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MODELING AND ANALYSIS OF PLANT DISEASE WITH DELAY AND LOGISTIC GROWTH OF INSECT VECTOR

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Abstract. Plants are essential for the survival of human beings. Plants can be subjected to diseases. Plant diseases are caused by pathogens such as fungi, bacteria and viruses. Most of these pathogens are transmitted by insect vectors. In this paper we formulate and analyze a delay differential equation model for plant disease by incorporating the incubation delay which is the time taken for a plant to become infected. The mathematical model is formulated by considering both the plant and the insect vector populations. The total plant population is taken as a constant and the insect vector population is taken as variable. It is assumed that the insect vector population is growing logistically in the environment. The existence and stability of equilibria of the model are discussed in detail. The basic reproduction number R_0 of the model is computed and it is observed that the disease-free equilibrium point is stable for all delay whenever $R_0 < 1$. When $R_0 > 1$ the endemic equilibrium point is stable in the absence of delay. We have estimated the length of delay which preserves the stability of endemic equilibrium point. So when the delay is less than a threshold value, the endemic equilibrium point is stable. At that threshold value we get Hopf bifurcation and system shows oscillatory behaviour. Here numerical simulation is also performed to support the analytical results.

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1. Introduction

We all depend on agricultural production for our food security and livelihoods. There are several factors which influence the crop production worldwide. One of them is plant pathogens, e.g. viruses, bacteria, fungi etc. which cause severe damage to plant production leading to economic loss and depressions in farmers. Mathematical modeling is an useful tool to understand the disease dynamics in plant population. Although there are plenty of mathematical models to predict the dynamics of human diseases, there are very few models to understand the disease dynamics in plant population. We have plant-specific diseases, so it is better to formulate suitable mathematical models by keeping a particular crop/plant in mind. Plants are also subjected to pesticides and proper use of pesticides is also necessary to have less damage to the crop productions. Mathematical modeling can help in formulating integrated pest management tools. In [1], authors formulated and analyzed a plant-insect herbivore-pesticide model by considering broccoli patches surrounded by different types of ground and exposed to different levels of insecticide spray. They fitted their model with experimental data and concluded that the interaction between pesticide sprays and weedy margins plays an important role in integrated pest management. The dynamics of plant disease model with continuous and impulsive control strategies is discussed in [2]. In [3], authors have considered a mathematical model for vectored plant disease by considering variable virus density. They also extend their proposed model by assuming that there are two competing viruses. They obtained the conditions to determine displacement and coexistence of the viruses. A non-autonomous plant disease model by considering latent period and periodicity in the model parameters is analyzed in [4]. Analysis of plant disease model in periodic environment and pulse roguing is demonstrated in [5]. In [6], authors have formulated and analyzed a mathematical model for mosaic disease in Jatropha curcas plantation by incorporating roguing and delay. Recently a plant disease model with delay is formulated and analyzed by Jackson and Chen-Charpentier [7] where they assumed that insect vectors can transit the virus from one plant to another. So in place of virus population, they considered insect vector population while formulating their mathematical model. They considered incubation delay in their model and compared the result of model with delay with the model without delay using numerical simulation. Later in [8], they extended their work by including predator population which can eat insect vectors. Here authors presented the theoretical implication of biological control of plant disease. In our present work we extend the model by [7, 8]. In [7], authors assumed that the insect vector population follows constant recruitment and death type demography. However for the analysis purpose they assumed that the total vector population is a constant equal to the upper limit of it. This assumption is valid if the vector population reaches to its saturation level very fast compared to the plant population. But this may not be a good assumption as it may depend on the species of plants and insect vectors. In [8], authors again considered the same constant recruitment and death type demography for vector population. Here they kept vector population as variable and discussed the existence and stability of disease-free equilibrium. Here the existence and stability of endemic equilibrium are demonstrated only through numerical simulation. In our proposed model we have assumed that the total insect population is variable and growing logistically in the environment. Here the density dependent birth and death rates of insect population when they are exposed to infection are following a specific demography as discussed in [9]. This type of demography is little hard to analyze but is more realistic. Here one can adjust the convex combination constant, the parameter which determines the level of density dependence of birth and death rates of the population under consideration. The carrying capacity of the insect vector population can also influence the disease dynamics [10].

This paper is organized as follows. In section 2, we formulate our mathematical model. In Section 3, we find the equilibria of the model and compute the basic reproduction number. In Section 4, we discuss the stability of different equilibria of the model in presence and absence of delay. Here we also discuss the existence of Hopf-bifurcation and obtain the critical value of the delay τ beyond which system shows oscillatory behaviour. In Section 5, we perform numerical simulation to support our analytical findings. Finally, we conclude our results in Section 6.

2. The Model

Here the total plant population ($M(t)$) is divided into three disjoint classes namely, susceptible plants S(t), infected plants $I(t)$ and recovered plants $R(t)$. Similarly, the total insect vector ($N(t)$) is divided into two classes namely, susceptible insect vectors $X(t)$ and infected insect vectors

