

STABILITY ANALYSIS OF A HOST-VECTOR TRANSMISSION MODEL FOR PINE WILT DISEASE WITH ASYMPTOMATIC CARRIER TREES

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ABSTRACT. A deterministic model for the spread of pine wilt disease with asymptomatic carrier trees in the host pine population is designed and rigorously analyzed. We have taken four different classes for the trees, namely susceptible, exposed, asymptomatic carrier and infected, and two different classes for the vector population, namely susceptible and infected. A complete global analysis of the model is given, which reveals that the global dynamics of the disease is completely determined by the associated basic reproduction number, denoted by \mathcal{R}_0 . If \mathcal{R}_0 is less than one, the disease-free equilibrium is globally asymptotically stable, and in such a case, the endemic equilibrium does not exist. If \mathcal{R}_0 is greater than one, the disease persists and the unique endemic equilibrium is globally asymptotically stable.

1. Introduction

Pine wilt disease (PWD) is vector-borne disease as known in plant pathology. There are some important wilting diseases of trees, such as the pine wilt disease and the red ring disease of palms, which are caused by nematodes that have intriguing association with insect vectors ([7]). Pine wilt is caused by the pinewood nematode, *Bursaphelenchus xylophilus* (Steiner and Buhrer) Nickle whose synonym is *Bursaphelenchus lignicolus* ([8], [15]). The nematode is transmitted by cerambycid beetles of the genus *Monochamus* which serve as vector ([14], [16], [21]). The pinewood nematode has spread to the Far East (Japan, China and Korea), North America (USA and Canada) and Portugal and has devastated pine forest ecosystems ([23]). Nowadays pine wilt disease (PWD) caused by the pine wood nematode is the most serious threat to pine forests systems worldwide. PWD was ranked first on the 1986 quarantine list published by the European Plant Protection Organization; quarantining has caused international disputes in the lumber trade ([17]).

Received May 5, 2016; Revised September 19, 2016.

2010 *Mathematics Subject Classification.* 92B05, 34D23.

Key words and phrases. mathematical model, pine wilt disease, basic reproduction number, global stability.

The vector beetle, *M. alternatus*, emerges from the dead pine trees carrying pathogenic nematodes in late May to early July, and feeds on young twigs of healthy pines. Through the feeding wounds of the vector beetle, nematodes invade the host pine trees. Nematode infection results in cessation of oleoresin exudation in 2-3 weeks as an early symptom of disease, after which nematode populations increase rapidly and spread through the wood tissue, causing the pine to wilt. Most trees die within the year of infection, showing reddish-brown foliage, though a small number of infected trees succumb to the disease early in the next year ([22]). PWD control has focused mainly on elimination of pine sawyer larvae inhabiting wilt pine trees, either by winter fumigation or by controlling the adult sawyers with aerial insecticide spray in summer. Despite intensive efforts to remove dead pine trees from the stands, newly dead trees tend to appear in the vicinity of the stumps of trees killed the previous year, and pine wilt disease recurs in the same pine stand every year ([6], [9])

An asymptomatic carrier tree could be defined as a tree that harbors *B. xylophilus* but shows no apparent symptoms such as needle chlorosis or browning, and may or may not cease oleoresin exudation. They could play a significant role as attractants for *M. alternatus* that could then transmit *B. xylophilus* to neighboring trees. The spread and occurrence of PWD are influenced by many factors, including meteorological conditions, the flight ability of vector beetles, soil eutrophication, topographic conditions, mycorrhizal relationships, asymptomatic carrier trees, and human activities ([4], [5]).

Recently, many mathematical models have been used to investigate the transmission dynamics of pine wilt disease ([19], [20]). In these papers, authors have worked on modeling of population dynamics of the vector beetle (*Monochamus alternatus*) and the pine tree to explore expansion of the disease using an integro-difference equation with a dispersal kernel that describes beetle mobility. Furthermore, there are many mathematical works which describe the host-vector relationship between pine trees and pine sawyer beetles by means of ordinary differential equations ([11], [12], [13], [18]).

