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ORIGINAL ARTICLE

Modelling the Effect of Incubation and Latent Periods on the Dynamics of Vector-Borne Plant Viral Diseases

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Abstract

Most of the plant viral diseases spread through vectors. In case of the persistently transmitted disease, there is a latent time of infection inside the vector after acquisition of the virus from the infected plant. Again, the plant after getting infectious agent shows an incubation time after the interaction with an infected vector before it becomes diseased. The goal of this work is to study the effect of both incubation delay and latent time on the dynamics of plant disease, and accordingly a delayed model has been proposed. The existence of the equilibria, basic reproductive number ($\mathcal{R}_0$) and stability of equilibria have been studied. This study shows the relevance of the presence of two time delays, which may lead to system stabilization.

Keywords Time delay model · Basic reproduction number · Stability · Bifurcation

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1 Introduction

Plant disease epidemiology is the scientific study of how diseases affect plant populations and the subsequent application of the results of this study to control plant diseases. Spatio-temporal plant epidemiological models can provide important mathematical and statistical information about probable ways of disease propagation. Plant epidemiological models became popular in the mid-twentieth century (Van der Plank 1963). This kind of models has shown its usefulness in terms of both theoretical (especially where detailed laboratory or field experiment is not feasible) and practical applications (Bailey 1975), for example, in Cassava Mosaic Disease (Holt et al. 1997), Potato Late Blight (Bruhn and Fry 1981), Pine Wilt Disease (Yoshimura et al. 1999), etc. Later, the development of sophisticated tools to study nonlinear dynamics and to perform exhaustive numerical simulations helped to study complicated ecological problems (Villa 1992). This further boosted the development of more realistic and complex models to eradicate plant disease.

The basic backbone of the plant epidemiological system is modelling the interactions between healthy and infected plant populations, which can occur either directly or through a vector. The healthy plants become infected when an infected vector feeds on them. Similarly, the non-infected vector becomes infected when it feeds on the infected plant. The vector-borne plant disease can be further divided into three types depending on the residence time of the infectious agent in the vector: persistent, semi-persistent and non-persistent (Hohn 2007; Anguelov et al. 2012). In case of persistent transmission, viruses are first internalized by the vector during feeding on plant sap of infected plants. Then, the viruses cross over haemolymph from the gut and ultimately reach the salivary gland to be released into the plant tissue. So unlike the other two modes of transmission, a long time lag exists between the infection of a vector with the virus and the time to become infectious, in the persistent mode of transmission (Dietzgen et al. 2016; Hohn 2007). This time lag is called *latent time* or *latent period* of infection in *vector*. Similarly, a healthy plant after infection becomes infectious after a certain time lag called *latent period* in the *plant* (Madden et al. 2007). A longer time is needed for the appearance of disease symptoms after infection, called *incubation period* or *incubation time* (Van der Plank 1963). The incubation period varies according to plant species (Kern 1956). For example, in African Cassava Mosaic Virus (ACMV), Beet Mosaic Virus, Bean Golden Mosaic Virus and Tobacco Mosaic Virus the incubation periods are 3–5 weeks (Legg 2008), 7–15 days (Smrž 1972), 5–6 days (Haber et al. 1991) and 5h (Siegel et al. 1957), respectively. It is important to note that incubation period and latent period in plant are different. But expression of disease symptoms has positive correlation with the disease transmission (Rimbaud et al. 2015). Moreover, latent period is difficult to assess, while disease symptom is easier to note by observation. Thus, only the incubation period is considered in our analysis for the model development.

Mosaic disease and leaf curl disease are two of the most common vector-borne viral diseases for the agricultural crops. These are caused by hemipteran vectors, such as whitefly (*Bemisia* sp.), which causes many viral diseases of cassava, *Jatropha*, cotton, tomato, tobacco and other plants. Whitefly transmits most of the disease in a recurrent manner, i.e. a latent period is observed oftenly (Duffus 1987). Unfortunately, not too

much information is available in the literature about the incubation and latent time of infection for the various kinds of persistently transmitted disease. The value of both kinds of delay differs with both the viral agents and the host plant species. It also varies with different whitefly species or races for the same virus and host plants. The possible reasons for the variability of these delays are genotype complexity, phenotypic heterogeneity, phenotypic plasticity and climatic variation (Suffert and Thompson 2018). In most cases, the incubation period in plants is longer (from some days to few weeks) than the latent period in vector (from a few hours to a day). In the case of ACMV for instance, the latent period is 6 hours and the incubation period is 3–5 weeks (Legg 2008).

Models using ordinary differential equations (ODE) are unable to account for the incubation period or the latent period. However, models formulated via delay differential equations (DDEs) allow to incorporate the delay in the system. It can describe the dynamics of the system when their evolution depends on past times. Delays represent one of the most powerful mathematical modelling approaches and prove very useful in various fields of science and technology whenever time lag responses exist (Jiao and Chen 2008). DDE models are more realistic as compared to ODE models at the cost of increasing complexity. Mathematical models with delay differential equations have been widely used in studying prey–predator systems (Ruan 2001; Pal et al. 2012). Considering delay, various dynamic behaviours such as instability, oscillations and bifurcation can be studied.

Van der Plank (1963) first incorporated DDE with a single delay in plant epidemics. Cooke (1979) proposed a model with the incubation period for vector-borne infectious disease, which considered the susceptible and infected individual as state variables. Zhang (2012) introduced the plant incubation period as a delay in a plant disease model proposed by Meng and Li (2010), and changes in the dynamics of the model are observed. Recently, a model for the dynamics of soil-borne plant disease taking a time delay due to the latent period of inoculum/vectors has been proposed and analysed (Buonomo and Cerasuolo 2014).

A compartment for latently infected plant population has been incorporated in an ODE model for the impact of roguing and replanting in plant disease eradication to include the latency in plant disease (Chan and Jeger (1994)). The model includes healthy, latently infected, infectious and post-infectious classes of plants, but it does not consider any vector compartment. Another model with healthy plant, infected plant, susceptible vector and healthy vector was proposed in Holt et al. (1997), but no delays were incorporated. Jackson and Chen (2016) modified the model proposed by Shi et al. (2014) for vector-borne plant disease by including multiple delays for the incubation periods of plants and latent period in vectors. Changes in the dynamics of the system solutions are observed, and the threshold value for delay-induced destabilization is derived. A more recent article (Li et al. 2018), further modified the model of Jackson and Chen (2016), performed Hopf bifurcation analysis by choosing incubation and latent period as parameters. Further important features of the dynamics that could induce misleading conclusions have been revealed in Banerjee and Takeuchi (2017). Large delay can stabilize a system or make the system disease-free (Buonomo and Cerasuolo 2014). However, vector-borne plant disease models without delay can show stability switches, periodic oscillations, transcritical bifurcations, just by changes in

their parameters (Buonomo and Cerasuolo 2014; Venturino et al. 2016; Holt et al. 1997). Thus, the analysis of the undelayed model cannot be ignored as in Jackson and Chen (2016) and Li et al. (2018). It proves to be crucial to assess the persistence of infection for disease control, and the construction of vector-borne plant disease models with time delays requires a clear understanding of the relevant biological mechanism.

In this article, a mathematical model is formulated for the dynamics of persistently transmitted vector-borne viral plant disease. The effect of both incubation delay and latent period on the dynamics of the diseases is analysed. The existence of equilibria and their stability are analysed. In both delayed and non-delayed systems, stability switches have been observed analytically and numerically as well. For numerical simulations, we have chosen the set of parameters for the case of mosaic disease in Cassava and in this manuscript we have discussed the dynamical regimes that can be exhibited in the model for plant mosaic disease. Regions of stability of equilibria are also identified, which can help in analysing the systems dynamics.

