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Research Article

Analysis of Pine Wilt Disease Model with Nonlinear Incidence and Horizontal Transmission

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The deterministic pine wilt model with vital dynamics to determine the equilibria and their stability by considering nonlinear incidence rates with horizontal transmission is analyzed. The complete global analysis for the equilibria of the model is discussed. The explicit formula for the reproductive number is obtained and it is shown that the "disease-free" equilibrium always exists and is globally asymptotically stable whenever $R_0 \leq 1$. Furthermore, the disease persists at an "endemic" level when the reproductive number exceeds unity.

1. Introduction

Pine wilt, a fatal disease of commonly planted pines brought on by the pinewood nematode (*Bursaphelenchus xylophilus*), causes changes to ecosystem and destructs the variety of ecosystem. Pine trees affected by pine wilt disease usually die within few months. Symptoms of pine wilt disease normally appear in late spring or summer. The most prominent symptom is the lack of resin exudation from barks wounds. The foliage becomes light grayish green, then becomes yellow, and finally it becomes reddish brown. The tree succumbs to the disease at this stage. The affected trees totally lack resin and their wood becomes dry.

The long-horned pine sawyer beetles (*Monochamus alternatus*) are the main culprits for the spread of pinewood nematodes from infected pines to healthy or stressed pines. When new adult beetles emerge in spring, they locate a living host tree to feed on the bark of the young branches and transfer nematodes to the healthy trees through the feeding wounds produced by these sawyers. This transmission is referred to as primary transmission. The transmission of the nematodes during egg-laying activities in freshly cut timber or dying trees is referred to as secondary transmission. Nematodes, introduced during primary transmission, migrate to the resin canals of their hosts and kill these cells rendering them ineffective due to which a susceptible host can wilt and die within weeks of being infested upon the availability of favorable conditions to disease development. The principle of the *Bursaphelenchus xylophilus* transmission and disease dissemination is reviewed by Evans et al. [1]. Pine wilt particularly kills Scots pine within few weeks to few months. Some other pine species as Austrian (*Pinus nigra*), jack (*P. banksiana*), mogo (*P. mugo*), and red (*P. resinosa*) pines are occasionally killed by pine wilt.

Mathematical modeling became a considerably important tool in the study of epidemiology because it helps us to understand the observed epidemiological patterns and disease control and provides understanding of the underlying mechanisms which influence the spread of disease and may suggest control strategies. The incidence of a disease is defined as new cases occurring per unit time. It plays a vital role in mathematical epidemiology. The classical epidemiological models are developed by the assumption of bilinear incidence rate βSI and the standard incidence rate $\beta SI/N$, where β is the transmission probability per contact and S, I are susceptible and infected individuals, respectively. However, there are several reasons that require modification in these incidence rates. For example, the assumption of homogeneous mixing may be invalid and in this case a model having a particular form of nonlinear transmission may be incorporated for heterogeneous mixing and the necessary population structure.

The saturation effects may also require a nonlinear incidence rate because if the proportion of the infected population is high enough so that exposure to the disease agent is virtually certain, then transmission rate may react more slowly than linear in order to increase the number of infected individuals. Capasso and Serio [2] who studied the cholera epidemic spread in Bari in 1973 and introduced the saturated incidence rate $g(I)S$ in the epidemic model observed this effect. A variety of nonlinear incidence rates has been utilized in epidemic models [3–7]. An epidemic model with nonlinear incidence is proposed in [8], in which the authors described the dynamics of diseases spread by vector mosquitoes such as malaria, dengue, and yellow fever.

The incidence of pine wilt disease depends on beetles' density because pine sawyer beetles are the source of transmission of pinewood nematode. This incidence may approach its saturation level at very high beetle densities. The adult female pine sawyer attempts to avert from erstwhile oviposition scants. It approaches another tree before the saturation point of oviposition is reached. Thus the isolation of infected individuals results in the decrease in the number of contacts between the susceptible and infected individuals at high infective levels. These observations inspire to consider nonlinearities in the incidence rates.

In this paper, based on the ideas posed in [9–11], a pine wilt disease model considering a direct mode of transmission as well as nonlinear incidence rate is formulated. The aim of this paper is to establish stability properties of equilibria and the threshold parameter R_0 that completely determines the existence of endemic or disease-free equilibrium. If $R_0 \leq 1$, the disease-free equilibrium is globally asymptotically stable. If $R_0 > 1$, a unique endemic equilibrium exists and is globally asymptotically stable. The rest of the paper is organized as follows. In Section 2, the description of the extended mathematical model is presented. Section 3 is devoted to the existence of equilibria. In Section 4, the Lyapunov function theory is used to show global stability of disease-free equilibrium and geometric approach is used to prove global stability of endemic equilibrium in Section 5. Discussions and simulations are done in Section 6.

2. Model Description

The pine population, with total population size denoted by $N_h(t)$, is subdivided into two mutually exclusive compartments: susceptible pine trees $S_h(t)$ and infectious pine trees $I_h(t)$. Thus, $N_h(t) = S_h(t) + I_h(t)$. The emission of oleoresin from susceptible host pines behaves like a physical barrier for beetle oviposition. Beetles can oviposit on the infected pine trees because these trees cease oleoresin. Since there are no cures for pine wilt once a susceptible tree becomes infested with pinewood nematodes, the recovered class $R_h(t)$ has not been considered.