In this paper, we extend the model presented in ([12], [13]) by taking into account the role of asymptomatic carrier trees. We treat a PWD transmission model with asymptomatic carrier trees. We shall investigate the global stability of both the disease-free equilibrium and endemic equilibrium by constructing suitable Lyapunov functions. If \mathcal{R}_0 is less than one, the disease-free equilibrium is globally asymptotically stable, and in such a case, the endemic equilibrium does not exist. If \mathcal{R}_0 is greater than one, the disease persists and the unique endemic equilibrium is globally asymptotically stable. The dynamics of the host pine trees and vector beetles is described by SEAI and SI models, respectively.

The organization of the paper is as follows. In Section 2, a model for the dynamics of pine wilt disease is formulated. The stability of disease free equilibrium and the stability of endemic equilibrium are investigated in Sections 3 and 4, respectively. Lastly, we give a brief discussion of our results in Section 5.

2. Formulation of the model

In this section, we formulate a mathematical model for the spread of pine wilt disease with asymptomatic carrier trees in the host pine population. We formulate a mathematical model for PWD in the host pine and vector beetles population with total population size at time t , given by N_h and N_v , respectively. The total host population is divided into four subclasses: susceptible host pine tree, exposed pine tree, asymptomatic carrier host pine tree and infected host pine tree denoted by S_h , E_h , A_h , I_h , respectively. Susceptible host pines are those trees which are healthy and have the potential to be infected by the nematode. Healthy trees emit oleoresin, which acts as a physical barrier to beetle oviposition. Exposed host pine trees are the ones which have been infected by the nematode, but still possess the capability for oleoresin production. An asymptomatic carrier host pine tree shows no apparent symptoms and may or may not cease oleoresin exudation. The infected host pine trees are those trees which have been infected by the nematode and have lost the ability to exude oleoresin, and hence, beetles can oviposit on them. The susceptible adult beetles who do not carry pinewood nematode at time t are denoted by S_v , and the infective adult beetles who carry pinewood nematode at time t are given by I_v . Thus, $N_h = S_h + E_h + A_h + I_h$. The pine sawyer vector population N_v has subclasses S_v and I_v for the susceptible and infected subclasses, respectively. Thus $N_v = S_v + I_v$.

The parameters used in the system are as follows: the parameter Λ_h is the constant increase rate of pine tree at time t and Λ_v is the constant emergence rate of adult beetles at time t during the period of emergence. The per capita natural death rate of pine trees and beetles (as vectors) is given by μ_h and μ_v , respectively.

The parameter α represents the transmission rate per contact with infected vectors during maturation feeding. The parameter ϕ is the average number of contact per day of the vector adult beetles during maturation feeding period. The incidence of new infections via this route is given by the mass actions term $\alpha\phi S_h I_v$. The transmission probability by which infected beetles transmit nematodes by oviposition is denoted by β and ψ is the average number of contact per day when adult beetles oviposit. The parameter θ is the probability that susceptible host pine trees cease oleoresin exudation without being infected by the nematode. In the model, the term $\beta\psi\theta S_h I_v$ denotes the rate at which the host pine trees cease oleoresin exudation without being infected by the nematode. The parameter γ is the rate at which adult beetles have pinewood nematode when the beetles escape from dead trees. The incidence terms for vector populations is given by the mass action term $\gamma I_h S_v$. The constant per-capita rate, k , models progression from the exposed class E_h to the either the A_h or I_h infected class; the constant ρ ($0 < \rho < 1$) budgets the rate of progress of individuals which move to either the I_h class or to the asymptomatic carrier

trees A_h at the per capita rate k . The flow chart of the disease dynamics is presented in Figure 1.

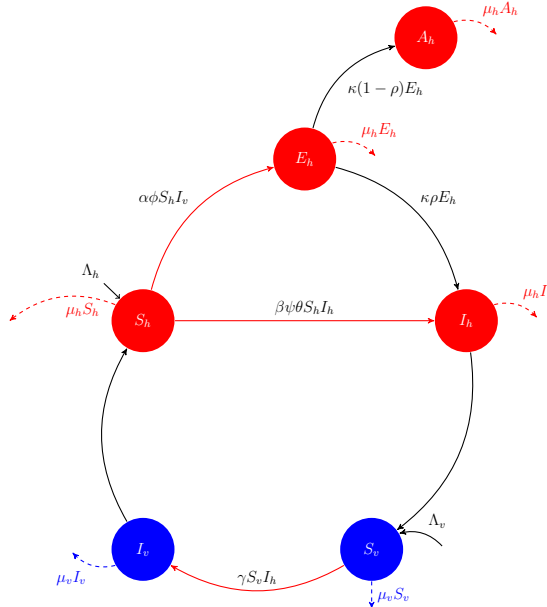


FIGURE 1. The flow diagram for pine wilt transmission illustrating the transmission of disease in trees and vectors. The continuous red line represents transmission rate of infection, the dashed line represents the death rates, Λ_h and Λ_v are recruitment rates of trees and vectors, respectively, while the continuous black line represents the transition between different compartments.