The present work is organised as follows. The mathematical model is presented in Sect. 2. The analysis of equilibria is performed in Sect. 3. Hopf bifurcation for the non-delayed model is presented in Sect. 4. Numerical simulations are performed and important results are identified in Sect. 5. Finally, a discussion concludes the paper in Sect. 6.

2 The Mathematical Model

Plant and vector populations are considered without explicitly including the mosaic virus as a component of the model. Here, $S(t)$ and $I(t)$ denote the healthy and infected plant (here the term 'infected plant' actually indicates the diseased plants) densities, respectively. Uninfected and infected whitefly populations are denoted as $U(t)$ and $V(t)$, respectively.

Due to the finite area of the plantation and limitation of natural resources, for healthy plants, logistic growth with the net growth rate r and carrying capacity K are assumed (Holt et al. 1997). A successful contact between an infected vector with a healthy plant makes it infected. Let λ be the transmission rate between an infected vector and a susceptible plant, then λSV is the number of susceptible individuals leaving the susceptible compartment to the infected compartment due to new cases of infection.

Polyphagous pest, such as whitefly, shows host shift in response to the different biological and environmental situations. They usually migrate in large numbers between different cultivation fields (Horowitz 1986; Henneberry and Castle 2001). They also breed in the cultivation field. Their life history follows the Holling type III survivorship curve, because they show a high mortality in their early life stages (Henneberry and Castle 2001). Both the adult and nymph whiteflies are capable of transmitting disease.

Crops are usually cultivated at specific times of every year. Most of the crops are harvested a few months after plantation. A small number of vectors migrate from nearby or distant patches and then reproduce within the vegetative land. The growth of vectors initiates due to migration from some other patch and then grows due to reproduction at the concerned patch or vegetative land. The seasonal fluctuation in vector population is also neglected for the same reason.

In this model, an open system is considered. The rate at which the vector enters the system by birth and migration (here 'migration' means, net migration, i.e. *immigration-emigration*), is assumed to be a constant, Π. No vertical transmission of the virus is assumed, and a vector cannot transmit the virus to another vector. In addition, vectors do not get killed by the virus nor do they defend against it. The vector keeps the virus for its lifespan and does not recover. The infective insects do not get sick from the virus, but they act as carriers (Jackson and Chen 2016). Let μ be the plant natural death rate and d be the mortality rate of the vector. The mortality rate of plants due to infection is assumed to be higher than the natural mortality in plant. Additional mortality of infected plants due to infection is denoted by m_1. Therefore, the total mortality rate of infected plant is $m = \mu + m_1$. The transmission rate between infected plant I and uninfected vector U is denoted by β. Hence, $\beta U I$ represents the number of uninfected vectors (U) entering into the infected vector (V) compartment.

In reality, the infection of both plants and vectors is a delayed process. Let $\tau_1 \in R^+$ be the discrete time required for a healthy plant to become infected after successful infection, i.e. incubation time. More precisely, the transmission of infection at time t is described by the term $\lambda e^{-m\tau_1} S(t-\tau_1)V(t-\tau_1)$, where μ and λ are the positive constants described earlier. The term $e^{-m\tau_1}$ represents the survival probability of a healthy plant through the incubation time $[t-\tau_1, t]$, i.e. the amount of susceptible plants that came in contact with infected vector at time $t-\tau_1$ ultimately surviving up to time t to become infected plants.

Again, $\tau_2 \in R^+$ is the latent period in vector. Thus, the transmission of infection at time t is described by the term $\beta e^{-d\tau_2} U(t-\tau_2)I(t-\tau_2)$, where $e^{-d\tau_2}$ represents the survival probability of the vector through the latent time $[t-\tau_2, t]$. It is the amount of uninfected vectors that came in contact with infected vector at time $t-\tau_2$ and that ultimately survive until time t to become infected vector.

From the above assumptions, the mathematical model takes the following form:

$$\begin{aligned}
\frac{dS}{dt} &= rS\left[1 - \frac{S+I}{K}\right] - \lambda SV,\\
\frac{dI}{dt} &= \lambda e^{-m\tau_1} S(t-\tau_1)V(t-\tau_1) - mI,\\
\frac{dU}{dt} &= \Pi - \beta U I - dU,\\
\frac{dV}{dt} &= \beta e^{-d\tau_2} U(t-\tau_2)I(t-\tau_2) - dV,
\end{aligned} \tag{1}$$

where $m = \mu + m_1$ and the initial biological conditions are:

$$S(\gamma) > 0, \quad U(\gamma) > 0, \quad I(\gamma) > 0, \quad V(\gamma) > 0; \quad \gamma \in [-\tau, 0], \quad \tau = \max\{\tau_1, \tau_2\}. \tag{2}$$

3 Existence of Equilibria and Stability of Disease-Free Equilibrium

Note that the presence of Π in the growth equation of U prevents us to get an equilibrium of the form $(K, 0, 0, 0)$. Thus, the system (1) admits three nontrivial equilibria, namely the plant-free equilibrium, $E_1 = (0, 0, U_1, 0)$, the disease-free equilibrium, $E_2 = (K, 0, U_2, 0)$ where $U_1 = U_2 = \frac{\Pi}{d}$, and the endemic equilibrium, $E^* = (S^*, I^*, U^*, V^*)$. Here,

$$\begin{aligned} S^* &= \frac{md(d + \beta e^{-d\tau_2} I^*)}{\lambda\beta e^{-(m\tau_1 + d\tau_2)}\Pi}, \\ U^* &= \frac{\Pi}{d + \beta e^{-d\tau_2} I^*}, \\ V^* &= \frac{\Pi\beta e^{-d\tau_2} I^*}{d(d + \beta e^{-d\tau_2} I^*)}, \end{aligned} \tag{3}$$

where I^* is the positive root of

$$H(I) = \alpha_1 I^2 + \alpha_2 I + \alpha_3 = 0, \tag{4}$$

with

$$\begin{aligned} \alpha_1 &= -r\beta e^{-d\tau_2} - \frac{rmd\beta e^{-d\tau_2}}{\lambda\Pi} < 0, \\ \alpha_2 &= rK\beta e^{-d\tau_2} - \frac{2rmd^2}{\lambda\Pi} - dr - K\Pi\beta\lambda e^{-(m\tau_1 + d\tau_2)}, \\ \alpha_3 &= drK - \frac{rmd^3}{\Pi\beta\lambda e^{-(m\tau_1 + d\tau_2)}}. \end{aligned}$$

For $\alpha_3 > 0$, one positive root is ensured. If $\alpha_3 < 0$, $\Delta = \alpha_2^2 - 4\alpha_1\alpha_3 > 0$ and $\alpha_2 > 0$, then two positive roots exist.

The basic reproduction number $\mathcal{R}_0$ of the delayed system is

$$\mathcal{R}_0 = \frac{\Pi k\lambda\beta e^{-(m\tau_1 + d\tau_2)}}{md^2}. \tag{5}$$

A brief description on how this can be obtained is provided in 'Appendix A'.