The total vector population at any time t is denoted by $N_v(t) = S_v(t) + I_v(t)$, where $S_v(t)$ denotes the susceptible adult beetles that do not have any pinewood nematode at time t and $I_v(t)$ denotes the infected adult beetles carrying pinewood nematode at time t when they emerge from dead pine trees. After emergence from the dead tree, beetles choose a healthy tree for sufficient feeding and transmit nematodes into the tree. These nematodes move through the feeding wounds and approach the xylem of the tree. When beetles are in oviposition they choose dying or dead tree and transmit nematode when they lay eggs in slits in bark. Nematodes enter these slits, feed on wood cells or fungi, and reproduce themselves. Before beetle's emergence from dead tree the nematodes attach with the tracheae of its respiratory system. The following assumptions are made in formulating the mathematical model.

(i) The exploitation rate of pine trees infected with *Bursaphelenchus xylophilus* is greater than the normal and susceptible pine trees.

(ii) The susceptible beetles receive nematodes directly from infectious ones through mating.

(iii) Adult beetles emerging from infected trees have pinewood nematode.

(iv) The infected vectors transmit the nematode during maturation feeding as well as via oviposition.

Under these assumptions, the vector-host model with nonlinear incidence can be described by the following system of differential equations:

$$\begin{aligned}
\frac{dS_h}{dt} &= \Pi_h - \frac{\delta_1 S_h I_v}{1+\alpha_1 I_v} - \frac{\delta_2 \theta S_h I_v}{1+\alpha_1 I_v} - \mu_h S_h,\\
\frac{dI_h}{dt} &= \frac{\delta_1 S_h I_v}{1+\alpha_1 I_v} + \frac{\delta_2 \theta S_h I_v}{1+\alpha_1 I_v} - \omega I_h,\\
\frac{dS_v}{dt} &= \Pi_v - \frac{\beta_1 S_v I_h}{1+\alpha_2 I_h} - \beta_2 S_v I_v - \mu_v S_v,\\
\frac{dI_v}{dt} &= \frac{\beta_1 S_v I_h}{1+\alpha_2 I_h} + \beta_2 S_v I_v - \mu_v I_v,
\end{aligned} \tag{1}$$

where Π_h is the constant increase rate of pines, Π_v is the constant input rate of vectors, and μ_v is the mortality rate of vectors. The exploitation rate of susceptible pines is μ_h whereas the isolation and felling rate of infected pines is ω. The transmission between susceptible pines and infected vectors occurs when infected beetles lay eggs on those dead pines that die of natural causes or through the maturation feeding of infected vectors; the incidence terms for these transmissions are $\delta_2\theta S_h I_v/(1+\alpha_1 I_v)$ and $\delta_1 S_h I_v/(1+\alpha_1 I_v)$, respectively. The parameter θ is the probability by which susceptible pines die of natural causes and cease oleoresin exudation without being infected by the nematode, and δ_2 indicates the rate at which infected vectors transmit the nematode via oviposition whereas δ_1 denotes transmission rate per contact during maturation feeding. The transmission between susceptible vectors and infected hosts occurs when adult beetles emerge from dead pine trees. This transmission is denoted by $\beta_1 S_v I_h/(1+\alpha_2 I_h)$, where β_1 is the rate at which adult beetles carry the pinewood nematode when they emerge from dead trees. The parameters α_1 and α_2 determine the level at which the infection is saturated. The

beetles transmit nematodes directly through mating. The incidence term for this transmission is $\beta_2 S_v I_v$, where β_2 is the transmission rate among beetles during mating. All parameters are assumed to be positive.

The total dynamics of vector population satisfy the following equation:

$$\frac{dN_v}{dt} = \Pi_v - \mu_v N_v. \tag{2}$$

This leads to $N_v \to \Pi_v/\mu_v$ as $t \to \infty$. Thus, the system (1) is reduced to the following system of differential equations:

$$\begin{aligned}
\frac{dS_h}{dt} &= \Pi_h - \frac{\delta_1 S_h I_v}{1+\alpha_1 I_v} - \frac{\delta_2 \theta S_h I_v}{1+\alpha_1 I_v} - \mu_h S_h,\\
\frac{dI_h}{dt} &= \frac{\delta_1 S_h I_v}{1+\alpha_1 I_v} + \frac{\delta_2 \theta S_h I_v}{1+\alpha_1 I_v} - \omega I_h,\\
\frac{dI_v}{dt} &= \beta_1 \left(\frac{\Pi_v}{\mu_v} - I_v\right) \frac{I_h}{1+\alpha_2 I_h} + \beta_2 \left(\frac{\Pi_v}{\mu_v} - I_v\right) I_v - \mu_v I_v.
\end{aligned} \tag{3}$$

Considering ecological significance, we study system (3) in the closed set $\Omega = \{(S_h, I_h, I_v) : \Pi_h/\omega \le S_h + I_h \le \Pi_h/\mu_h, 0 \le I_v \le \Pi_v/\mu_v\}$. It can be easily verified that Ω is positively invariant with respect to (3).

