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An epidemic model of a vector-borne disease with direct transmission and time delay

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Abstract

This paper considers an epidemic model of a vector-borne disease which has direct mode of transmission in addition to the vector-mediated transmission. The incidence term is assumed to be of the bilinear mass-action form. We include both a baseline ODE version of the model, and, a differential-delay model with a discrete time delay. The ODE model shows that the dynamics is completely determined by the basic reproduction number R_0. If $R_0 \leqslant 1$, the disease-free equilibrium is globally stable and the disease dies out. If $R_0 > 1$, a unique endemic equilibrium exists and is locally asymptotically stable in the interior of the feasible region. The delay in the differential-delay model accounts for the incubation time the vectors need to become infectious. We study the effect of that delay on the stability of the equilibria. We show that the introduction of a time delay in the host-to-vector transmission term can destabilize the system and periodic solutions can arise through Hopf bifurcation.

Keywords: Epidemic models; Vector-borne disease; Equilibrium analysis; Stability; Threshold; Time delay; Hopf bifurcation

1. Introduction

Vector-borne diseases are infectious diseases caused by viruses, bacteria, protozoa or rickettsia which are primarily transmitted by disease transmitting biological agents (anthropoids), called vectors, who carry the disease without getting it themselves. Globally, malaria is the most prevalent vector-borne disease whose vectors are the mosquitoes. The mosquitoes are vectors of a number of infectious diseases most prominent among which are dengue (the second most important vector-borne disease), yellow fever, St Louis Encephalitis, Japanese Encephalitic, and West Nile Fever, caused by the West Nile Virus. Other vectors are the assassin bugs, causing the Chagas disease, fleas transmitting the plague from its normal host (wild rodents and other small mammals) to humans, or from human to human, and ticks which transmit the most prevalent vector-borne disease in North America (Harrus and Baneth [1])—the Lyme disease, but also tick-borne Encephalitis and others. Ample evidences now exist that in the last 20–30 years vector-borne dis-

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eases have emerged in new locations (Marfin and Gubler [2]) or reemerged as a significant health problem after being put under control for most of the world, except Africa, in the 1950s and 1960s. A number of factors that contribute to the upsurgence of vector-borne diseases have been repeatedly pointed out and discussed (Molyneux [3], Gubler [4], Harrus and Baneth [1]). Such factors include (1) the ability of the anthropoids to adapt to new habitats, (2) development of insecticide and drug-resistant vectors, (3) global and rapid human movement (by jet airplanes), (4) building widespread irrigation and water-impoundment, (5) civil unrest and wars which lead to displacement of large masses of people who live for long periods of time under poor conditions, (6) rapid urbanization which concentrates many host on small area, (7) change in policies that took away resources for vector-control measures. The impact of climate change and global warming is a topic of significant debate (Watson et al. [5], Khasnis and Nettleman [6]). Most of the above factors are a part of the global change linked to human vulnerability in the review by Sutherst [7]. The emergence and reemergence of vector-borne diseases have promoted interest in their mathematical modeling. Rogers [8] reviews the early modeling efforts and McKenzie [9] discusses what makes mathematical models, in particular those for malaria, useful. Most of the models are related to a specific vector-borne disease, most often malaria and dengue. Dynamical models of malaria in the recent years are more often associated with the within-host dynamics of the parasite. Rodríguez and Torres-Sorando [10] present a finite-number of localities model based on the Ross–MacDonald classic model of malaria dynamics. Ishikawa et al. [11] use a mathematical model to evaluate the impact from the programs of selective mass drug administration (MDA) and vector control through permethrin-treated bednets.

Drug-resistance of malaria in the human host is becoming an increasing problem and has been addressed in Koella and Antia [12] (see also review by Mackinnon [13]). Convolution between malaria and its host has been well documented and has been investigated through mathematical models in several articles (Koella and Boete [14], Feng et al. [15]). Incidence of dengue has increased and dengue is now endemic in more than 100 countries (Calisher [16]). Furthermore, there is evidence that selection for more virulent dengue viruses occurs (Cologna et al. [17]).