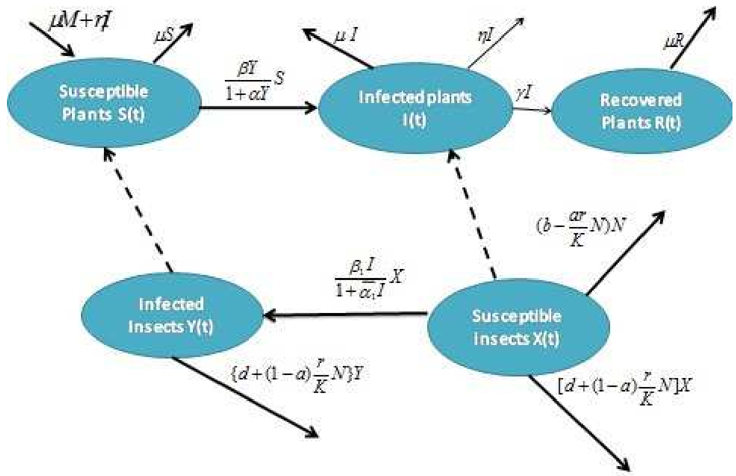

FIGURE 1. Flow diagram of plant disease dynamics

$Y(t)$. As infected vectors do not feel sick or die due to infection, so recovered insect class is not incorporated in this model. The virus travel from one plant to another through insect vectors. Here the total plant population is assumed to be a constant. When an infected plant dies due to disease, it is replaced by new plant. The insect population is assumed to grow logistically which is more realistic. When an infected vector feeds/walks on susceptible plant, it transmits virus/bacteria to the plant. Similarly, when a susceptible insect feeds/walks on infected plant, it acquires bacteria/virus. Keeping in view of these facts we formulate our model as follows:

$$
\begin{aligned}
\frac{dS}{dt} &= \mu(M-S)+\eta I-\frac{\beta YS}{1+\alpha Y}, \\
\frac{dI}{dt} &= \frac{\beta YS}{1+\alpha Y}-(\gamma+\mu+\eta)I \\
\frac{dR}{dt} &= \gamma I-\mu R \\
\frac{dX}{dt} &= \left(b-\frac{ar}{K}N\right)N-\left(d+(1-a)\frac{r}{K}N\right)X-\left(\frac{\beta_1 I}{1+\alpha_1 I}\right)X \\
\frac{dY}{dt} &= \left(\frac{\beta_1 I}{1+\alpha_1 I}\right)X-\left(d+(1-a)\frac{r}{K}N\right)Y.
\end{aligned} \tag{2.1}
$$

$$S(0)>0,\ I(0)\geq 0,\ R(0)\geq 0,\ X(0)>0, Y(0)\geq 0.$$

The description of parameters are given in Table 1 and the schematic flow diagram of our model is shown in Figure 1. As $S+I+R=M$ is a constant, so considering S and I is enough to understand the dynamics of plant population. Also we have $X+Y=N$, and we consider

TABLE 1. Description of parameters and their values

Parameter	Description	Value	Reference
M	Total plant host population	200	[7]
K	Carrying capacity of the insect vector population	100	[7]
β	Infection rate of plants due to vectors	0.003	Assumed
β_1	Infection rate of vectors due to plants	0.003	Assumed
α	Saturation constant of plants due to vectors	0.01	[7]
α_1	Saturation constant of vectors due to plants	0.01	Assumed
μ	Natural death rate of plants	0.01	[7, 8]
γ	Recovery rate of plants	0.01	[7, 8]
η	Death rate of infected plants due to the disease	0.01	Assumed
b	Natural birth rate constant for insect population	0.2	Assumed
d	Natural death rate constant for insect population	0.1	[7]
$b-d=r$	Growth rate constant for insects population	0.1	Assumed
$0\leq a\leq 1$	Convex combination constant	0.8	Assumed

the differential equations corresponding to Y and N in place of X and Y as it makes analysis simpler. Additionally, we incorporate incubation delay (τ) in the plants becoming infected. So we modify our model (2.1) and write the delay differential equation model as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \mu(M-S)+\eta I-\frac{\beta Y(t-\tau)S(t-\tau)}{1+\alpha Y(t-\tau)},\\
\frac{dI}{dt} &= \frac{\beta Y(t-\tau)S(t-\tau)}{1+\alpha Y(t-\tau)}-(\gamma+\mu+\eta)I\\
\frac{dN}{dt} &= rN\left(1-\frac{N}{K}\right)\\
\frac{dY}{dt} &= (\frac{\beta_1 I}{1+\alpha_1 I})(N-Y)-(d+(1-a)\frac{r}{K}N)Y.
\end{aligned} \tag{2.2}$$

The initial conditions for the system (2.2) are given by $S(\theta)=\phi_1(\theta),\ I(\theta)=\phi_2(\theta),\ N(\theta)=\phi_3(\theta),\ Y(\theta)=\phi_4(\theta),\ \phi_1(\theta)\geq 0,\ \phi_2(\theta)\geq 0,\ \phi_3(\theta)\geq 0,\ \phi_4(\theta)\geq 0$, where $\phi_1(\theta),\ \phi_2(\theta),\ \phi_3(\theta),\ \phi_4(\theta)\in\mathscr{C}([-\tau,0],\mathscr{R}^4_+)$, the Banach space of continuous functions mapping the interval $[-\tau,0]$ into $\mathscr{R}^4_+$ where $\mathscr{R}^4_+=\{(x_1,x_2,x_3,x_4): x_i\geq 0,\ i=1,2,3,4\}$.

3. Existence of Equilibria and the Basic Reproduction Number

The system (2.2) has three equilibria, namely

i) the boundary equilibrium $E_0 = (M,0,0,0)$,

ii) the disease-free equilibrium $E_1 = (M,0,K,0)$ and

iii) the endemic equilibrium $E_2 = (S^*, I^*, N^*, Y^*)$ where,

$$\begin{aligned}
S^* &= \frac{(\gamma+\mu+\eta)\delta\left[\delta+\frac{\eta}{\mu}\delta+\beta_1 M(1+\alpha K)+\delta\alpha_1 M\right]}{\left[\beta\beta_1 K\delta+\frac{\beta\beta_1 K\delta\eta}{\mu}+(\gamma+\mu+\eta)\delta\{\beta_1+\delta\alpha_1+\alpha_1\beta_1 K\}\right]},\\
Y^* &= \frac{(M-S^*)\beta_1 K}{\left(\delta+\frac{\eta}{\mu}\delta\right)+\beta_1(M-S^*)+\alpha_1\delta(M-S^*)},\\
I^* &= \frac{\beta Y^* S^*}{(\gamma+\mu+\eta)(1+\alpha Y^*)},\ N^* = K,
\end{aligned}$$