3. Existence of Equilibria

The dynamics of the disease are described by the threshold quantity R_0 which is called the reproduction number defined as "the average number of secondary infections produced by an infected individual in a completely susceptible population." It is one of the most useful threshold parameters that characterizes mathematical problems related to infectious diseases. This metric helps to determine whether or not an infectious disease will spread through a population. The basic reproduction number of model (3) is given by

$$R_0 = \frac{\beta_2 \Pi_v}{\mu_v^2} + \frac{\beta_1 \Pi_v}{\mu_v^2} \frac{\Pi_h}{\mu_h \omega} \left(\delta_1 + \theta \delta_2\right). \tag{4}$$

Direct calculation shows that for $R_0 \le 1$, there is only disease-free equilibrium $E_0(\Pi_h/\mu_h, 0, 0)$ and for $R_0 > 1$, there is an additional equilibrium $E^*(S_h^*, I_h^*, I_v^*)$ which is called endemic equilibrium, with

$$\begin{aligned}
S_h^* &= \frac{\Pi_h - \omega I_h^*}{\mu_h},\\
I_h^* &= \left(\Pi_h I_v^* \left[\delta_1 + \delta_2 \theta + \left(\delta_1 \alpha_1 + \delta_2 \theta \alpha_1\right) I_v^*\right]\right)\\
&\quad \times \left(\left[\left(\alpha_1 \mu_h + \delta_1 + \delta_2 \theta\right) \alpha_1 \omega I_v^{*2}\right.\right.\\
&\qquad \left.\left. + \left(\delta_1 + \delta_2 \theta + 2\alpha_1 \mu_h\right) \omega I_v^* + \omega \mu_h\right]\right)^{-1},
\end{aligned} \tag{5}$$

and I_v^* is the root of the following equation:

$$A I_v^{*3} + B I_v^{*2} + C I_v^* + D = 0, \tag{6}$$

where,

$$\begin{aligned}
A &= \Pi_h \mu_v \alpha_2 \beta_2 \left(\alpha_1 \delta_1 + \theta \alpha_1 \delta_2\right),\\
B &= \theta \omega \alpha_1 \beta_2 \delta_2 \mu_v + \theta \alpha_1 \beta_1 \delta_2 \Pi_h \mu_v\\
&\quad + \theta \alpha_2 \beta_2 \delta_2 \Pi_h \mu_v + \omega \alpha_1 \alpha_1 \beta_2 \mu_h \mu_v,\\
C &= \omega \beta_2 \mu_h \mu_v + \alpha_1 \beta_1 \delta_1 \Pi_h \Pi_v + \theta \alpha_1 \beta_1 \delta_2 \Pi_h \Pi_v,\\
D &= \omega \mu_h \mu_v^2 \left(1 - R_0\right).
\end{aligned} \tag{7}$$

From (7), we see that $R_0 > 1$ if and only if $D < 0$. Since A, B, and C are always positive, there will be zero or unique positive endemic equilibrium accordingly as $R_0 \le 1$ or $R_0 > 1$. Thus we have the following theorem.

Theorem 1. *System* (3) *always has the infection-free equilibrium* E_0. *If* $R_0 > 1$, *system* (3) *has a unique endemic equilibrium* $E^*(S_h^*, I_h^*, I_v^*)$ *defined by* (5) *and* (6).

4. Stability of Disease-Free Equilibrium

Here, we analyze stability of disease-free equilibrium $E_0(\Pi_h/\mu_h, 0, 0)$ for system (3). The linearization of the system (3) at E_0 results in the following characteristic equation:

$$\left(-\mu_h - \lambda\right) \left[\lambda^2 + \lambda \left(\omega + \mu_v - \frac{\beta_2 \Pi_v}{\mu_v}\right) + \omega \mu_v \left(1 - R_0\right)\right] = 0. \tag{8}$$

The characteristic equation (8) has one eigenvalue $-\mu_h$. The other eigenvalues can be found by the equation

$$\lambda^2 + a\lambda + b = 0, \tag{9}$$

where $a = \omega + \mu_v - (\beta_2 \Pi_v/\mu_v)$ and $b = \omega \mu_v (1 - R_0)$.

We observe that the roots of the quadratic equation (9) have negative real parts if $R_0 < 1$. If $R_0 = 1$, one root of (9) is 0. This fact does not guarantee that all eigenvalues have negative real parts. It will only be possible in case of real roots. If $R_0 > 1$, one of the roots of (9) has positive real part. The above discussion leads to the following theorem.

Theorem 2. *The disease-free equilibrium of system* (3) *is locally asymptotically stable in* Ω *if* $R_0 < 1$ *and it is unstable if* $R_0 > 1$.

Now, we analyze the global behavior of the disease-free equilibrium E_0. The following theorem provides the global property of the system.

Theorem 3. *If* $R_0 \le 1$, *then the infection-free equilibrium* E_0 *is globally asymptotically stable in the interior of* Ω.

Proof. The following Lyapunov function is proposed to establish the global stability of disease-free equilibrium:

$$L = \beta_1 \frac{\Pi_v}{\omega} I_h + \mu_v I_v. \tag{10}$$

Taking the time derivative of L along the solutions of (3), we have

$$\begin{aligned}
L' &= \beta_1 \frac{\Pi_v}{\omega} I_h' + \mu_v I_v' \\
&= \beta_1 \frac{\Pi_v}{\omega} \left(\frac{\delta_1 S_h I_v}{1+\alpha_1 I_v} + \frac{\delta_2 \theta S_h I_v}{1+\alpha_1 I_v} - \omega I_h \right) \\
&\quad + \mu_v \left[\beta_1 \left(\frac{\Pi_v}{\mu_v} - I_v \right) \frac{I_h}{1+\alpha_2 I_h} + \beta_2 \left(\frac{\Pi_v}{\mu_v} - I_v \right) I_v - \mu_v I_v \right] \\
&\le \beta_1 \frac{\Pi_v}{\omega} \left(\delta_1 S_h I_v + \delta_2 \theta S_h I_v - \omega I_h \right) \\
&\quad + \mu_v \left[\left(\beta_1 \frac{\Pi_v}{\mu_v} \frac{I_h}{1+\alpha_2 I_h} - \beta_1 \frac{I_v I_h}{1+\alpha_2 I_h} \right) \right. \\
&\qquad \left. + \left(\beta_2 \frac{\Pi_v}{\mu_v} I_v - \beta_2 I_v I_v \right) \right] - \mu_v^2 I_v \\
&< \left(\delta_1 + \delta_2 \theta \right) \beta_1 \frac{\Pi_v}{\omega} \frac{\Pi_h}{\mu_h} I_v + \mu_v \beta_2 \frac{\Pi_v}{\mu_v} I_v - \mu_v^2 I_v \\
&\quad - \beta_1 \Pi_v I_h + \beta_1 \Pi_v I_h - \mu_v \beta_1 I_v \frac{I_h}{1+\alpha_2 I_h} - \mu_v \beta_2 I_v^2 \\
&= I_v \left[\mu_v^2 \left(R_0 - 1 \right) - \mu_v \beta_1 \frac{I_h}{1+\alpha_2 I_h} - \mu_v \beta_2 I_v \right] \le 0.
\end{aligned} \tag{11}$$