Thus, for $\mathcal{R}_0 > 1$, a unique coexistence equilibrium E^* exists. Also, it can be shown that E_2 is stable for $\mathcal{R}_0 < 1$ and unstable for $\mathcal{R}_0 > 1$. Transcritical bifurcation occurs at $\mathcal{R}_0 = 1$ (see 'Appendix B'). The plant-free equilibrium E_1 is always unstable.

We can determine the minimum value of the delays for which E_0 is stable:

$$m\tau_1 + d\tau_2 > \ln\left(\frac{\Pi k\lambda\beta}{md^2}\right) = \ln\mathcal{R}_0, \tag{6}$$

where $\mathcal{R}_0$ is the basic reproduction number of the system without delay.

Remark 1 Note that $\mathcal{R}_0$ depends on d and m; hence, mortalities are important parameters in the infection eradication process. Further, $\mathcal{R}_0$ is monotonically decreasing with increasing m. This suggests that disease eradication, represented here by the stable disease-free steady state E_2, is only possible if $\mathcal{R}_0 < 1$. The disease-free state can be achieved by increasing roguing (the rate of removal of infected plant), reducing vector population by insecticides (Venturino et al. 2016; Al Basir et al. 2018) or introducing predators in the system or other biological control methods (Shun-xiang et al. 2001; Moore et al. 2009; Okamoto and Amarasekare 2012).

4 Stability of Endemic Equilibrium and Hopf Bifurcation

The Jacobian matrix evaluated at the steady point $e^*(S^*, I^*, U^*, V^*)$ gives us the following characteristic equation:

$$| \xi I_4 - M - e^{-\xi\tau_1} D - e^{-\xi\tau_2} E | = 0,$$

where I_4 denotes the 4×4 identity matrix and M, D, E are defined as follows:

$$M = [m_{ij}] = \begin{bmatrix} -\frac{rS^*}{K} & -\frac{rS^*}{K} & 0 & -\lambda S^* \\ 0 & -m & 0 & 0 \\ 0 & -\beta U^* & -\beta I^* - d & 0 \\ 0 & 0 & 0 & -d \end{bmatrix}$$

and

$$D = \begin{bmatrix} 0 & 0 & 0 & 0 \\ \lambda e^{-m\tau_1} V^* & 0 & 0 & \lambda e^{-m\tau_1} S^* \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

$$E = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \beta e^{-d\tau_2} U^* & \beta e^{-d\tau_2} I^* & 0 \end{bmatrix}.$$

The characteristic equation can be explicitly written as

$$\begin{aligned} \phi(\xi, \tau_1, \tau_2) = {} & \xi^4 + a_1\xi^3 + a_2\xi^2 + a_3\xi + a_4 \\ & + e^{-\xi(\tau_1+\tau_2)}[p_1\xi^2 + p_2\xi + p_3] + e^{-\xi\tau_1}[b_1\xi^2 + b_2\xi + b_3] = 0, \end{aligned}$$

where

$$a_1 = \frac{rS^*}{K} + m + \beta I^* + 2d,$$

$$a_2 = \frac{rmS^*}{K} + \frac{rS^*(\beta I^* + d)}{K} + m(\beta I^* + d) + \frac{rdS^*}{K} + md + d(\beta I^* + d),$$
$$a_3 = \frac{rmS^*(\beta I^* + d)}{K} + \frac{rmdS^*}{K} + \frac{rdS^*(\beta I^* + d)}{K} + md(\beta I^* + d),$$
$$a_4 = \frac{rdmS^*(\beta I^* + d)}{K},$$

and

$$b_1 = \frac{r\lambda e^{-m\tau_1} V^* S^*}{K},$$
$$b_2 = \frac{rS^*\lambda e^{-m\tau_1} V^*(\beta I^* + d)}{K} + \frac{rdS^*\lambda e^{-m\tau_1} V^*}{K},$$
$$b_3 = \frac{rS^*\lambda e^{-m\tau_1} V^*(\beta I^* + d)d}{K},$$
$$p_1 = -\lambda e^{-m\tau_1} S^* \beta e^{-d\tau_2} U^*$$
$$p_2 = \lambda\beta e^{-(m\tau_1 + d\tau_2)} \left[\lambda S^* V^* U^* - \frac{rS^{*2}U^*}{K} - dS^* U^* \right],$$
$$p_3 = \lambda\beta e^{-(m\tau_1 + d\tau_2)} \left[\lambda S^* V^* dU^* - \frac{rS^{*2}dU^*}{K} \right].$$

Now, we consider the stability of E^* for four different cases, namely for no delay, only for incubation delay τ_1 with $\tau_2 = 0$, only for latent delay τ_2 with $\tau_1 = 0$ and both incubation delay τ_1 and latent delay τ_2 that are non-zero.

Case I $\tau_1 = 0 = \tau_2$.

Note that in this case $a_{11} = -rS^*K^{-1}$ and $a_{33} = -\Pi(U^*)^{-1}$, so that $\sigma_1 = -\mathrm{tr}(A(E^*)) > 0$.

The remaining Routh–Hurwitz conditions for the characteristic equation

$$\rho^4 + \sigma_1\rho^3 + \sigma_2\rho^2 + \sigma_3\rho + \sigma_4 = 0 \tag{7}$$

give the following requirements for stability of E^*

$$\sigma_4 > 0, \quad \sigma_1\sigma_2 - \sigma_3 > 0, \quad (\sigma_1\sigma_2 - \sigma_3)\sigma_3 - \sigma_1^2\sigma_4 > 0, \tag{8}$$

where

$$\sigma_2 = r\lambda K^{-1} S^* V^* - \lambda\beta S^* U^* + r\beta K^{-1} I^* S^* + 2rS^* dK^{-1} + \beta I^* d + d^2 + mrK^{-1} S^* + \beta I^* m + 2dm,$$
$$\sigma_3 = \lambda^2\beta K^{-1} S^* V^* U^* - \lambda\beta K^{-1} r(S^*)^2 U^* + rS^*\lambda V^*\beta I^* + 2rK^{-1} d\lambda S^* V^* - \lambda S^*\beta U^* d + rdK^{-1}\beta S^* I^* + rS^* K^{-1} d^2 + r\beta m K^{-1} S^* I^* + 2rK^{-1} dmS^* + \beta I^* dm + d^2 m,$$

$$\sigma_4 = -r\lambda\beta K^{-1}(S^*)^2U^*d + \lambda^2 d\beta S^*V^*U^* + rS^*K^{-1}\lambda V^*\beta I^*d + rK^{-1}\lambda d^2 S^*V^* + rdm\beta K^{-1}S^*I^* + rd^2mK^{-1}S^*. \quad (9)$$

Due to the complicated structures of the expressions of the coefficients of the characteristic equation, it is quite difficult to find the stability condition explicitly in terms of any parameter.

For the Hopf bifurcation at endemic equilibrium E^*, we consider λ as the bifurcation parameter. A Hopf bifurcation will occur if the characteristic equation (7) has two complex conjugate eigenvalues for $\lambda = \lambda^* \in (0, \infty)$ and the other two eigenvalues have negative real parts. Moreover, the transversality condition

$$\left.\frac{d\mathrm{Re}\rho(\lambda)}{d\lambda}\right|_{\lambda^*} \neq 0$$

needs to be satisfied. We have the following result (Hassard et al. 1981; Kuznetsov 2013; Venturino et al. 2016).

