_0 & = -b_0\cos(\omega\tau) - b_1\omega\sin(\omega\tau), \\ -\omega^3 + a_1\omega & = -b_1\omega\cos(\omega\tau) + b_0\sin(\omega\tau). \end{array} \tag{27}$$

Squaring and adding both equations, we obtain

$$\omega^6 + T_1\omega^4 + T_2\omega^2 + T_3 = 0, \tag{28}$$

where

$$T_1 = a_2^2 - 2a_1, \quad T_2 = a_1^2 - 2a_0a_2 - b_1^2, \quad T_3 = a_0^2 - b_0^2.$$

Depending on the sign of T_i, $i = 1, 2, 3$, equation (28) will admit one or two positive real roots. For example, if $a_0 < b_0$ then $T_3 < 0$ and (28) can only have one positive real root ω_0, that is the characteristic equation (18) admits a pair of purely imaginary roots in the form $\pm i\omega_0$. It is important to notice that $\omega = \omega(\tau)$ and that there exists a set $I \subseteq \mathbf{R}$ such that if $\tau \in I$ then $\omega(\tau)$ is a positive root of (28) otherwise $\omega(\tau)$ is not definite. From (27) we know that $\tau = \tau_{0n}$ corresponding to $\omega = \omega_0(\tau)$ is defined as

$$\tau_{0n}(\tau) = \frac{1}{\omega_0(\tau)}\left(\frac{\omega b_1(a_0 - \omega^2 a_2) - b_0(a_1\omega - \omega^3)}{b_0(a_0 - \omega^2 a_2) + \omega b_1(a_1\omega - \omega^3)} + 2n\pi\right),$$

and that the imaginary roots of the characteristic equation (18) crosses the imaginary axis from left to right if $\delta(\tau_0) > 0$, and from right to left if $\delta(\tau_0) < 0$ [4], with

$$\delta(\tau_0) = \operatorname{sign}\left\{\frac{d\Re e\lambda}{d\tau}\Big|_{\lambda=i\omega_0(\tau_0)}\right\}.$$

Due to the complexity of computations we will not prove this result analytically, but with the aid of numerical simulations we will show that when E_2 is unstable and there exists a stable limit cycle, then for large values of the delay a Hopf bifurcation occurs with the disappearance of the stable periodic orbit and the stabilisation of the equilibrium.

Remark 3. If $T_3 > 0$ and (28) has only one real root, then the root will be negative. This implies that the delay does not change the stability properties of E_2. If $T_3 > 0$ and (28) has three real roots, then two of them will be positive if and only if $T_1 < 0$ or $T_1 = 0$, $T_2 < 0$. Finally, if $T_3 < 0$ then at least one real root of (28) will be positive. In the last two cases the method described above can be applied and stability switches can happen.

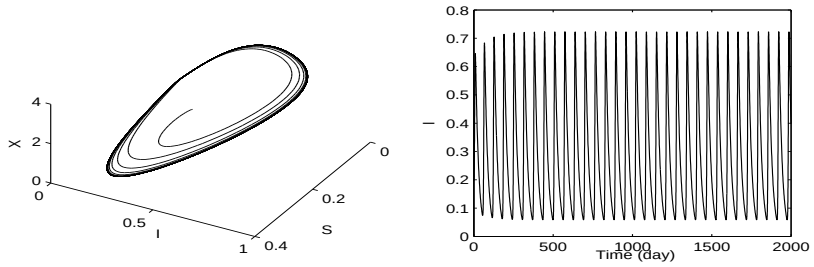

FIGURE 2. Temporal behaviour of the infected host and corresponding three-dimensional phase of system (4) obtained by using the following parameters: $\tau = 0$, $\beta_p = 0.3$, $\beta_s = 0.5$, $\mu = 0.05$ and $c = 0.15$. In this case E_2 is unstable, the conditions of Corollary 2 hold, and system (4) has a periodic orbit.

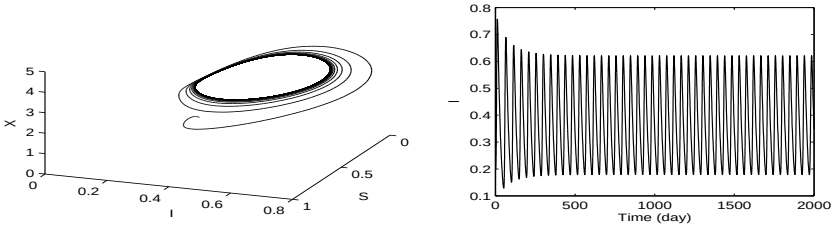

FIGURE 3. Temporal behaviour of the infected host and corresponding three-dimensional phase of system (4) obtained by using the following parameters: $\tau = 4$, $\beta_p = 0.3$, $\beta_s = 0.5$, $\mu = 0.05$ and $c = 0.15$. In this case $0 < \tau < \tau_0$, E_2 is unstable and system (4) has a periodic orbit.