The following system of coupled nonlinear differential equations, derived on the basis of parameter definitions and assumptions, describe the dynamics of the PWD:

$$\begin{aligned}
 \frac{dS_h}{dt} &= \Lambda_h - \alpha\phi S_h I_v - \beta\psi\theta S_h I_h - \mu_h S_h, \\
 \frac{dE_h}{dt} &= \alpha\phi S_h I_v - (k + \mu_h)E_h, \\
 \frac{dA_h}{dt} &= k(1 - \rho)E_h - \mu_h A_h, \\
 \frac{dI_h}{dt} &= k\rho E_h + \beta\psi\theta S_h I_h - \mu_h I_h, \\
 \frac{dS_v}{dt} &= \Lambda_v - \gamma I_h S_v - \mu_v S_v, \\
 \frac{dI_v}{dt} &= \gamma I_h S_v - \mu_v I_v.
 \end{aligned}
 \tag{1}$$

In our proposed model (1), the total population of pine trees and beetles is $S_h + E_h + A_h + I_h = \frac{\Lambda_h}{\mu_h}$, and $S_v + I_v = \frac{\Lambda_v}{\mu_v}$, respectively, for all $t \geq 0$, provided that $S_h(0) + E_h(0) + A_h(0) + I_h(0) = \frac{\Lambda_h}{\mu_h}$, and $S_v(0) + I_v(0) = \frac{\Lambda_v}{\mu_v}$.

The feasible region for system (1) is R_+^6 (the positive orthant of R^6). The model (1) is obviously well-posed. The total host population dynamics is given by $\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h$. The given initial conditions make sure that $N_h(0) \geq 0$. The total dynamics of vector population is $\frac{dN_v}{dt} = \Lambda_v - \mu_v N_v$. We know $N_h(t) \rightarrow \frac{\Lambda_h}{\mu_h}$, $N_v(t) \rightarrow \frac{\Lambda_v}{\mu_v}$. Obviously,

$$\Gamma = \left\{ (S_h, E_h, A_h, I_h, S_v, I_v) \in R_+^6 \mid S_h + E_h + A_h + I_h \leq \frac{\Lambda_h}{\mu_h}, S_v + I_v \leq \frac{\Lambda_v}{\mu_v} \right\}$$

is positively invariant [2], system (1) is dissipative and the global attractor is contained in Γ . On Γ , we have $S_h = \frac{\Lambda_h}{\mu_h} - E_h - A_h - I_h$ and $S_v = \frac{\Lambda_v}{\mu_v} - I_v$. Hence we will study the following four-dimensional nonlinear system:

$$\begin{aligned} \frac{dE_h}{dt} &= \alpha\phi \left(\frac{\Lambda_h}{\mu_h} - E_h - A_h - I_h \right) I_v - (k + \mu_h)E_h, \\ \frac{dA_h}{dt} &= k(1 - \rho)E_h - \mu_h A_h, \\ \frac{dI_h}{dt} &= k\rho E_h + \beta\psi\theta \left(\frac{\Lambda_h}{\mu_h} - E_h - A_h - I_h \right) I_v - \mu_h I_h, \\ \frac{dI_v}{dt} &= \gamma I_h \left(\frac{\Lambda_v}{\mu_v} - I_v \right) - \mu_v I_v, \end{aligned} \tag{2}$$

in the invariant region

$$\Omega = \left\{ (E_h, A_h, I_h, I_v) \in R_+^4 \mid 0 \leq E_h + A_h + I_h \leq \frac{\Lambda_h}{\mu_h}, 0 \leq I_v \leq \frac{\Lambda_v}{\mu_v} \right\}.$$