Theorem 1 *For $\tau_1 = 0 = \tau_2$, the endemic equilibrium E^* undergoes a Hopf bifurcation at $\lambda = \lambda^*$, where $\lambda^* \in (0, \infty)$ if and only if*

$$\begin{aligned} &\text{i. } \Phi(\lambda^*) = 0 \text{ and } \sigma_1\sigma_2 - \sigma_3 > 0, \\ &\text{ii. } \sigma_1^3\sigma_2'\sigma_3(\sigma_1 - 3\sigma_3) > 2(\sigma_2\sigma_1^2 - 2\sigma_3^2)(\sigma_3'\sigma_1^2 - \sigma_1'\sigma_3^2). \end{aligned} \quad (10)$$

$\sigma_i, i = 2, 3, 4$, are defined in (9) and primes denote derivative with respect to λ. The complex conjugate eigenvalues are purely imaginary at $\lambda = \lambda^*$, and other two eigenvalues of (7) have negative real parts, and Φ is the continuously differentiable function $\Phi : (0, \infty) \to R$ of λ with

$$\Phi(\lambda) := \sigma_1(\lambda)\sigma_2(\lambda)\sigma_3(\lambda) - \sigma_3^2(\lambda) - \sigma_4(\lambda)\sigma_1^2(\lambda).$$

Case II When $\tau_1 > 0, \tau_2 = 0$.

In this case, the characteristic equation becomes:

$$\phi(\xi, \tau_1) = \xi^4 + C_1\xi^3 + C_2\xi^2 + C_3\xi + C_4 + \mathrm{e}^{-\xi\tau_1}[D_1\xi^2 + D_2\xi + D_3] = 0, \quad (11)$$

where

$$\begin{aligned} &C_1 = (a_1 + b_1), \quad C_2 = (a_2 + p_2), \\ &C_3 = (a_3 + b_3), \quad C_4 = (a_4 + b_4), \\ &D_1 = p_1, \quad D_2 = p_2 + p_1, \quad D_3 = p_2 + p_3. \end{aligned}$$

For $\tau_1 > 0$, (11) will have infinitely many roots. To determine the nature of the stability, the sign of the real parts of the roots of the characteristic equation (11) is required. A necessary condition for the stability changes of E^* is that the characteristic

equation (11) has purely imaginary solutions. Let $i\theta$, $\theta \in \mathbf{R}$, be a root of equation (11). Substituting $\rho = i\theta$ in (11) and then separating real and imaginary parts, we get:

$$D_1 \sin\theta\tau_1 + D_2 \cos\theta\tau_1 = -\theta^4 + C_2\theta^2 - C_4, \tag{12}$$
$$D_1 \cos\theta\tau_1 - D_2 \sin\theta\tau_1 = C_1\theta^3 - C_3\theta. \tag{13}$$

Squaring and adding the above two equations, and substituting $\theta^2 = l$, we obtain:

$$l^4 + \omega_1 l^3 + \omega_2 l^2 + \omega_3 l + \omega_4 = 0. \tag{14}$$

Here,

$$\omega_1 = {C_1}^2 - 2C_2, \quad \omega_2 = C_2^2 + 2C_4 - 2C_1C_3,$$
$$\omega_3 = -2C_2C_4 + C_3^2 - D_1^2, \quad \omega_4 = C_4^2 - (D_1^2 + D_2^2).$$

Lemma 1 *If $\omega_4 < 0$, then (14) has at least one positive root.*

Proof Let us define $H(l)$ as follows.

$$H(l) = l^4 + \omega_1 l^3 + \omega_2 l^2 + \omega_3 l + \omega_4. \tag{15}$$

Thus, $H(0) = \omega_4 < 0$ and $\lim_{l\to\infty} H(l) = \infty$. Hence, there exists an $l_0 \in (0, \infty)$ so that $H(l_0) = 0$ and this completes the proof. □

From (15), we have

$$H'(l) = 4l^3 + 3\omega_1 l^2 + 2\omega_2 l + \omega_3. \tag{16}$$

We now assume that $H'(l) = 0$ and define $z = l + \frac{3\omega_1}{4}$; then, (16) becomes

$$z^3 + \Lambda_1 z^2 + \Lambda_2 = 0, \tag{17}$$

where

$$\Lambda_1 = \frac{\omega_2}{2} - \frac{3\omega_1^2}{16},$$
$$\Lambda_2 = \frac{\omega_1^3}{32} - \frac{\omega_1\omega_2}{8} + \omega_3.$$

Let us set

$$\delta = \frac{\Lambda_2^2}{4} + \frac{\Lambda_1^3}{27}, \quad \Gamma = \frac{-1 + \sqrt{3i}}{2},$$
$$z_1 = \sqrt[3]{-\frac{\Lambda_2}{2} + \sqrt{\delta}} + \sqrt[3]{-\frac{\Lambda_2}{2} - \sqrt{\delta}},$$

$$z_2 = \Gamma \sqrt[3]{-\frac{\Lambda_2}{2} + \sqrt{\delta}} + \Gamma^2 \sqrt[3]{-\frac{\Lambda_2}{2} - \sqrt{\delta}},$$
$$z_3 = \Gamma^2 \sqrt[3]{-\frac{\Lambda_2}{2} + \sqrt{\delta}} + \Gamma \sqrt[3]{-\frac{\Lambda_2}{2} - \sqrt{\delta}}, \quad \text{and } l_r = z_r - \frac{3\Lambda_1}{4}, r = 1, 2, 3.$$

The roots of Eq. (14) have negative real parts if and only if the Routh–Hurwitz criterion is satisfied. In such case, (11) does not have purely imaginary roots. Thus, we have the following result.

Proposition 1 *Suppose that the system without delay is stable. The endemic equilibrium E^* is locally asymptotically stable for all $\tau_1 > 0$ if the following conditions are satisfied:*

$$\omega_1 > 0, \quad \omega_4 > 0, \quad \omega_1\omega_2 - \omega_3 > 0, \quad (\omega_1\omega_2 - \omega_3)\omega_3 - \omega_1^2\omega_4 > 0.$$

Lemma 2 *Suppose that $\omega_4 < 0$,*

(i) *If $\delta \geq 0$, then (14) has positive roots if and only if $l_1 > 0$ and $H(l_1) < 0$;*
(ii) *If $\delta < 0$, then (14) has positive root if and only if there exists at least one $l^* \in \{l_1, l_2, l_3\}$, such that $l^* > 0$ and $H(l^*) \leq 0$, where, l_1, l_2, l_3 are critical points of $H(l)$.*

Proof (i) If $\delta \geq 0$, (17) has a unique real root z_1 and thus (16) has unique real root l_1. Since $H(l)$ is a differentiable function and $\lim_{l\to\infty} H(l) = \infty$, l_1 is the unique minimum point of $H(l)$. The sufficient condition follows directly. So, we prove the necessary condition only.
Here, either $l_1 \leq 0$ or $l_1 > 0$ and $H(l_1) > 0$. In the former case, $H(0) = \omega_4 > 0$ is the minimum of $H(l)$ for $l \geq 0$. Thus, $H(l)$ has no positive real zeros. In the second case, $l_1 > 0$ and $H(l_1) > 0$, since $\min_{l>0} H(l) = H(l_1) > 0$. Thus here too, $H(l)$ has no positive real zeros.