Mathematical models have been used to evaluate the impact of ultra-low volume insecticide on the dengue epidemic (Newton and Reiter [18], Esteva and Vargas [19]), to study the interaction of the serotype (Feng and Velasco-Hernandez [20], Esteva and Vargas [21,22], Ferguson et al. [23]), to infer the implication of cross-immunity enhancement of transmission (Ferguson et al. [24]) or mortality (Kawaguchi et al. [25]) on the dynamics multiple-strain dengue pathogens, to explore the impact of mechanical transmission of dengue from the mosquito to the human host and vertical transmission of the virus in the mosquitoes on the dynamics of the dengue disease (Esteva and Vargas [26]). The importance of host age-structure for the determination of the transmission rates of dengue has been pointed out in several articles (Pongsumpun and Tang [27], Ferguson et al. [23]). Vaccine against dengue is not yet available (see Chaturvedi et al. [28] for overview of current state of vaccine development) but its potential impact is investigated in Derouich et al. [29]. The spatial dynamics of the transmitting mosquitoes only was investigated in Takahashi et al. [30]. A recent article discusses control strategies, such as the introduction of sterile mosquitoes, on the dynamics of the vector (Esteva and Mo Yang [31]).

The recent transfer and invasion of the West Nile virus to the North America have lead to several recent mathematical models of its epidemiology. Cruz-Pacheco et al. [32] model the dynamics by only taking into account the vector and the avian population as a host while Bowman et al. also take into account the human host in addition to the vector and the avian host (Bowman et al. [33]). The complex interaction of the ticks that transmit Lyme disease and possible multiple hosts was also investigated through mathematical models (Porco [34], Caraco et al. [35]). More general models of tick-borne diseases are also discussed (Gosh and Pugliese [36], Rosa et al. [37], Mwambi [38]). The impact of more complex factors as climate and space distribution is addressed through simulative models (Brownstein et al. [39]).

We consider in this article a Ross–MacDonald type model where the population of the vector is described by a system for the susceptible and infected vector while the dynamics of the host is described by an SIR model. The ODE version is introduced and analyzed in Section 2. We extend this model to include a fixed delay in the system for the vector in Section 3. Takeuchi et al. [40] consider a similar differential-delay model for vector-borne diseases but the main result there is the global stability of the endemic equilibrium under appropriate conditions, while we are interested in Hopf bifurcation and the presence of sustained oscillations. While the main mode of transmission in vector-borne diseases is through the vector, and that is the only way of transmission included in Takeuchi et al. [40], evidence exists that direct transmission is possible through blood transfusion, vertically or through needle stick injury. Such alternative modes of transmission have been reported in malaria and in Chagas diseases and direct transmission in addition to vector transmission has been incorporated in a recent age-since-infection structured model of Chagas diseases (Inaba and Sekine [41]).

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2. The ODE model

In this section, we present an ODE version of a model for spread of a vector-transmitted disease in a host population. The total host population size at time t, given by $N_1(t)$, is partitioned into subclasses of individuals who are susceptible, infectious and recovered, with sizes denoted by $S(t)$, $I(t)$ and $R(t)$, respectively. Furthermore, the host population dies at a natural death rate μ_1. In addition, the host population is recruited at a rate b_1. We assume that vertical transmission in the host population does not occur so that all newly recruited individuals are susceptible. The per capita recovery rate of the hosts is given by γ. The recovered individuals are assumed to acquire permanent immunity and there is no transfer from the R class back to the S class. Susceptible hosts can get infected via two routes of transmission—directly, through a contact with an infected individual (possibly as a result of blood transfusion), and through being bitten by an infectious vector. We denote the rate of direct transmission by λ_1 so that the incidence of new infections via this route is given by the mass action term $\lambda_1 S(t)I(t)$. Furthermore, we denote the biting rate that a pathogen-carrier vector has of susceptible hosts as λ_2 and the incidence of new infections transmitted by the vectors is given again by a mass action term $\lambda_2 S(t)V(t)$.

The following differential equations, derived based on the basic assumptions, give the dynamics of the disease in the host population:

$$\begin{cases} \dfrac{dS(t)}{dt} = b_1 - \lambda_1 S(t)I(t) - \lambda_2 S(t)V(t) - \mu_1 S(t), \\ \dfrac{dI(t)}{dt} = \lambda_1 S(t)I(t) + \lambda_2 S(t)V(t) - \gamma I(t) - \mu_1 I(t), \\ \dfrac{dR(t)}{dt} = \gamma I(t) - \mu_1 R(t), \end{cases} \tag{2.1}$$