The values of E_h and S_v can be determined correspondingly by $S_h = \frac{\Lambda_h}{\mu_h} - E_h - A_h - I_h$ and $S_v = \frac{\Lambda_v}{\mu_v} - I_v$, respectively. Possible equilibria of the system (2) and their stability are explored here. The equilibria for our model are determined by setting the right hand side of the model (2) equal to zero. The system (2) admits two equilibrium points namely $E_0 = (0, 0, 0, 0)$ and $E_1 = (E_h^*, A_h^*, I_h^*, I_v^*)$, with

$$\begin{aligned} E_h^* &= \frac{\Lambda_h \alpha \phi}{(k + \mu_h)(\mu_h + \alpha\phi I_v^* + \beta\psi\theta I_v^*)} I_v^*, \\ A_h^* &= \frac{\Lambda_h \alpha \phi k(1 - \rho)}{\mu_h(k + \mu_h)(\mu_h + \alpha\phi I_v^* + \beta\psi\theta I_v^*)} I_v^*, \\ I_h^* &= \frac{\mu_v I_v^*}{\gamma \left(\frac{\Lambda_v}{\mu_v} - I_v^* \right)}, \end{aligned} \tag{3}$$

$$I_v^* = \frac{\mu_h^2 \mu_v^2 (k + \mu_h) (R_0 - 1)}{\mu_v \left\{ \Lambda_h \gamma \{ (k + \mu_h) \beta \psi \theta + \alpha \phi k \rho \} + \mu_h \mu_v (k + \mu_h) (\alpha \phi + \beta \psi \theta) \right\}},$$

where

$$\mathcal{R}_0 = \frac{\gamma \Lambda_h \Lambda_v}{\mu_h^2 \mu_v^2} \left(\beta \psi \theta + \frac{\alpha \phi k \rho}{k + \mu_h} \right).$$

The threshold quantity \mathcal{R}_0 is called the basic reproduction number, which is defined as the average number of secondary infections produced by an infected individual in a completely susceptible population. The carriers in our system can have a great effect on \mathcal{R}_0 . To see the effect of κ on \mathcal{R}_0 , straightforward computation gives

$$\frac{\partial \mathcal{R}_0}{\partial \kappa} = \frac{\gamma \Lambda_h \Lambda_v}{\mu_h \mu_v^2} \left(\frac{\alpha \phi \rho}{(k + \mu_h)^2} \right)$$

and thus $\frac{\partial \mathcal{R}_0}{\partial \kappa} > 0$. Similarly, \mathcal{R}_0 is an increasing function of β and θ . From this analysis we see that a higher probability to develop carriage will increase \mathcal{R}_0 . Since the model deals with tree and beetle populations, it can easily be seen that every state variable will remain nonnegative for non-negative initial conditions (i.e., for all $t \geq 0$, all the state variables and parameters of the model are non-negative). Mathematical analysis of the model is carried out below.

3. Stability of the disease-free equilibrium

In this section, we study the stability of disease-free equilibrium. The local asymptotic stability of the disease-free equilibrium is stated in the following theorem.

Theorem 3.1. *The disease free equilibrium E_0 of model (2) is locally asymptotically stable whenever $\mathcal{R}_0 < 1$.*

Proof. We linearize the system (2) around the disease free equilibrium E_0 . The matrix of linearization at E_0 is given by

$$J(E_0) = \begin{pmatrix} -(k + \mu_h) & 0 & 0 & \frac{\alpha \phi \Lambda_h}{\mu_h} \\ k(1 - \rho) & -\mu_h & 0 & 0 \\ k\rho & 0 & -\mu_h & \frac{\beta \psi \theta \Lambda_h}{\mu_h} \\ 0 & 0 & \frac{\gamma \Lambda_v}{\mu_v} & -\mu_v \end{pmatrix}.$$

One eigenvalue of $J(E_0)$ is $-\mu_h < 0$. The other three eigenvalues are the roots of the following equation

$$(4) \quad \lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0,$$

where

$$\begin{aligned} a_1 &= k + 2\mu_h + \mu_v, \\ a_2 &= (\mu_h + \mu_v)(\mu_h + k) + \mu_h \mu_v - \frac{\beta \psi \theta \gamma \Lambda_h \Lambda_v}{\mu_h \mu_v}, \\ a_3 &= \mu_h \mu_v (k + \mu_h) (1 - \mathcal{R}_0). \end{aligned}$$