(ii) If $\delta < 0$, (17) has only three roots l_1, l_2, l_3, i.e. (16) has only three roots l_1, l_2, l_3 and at least one of them is real. We assume that l_1, l_2, l_3 are all real, i.e. $H(l)$ has at most three stationary points at l_1, l_2, l_3. The remaining part of the proof is similar to the one of (i). □

Now, suppose that (15) has positive roots that are denoted by l_r^*, $r = 1, 2, 3, 4$. Then, (16) has four positive roots, $\theta_r = \sqrt{z_r^*}$, $r = 1, 2, 3, 4$. From Eq. (12), the value of τ_1 is

$$\tau_{1_k}^n = \frac{1}{\theta_k} \cos^{-1}\left[\frac{\theta_0^2 D_2[C_1\theta_0^2 - C_3] + (D_1\theta_0^2 - D_3)[\theta_0^4 - C_2\theta_0^2 + C_4]}{(D_1\theta_0^2 - D_3)^2 + D_2^2\theta_0^2}\right] + \frac{2\pi n}{\theta_k}, k = 1, 2, 3, 4 \text{ and } n = 0, 1, 2, 3, \ldots \tag{18}$$

Thus, $\pm i\theta_k$ is a pair of purely imaginary roots of (11). Let

$$\tau_1^* = \tau_{1_{k_0}}^n = \min_{n\geq 0,\ 1\leq k\leq 4} \{\tau_{1_k}^n\}, \quad \theta_0 = \theta_{k_0}, \quad l_0 = l_{k_0}^*. \tag{19}$$

Lemma 3 *Suppose that the non-delayed system is stable, i.e. (8) holds. Now,*

(i) *all roots of (11) have negative real parts if one of the following conditions hold:*

(a) $\omega_4 < 0$,
(b) $\omega_4 \geq 0$, $\delta \geq 0$, $l_1 > 0$ *and* $H(l_1) \leq 0$,
(c) $\omega_4 \geq 0$, $\delta < 0$, *and there exists a* $l^* \in \{l_1, l_2, l_3\}$ *such that* $l^* > 0$ *and* $H(l^*) \leq 0$;

(ii) *if the conditions (a) to (c) of (i) are not satisfied, then all roots of (11) have negative real parts for all* $\tau_1 \geq 0$.

From Lemmas 2 and 3, we can say that if the conditions (a) to (c) are not satisfied, then (11) has no roots with zero real part for all $\tau_1 \geq 0$. If any one of (a), (b) and (c) holds, then Eq. (11) has roots with zero real part and τ_1^* is the minimum value of τ_1 so that (11) has purely imaginary roots (Cao and Xiao 2007).

Lemma 4 *Suppose* $H'(l_r^*) \neq 0$. *If* $\tau_1 = \tau_1^*$, *then* $\pm i\xi_0$ *is a pair of purely imaginary roots of (11). Moreover, if the conditions in (i) of Lemma 3 are satisfied, then the sign of* $\frac{\mathrm{d}(Re\ \xi)}{\mathrm{d}\tau_1}$ *is consistent with that of* $H'(l_r^*)$.

Proof Differentiating (11) with respect to τ_1 and after some algebraic calculations, we get:

$$\frac{\mathrm{d}\tau_1}{\mathrm{d}\xi} = \frac{4\xi^3 + 3(C_1\xi^2 + 2C_2\xi + C_3)}{D_2\xi^3 + D_3\xi^2 + D_4\xi} \mathrm{e}^{\xi\tau_1} + \frac{2D_2\xi + D_3}{D_2\xi^3 + D_3\xi^2 + D_4\xi} - \frac{\tau_1}{\xi}.$$

Now, using the relation (12), after a simple calculation, one obtains:

$$\begin{aligned} \mathrm{Sgn}\left[\frac{\mathrm{d}(Re\ \xi)}{\mathrm{d}\tau_1}\right]_{\tau_1=\tau_1^*} &= \mathrm{Sgn}\left[\mathrm{Re}\left(\frac{\mathrm{d}\xi}{\mathrm{d}\tau_1}\right)^{-1}\right]_{\xi=i\xi_0}, \\ &= \mathrm{Sgn}\left[\frac{4\xi_0^6 + \omega_1\xi_0^4 + \omega_2\xi_0^2 + \omega_3}{D_3^2\xi_0^2 + [-D_2\xi_0^2 + D_4]^2}\right], \end{aligned} \tag{20}$$

Since $D_3^2\xi_0^2 + [-D_2\xi_0^2 + D_4]^2$ is always positive, from Eq. (19) we have that $H'(l_0) \neq 0$ if $4\xi_0^6 + \omega_1\xi_0^4 + \omega_2\xi_0^2 + \omega_3 \neq 0$, i.e. it follows that

$$\mathrm{Sgn}\left[\frac{\mathrm{d}(\mathrm{Re}\ \xi)}{\mathrm{d}\tau_1}\right]_{\tau_1=\tau_1^*} \neq 0.$$

□

Thus, from Lemmas 2, 3 and 4, we have the following theorem:

Theorem 2 *If the conditions (a)–(c) of Lemma 3 are not satisfied, then (11) has no roots with zero real part for all $\tau_1 \geq 0$. If one of (a), (b) and (c) holds, Eq. (11) has negative real part roots when $\tau_1 \in [0, \tau_1^*)$, when $\tau_1 = \tau_1^*$ and $[\frac{\mathrm{d}(\mathrm{Re}\xi}{\mathrm{d}\tau_1}]_{\tau_1=\tau_1^*} \neq 0$, then (11) has purely imaginary roots $\pm i\xi_0$ and all other roots have negative real parts and the system undergoes a Hopf bifurcation at $\tau_1 = \tau_1^*$.*

Case III When $\tau_1 = 0, \tau_2 > 0$.

The characteristic equation in this case takes the following form:

$$\begin{aligned}\phi(\xi, \tau) = \xi^4 + A_1\xi^3 + A_2\xi^2 + A_3\xi + A_4 \\ +\mathrm{e}^{-\xi\tau_2}[B_1\xi^2 + B_2\xi + B_3] = 0.\end{aligned} \tag{21}$$

Here, $A_1 = a_1 + b_1$, $A_2 = (a_2 + b_2)$, $A_3 = (a_3 + b_3)$, $A_4 = (a_4 + b_3)$, $B_1 = c_1$, $B_2 = c_2 + p_1$, $B_3 = c_3 + p_2$, $B_4 = c_4 + p_3$. The rest of the analysis is similar to Case II.

Case IV $\tau_1 > 0, \tau_2 > 0$.

We state the following theorem without proof.

Theorem 3 *Suppose that the endemic equilibrium E^* is asymptotically stable for $\tau_2 \in (0, \tau_2^*)$. Now, if $\omega_4 < 0$ holds, then there exists τ^* for which stability switch occurs at E^* when τ_1 passes the critical value τ^*. Furthermore, E^* will undergo a Hopf bifurcation when $\tau_1 = \tau^*$, provided that*

$$\left[\frac{\mathrm{d}(\mathrm{Re}\xi)}{\mathrm{d}\tau_1}\right]_{\tau_1=\tau^*} > 0.$$

A similar result can be proved for τ_2 when $\tau_1 \in (0, \tau_1^*)$.

Remark 2 At the endemic equilibrium E^*, the characteristic equation (7) has delay-dependent coefficients (i.e. for $\tau_i, i = 1, 2$) and it is quite involved. Therefore, it is difficult to analytically obtain information on the nature of the eigenvalues and on the conditions for occurrence of stability switches. But the nature of the eigenvalues can be investigated at the endemic state through numerical simulations.

In 'Appendix C', we have used results from Lemmas 1 and 2 to compute the critical value of the time delay τ_1.

5 Numerical Results

In this section, numerical simulations are presented to validate the analytical findings. First, the analysis of the system without delay and then the system with delay is performed.