and $\delta = d+(1-a)r$. We find the basic reproduction number R_0 by following the next generation matrix methods as discussed in [11, 12]. We consider only the infected compartment I and Y and follow the same notation as in [11, 12]. The matrix $\mathscr{F}$ and $\mathscr{V}$ for our model is given by:

$$\mathscr{F} = \begin{pmatrix} \beta YS \\ \frac{\beta_1 I(N-Y)}{1+\alpha_1 I}) \end{pmatrix} \text{ and } \mathscr{V} = \begin{pmatrix} (\gamma+\mu+\eta)I \\ \left\{d+(1-a)\frac{r}{K}\right\}Y \end{pmatrix}$$

. F= Jacobian of $\mathscr{F}$ at $E_1 = \begin{pmatrix} 0 & \beta M \\ \beta_1 K & 0 \end{pmatrix}$

and V=Jacobian of $\mathscr{V}$ at $E_1 = \begin{pmatrix} \gamma+\mu+\eta & 0 \\ 0 & \{d+(1-a)r\} \end{pmatrix}$,

and it follows that

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\beta M}{d+(1-a)r} \\ \frac{\beta_1 K}{\gamma+\mu+\eta} & 0 \end{pmatrix}.$$

The largest eigenvalue of FV^{-1} is called the basic reproduction number R_0 and is obtained as follows:

$$R_0 = \sqrt{\frac{\beta\beta_1 MK}{\{d+(1-a)r\}(\gamma+\mu+\eta)}}.$$

4. Stability Analysis

Following [13], the Jacobian matrix of the system (2.2) is given by

$$J = \begin{pmatrix} -\mu - \frac{\beta Y_\tau e^{-\lambda\tau}}{1+\alpha Y_\tau} - \lambda & \eta & 0 & -\frac{\beta S_\tau e^{-\lambda\tau}}{(1+\alpha Y_\tau)^2} \\ \frac{\beta Y_\tau e^{-\lambda\tau}}{1+\alpha Y_\tau} & -(\gamma+\mu+\eta+\lambda) & 0 & \frac{\beta S_\tau e^{-\lambda\tau}}{(1+\alpha Y_\tau)^2} \\ 0 & 0 & r - \frac{2rN}{K} - \lambda & 0 \\ 0 & \frac{(N-Y)\beta_1}{(1+\alpha_1 I)^2} & m_{43} & m_{44} - \lambda \end{pmatrix}$$

where

$$m_{43} = \frac{\beta_1 I}{1+\alpha_1 I} - (1-a)\frac{r}{K}Y,$$
$$m_{44} = \frac{-\beta_1 I}{1+\alpha_1 I} - \{d + (1-a)\frac{r}{K}N\}.$$

The characteristic equation is given by [13]

$$det(J) = 0.$$

Theorem 4.1. *The boundary equilibrium $E_0(M,0,0,0)$ is always unstable.*

Proof. The Jacobian matrix evaluated at $E_0(M,0,0,0)$ is given by

$$\begin{pmatrix} -\mu-\lambda & \eta & 0 & -\beta M e^{-\lambda\tau} \\ 0 & -(\gamma+\mu+\eta+\lambda) & 0 & \beta M e^{-\lambda\tau} \\ 0 & 0 & r-\lambda & 0 \\ 0 & 0 & 0 & -d-\lambda \end{pmatrix}.$$

The eigenvalues of the above matrix are r, $-\mu$,$-(\gamma+\mu+\eta)$ and $-d$. As one of the eigenvalues is positive so the equilibrium E_0 is unstable.

Theorem 4.2. *The disease-free equilibrium $E_1 = (M,0,K,0)$ is locally asymptotically stable for $R_0 < 1$.*

Proof. The Jacobian matrix evaluated at $E_1(M,0,K,0)$ is given by

$$\begin{pmatrix} -\mu-\lambda & \eta & 0 & -\beta M e^{-\lambda\tau} \\ 0 & -(\gamma+\mu+\eta+\lambda) & 0 & \beta M e^{-\lambda\tau} \\ 0 & 0 & -r-\lambda & 0 \\ 0 & K\beta_1 & 0 & -\{d+(1-a)r\}-\lambda \end{pmatrix}$$

Clearly, two eigenvalues of the above matrix are $-\mu$ and $-r$ and the remaining eigenvalues are given by the roots of the following non-linear equation:

$$\lambda^2+\lambda\left((\mu+\gamma+\eta)+\{d+(1-a)r\}\right)+\{d+(1-a)r\}(\mu+\gamma+\eta)-\beta\beta_1 MKe^{-\lambda\tau}=0. \quad (4.1)$$

When $\tau=0$, last equation becomes a quadratic equation. The coefficient of λ is positive and hence the condition for roots to have negative real parts is given by

$$\frac{\beta\beta_1 MK}{\{d+(1-a)r\}(\mu+\gamma+\eta)}<1.$$

This corresponds to $R_0^2<1$, i.e. $R_0<1$. Hence the equilibrium E_1 is locally asymptotically stable for $R_0<1$ in the absence of delay.
When $\tau\neq 0$, let us assume $\lambda=i\omega$. Substituting $\lambda=i\omega$ in the equation (4.1) and equating the real and imaginary parts we get the following two equations:

$$-\omega^2+(\gamma+\mu+\eta)\{d+(1-a)r\}=\beta\beta_1 MK\cos\omega\tau,$$

$$\omega\{(\gamma+\mu+\eta)+d+(1-a)r\}=-\beta\beta_1 MK\sin\omega\tau.$$

Now squaring and adding the last two equations, we get the following biquadratic equation:

$$\omega^4+[(\gamma+\mu+\eta)^2+\{d+(1-a)r\}^2]\omega^2+(\gamma+\mu+\eta)^2\{d+(1-a)r\}^2-(\beta\beta_1 MK)^2=0.$$