If $\mathcal{R}_0 < 1$, then $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1 a_2 > a_3$. Thus, according to the Routh-Hurwitz criterion ([1]), all of the eigenvalues of the characteristic equation 4 have negative real parts. Hence the disease free equilibrium E_0 is locally asymptotically stable. \square

Theorem 3.1 also implies that pine wilt disease can be eliminated from the tree population (when $R_0 < 1$) if the initial sizes of the subpopulations of the model are in the basin of attraction of the disease-free equilibrium (E_0). To ensure that disease elimination is independent of the initial sizes of the subpopulations, it is necessary to show that the disease-free equilibrium is globally asymptotically stable if $R_0 < 1$. This is explored in the following theorem.

Theorem 3.2. *The disease free equilibrium E_0 of model (2) is globally asymptotically stable whenever $\mathcal{R}_0 < 1$.*

Proof. To establish the global stability of the disease-free equilibrium, we define the following Lyapunov function in Ω .

$$(5) \quad L(t) = b_1 E_h + b_2 I_h + b_3 I_v$$

with

$$(6) \quad \begin{aligned} b_1 &= \frac{\Lambda_v \gamma k \rho}{\mu_h \mu_v^2 (k + \mu_h)}, \\ b_2 &= \frac{\Lambda_v \gamma}{\mu_h \mu_v^2}, \\ b_3 &= \frac{1}{\mu_v}. \end{aligned}$$

The time derivative of L (where a prime represents differentiation with respect to t) is given by

$$(7) \quad \begin{aligned} L'(t) &= b_1 E'_h + b_2 I'_h + b_3 I'_v \\ &\leq \frac{\Lambda_v \gamma k \rho}{\mu_h \mu_v^2 (k + \mu_h)} \left\{ \frac{\Lambda_v \alpha \phi}{\mu_h} I_v - (k + \mu_h) E_h \right\} \\ &\quad + \frac{\Lambda_v \gamma}{\mu_h \mu_v^2} \left\{ k \rho E_h + \frac{\Lambda_h \beta \psi \theta}{\mu_h} I_v - \mu_h I_h \right\} + \frac{1}{\mu_v} \left\{ \frac{\Lambda_v \gamma}{\mu_v} I_h - \mu_v I_v \right\} \\ &= (\mathcal{R}_0 - 1) I_v \leq 0. \end{aligned}$$

Thus $L'(t) \leq 0$ if $\mathcal{R}_0 < 1$ and $L' = 0$ if and only if $I = 0$. Therefore, the largest compact invariant set in $\{(E_h, A_h, I_h, I_v) \in \Omega \mid L' = 0\}$ is the singleton $\{E_0\}$. Hence LaSalle’s invariance principle ([10]) implies that E_0 is globally asymptotically stable in Ω . This completes the proof. \square

The epidemiological implication of the above result is that PWD will be eliminated from the population if \mathcal{R}_0 can be brought to (and maintained at) a value less than unity.

4. Stability of the endemic equilibrium

In this section, we prove the global stability of the endemic equilibrium E_1 , when the basic reproduction number R_0 is greater than the unity. First, we will prove the following result.

Theorem 4.1. *If $R_0 > 1$, then system (2) is uniformly persistent, i.e., there exists $c > 0$ (independent of initial conditions), such that $\liminf_{t \rightarrow \infty} E_h(t) \geq c$, $\liminf_{t \rightarrow \infty} A_h(t) \geq c$, $\liminf_{t \rightarrow \infty} I_h(t) \geq c$, $\liminf_{t \rightarrow \infty} I_v(t) \geq c$.*