Thus $L'(t)$ is negative if $R_0 \le 1$. When $R_0 < 1$, the derivative $L' = 0$ if and only if $I_v = 0$, while in the case $R_0 = 1$, the derivative $L' = 0$ if and only if $I_h = 0$ or $I_v = 0$. Consequently, the largest compact invariant set in $\{(S_h, I_h, I_v \in \Omega), L' = 0\}$, when $R_0 \le 1$, is the singleton E_0. Hence, by LaSalle's invariance principle [12], E_0 is globally asymptotically stable in Ω. This completes the proof. □

5. Stability of Endemic Equilibrium

In this section, we will discuss global stability of endemic equilibrium E^* in the feasible region Ω. This is done through the geometrical approach applied by Li and Muldowney [13]. We summarize this approach below.

Consider a C^1 map $f : x \mapsto f(x)$ from an open set $D \subset R^n$ to R^n such that each solution $x(t, x_0)$ to the differential equation

$$x' = f(x) \tag{12}$$

is uniquely determined by the initial value $x(0, x_0)$. We have the following assumptions:

(H_1) D is simply connected;

(H_2) there exists a compact absorbing set $K \subset D$;

(H_3) Equation (12) has unique equilibrium $\overline{x}$ in D.

Let $P : x \mapsto P(x)$ be a nonsingular $\binom{n}{2} \times \binom{n}{2}$ matrix-valued function which is C^1 in D and a vector norm $|\cdot|$ on R^N, where $N = \binom{n}{2}$. Let μ be the Lozinskiĭ measure with respect to the $|\cdot|$. Define a quantity $\overline{q}_2$ as

$$\overline{q}_2 = \limsup_{t \to \infty} \sup_{x_0 \in K} \frac{1}{t} \int_0^t \mu \left(B \left(x \left(s, x_0 \right) \right) \right) ds, \tag{13}$$

where $B = P_f P^{-1} + P J^{[2]} P^{-1}$, the matrix P_f is obtained by replacing each entry p of P by its derivative in the direction of f, $(p_{ij})_f$, and $J^{[2]}$ is the second additive compound matrix of the Jacobian matrix J of (12). The following result has been established by Li and Muldowney [13].

Theorem 4. *Suppose that H_1, H_2, and H_3 hold; the unique endemic equilibrium E^* is globally stable in Ω if $\overline{q}_2 < 0$.*

Obviously Ω is simply connected and E^* is a unique endemic equilibrium for $R_0 > 1$ in Ω. To apply the result of the above theorem for global stability of endemic equilibrium E^*, we first prove the uniform persistence of (3) when the threshold parameter $R_0 > 1$, by applying the acyclicity theorem (see [14]).

Definition 5 (see [15]). The system (3) is uniformly persistent; that is, there exists $c > 0$ (independent of initial conditions), such that $\liminf_{t \to \infty} S_h \ge c$, $\liminf_{t \to \infty} I_h \ge c$, and $\liminf_{t \to \infty} I_v \ge c$.

Let X be a locally compact metric space with metric d and let Ω be a closed nonempty subset of X with boundary $\partial\Omega$ and interior Ω°. Clearly, Ω° is a closed subset of Ω. Let Φ_t be a dynamical system defined on Ω. A set B in X is said to be invariant if $\Phi(B, t) = B$. Define $M_\partial := \{x \in \Omega : \Phi(t, x) \in \Omega, \text{for all } t \ge 0\}$.

Lemma 6 (see [14]). *Assume that*

(a) *Φ_t has a global attractor;*

(b) *there exists $M = \{M_1, \ldots, M_k\}$ of pairwise disjoint, compact, and isolated invariant set on $\partial\Omega$ such that*

(1) $\bigcup_{x \in M_\partial} \omega(x) \subseteq \bigcup_{j=1}^k M_j$;

(2) *no subsets of M form a cycle on $\partial\Omega$;*

(3) *each M_j is also isolated in Ω;*

(4) *$W^s(M_j) \cap \Omega^\circ = \phi$ for each $1 \le j \le k$, where $W^s(M_j)$ is stable manifold of M_j. Then Φ_t is uniformly persistent with respect to Ω.*