Figure 1 shows the changes in steady-state values of the infected plant and vectors with λ for $\tau = 0$. The endemic steady state E^* becomes feasible for some minimum values of λ that correspond to $\mathcal{R}_0 = 1$, while for values of λ just above this critical

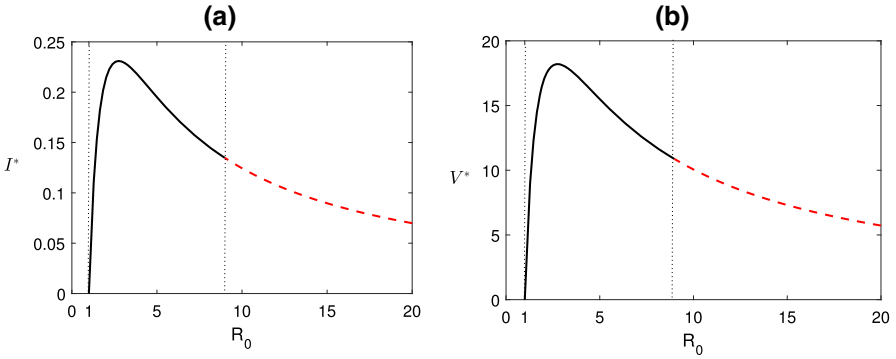

Fig. 1 Hopf bifurcation: steady-state values of infected plant (I^*) and infected vector (V^*) plotted as a function of $\mathcal{R}_0$. Parameter values: $\Pi = 40$, $r = 0.05$, $K = 1$, $\beta = 0.03$, $d = 0.12$, $m = 0.032$ and $\lambda \in (0, 0.00345]$. Solid line corresponds to a stable steady state, while a dashed line denotes an unstable steady state

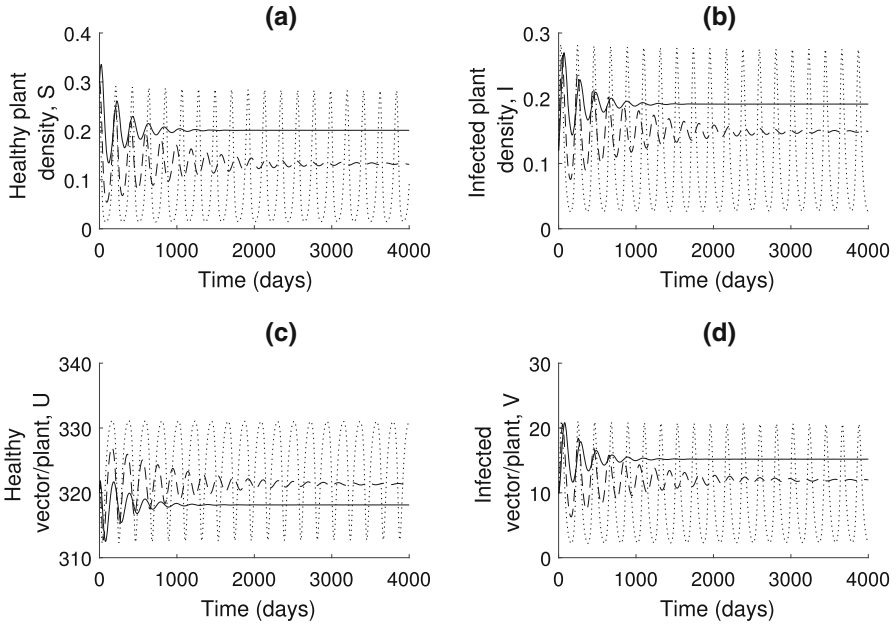

Fig. 2 Numerical solution of the system without delay for $\lambda = 0.002$ (solid line) and $\lambda = 0.003$ (dashed line) and $\lambda = 0.0045$ (dotted line) taking parameter values as in Fig. 1

value, the steady state E^* is stable. Further increment in the disease transmission rate λ causes loss of stability for the endemic steady state via a Hopf bifurcation (which verifies the results of Theorem 1).

In Fig. 2, we consider the set of the parameters (given in Table 1) for which the interior equilibrium E^* exists, namely $\mathcal{R}_0 > 1$. The non-delayed system is stable in nature for $\lambda = 0.002$, oscillates with decaying magnitude for $\lambda = 0.003$ (dashed line) and has a periodic solution for $\lambda = 0.0045$ (dotted lines). Occurrence of a periodic solution means that there exists a pair of imaginary roots of the characteristic equation

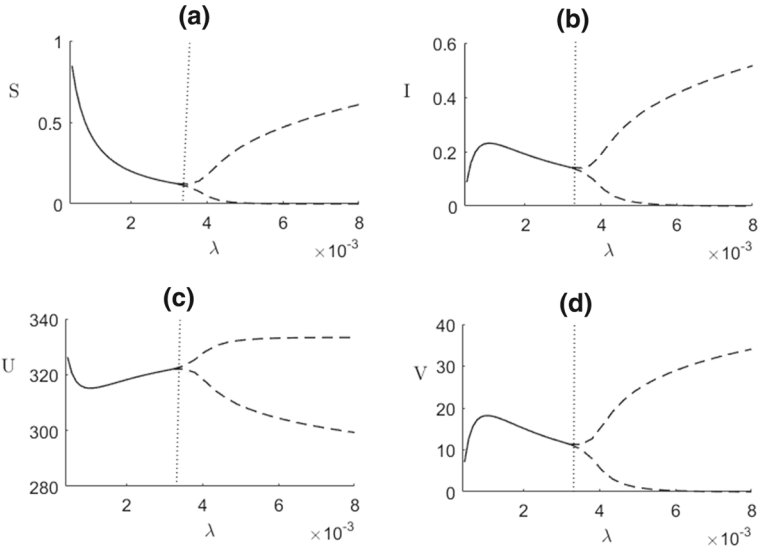

Fig. 3 Bifurcation diagram of the system without delay: steady-state values of all population plotted as a function of λ. The other parameters, taken from Table 1, are kept fixed. In this figure, we have plotted the minima and maxima of the corresponding periodic solutions around the unstable endemic steady state (dashed line); stable endemic steady points are shown using solid lines. The critical value of λ is $\lambda^* = 0.003233$

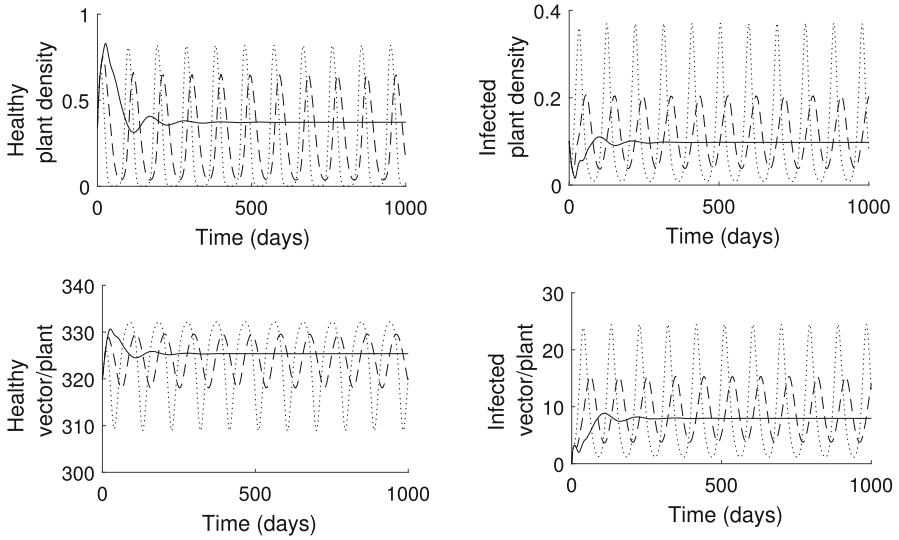

Fig. 4 Numerical solution of the system for different τ_1 when $\tau_2 = 0$ and $\lambda = 0.02$. The other parameters are taken from Fig. 4. Solid lines denote $\tau_1 = 18$, dashed lines represent $\tau_1 = 12$, and dotted line denotes $\tau_1 = 6$ days. Parameters are: $\Pi = 40$, $r = 0.3$, $k = 1$, $\beta = 0.03$, $\lambda = 0.012$, $d = 0.12$, $m = 0.1$