Proof. Let π be a semidynamical system (2) in $(R_0^+)^4$, χ be a locally compact metric space and $\Omega_0 = \{(E_h, A_h, I_h, I_v) \in \Gamma \mid I_v = 0\}$. The set Γ_0 is a compact subset of Ω_0 and Ω/Ω_0 is positively invariant set of system (2). Let $P : \chi \rightarrow R_0^+$ be defined by $P(E_h, A_h, I_h, I_v) = I_v$ and set $S = \{(E_h, A_h, I_h, I_v) \in \Omega \mid P(E_h, A_h, I_h, I_v) < \rho\}$, where ρ is sufficiently small so that $R_0(1 - \frac{\mu_v}{\Lambda_v}\rho) > 1$. Assume that there is a solution $x \in S$ such that for each $t > 0$, we have $P(\pi(x, t)) < P(x) < \rho$. Let us consider the following:

$$L(t) = \frac{\Lambda_h(\alpha\phi k\rho + \beta\psi\theta(k + \mu_h))(1 - \delta^*)}{\mu_h\mu_v(k + \mu_h)}I_v + I_h,$$

where $\delta^* > 0$ is a sufficiently small constant so that $R_0(1 - \frac{\mu_v}{\Lambda_v}\rho)(1 - \delta^*) > 1$. By a direct calculation, we have

$$L'(t) \geq \mu_h \left[\frac{\Lambda_h\Lambda_v\gamma(\alpha\phi k\rho + \beta\psi\theta(k + \mu_h))(1 - \delta^*)(1 - \frac{\mu_v\rho}{\Lambda_v})}{\mu_h^2\mu_v^2(k + \mu_h)} - 1 \right] I_h + \frac{\Lambda_h(\alpha\phi k\rho + \beta\psi\theta(k + \mu_h))\delta^*}{\mu_h^2(k + \mu_h)}I_v.$$

Let

$$\delta = \min \left\{ \mu_1 \left[\frac{\Lambda_h\Lambda_v\gamma(\alpha\phi + \beta\psi\theta)(1 - \delta^*)(1 - \frac{\mu_2\rho}{\Lambda_v})}{\mu_1^2\mu_2^2(k + \mu_h)} - 1 \right], \frac{\mu_h\delta^*}{\mu_v(1 - \delta^*)} \right\} > 0.$$

Thus, we have

$$(8) \quad L'(t) \geq \delta L(t).$$

The above inequality (8) implies that $L(t) \rightarrow \infty$ as $t \rightarrow \infty$. However, $L(t)$ is bounded on the set Γ . According to Theorem 1 in ([3]), we complete the proof of Theorem 4.1. □

Now, we investigate the global asymptotic stability of the unique endemic equilibrium E_1 when $\mathcal{R}_0 > 1$. The global asymptotic stability of the endemic equilibrium is proved below.

Theorem 4.2. *If $\mathcal{R}_0 > 1$, the endemic equilibrium E_1 of model (2) is globally asymptotically stable in Ω .*

Proof. We propose the following Lyapunov function

$$\begin{aligned}
 \mathcal{F}(S_h, E_h, I_h, S_v, I_v) = & c_1 \left(S_h - S_h^* - S_h^* \ln \frac{S_h}{S_h^*} \right) + c_2 \left(E_h - E_h^* - E_h^* \ln \frac{E_h}{E_h^*} \right) \\
 (9) \quad & + c_3 \left(I_h - I_h^* - I_h^* \ln \frac{I_h}{I_h^*} \right) + c_4 \left(S_v - S_v^* - S_v^* \ln \frac{S_v}{S_v^*} \right) \\
 & + c_5 \left(I_v - I_v^* - I_v^* \ln \frac{I_v}{I_v^*} \right),
 \end{aligned}$$

where

$$\begin{aligned}
 c_1 = & \frac{k\rho E_h^* + \beta\psi\theta S_h^* I_v^*}{S_h^* I_v^* (\alpha\phi + \beta\psi\theta)}, \\
 c_2 = & \frac{k\rho E_h^*}{\alpha\phi S_h^* I_v^*}, \\
 (10) \quad c_3 = & 1, \\
 c_4 = c_5 = & \frac{k\rho E_h^* + \beta\psi\psi\theta S_h^* I_v^*}{\gamma I_h^* S_v^*}.
 \end{aligned}$$

The system (2) satisfy the following relations at equilibrium point

$$\begin{aligned}
 \Lambda_h = & \alpha\phi S_h^* I_v^* + \beta\psi\theta S_h^* I_v^* + \mu_h S_h^*, \\
 k + \mu_h = & \frac{\alpha\phi S_h^* I_v^*}{E_h^*}, \\
 (11) \quad \mu_h = & \frac{k\rho E_h^* + \beta\psi\theta S_h^* I_v^*}{I_h^*}, \\
 \Lambda_v = & \gamma I_h^* S_v^* + \mu_v S_v^*, \\
 \mu_v = & \frac{\gamma I_h^* S_v^*}{I_v^*}.
 \end{aligned}$$