where $V(t)$ is the number of vectors at time t who carry the pathogen. The second component of the vector population is the number of pathogen-free (susceptible) vectors at time t, given by $M(t)$. The total size of the vector population at time t, given by $N_2(t)$, is subdivided into those two vector-population classes, the susceptible vectors and infectious vectors. The vector population dies at a natural death rate μ_2. In addition, the vector population is recruited at a birth rate b_2. Although evidence exists that the pathogen of several vector-borne diseases (e.g. West Nile fever and yellow fever and Lyme disease), can be transmitted from (female) parent to offspring in the vector population, we will assume that all newborn vectors are susceptible and vertical transmission can be neglected. Susceptible vectors start carrying the pathogen after getting into contact (biting) an infective host at a rate λ_3 so that the incidence of newly infected vectors is given by a mass action term $\lambda_3 M(t)I(t)$.

In contrast to the host population, once the vectors become carriers of the microparasite, they carry it for life. The system that describes the dynamics of the vectors is given by

$$\begin{cases} \dfrac{dM(t)}{dt} = b_2 - \lambda_3 M(t)I(t) - \mu_2 M(t), \\ \dfrac{dV(t)}{dt} = \lambda_3 M(t)I(t) - \mu_2 V(t). \end{cases} \tag{2.2}$$

We make some reasonable technical assumptions on the parameters of the model, namely that $\gamma > 0$, $\mu_j > 0$ and $b_j > 0$ for $j = 1, 2$. The above systems for the host population and the vector are also equipped with initial conditions as follows: $S(0) = S_0$, $I(0) = I_0$, $R(0) = \bar{R}_0$, $M(0) = M_0$ and $V(0) = V_0$.

The total host population size $N_1(t)$ can be determined by $N_1(t) = S(t) + I(t) + R(t)$ or from the differential equation

$$N_1' = b_1 - \mu_1 N_1,$$

which is derived by adding the equations in (2.1). The total number of vectors $N_2(t)$ can be determined by $N_2(t) = M(t) + V(t)$ or from the differential equation

$$N_2' = b_2 - \mu_2 N_2,$$

which is derived by adding the equations in (2.2).

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It is easily seen that both for the host population and for the vector population the corresponding total population sizes are asymptotically constant: $\lim_{t\to\infty} N_1(t) = \frac{b_1}{\mu_1}$, and $\lim_{t\to\infty} N_2(t) = \frac{b_2}{\mu_2}$. This implies that in our model we assume without loss of generality that $N_1(t) = \frac{b_1}{\mu_1}$, $N_2(t) = \frac{b_2}{\mu_2}$ for all $t \geqslant 0$, provided that $S_0 + I_0 + \bar{R}_0 = \frac{b_1}{\mu_1}$, $M_0 + V_0 = \frac{b_2}{\mu_2}$.

Previous results (Thieme [42]) imply that the dynamics of systems (2.1) and (2.2) are qualitatively equivalent to the dynamics of system given by

$$\begin{cases} \dfrac{dS(t)}{dt} = b_1 - \lambda_1 S(t) I(t) - \lambda_2 S(t) V(t) - \mu_1 S(t), \\ \dfrac{dI(t)}{dt} = \lambda_1 S(t) I(t) + \lambda_2 S(t) V(t) - \gamma I(t) - \mu_1 I(t), \\ \dfrac{dV(t)}{dt} = \lambda_3 \left(\dfrac{b_2}{\mu_2} - V(t) \right) I(t) - \mu_2 V(t). \end{cases} \tag{2.3}$$

The values of R and M can be determined correspondingly by $R = \frac{b_1}{\mu_1} - S - I$ and $M = \frac{b_2}{\mu_2} - V$ or from $R' = \gamma I - \mu_1 R$ and $M' = b_2 - \lambda_3 M I - \mu_2 M$, respectively.

For biological reasons we need the solutions non-negative. Mathematical properties of the solutions lead us to study the system (2.3) in the closed set

$$\Gamma = \left\{ (S, I, V) \in \mathbf{R}^3_+ \;\middle|\; 0 \leqslant S + I \leqslant \frac{b_1}{\mu_1},\ 0 \leqslant V \leqslant \frac{b_2}{\mu_2},\ S \geqslant 0,\ I \geqslant 0 \right\},$$

where $\mathbf{R}^3_+$ denotes the non-negative cone of $\mathbf{R}^3$ including its lower dimensional faces. It can be verified that Γ is positively invariant with respect to (2.3). We denote by $\partial\Gamma$ and Γ^o the boundary and the interior of Γ in $\mathbf{R}^3$, respectively. Direct calculation shows that system (2.3) has the disease-free equilibrium $E_0 = (\frac{b_1}{\mu_1}, 0, 0) \in \partial\Gamma$, in the non-negative cone $\mathbf{R}^3_+$.