Proof. We have $\Omega = \{(S_h, I_h, I_v) : \Pi_h/\mu_h \le S_h + I_h \le \Pi_h/\omega, 0 \le I_v \le \Pi_v/\mu_v\}$, $\Omega^\circ = \{(S_h, I_h, I_v) : S_h, I_h, I_v > 0\}$, $\partial\Omega = \Omega/\Omega^\circ$. Obviously, $M_\partial = \partial\Omega$. Since Ω is bounded and positively invariant there exists a compact set M in which all solutions of system (3) initiated in Ω ultimately enter and remain forever. On S_h-axis we have $S_h' = \Pi_h - \mu_h S_h$ which means $S_h \to \Pi_h/\mu_h$ as $t \to \infty$. Thus E_0 is the only omega limit point on $\partial\Omega$; that is, $\omega(x) = E_0$ for all $x \in M_\partial$. Furthermore $M = E_0$ is a covering of $\Omega = \bigcup_{x \in M_\partial} \omega(x)$ because all solutions initiated on the S_h-axis converge to E_0. Also E_0 is isolated and acyclic. This verifies that hypotheses

(1) and (3) hold. When $R_0 > 1$, the "disease-free" equilibrium (DFE) E_0 is unstable from theorem (3) and also $W^s(M) = \partial\Omega$. Hypotheses (4) and (5) hold. There always admits a global attractor due to ultimate boundedness of solutions. □

The boundedness of Ω and the above lemma imply that (3) has a compact absorbing set $K \subset \Omega$ [15]. Now we will prove that the quantity $\bar{q}_2 < 0$. We choose a suitable vector norm $|\cdot|$ in R^3 and a 3×3 matrix valued function

$$P(x) = \begin{bmatrix} 1 & 0 & 0 \\ 0 & \frac{I_h}{I_v} & 0 \\ 0 & 0 & \frac{I_h}{I_v} \end{bmatrix}. \tag{14}$$

Obviously P is C^1 and nonsingular in the interior of Ω. Linearizing system (3) about an endemic equilibrium E^* gives the following Jacobian matrix:

$$J = \begin{bmatrix} -\mu_h - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} & 0 & -S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} - S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2} \\ I_v\frac{\delta_1}{1+\alpha_1 I_v} + I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} & -\omega & S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2} \\ 0 & \frac{\beta_1}{\mu_v(1+\alpha_2 I_h)^2}(\Pi_v - I_v\mu_v) & \frac{\beta_2}{\mu_v}(\Pi_v - 2I_v\mu_v) - \mu_v - I_h\frac{\beta_1}{1+\alpha_2 I_h} \end{bmatrix}. \tag{15}$$

The second additive compound matrix of $J(E^*)$ is given by

$$J^{[2]} = \begin{bmatrix} b_{11} & S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2} & S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2} \\ \frac{\beta_1}{\mu_v(1+\alpha_2 I_h)^2}(\Pi_v - I_v\mu_v) & b_{22} & 0 \\ 0 & I_v\frac{\delta_1}{1+\alpha_1 I_v} + I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} & b_{33} \end{bmatrix}, \tag{16}$$

where

$$\begin{aligned} b_{11} &= -\mu_h - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} - \omega, \\ b_{22} &= -\mu_h - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} \\ &\quad + \frac{\beta_2}{\mu_v}(\Pi_v - 2I_v\mu_v) - \mu_v - I_h\frac{\beta_1}{1+\alpha_2 I_h}, \\ b_{33} &= -\omega + \frac{\beta_2}{\mu_v}(\Pi_v - 2I_v\mu_v) - \mu_v - I_h\frac{\beta_1}{1+\alpha_2 I_h}. \end{aligned} \tag{17}$$

The matrix $B = P_f P^{-1} + PJ^{[2]}P^{-1}$ can be written in block form as $B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}$, with

$$\begin{aligned} B_{11} &= -\mu_h - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} - \omega, \\ B_{12} &= \begin{pmatrix} \frac{I_v}{I_h}\left(S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2}\right), \\ \frac{I_v}{I_h}\left(S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2}\right) \end{pmatrix}, \\ B_{21} &= \begin{pmatrix} \frac{I_h}{I_v}\frac{\beta_1}{\mu_v(1+\alpha_2 I_h)^2}(\Pi_v - I_v\mu_v) \\ 0 \end{pmatrix}, \\ B_{22} &= \begin{pmatrix} M_{22} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v} & 0 \\ I_v\frac{\delta_1}{1+\alpha_1 I_v} + I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} & M_{33} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v} \end{pmatrix}, \end{aligned} \tag{18}$$

where

$$\begin{aligned} M_{22} &= -\mu_h - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} \\ &\quad + \frac{\beta_2}{\mu_v}(\Pi_v - 2I_v\mu_v) - \mu_v - I_h\frac{\beta_1}{1+\alpha_2 I_h}, \\ M_{33} &= -\omega + \frac{\beta_2}{\mu_v}(\Pi_v - 2I_v\mu_v) - \mu_v - I_h\frac{\beta_1}{1+\alpha_2 I_h}. \end{aligned} \tag{19}$$

Consider the norm in R^3 as $|(u, v, w)| = \max(|u|, |v| + |w|)$, where (u, v, w) denotes the vector in R^3. The Lozinskiĭ

measure with respect to this norm is defined as $\mu(B) \leq \sup(g_1, g_2)$, where

$$g_1 = \mu_1(B_{11}) + |B_{12}|, \qquad g_2 = \mu_1(B_{22}) + |B_{21}|. \tag{20}$$

From system (3) we can write

$$\begin{aligned}
\frac{I_h'}{I_h} &= \frac{I_v}{I_h}\left(S_h\frac{\delta_1}{1+\alpha_1 I_v} + S_h\theta\frac{\delta_2}{1+\alpha_1 I_v}\right) - \omega,\\
\frac{I_v'}{I_v} &= \frac{I_h}{I_v}\frac{\beta_1}{\mu_v(1+\alpha_2 I_h)}(\Pi_v - \mu_v I_v) + \frac{\beta_2}{\mu_v}(\Pi_v - \mu_v I_v) - \mu_v.
\end{aligned} \tag{21}$$