Since, the arithmetic mean is greater than or equal to the geometric mean, we have

$$\begin{aligned}
 (12) \quad \frac{S_h^*}{S_h} + \frac{S_h}{S_h^*} \frac{E_h^*}{E_h} \frac{I_v}{I_v^*} + \frac{E_h}{E_h^*} \frac{I_h^*}{I_h} + \frac{I_h}{I_h^*} \frac{S_v}{S_v^*} \frac{I_v^*}{I_v} + \frac{S_v^*}{S_v} & \geq 5, \\
 \frac{S_h^*}{S_h} + \frac{S_h}{S_h^*} \frac{I_h^*}{I_h} \frac{I_v}{I_v^*} + \frac{I_h}{I_h^*} \frac{S_v}{S_v^*} \frac{I_v^*}{I_v} + \frac{S_v^*}{S_v} & \geq 4.
 \end{aligned}$$

Hence

$$\begin{aligned}
 \mathcal{F}' = & -\mu_h \frac{k\rho E_h^* + \beta\psi\theta S_h^* I_v^*}{S_h^* I_v^* (\alpha\phi + \beta\psi\theta)} \frac{(S_h - S_h^*)^2}{S_h} - \mu_v \frac{k\rho E_h^* + \beta\psi\psi\theta S_h^* I_v^*}{\gamma I_h^* S_v^*} \frac{(S_v - S_v^*)^2}{S_v} \\
 (13) \quad & + k\rho \left(5 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*} \frac{E_h^*}{E_h} \frac{I_v}{I_v^*} - \frac{E_h}{E_h^*} \frac{I_h^*}{I_h} - \frac{I_h}{I_h^*} \frac{S_v}{S_v^*} \frac{I_v^*}{I_v} - \frac{S_v^*}{S_v} \right) \\
 & + \beta\psi\theta S_h^* I_v^* \left(4 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*} \frac{I_h^*}{I_h} \frac{I_v}{I_v^*} - \frac{I_h}{I_h^*} \frac{S_v}{S_v^*} \frac{I_v^*}{I_v} - \frac{S_v^*}{S_v} \right).
 \end{aligned}$$

Thus, it follows from (12) and (13) that $\mathcal{F}' \leq 0$ in Ω . The equality $\mathcal{F}' = 0$ holds if and only if $S_h = S_h^*$, $A_h = A_h^*$, $E_h = E_h^*$, $I_h = I_h^*$, $S_v = S_v^*$, $I_v = I_v^*$ in Ω . The maximal compact invariant set in $\{(S_h, A_h, E_h, I_h, S_v, I_v) \in \Omega \mid \mathcal{F}' = 0\}$ is $\{E_1\}$ when $\mathcal{R}_0 > 1$. From the LaSalle's Invariant Principle, we have the unique endemic equilibrium E_1 of system (2) is globally asymptotically stable for $\mathcal{R}_0 > 1$. \square

The epidemiological implication of Theorem 3.3 is that PWD would persist in the population if $\mathcal{R}_0 > 1$.

5. Conclusions

In this paper, a deterministic model for the transmission dynamics of pine wilt disease with asymptomatic carrier trees is developed and analyzed. The basic reproduction number of the model is obtained. The global dynamics of pine wilt disease with asymptomatic carrier trees in the host pine population is completely determined by the associated basic reproduction number. If $\mathcal{R}_0 < 1$, the disease-free equilibrium is globally asymptotically stable, so the disease always dies out. If $\mathcal{R}_0 > 1$, the disease-free equilibrium becomes unstable while the endemic equilibrium emerges as the unique positive equilibrium and it is shown to be globally asymptotically stable in the interior of the feasible region, and once the disease appears, it eventually persists at the unique endemic equilibrium level. Also, it is observed that a higher carriage increases the basic reproduction number. That is, a suitable reduction in the carriage can effectively bring \mathcal{R}_0 below 1 and hence alter the asymptotic dynamics of the disease from globally stable endemicity to a disease-free steady state.

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