Assuming $\omega^2=u$, we have

$$u^2+[(\gamma+\mu+\eta)^2+\{d+(1-a)r\}^2]u+(\gamma+\mu+\eta)^2\{d+(1-a)r\}^2(1-R_0^2)=0.$$

Clearly, for $R_0<1$, the last quadratic is not having any positive root and this implies that real $\omega>0$ does not exist. Hence the equation (4.1) will not have purely imaginary roots for $R_0<1$. This implies that the equation (4.1) has all the roots with negative real parts. Thus the equilibrium point $E_1(M,0,K,0)$ is locally asymptotically stable for all delay whenever $R_0<1$.
Theorem 4.3. *The endemic equilibrium $E_2=(S^*,I^*,N^*,Y^*)$ is locally asymptotically stable whenever it exists in the absence of delay.*

Proof. The Jacobian of the system (2.2) at the endemic equilibrium $E_2(S^*, I^*, N^*, Y^*)$ is,

$$\begin{pmatrix} -\mu-\frac{\beta Y^* e^{-\lambda\tau}}{1+\alpha Y^*}-\lambda & \eta & 0 & -\frac{\beta S^* e^{-\lambda\tau}}{(1+\alpha Y^*)^2} \\ \frac{\beta Y^* e^{-\lambda\tau}}{1+\alpha Y^*} & -(\gamma+\mu+\eta+\lambda) & 0 & \frac{\beta S^* e^{-\lambda\tau}}{(1+\alpha Y^*)^2} \\ 0 & 0 & -(r+\lambda) & 0 \\ 0 & \frac{(K-Y^*)\beta_1}{(1+\alpha_1 I^*)^2} & m_{43}^* & m_{44}^*-\lambda \end{pmatrix},$$

where

$$m_{43}^* = \frac{\beta_1 I^*}{1+\alpha_1 I^*} - (1-a)\frac{r}{K}Y^*$$

$$m_{44}^* = -\left(\frac{\beta_1 I^*}{1+\alpha_1 I^*} + \{d+(1-a)r\}\right)$$

The characteristic polynomial of the above matrix is given by

$$(\lambda+r)\left[\lambda^3 + B_1\lambda^2 + B_2\lambda + B_3 + \mathrm{e}^{-\lambda\tau}(\mathrm{C}_1\lambda^2 + \mathrm{C}_2\lambda + \mathrm{C}_3)\right] = 0, \tag{4.2}$$

where

$$\begin{aligned}
B_1 &= \left(\gamma + 2\mu + \eta + \{d+(1-a)r\} + \frac{\beta_1 I^*}{1+\alpha_1 I^*}\right) > 0 \\
C_1 &= \frac{\beta Y^*}{1+\alpha Y^*} > 0 \\
B_2 &= \left(\frac{\beta_1 I^*}{1+\alpha_1 I^*} + \{d+(1-a)r\}\right)(\gamma+2\mu+\eta) + \mu(\gamma+\mu+\eta) > 0 \\
C_2 &= \left(\frac{\beta_1 I^*}{1+\alpha_1 I^*} + \{d+(1-a)r\}\right)\frac{\beta Y^*}{1+\alpha Y^*} + (\gamma+\mu)\frac{\beta Y^*}{1+\alpha Y^*} - \frac{\beta\beta_1 S^*(K-Y^*)}{(1+\alpha Y^*)^2(1+\alpha_1 I^*)^2} \\
B_3 &= \left(\frac{\beta_1 I^*}{1+\alpha_1 I^*} + \{d+(1-a)r\}\right)\mu(\mu+\gamma+\eta) > 0 \\
C_3 &= \left(\frac{\beta_1 I^*}{1+\alpha_1 I^*} + \{d+(1-a)r\}\right)\left(\frac{\beta Y^*}{1+\alpha Y^*}\right)(\gamma+\mu) - \frac{\beta\beta_1\mu S^*(K-Y^*)}{(1+\alpha Y^*)^2(1+\alpha_1 I^*)^2}
\end{aligned}$$

When $\tau = 0$, the characteristic equation reduces to

$$(\lambda+r)\left[\lambda^3 + (B_1+C_1)\lambda^2 + (B_2+C_2)\lambda + (B_3+C_3)\right] = 0.$$

Clearly, one eigenvalue is $-r$. Using Routh-Hurwitz criterion the roots of the cubic equation will have negative real parts if

$$B_3 + C_3 > 0 \text{ and} (B_1+C_1)(B_2+C_2) - (B_3+C_3) > 0. \tag{4.3}$$

Using the equations corresponding to endemic equilibrium point, it is easy to verify that the condition stated in (4.3) hold. Hence the endemic equilibrium point E_2 is locally asymptotically stable when the delay $\tau = 0$.