Since B_{11} is a scalar, its Lozinskiĭ measure with respect to any vector norm in R^1 will be equal to B_{11}. Thus

$$\begin{aligned}
B_{11} &= -\mu_h - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} - \omega,\\
|B_{12}| &= \frac{I_v}{I_h}\left(S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2}\right),
\end{aligned} \tag{22}$$

and g_1 will become

$$\begin{aligned}
g_1 &= -\mu_h - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v}\\
&\quad - \omega + \frac{I_v}{I_h}\left(S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2}\right)\\
g_1 &= \frac{I_h'}{I_h} - \mu_h - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v}.
\end{aligned} \tag{23}$$

Also $|B_{21}| = (I_h/I_v)(\beta_1/\mu_v(1+\alpha_2 I_h)^2)(\Pi_v - I_v\mu_v)$, $|B_{12}|$ and $|B_{21}|$ are the operator norms of B_{12} and B_{21} which are mapping from R^2 to R and from R to R^2, respectively, and R^2 is endowed with the l_1 norm. $\mu_1(B_{22})$ is the Lozinskiĭ measure of 2×2 matrix B_{22} with respect to l_1 norm in R^2. Consider

$$\begin{aligned}
\mu_1(B_{22}) &= \sup\left\{M_{22} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v} + I_v\frac{\delta_1}{1+\alpha_1 I_v}\right.\\
&\qquad \left. + I_v\theta\frac{\delta_2}{1+\alpha_1 I_v}, M_{33} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v}\right\}\\
&= \sup\left\{-\mu_h - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v}\right.\\
&\qquad + \frac{\beta_2}{\mu_v}(\Pi_v - 2I_v\mu_v) - \mu_v - I_h\frac{\beta_1}{1+\alpha_2 I_h}\\
&\qquad + \frac{I_h'}{I_h} - \frac{I_h}{I_v}\frac{\beta_1}{\mu_v(1+\alpha_2 I_h)}(\Pi_v - \mu_v I_v)\\
&\qquad - \frac{\beta_2}{\mu_v}(\Pi_v - \mu_v I_v) + \mu_v + I_v\frac{\delta_1}{1+\alpha_1 I_v}\\
&\qquad \left. + I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} - \omega + \frac{\beta_2}{\mu_v}(\Pi_v - 2I_v\mu_v)\right.\\
&\qquad - \mu_v - I_h\frac{\beta_1}{1+\alpha_2 I_h} + \frac{I_h'}{I_h}\\
&\qquad - \frac{I_h}{I_v}\frac{\beta_1}{\mu_v(1+\alpha_2 I_h)}(\Pi_v - \mu_v I_v)\\
&\qquad \left. - \frac{\beta_2}{\mu_v}(\Pi_v - \mu_v I_v) + \mu_v\right\},
\end{aligned}$$

$$\begin{aligned}
\mu_1(B_{22}) &= \frac{I_h'}{I_h} - \frac{\beta_2}{\mu_v}(I_v\mu_v)\\
&\quad - \frac{I_h}{I_v}\frac{\beta_1}{\mu_v(1+\alpha_2 I_h)}(\Pi_v - \mu_v I_v) - \widetilde{\beta}_1,
\end{aligned} \tag{24}$$

where

$$\widetilde{\beta}_1 = \min\left\{I_h\frac{\beta_1}{1+\alpha_2 I_h} + \mu_h, \omega + I_h\frac{\beta_1}{1+\alpha_2 I_h}\right\}. \tag{25}$$

Hence $g_2 \leq (I_h'/I_h) - (\beta_2/\mu_v)(I_v\mu_v) - \widetilde{\beta}_1$.

Thus

$$\begin{aligned}
\mu(B) &= \sup\{g_1, g_2\}\\
&\leq \sup\left\{\frac{I_h'}{I_h} - \mu_h - I_v\frac{\delta_1}{1+\alpha_1 I_v}\right.\\
&\qquad \left. - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v}, \frac{I_h'}{I_h} - \frac{\beta_2}{\mu_v}(I_v\mu_v) - \widetilde{\beta}_1\right\},\\
\mu(B) &\leq \frac{I_h'}{I_h} - \widetilde{\beta}_2,
\end{aligned} \tag{26}$$

where

$$\widetilde{\beta}_2 = \min\left\{\mu_h + I_v\frac{\delta_1}{1+\alpha_1 I_v} + I_v\theta\frac{\delta_2}{1+\alpha_1 I_v}, \frac{\beta_2}{\mu_v}(I_v\mu_v) + \widetilde{\beta}_1\right\}. \tag{27}$$

Since (3) is uniformly persistent when $R_0 > 1$, so for $T > 0$ such that $t > T$ implies $I_h(t) \geq c, I_v(t) \geq c$ and $(1/t)\log I_h(t) < \widetilde{\beta}_2/2$ for all $(S_h(0), I_h(0), I_v(0)) \in K$.

Thus

$$\frac{1}{t}\int_0^t \mu(B)\,dt < \frac{\log I_h(t)}{t} - \widetilde{\beta}_2 < -\frac{\widetilde{\beta}_2}{2}, \tag{28}$$

for all $(S_h(0), I_h(0), I_v(0)) \in K$, which further implies that $\overline{q}_2 < 0$. Therefore all the conditions of Theorem 4 are satisfied. Hence unique endemic equilibrium E^* is globally stable in Ω.