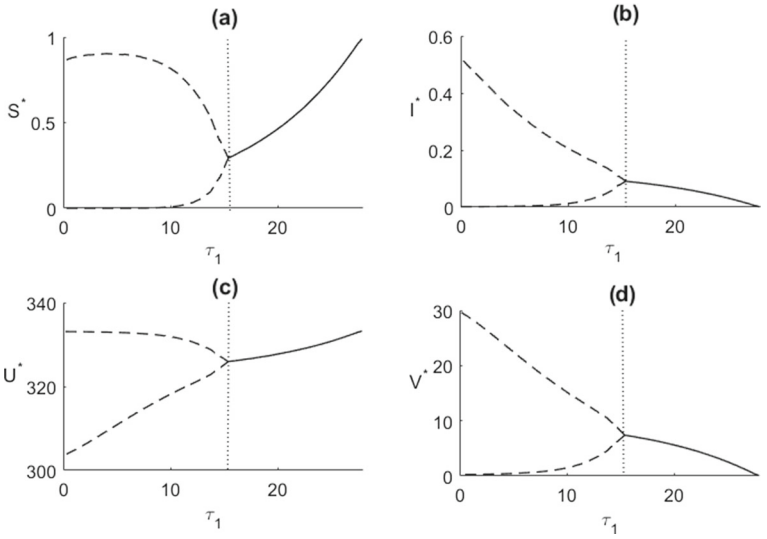

Fig. 5 Bifurcation diagram of delayed system taking τ_1 as bifurcation parameter with $\tau_2 = 0$. The other parameters are the same as in Fig. 4 except $\lambda = 0.025$. The minima and maxima of the corresponding periodic solutions (dashed lines) are plotted around the unstable endemic steady state, and the solid line indicates stable endemic steady states. Critical value of τ_1, i.e. $\tau_1^* = 15.38$

Table 1 Values of the parameters used in the numerical simulations

Parameters	Description	Values	Unit	References
r	Plants net growth rate	0.3	day^{-1}	Holt et al. (1997)
K	Carrying capacity	1	m^{-2}	Holt et al. (1997)
λ	Disease transmission rate between infected vector and healthy plant	0.025	$\text{vector}^{-1}\ \text{day}^{-1}$	Holt et al. (1997)
μ	Plants natural mortality rate	0.1	day^{-1}	Rakshit et al. (2019) and Jackson and Chen (2016)
m_1	Additional mortality of infected plants	0.01	day^{-1}	Jackson and Chen (2016)
Π	Cumulative growth rate of vector population by birth or immigration	40	day^{-1}	Jackson and Chen (2016)
β	Disease transmission rate between infected vector and healthy plant	0.03	$\text{vector}^{-1}\ \text{day}^{-1}$	Holt et al. (1997)
d	Vector mortality rate	0.12	day^{-1}	Holt et al. (1997)

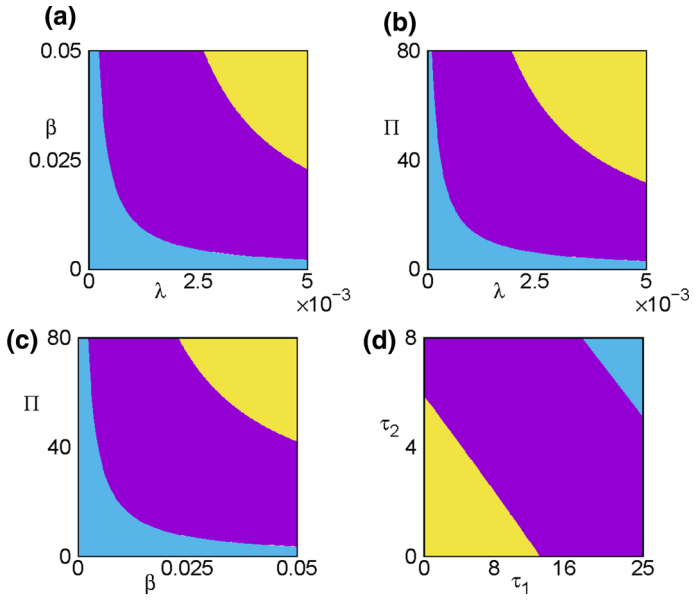

Fig. 6 Stability of steady states of the system (1): **a** in $\lambda - \beta$, **b** in $\lambda - \Pi$, **c** in $\beta - \Pi$, and **d** in $\tau_1 - \tau_2$ parameter planes. Disease-free equilibrium E_2 is stable in sky blue region, endemic equilibrium E^* is stable in violet region and unstable in yellow region. This figure is plotted for parameter values from Table 1. In plots **a** to **c**, the values of τ_1 and τ_2 are taken as $\tau_1 = \tau_2 = 0$ (Color figure online)

at the endemic equilibrium, i.e. the endemic equilibrium is not asymptotically stable. The system tends towards a stable endemic steady state for lower values of λ and oscillates around this steady state for higher λ as shown in Fig. 3.

In Fig. 4, the solution of the delayed system is plotted using the parameters from Fig. 1 except $\lambda = 0.02$. System without delay shows periodic oscillations but for $\tau_1 = 18$ and $\tau_2 = 0$, E^* is stable. We have only analysed (using Theorem 2) the stability changes of endemic equilibrium when it is stable in the absence of delay that is for $\tau_1 = \tau_2 = 0$. Numerically, we have shown that the endemic equilibrium is unstable for $\tau_1 = \tau_2 = 0$ and $\lambda = 0.02$. But for $\lambda = 0.02$, $\tau_1 = 18$ and $\tau_2 = 0$, the system settles to the endemic equilibrium. This means that large delays can stabilise the unstable system behaviour (Figs. 4 and 5).

The bifurcation diagram of the delayed system is shown in Fig. 5. Stability changes (from unstable to stable) when τ_1 crosses the critical value $\tau_1^* = 15.38$ days. Regions of stability of different equilibria are shown in Fig. 6. The region where E^* loses its stability and each population bifurcates into periodic solution at E^* are identified. Figure 6a shows that for the lower value of the infection rates, disease-free steady state E_2 is stable in the sky blue region (since $\mathcal{R}_0 < 1$). Moreover, E^* is not feasible here. In the violet region, E^* is stable but loses its stability and bifurcates into periodic solution in the yellow region. Similar description is valid for Fig. 6b and c. If the delays are large, the coexistence equilibria E^* seem stable in spite of high infection rates. Figure 6d shows the region of stability in (τ_1, τ_2)-parameter plane. Large delays seem to imply that the coexistence equilibrium E^* is stable, in spite of the high infection rate although they might not be biologically realistic. When the delay crosses the threshold

value, the endemic equilibrium becomes infeasible and the disease-free equilibrium becomes stable.