Now we consider the case when the delay $\tau > 0$. We know that one eigenvalue is $-r$. For the stability change the characteristic equation should have a pair of purely imaginary root. To check whether the characteristic equation (4.2) has purely imaginary roots or not, we put $\lambda = i\omega$ in the remaining cubic equation and separate real and imaginary parts. This gives the following equations:

$$\begin{aligned} (B_3 - B_1\omega^2) &= (C_1\omega^2 - C_3)\cos\omega\tau - C_2\omega\sin\omega\tau \\ (\omega^3 - B_2\omega) &= (C_1\omega^2 - C_3)\sin\omega\tau + C_2\omega\cos\omega\tau \end{aligned} \quad (4.4)$$

Squaring and adding the above equations, and writing $\omega^2 = u$ we get the following cubic equation in u,

$$F(u) = u^3 + d_1u^2 + d_2u + d_3 = 0, \quad (4.5)$$

where

$$\begin{aligned} d_1 &= B_1^2 - 2B_2 - C_1^2 \\ d_2 &= B_2^2 - 2B_1B_3 - C_2^2 + 2C_1C_3 \\ d_3 &= B_3^2 - C_3^2 \end{aligned} \quad (4.6)$$

It is easy to observe that if the coefficients $(d_i's)$ in $F(u)$ satisfy the conditions of Routh-Hurwitz criterion, then the equation (4.5) will not have any positive real root, i.e. we will not get any positive value of ω, which satisfy the transcendental equations stated in (4.4). In this case the result is summarized in the following theorem.

Theorem 4.4. *If the coefficients d_1, d_2, d_3 in $F(u)$ satisfy the conditions of Routh-Hurwitz criterion, then the interior equilibrium $E_2(S^*, I^*, N^*, Y^*)$ of the system (2.2), if exists, is asymptotically stable for all delay $\tau > 0$, provided it is stable in absence of delay.*

Theorem 4.5. *If the coefficients d_1, d_2, d_3 in $F(u)$ satisfy the conditions of Routh-Hurwitz criterion, and the endemic equilibrium point $E_2(S^*, I^*, N^*, Y^*)$ is unstable at $\tau = 0$, then it will remain unstable for all $\tau \geq 0$.*

We have the following results for the roots of a cubic polynomial [14].

Lemma 4.1. *For the polynomial equation* $u^3+d_1u^2+d_2u+d_3=0$,

(i) If $d_3<0$, *the equation has at least one positive root;*

(ii) If $d_3\geq 0$ *and* $\triangle=d_1^2-3d_2\leq 0$, *the equation has no positive root;*

(iii) If $d_3\geq 0$ *and* $\triangle=d_1^2-3d_2>0$, *the equation has positive roots if and only if* $u_1^*=\dfrac{-d_1+\sqrt{\triangle}}{3}>0$ *and* $F(u_1^*)\leq 0$, *where* $F(u)=u^3+d_1u^2+d_2u+d_3$.

So using the last lemma if the equation $F(u)=0$ has a positive zero, then the characteristic equation (4.2) has a pair of purely imaginary roots $\pm i\omega$ (say). In this case eliminating $\sin\omega\tau$ from the equations in (4.4), we get

$$\tau=\tau_n^* \text{ (say) } =\frac{1}{\omega}cos^{-1}\left[\frac{(\omega^3-B_2\omega)C_2\omega+(B_1\omega^2-B_3)(C_3-C_1\omega^2)}{(C_2^2\omega^2+(C_3-C_1\omega^2)^2}\right]+\frac{2n\pi}{\omega},\ (n=0,1,2\ldots).$$

This result is summarized in the following theorem.

Theorem 4.6. *The endemic equilibrium point* E_2 *of the system (2.2) is conditionally stable if and only if all the roots of the characteristic equation (4.2) have negative real parts at* $\tau=0$ *and there exist some positive value of the delay* τ *such that the characteristic equation (4.2) has a pair of purely imaginary roots* $\pm i\omega_0$ *(say). The system will undergo a stability change for an infinite number of values of* τ *say* τ_n^*, *where*

$$\tau_n^*=\frac{1}{\omega_0}cos^{-1}\left[\frac{(\omega_0^3-B_2\omega_0)C_2\omega_0+(B_1\omega_0^2-B_3)(C_3-C_1\omega_0^2)}{(C_2^2\omega_0^2+(C_3-C_1\omega_0^2)^2}\right]+\frac{2n\pi}{\omega_0},\ (n=0,1,2\ldots).$$

Now in order to verify the transversality condition for the existence of Hopf-bifurcation we need to prove that $\frac{d\xi}{d\tau}\neq 0$ at $\xi=0$, where $\lambda(\tau)=\xi(\tau)+i\omega(\tau)$ and λ is the root of the following cubic equation which is coming from the characteristic equation (4.2):

$$\lambda^3+B_1\lambda^2+B_2\lambda+B_3+\mathrm{e}^{-\lambda\tau}(\mathrm{C}_1\lambda^2+\mathrm{C}_2\lambda+\mathrm{C}_3)=0$$

Differentiating it with respect to τ we get,

$$(3\lambda^2+2B_1\lambda+B_2)\frac{d\lambda}{d\tau}+e^{-\lambda\tau}(2C_1\lambda+2C_2)\frac{d\lambda}{d\tau}+(C_1\lambda^2+C_2\lambda+C_3)(-\tau e^{-\lambda\tau}\frac{d\lambda}{d\tau}-\lambda e^{-\lambda\tau})=0$$

$$\{(3\lambda^2+2B_1\lambda+B_2)+e^{-\lambda\tau}(2C_1\lambda+C_2)-\tau e^{-\lambda\tau}(C_1\lambda^2+C_2\lambda+C_3)\}\frac{d\lambda}{d\tau}=\lambda e^{-\lambda\tau}(C_1\lambda^2+C_2\lambda+C_3)$$

which implies,

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{(3\lambda^2+2B_1\lambda+B_2)e^{\lambda\tau}}{\lambda(C_1\lambda^2+C_2\lambda+C_3)} + \frac{(2C_1\lambda+C_2)}{\lambda(C_1\lambda^2+C_2\lambda+C_3)} - \frac{\tau}{\lambda}.$$