The dynamics of the disease is described by the quantity

$$R_0 = \frac{b_1}{\mu_1} \left(\frac{\lambda_2}{(\gamma + \mu_1)} \frac{\lambda_3}{\mu_2} \frac{b_2}{\mu_2} + \frac{\lambda_1}{(\gamma + \mu_1)} \right).$$

The quantity R_0 is called the basic reproduction number [43] or the contact number [44]. It represents the average number of secondary infections that single infectious host can generate in a totally susceptible population of hosts and vectors. The reproduction number consists of two terms. The first term gives the number of secondary infections that one infectious host can generate only through vector transmission. To see this notice that there are $\frac{b_2}{\mu_2}$ susceptible vectors, and $\lambda_3 \frac{b_2}{\mu_2}$ will become infected per unit of time and will stay infected for $\frac{1}{\mu_2}$ time units, so that the total number of infected vectors from one infectious host will be $\frac{\lambda_3}{\mu_2} \frac{b_2}{\mu_2}$. Since there are $\frac{b_1}{\mu_1}$ susceptible hosts, these infectious vectors will bite and transmit the pathogen to $\lambda_2 \frac{b_1}{\mu_1}$ of them per unit of time. The total time spent as infectious individual is $\frac{1}{\gamma+\mu_1}$ so that the total number of secondary infected hosts through the vector-transmission is $\frac{\lambda_2}{(\gamma+\mu_1)} \frac{b_1}{\mu_1} \frac{\lambda_3}{\mu_2} \frac{b_2}{\mu_2}$. The second term of the reproduction number gives the number of secondary infections that one infectious individual can generate in a susceptible population only through direct transmission. To see that we notice that since there are $\frac{b_1}{\mu_1}$ susceptible hosts, one infectious host can infect through direct transmission only $\lambda_1 \frac{b_1}{\mu_1}$ of them per unit of time, and since they spend $\frac{1}{\gamma+\mu_1}$ time units infectious, the total number of secondary infections is $\frac{\lambda_1}{\gamma+\mu_1} \frac{b_1}{\mu_1}$.

The reproduction number R_0 controls the number of equilibria of the system (2.3). If $R_0 \leqslant 1$, then the disease-free equilibrium E_0 is the only equilibrium in Γ. If $R_0 > 1$, the disease-free equilibrium E_0 is still present, but there is also a unique endemic equilibrium E^* which exists in Γ^o.

2.1. The disease-free equilibrium and its stability

In this section we study the local stability of the disease-free equilibrium E_0 in the two cases when the reproduction number $R_0 < 1$ and when $R_0 > 1$.

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Theorem 2.1.1. *The disease-free equilibrium E_0 of* (2.3) *is locally asymptotically stable in Γ if $R_0 < 1$. The disease-free equilibrium E_0 of* (2.3) *is unstable if $R_0 > 1$.*

Proof. Linearizing the system (2.3) at the equilibrium E_0, we obtain the corresponding characteristic equation. According to the Hurwitz criterion, if $R_0 < 1$ it has only roots with negative real parts and the disease-free equilibrium E_0 is locally asymptotically stable. Otherwise, it has one positive root and E_0 is unstable if $R_0 > 1$. This completes the proof. □

Theorem 2.1.2. *If $R_0 \leqslant 1$, then the disease-free equilibrium E_0 of* (2.3) *is globally asymptotically stable in Γ.*

Proof. To see the global stability of the disease-free equilibrium we introduce a new dependent variable $L = \mu_1 I + \frac{\lambda_2 b_1}{\mu_2} V$. Clearly, $L \geqslant 0$ along the solutions of the system (2.3) and is zero if and only if both I and V are zero. The derivative of L along the solutions of (2.3) is