6 Discussion and Conclusion

Knowledge of the incubation period of infectious diseases is crucial for our understanding of epidemiological phenomena and for the design and implementation of appropriate prevention and control policies. Mathematical modelling techniques can help in the prediction of disease propagation in the agricultural realm.

The basic reproductive number is used to understand the dynamics of the model. In the case when $\mathcal{R}_0 < 1$, the introduction of delays is found to cause significant changes in the solution, including a longer time to approach the disease-free equilibrium point and initiation of oscillations. The delayed model is more realistic because it takes into account the time between the release of a factor, its absorption and subsequent effect. For the insect populations, a smaller change in the solution is noted. When $\mathcal{R}_0 > 1$, however, the endemic steady state becomes stable and the disease-free state becomes unstable. This is also verified through the numerical simulations. The natural death rate of the vector is chosen as the continuation parameter, since it is one value that can be modified by the use of pesticides, predators or other vector controlling means.

Both delays are present in the expression (5) of $\mathcal{R}_0$, so they affect the stability threshold of the disease-free equilibrium. In particular, from (5) a large value of either τ_1 or τ_2 is sufficient to reduce the value of $\mathcal{R}_0$ below the threshold 1. Numerically, we have also shown that large delays are able to stabilise a system oscillatory behaviour around the endemic state. For increasing values of the delay, the oscillations first damp towards the endemic equilibrium and then the system experiences a transition to the disease-free state, see Fig. 5.

The delayed model can be useful for agriculture researchers and workers. In order to be a useful model for field predictions, necessary data need to be collected by plant and insect researchers. By fitting the data to the simulation's outcomes, the model parameters can be tuned to fit a particular real-life situation.

Since the incubation delay of plants can artificially be increased using biocontrolling agents (Cook et al. 1999), genetic engineering (Liu et al. 2012; Niehaus et al. 1993), chemicals (Murphy et al. 2000), the findings in Fig. 6d could be used to drive the ecosystem toward the disease-free equilibrium. Thus, if the latency or incubation can be increased to the optimal level, for which $\mathcal{R}_0 < 1$, the disease can be eradicated from the system.

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Appendix A

We follow the method established in the paper by Heffernan et al. (2005) for calculating $\mathcal{R}_0$.

We consider the next-generation matrix G which comprises two parts, namely F and V, where

$$F = \left[\frac{\partial F_i(E_0)}{\partial x_j}\right] = \begin{bmatrix} 0 & \lambda K \\ \frac{\beta\Pi}{d} & 0 \end{bmatrix}$$
$$V = \left[\frac{\partial V_i(E_0)}{\partial x_j}\right] = \begin{bmatrix} m & 0 \\ 0 & d \end{bmatrix}$$

where F_i are the new infections, while the V_i transfers of infections from one compartment to another. E_0 is the disease-free equilibrium. We get $\mathcal{R}_0 = \frac{\Pi k\lambda\beta}{md^2}$, and it is the dominant eigenvalue of the matrix $G = FV^{-1}$.

Appendix B

We rewrite the model (1), without delay, as follows:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = rS\left(1 - \frac{S+I}{K}\right) - \lambda SV \equiv f_1(S, I, U, V), \tag{22a}$$
$$\frac{\mathrm{d}I}{\mathrm{d}t} = \lambda SV - mI \equiv f_2(S, I, U, V), \tag{22b}$$
$$\frac{\mathrm{d}U}{\mathrm{d}t} = \Pi - \beta UI - dU \equiv f_3(S, I, U, V), \tag{22c}$$
$$\frac{\mathrm{d}V}{\mathrm{d}t} = \beta UI - dV \equiv f_4(S, I, U, V), \tag{22d}$$

and consider λ as bifurcation parameter to check the change in stability of the disease-free equilibrium point $E_2(K, 0, \frac{\Pi}{d}, 0)$. Jacobian matrix for (22) evaluated at E_2 is given by

$$J(E_2) = \begin{bmatrix} -r & -r & 0 & -\lambda K \\ 0 & -m & 0 & \lambda K \\ 0 & -\frac{\beta\Pi}{d} & -d & 0 \\ 0 & \frac{\beta\Pi}{d} & 0 & -d \end{bmatrix}. \tag{23}$$

This matrix has a zero eigenvalue when $\lambda \equiv \lambda_* = \frac{d^2 m}{K\Pi\beta}$. The system undergoes a transcritical bifurcation as λ crosses λ_*. To prove the transversality condition of transcritical bifurcation, we use standard notations in Perko (1996). The system (22) can be written in compact form as

$$\frac{\mathrm{d}X}{\mathrm{d}t} = \mathbf{f}(X), \tag{24}$$

and the Jacobian matrix in (23) is denoted as $A = J(E_2)$. For $\lambda = \lambda_*$, the matrix A has a zero eigenvalue, eigenvectors corresponding to zero eigenvalue for the matrices

A and A^t are given by

$$W_1 = \begin{bmatrix} -\frac{r+m}{r} \\ 1 \\ -\frac{\Pi\beta}{d^2} \\ \frac{\Pi\beta}{d^2} \end{bmatrix},$$
$$W_2 = \begin{bmatrix} 0 \\ \frac{\Pi\beta}{md} \\ 0 \\ 1 \end{bmatrix}. \tag{25}$$

Partial differentiation of $\mathbf{f}$ with respect to λ gives

$$\mathbf{f}_\lambda = \begin{bmatrix} -SV \\ SV \\ 0 \\ 0 \end{bmatrix}, \tag{26}$$

and the Jacobian of $\mathbf{f}_\lambda$ can be calculated as follows:

$$D\mathbf{f}_\lambda = \begin{bmatrix} -V & 0 & 0 & -S \\ V & 0 & 0 & S \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}. \tag{27}$$

Hence, we can verify the transversality conditions for transcritical bifurcation as follows:

$$W_2^t\left[\mathbf{f}_\lambda(E_2, \lambda_*)\right] = 0, \tag{28a}$$
$$W_2^t\left[D\mathbf{f}_\lambda(E_2, \lambda_*)\, W_1\right] = \frac{\Pi^2\beta^2 K}{md^3} \neq 0, \tag{28b}$$
$$W_2^t\left[D^2\mathbf{f}(E_2, \lambda_*)\,(W_1, W_1)\right] = -\frac{2\beta^2\Pi}{d^2}\left(1 + \frac{\Pi d(r+m)}{rK\beta}\right) \neq 0. \tag{28c}$$

These ensure that the boundary equilibrium point E_2 loses stability through transcritical bifurcation.

Appendix C

For the values in Table 1, we get the four roots of Eq. (15) as: -0.0664, 0.0360 and $-0.0108 \pm 0.0430i$. We get one pair of complex root and the positive root as 0.0360. Here, $\omega_4 = -4.6897 \times 10^{-6} < 0$. According to Lemma 1, Eq. (15) will have at least one positive root.

Equation (16) has three roots: -0.0374 and $-0.0008 \pm 0.0073i$. We have got $\delta = 2.6996 \times 10^{-11} > 0$, $H(l) < 0$. According to Lemma 2, Eq. (15) has one positive root. Then, Eq. (11) has a pair of purely imaginary root. Finally, we have obtained $\theta_0 = 0.1897$ from (19), as well as the critical value of τ_1, namely $\tau_1^* = 15.38$, from relation (18).

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