Since $\lambda(\tau_0) = i\omega_0$ is a simple root of the characteristic equation (4.2), we can evaluate the expressions involved in the above derivative at $\tau = \tau_0$ as follows:

$$\begin{aligned} \left\{(3\lambda^2+2B_1\lambda+B_2)e^{\lambda\tau}\right\}|_{\tau=\tau_0} &= \delta_1 + i\delta_2, \\ \left\{\lambda(C_1\lambda^2+C_2\lambda+C_3)\right\}|_{\tau=\tau_0} &= \delta_3 + i\delta_4, \\ \{2C_1\lambda+C_2\}|_{\tau=\tau_0} &= \delta_5 + i\delta_6, \end{aligned} \tag{4.7}$$

where

$$\begin{aligned} \delta_1 &= (B_2-3\omega_0^2)\cos\omega_0\tau_0 - 2B_1\omega_0\sin\omega_0\tau_0 \\ \delta_2 &= 2B_1\omega_0\cos\omega_0\tau_0 + (B_2-3\omega_0^2)\sin\omega_0\tau_0 \\ \delta_3 &= -C_2\omega_0^2, \\ \delta_4 &= \omega_0(C_2-C_1\omega_0^2), \\ \delta_5 &= C_2, \\ \delta_6 &= 2C_1\omega_0. \end{aligned}$$

Now

$$\left(\frac{d\lambda}{d\tau}\right)^{-1}|_{\tau=\tau_0} = \left(\frac{d}{d\tau}Re\lambda(\tau_0)\right)^{-1} = \frac{\delta_1\delta_3+\delta_2\delta_4+\delta_5\delta_3+\delta_4\delta_6}{\delta_3^2+\delta_4^2}.$$

Using the equations in (4.4), we can rewrite above expression as follows:

$$\begin{aligned} \left(\frac{d\lambda}{d\tau}\right)^{-1}|_{\tau=\tau_0} &= \frac{\omega_0^2\left[3\omega_0^4+2(B_1^2-2B_2-C_1^2)\omega_0^2+(B_2^2-2B_1B_3+2C_1C_3-C_2^2)\right]}{\delta_3^2+\delta_4^2} \\ &= \frac{\omega_0^2}{\delta_3^2+\delta_4^2}(3u^2+2d_1u+d_2)|_{u=\omega_0^2} = \frac{\omega_0^2}{\delta_3^2+\delta_4^2}F'(u)|_{u=\omega_0^2} \end{aligned}$$

Therefore

$$sign\left[\left(\frac{d}{d\tau}Re\lambda(\tau_0)\right)\right] = sign\left[\left(\frac{d}{d\tau}Re\lambda(\tau_0)\right)^{-1}\right] = sign\left[\frac{\omega_0^2}{\delta_3^2+\delta_4^2}F'(u)|_{u=\omega_0^2}\right].$$

As $\delta_3^2+\delta_4^2 > 0$, $\omega_0^2 > 0$ and $F'(u)|_{u=\omega_0^2} \neq 0$, the $sign\left[\left(\frac{d}{d\tau}Re\lambda(\tau_0)\right)\right]$ will be determined by the $sign\left[F'(u)|_{u=\omega_0^2}\right]$.

We already have $Re(\lambda(\tau)) = \xi(\tau)$ and $\xi(\tau_0) = 0$. Therefore if $sign\left[F'(u)|_{u=\omega_0^2}\right] < 0$, then there exists a $\zeta > 0$ such that $\xi(\tau)$ is decreasing in $(\tau_0-\zeta, \tau_0)$ and $\xi(\tau) = 0$ at $\tau = \tau_0$. Hence for all

$\tau \in (\tau_0 - \zeta, \tau_0)$, $\xi(\tau) > 0$, which contradicts the fact that roots of the characteristic equation (4.2) have negative real parts for all $\tau \in [0, \tau_0]$ and $\tau = \tau_0$ is the minimum value of delay τ for which (4.2) will have purely imaginary roots. Hence *sign* $\left[F'(u)|_{u=\omega_0^2}\right] > 0$ which shows that there exists at least one $\lambda(\tau)$ with $\xi(\tau) > 0$ for $\tau > \tau_0$. Thus the transversality condition is satisfied. Hence we have the following Theorem.

Theorem 4.7. *The endemic equilibrium point E_2 is locally asymptotically stable if $\tau \in [0, \tau_0]$, and it is unstable for $\tau > \tau_0$. When $\tau = \tau_0$ a Hopf bifurcation occurs, i.e., a family of periodic solutions bifurcate from the infected steady state E_2 as τ passes through τ_0.*

5. Simulation

Here we simulate the system (2.2) for a set of parameters stated in Table 1. For this set of parameters the basic reproduction number $R_0 = 7.07$. The disease-free equilibrium point $E_1(200, 0, 100, 0)$ is unstable and the endemic equilibrium $E_2(25, 87.5, 100, 53.8)$ is stable in absence of delay. For this set of parameters the critical value of delay is obtained as $\tau_0 = 18.2027$. So the system is stable for all delay $\tau \in [0, 18.2027)$ and at $\tau = 18.2027$ it exhibits Hopf-bifurcation. This fact is demonstrated in Figures 2-5, where plots of susceptible plant, infected plant, susceptible insect and infected insect are plotted against time for different values of delay parameter τ. From these figure it is clear that for any $\tau > 18.2027$ the system will exhibit oscillations. Here oscillation is demonstrated using $\tau = 18.5$. The 2-d phase plot for susceptible plant verses infected plant is shown in Figure 6 for $\tau = 18.5$ where system is exhibiting a limit cycle. Next we repeat our simulation for different value of the carrying capacity K of insect population. It is found that with the increase in the carrying capacity K the critical value of the delay τ_0 decreases. This fact is demonstrated in Figures 7-10, where the carrying capacity K is changed to 150 and all other parameters are as mentioned in Table 1. For this set of parameters we get $R_0 = 8.66$ and the critical value of delay $\tau_0 = 13.434$. Hence in this situation system exhibits Hopf-bifurcation even at lesser value of delay. This implies that carrying capacity of the insect population also plays an important role in determining the dynamics of plant-insect populations.