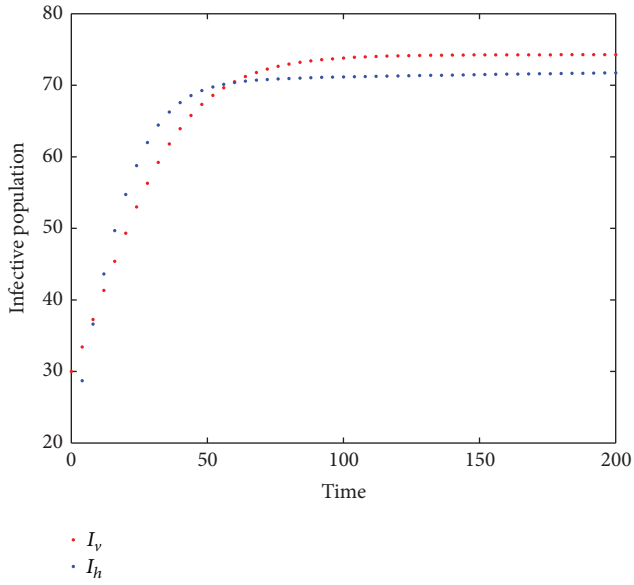

FIGURE 1: The infected population for $\Pi_h = 0.22$, $\Pi_v = 0.32$, $\mu_h = 0.00002$, $\omega = 0.003$, $\mu_v = 0.004$, $\beta_1 = 0.0004$, $\beta_2 = 0.00034$, $\delta_1 = 0.0016$, $\delta_2 = 0.00016$, $\theta = 0.00301$, $\alpha_1 = 0.001$, and $\alpha_2 = 0.001$.

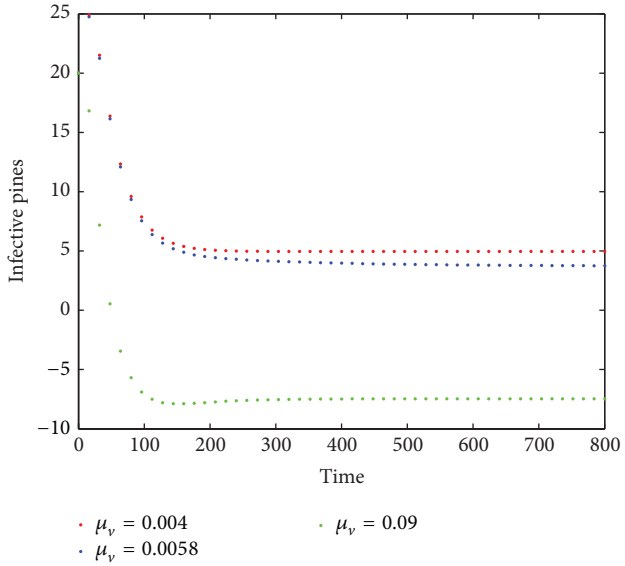

FIGURE 2: The effect of μ_v on infected pine trees for $\Pi_h = 0.22$, $\Pi_v = 0.32$, $\mu_h = 0.02$, $\omega = 0.03$, $\mu_v = 0.004, 0.0058, 0.09$, $\beta_1 = 0.0004$, $\beta_2 = 0.00034$, $\delta_1 = 0.0016$, $\delta_2 = 0.00016$, $\theta = 0.00301$, $\alpha_1 = 0.001$, and $\alpha_2 = 0.001$.

6. Discussions and Simulations

In this paper, pine wilt disease transmission model with nonlinear incidence rates and horizontal transmission is proposed and analyzed. The basic reproduction number, R_0, of the model is obtained and with the help of this reproduction number the asymptotic behavior of the model is discussed. The variation of infected hosts and infected vectors is shown in Figure 1. It is not meaningful to consider the saturation level when transmission occurred during mating. Thus bilinear incidence has been considered. By simple calculation we see that

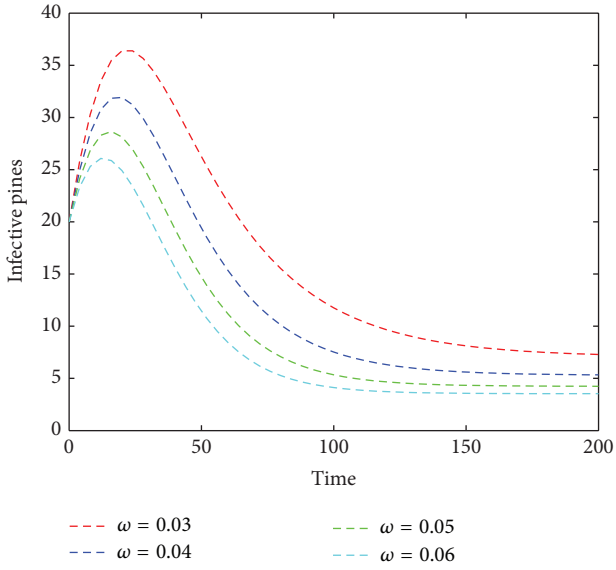

FIGURE 3: The effect of ω on infected pine trees for $\Pi_h = 0.22$, $\Pi_v = 0.32$, $\mu_h = 0.002$, $\omega = 0.03, 0.04, 0.05, 0.06$, $\mu_v = 0.004$, $\beta_1 = 0.0004$, $\beta_2 = 0.00034$, $\delta_1 = 0.0016$, $\delta_2 = 0.00016$, $\theta = 0.00301$, $\alpha_1 = 0.001$, and $\alpha_2 = 0.001$.