We consider the region of the parameter space of system (4) corresponding to the existence of the internal equilibrium, that is $R_0 > 1$. To illustrate the transition from the instability to the stability of E_2, we will initially assume $\tau = 0$ and then $\tau > \tau_0$. We consider the following parameter values: $\beta_p = 0.3$, $\beta_s = 0.5$, $\mu = 0.05$ and $c = 0.15$. In this way, for $\tau = 0$ we get $R_0 = 50$, $E_2 \equiv (0.02, 0.28, 1.87)$ is unstable and system (4) has a periodic orbit (see Figure 2). In this case equation (28) admits one or two positive real roots for $\tau \in [0, 5.344]$. In particular there exists a critical delay $\tau_0 = 5.3$ such that E_2 is unstable for $\tau < \tau_0$ (see Figure 3) and acquires stability by a Hopf bifurcation for $\tau > \tau_0$ as shown in Figure 4.

7. **Conclusions.** The main aim of this paper was to analyse how the latent time of the disease, represented by a time delay in the infectivity of the free-living inoculum, can affect the dynamics of (air-) soil-borne plant disease models. As widely mentioned in the previous sections, these type of models have been extensively studied by Gilligan and coworkers. In their works, they were mostly interested in conditions for cultural or biological control of the disease, and therefore of the pathogen [10, 11, 17]. Here, we studied a variant of the class of models introduced by Gubbins et al. [17]. In particular, we considered that the primary infection is

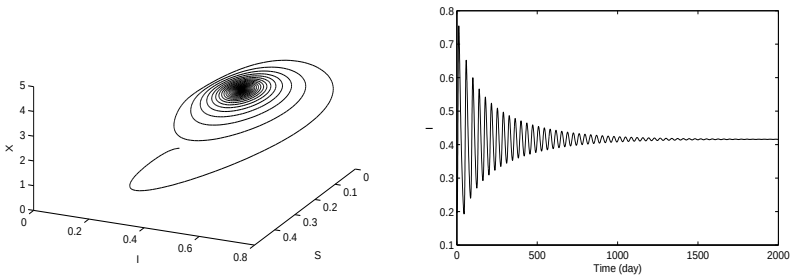

FIGURE 4. Temporal behaviour of the infected host and corresponding three-dimensional phase of system (4) obtained by using the following parameters: $\tau = 6$, $\beta_p = 0.3$, $\beta_s = 0.5$, $\mu = 0.05$ and $c = 0.15$. In this case $\tau > \tau_0$ and $\delta(\tau_0) < 0$, therefore the imaginary roots of the characteristic equation (18) crossed the imaginary axis from right to left causing the stabilisation of E_2.

not instantaneous but delayed. Therefore model (2) is given by delay differential equations with delay dependent parameters.

Regarding the disease-free equilibrium E_1, we showed that the classical stability–threshold holds true. If $R_0 < 1$, then the pathogens are unable to invade and the pathogen-free equilibrium is globally asymptotically stable, whereas if $R_0 > 1$ then E_1 becomes unstable (Theorem 3.2).

We have shown that the delay influences the delay-existence domain of the coexistence equilibrium E_2 (Remark 1). In other words, when the basic reproduction number is greater than unity and the infectious hosts are removed at a rate that is greater than the secondary infection rate (i.e., $\mu > \beta_s$) then the disease can't become endemic unless the length of the latent time is small enough. On the opposite, when $\mu < \beta_s$ the disease can become endemic for all sizes of the delay. Furthermore, if the coexistence equilibrium is unstable the delay can affect its stability properties, and the effect of the delay is stabilising (Figure 4). These results are biologically relevant because suggest that plant epidemics can be successful: (i) if pathogens have short latent periods for the primary infection, in case of slow diffusion of secondary infection; (ii) if the secondary infection rate is large enough and exceeds the removal rate of the infected host. In this last case a periodic behaviour of the epidemics will not occur with large delays (latency), e. g. due to the generation time for spore production, as in fungal pathogens.

Additionally, the delay is harmless in affecting the stability property of E_2 by failing to induce stability switches when E_2 is stable. On the contrary, when E_2 is unstable, and a periodic solution has emerged near the steady state, then large delays can be stabilising and cause the disappearance of the periodic orbit. This result seems to be particularly interesting. In fact, in [10] the authors found a reduction in the range of values ensuring the endemic stability. This circumstance is deemed to be due to the host logistic growth. We show that such reduction does not occur when the latency period is large enough.

Finally we analysed the case $\tau = 0$. We showed that near the criticality (at E_1 and $R_0 = 1$) there is a stable coexistence equilibrium (E_2) bifurcating from E_1, and E_1 changes from being stable to unstable. Nevertheless, E_2 can destabilise through Hopf bifurcation and an onset of oscillations occurs. Unfortunately, the condition

guaranteeing the Hopf bifurcation involves multiple parameters and for this reason does not allow a feasible biological interpretation.

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