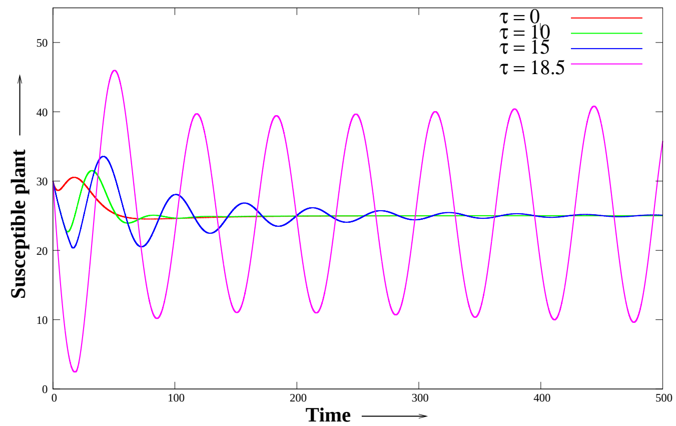

FIGURE 2. Variation of susceptible plants with time for different values of delay τ.

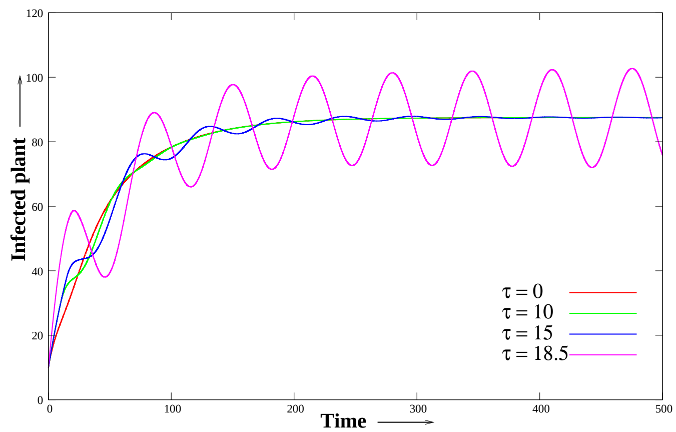

FIGURE 3. Variation of infected plants with time for different values of delay τ.

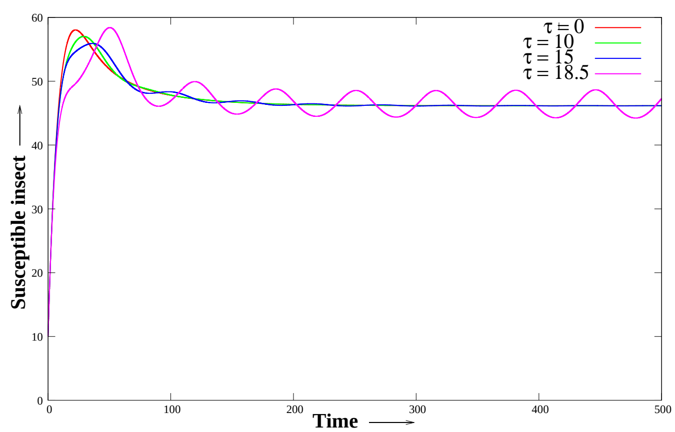

FIGURE 4. Variation of susceptible insects with time for different values of delay τ.

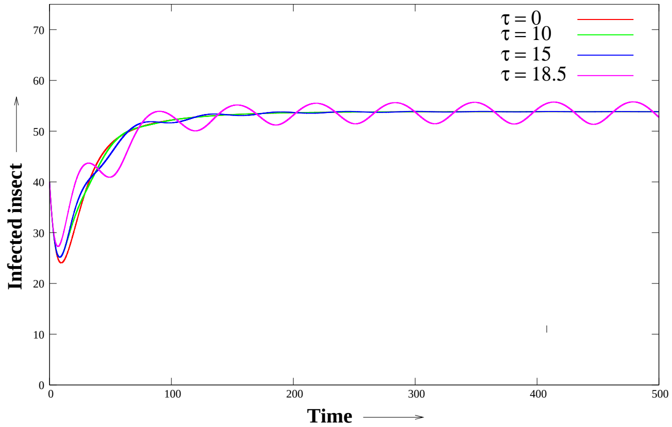

FIGURE 5. Variation of infected insects with time for different values of delay τ.

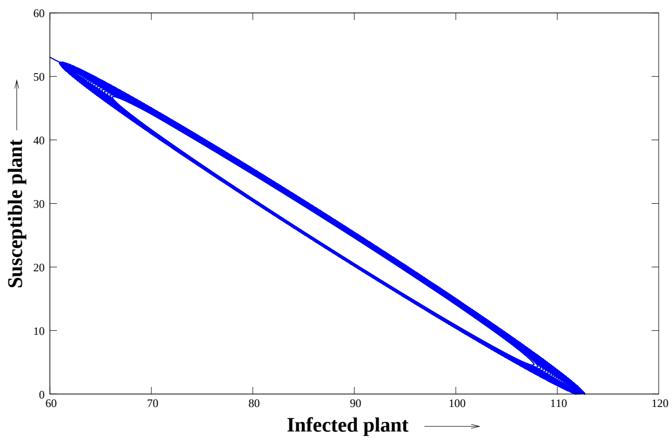

FIGURE 6. 2-d plot of susceptible plant verses infected plant for $\tau = 18.5$.