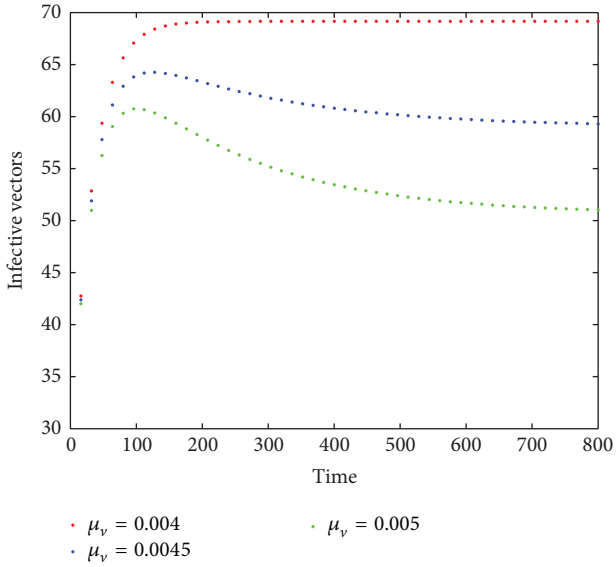

FIGURE 4: The effect of μ_v on infected vectors for $\Pi_h = 0.22$, $\Pi_v = 0.32$, $\mu_h = 0.02$, $\omega = 0.03$, $\mu_v = 0.004, 0.0058, 0.09$, $\beta_1 = 0.0004$, $\beta_2 = 0.00034$, $\delta_1 = 0.0016$, $\delta_2 = 0.00016$, $\theta = 0.00301$, $\alpha_1 = 0.001$, and $\alpha_2 = 0.001$.

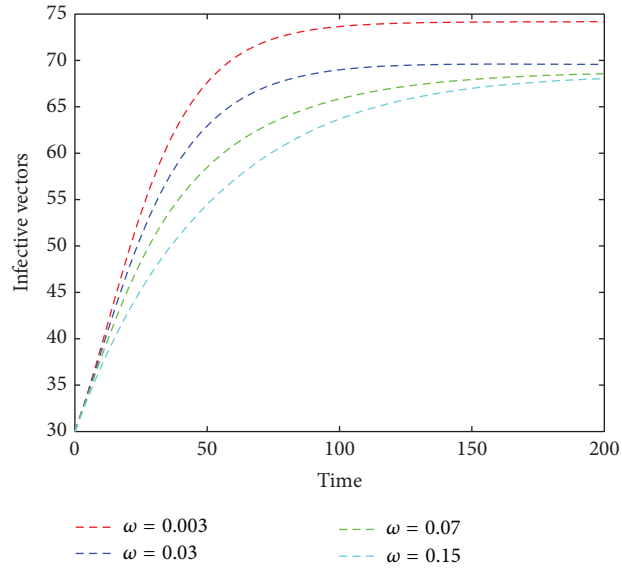

FIGURE 5: The effect of ω on infected vectors for $\Pi_h = 0.22$, $\Pi_v = 0.32$, $\mu_h = 0.002$, $\omega = 0.003, 0.03, 0.07, 0.15$, $\mu_v = 0.004$, $\beta_1 = 0.0004$, $\beta_2 = 0.00034$, $\delta_1 = 0.0016$, $\delta_2 = 0.00016$, $\theta = 0.00301$, $\alpha_1 = 0.001$, and $\alpha_2 = 0.001$.

$$\frac{\partial R_0}{\partial \mu_v} = -\frac{2}{\omega \mu_h \mu_v^3} \Pi_v \left(\omega \beta_2 \mu_h + \beta_1 \delta_1 \Pi_h + \theta \beta_1 \delta_2 \Pi_h\right) < 0,$$
$$\frac{\partial R_0}{\partial \omega} = -\frac{1}{\omega^2} \frac{\beta_1}{\mu_h} \frac{\Pi_h}{\mu_v^2} \Pi_v \left(\delta_1 + \theta \delta_2\right) < 0, \tag{29}$$

which means that R_0 is a decreasing function of μ_v and ω. The question arises, which parameter is more crucial in order to decrease the reproductive number? By using the definition given in [8] and parameter values $\Pi_h = 100$, $\Pi_v = 400$, $\beta_2 = 0.00034$, $\mu_v = 0.00054$, $\beta_1 = 0.4$, $\mu_h = 0.000274$, $\omega = 0.00137$, $\delta_1 = 0.01$, $\theta = 0.00304$, and $\delta_2 = 0.01$, we see that the sensitivity index of the reproductive number with respect to μ_v is -2 and with respect to ω is -1. It means that the most sensitive parameter for R_0 is μ_v. Increasing the mortality rate of *Monochamus alternatus* by 10% decreases R_0 by 20%. Also, increasing the exploitation rate of infected pines by 10% decreases R_0 by 10%. Thus control strategies, for example, setting out beetle traps, setting vertical wood traps, and using chemicals to kill sawyer beetles, by cutting down dead pine trees and disposing of them before the emergence of beetles can be useful for eradicating the disease.

The above mentioned measures are very effective to control pine wilt disease but they have not yet been practiced to eradicate pine wilt disease ultimately because these measures require more cost and labor and even entail danger of forest fires due to which most owners of forests hesitate to use these measures.

However, we can decrease the endemic level of the disease by increasing the parameters μ_v and ω. Figures 2, 3, 4, and 5 show different endemic levels of I_v and I_h with respect to the parameters μ_v and ω. We see that by increasing these parameters the infective levels of pine trees and vectors decrease.

Conflict of Interests

The author declares that there is no conflict of interests regarding the publication of this paper.

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