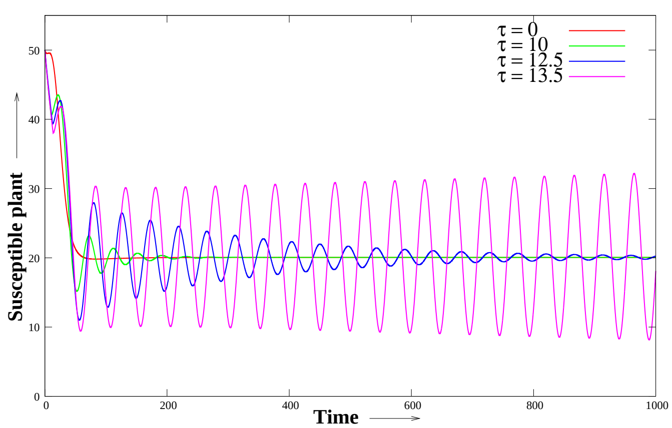

FIGURE 7. Variation of susceptible plants with time for different values of delay τ when the carrying capacity $K = 150$.

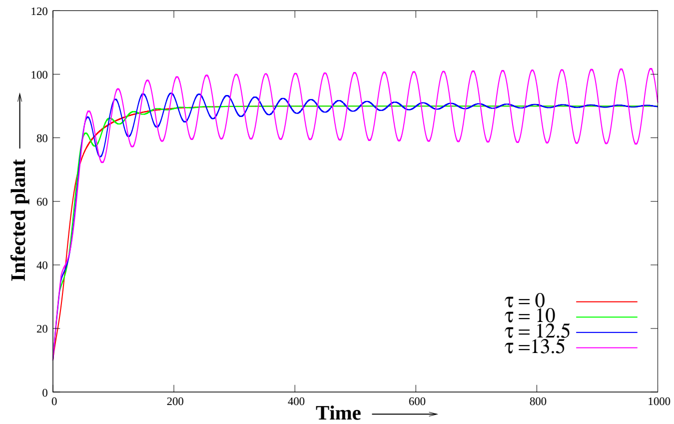

FIGURE 8. Variation of infected plants with time for different values of delay τ when the carrying capacity $K = 150$.

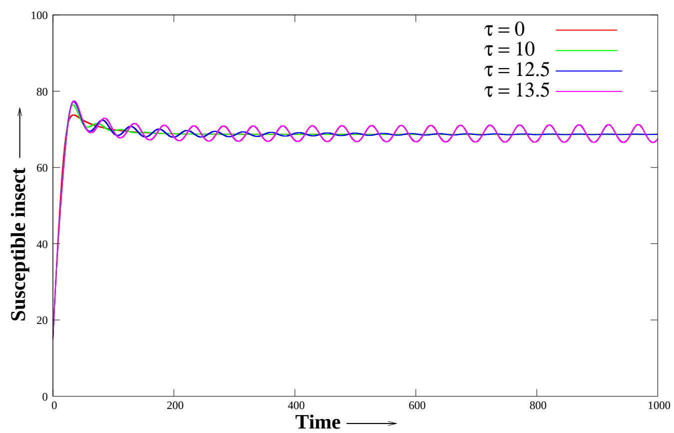

FIGURE 9. Variation of susceptible insects with time for different values of delay τ when the carrying capacity $K = 150$.

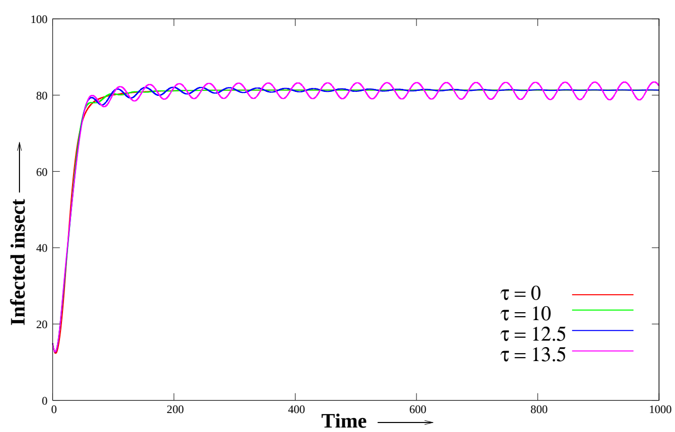

FIGURE 10. Variation of infected insects with time for different values of delay τ when the carrying capacity $K = 150$.

6. Conclusion

Here we propose and analyze a delay differential equation model for plant disease where virus is propagated by insect vector. It is assumed that insect vector population is variable and growing logistically in the environment. The proposed model has three equilibria, namely, the boundary equilibrium point (insect-free), disease-free equilibrium point and the endemic equilibrium point. The basic reproduction number is computed and the stability of different equilibria is discussed in detail. It is observed that the first equilibrium point is always unstable, the disease-free equilibrium is locally asymptotically stable for all delays whenever the basic reproduction number $R_0 < 1$, the endemic equilibrium point is locally asymptotically stable till delay is less than some critical value and is unstable beyond this critical value. At this critical value of delay, system undergoes through Hopf-bifurcation about the endemic equilibrium point. Identification of this critical value of delay is very important for practical problem as any control efforts to eliminate the infection may not work if system is undergoing through stable oscillations. It is found that the carrying capacity of the insect population also plays an important role as increase in this leads to decrease in the critical value of delay beyond which system exhibits oscillations. Numerical simulation is also performed to support our analytical findings. Present study may help in planning suitable control strategy to control the spread of disease in plant population. Here we have not considered biological control by introducing predator population. This we leave for our future work.

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Conflict of Interests

The authors declare that there is no conflict of interests.

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