$$\begin{aligned} L' &= \mu_1\lambda_1 SI + \mu_1\lambda_2 SV - \mu_1(\gamma+\mu_1)I + \frac{\lambda_2\lambda_3 b_1 b_2}{\mu_2^2} I - \frac{\lambda_2\lambda_3 b_1}{\mu_2} VI - \lambda_2 b_1 V \\ &\leqslant I\left[\lambda_1 b_1 - \mu_1(\gamma+\mu_1) + \frac{\lambda_2\lambda_3 b_1 b_2}{\mu_2^2} - \frac{\lambda_2\lambda_3 b_1}{\mu_2} V\right] = I\left[b_1\left(\frac{\lambda_2\lambda_3 b_2}{\mu_2^2} + \lambda_1\right) - \mu_1(\gamma+\mu_1) - \frac{\lambda_2\lambda_3 b_1}{\mu_2} V\right] \\ &= I\left[\mu_1(\gamma+\mu_1)(R_0 - 1) - \frac{\lambda_2\lambda_3 b_1}{\mu_2} V\right] \leqslant 0, \end{aligned}$$

where in the first inequality we have used the fact that $\mu_1\lambda_2 SV - \lambda_2 b_1 V < 0$ in Γ and, therefore, we have neglected it. In addition, the last inequality follows from the assumption that $R_0 \leqslant 1$. Furthermore, in the case $R_0 < 1$ the derivative $L' = 0$ if and only if $I = 0$, while in the case $R_0 = 1$ the derivative $L' = 0$ if and only if $I = 0$ or $V = 0$. Consequently, the largest compact invariant set in $\{(S, I, V) \in \Gamma\colon\ L' = 0\}$, when $R_0 \leqslant 1$, is the singleton $\{E_0\}$. LaSalle's invariance principle [45] then implies that E_0 is globally asymptotically stable in Γ. This proves the theorem. □

2.2. Existence of an endemic equilibrium and its stability

Throughout this subsection, we will assume $R_0 > 1$. We obtain the unique positive endemic equilibrium $E^* = (S^*, I^*, V^*) \in \Gamma^o$ in the non-negative cone $\mathbf{R}^3_+$ by using the Mean Value Theorem:

$$\begin{aligned} S^* &= \frac{b_1 - (\gamma+\mu_1)I^*}{\mu_1}, \\ V^* &= \frac{\lambda_3 b_2 I^*}{\mu_2(\mu_2 + \lambda_3 I^*)}, \end{aligned}$$

where I^* is uniquely determined by the following equation

$$\frac{b_1 - (\gamma+\mu_1)I}{\mu_1}\left[\frac{\lambda_1}{\gamma+\mu_1} + \frac{\lambda_2\lambda_3 b_2}{\mu_2(\gamma+\mu_1)(\lambda_3 I + \mu_2)}\right] = 1. \tag{2.4}$$

Next, we study the stability of the endemic equilibrium.

Theorem 2.2.1. *If $R_0 > 1$, then the endemic equilibrium E^* of* (2.3) *is locally asymptotically stable in Γ.*

Similar to the proof of Theorem 2.1.1, Theorem 2.2.1 can also be obtained easily. This proof is omitted.

3. The delay model

In this section, we introduce a time delay into the systems (2.1) and (2.2) to represent the incubation time that the vectors need to become infectious. The model for the host population is exactly as before:

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$$\begin{cases} \dfrac{dS(t)}{dt} = b_1 - \lambda_1 S(t)I(t) - \lambda_2 S(t)V(t) - \mu_1 S(t), \\ \dfrac{dI(t)}{dt} = \lambda_1 S(t)I(t) + \lambda_2 S(t)V(t) - \gamma I(t) - \mu_1 I(t), \\ \dfrac{dR(t)}{dt} = \gamma I(t) - \mu_1 R(t). \end{cases} \tag{3.1}$$

The time delay is introduced in the system describing the dynamics of the vector. At time t only susceptible vectors that have bitten an infectious host τ time units ago, that is at time $t-\tau$, become infectious, provided that they have survived the incubation period of τ units, given that they were alive at time $t-\tau$ when they bit the infectious host. Thus the incidence term of infectious vectors changes from $\lambda_3 M(t)I(t)$ to $\lambda_3 M(t-\tau)I(t-\tau)$. The system for the dynamics of the vector takes the form:

$$\begin{cases} \dfrac{dM(t)}{dt} = b_2 - \lambda_3 M(t-\tau)I(t-\tau) - \mu_2 M(t), \\ \dfrac{dV(t)}{dt} = \lambda_3 M(t-\tau)I(t-\tau) - \mu_2 V(t). \end{cases} \tag{3.2}$$

The systems (3.1) and (3.2) satisfy also the initial conditions: $S(\theta) = S_0$, $I(\theta) = I_0$, $R(\theta) = \bar{R}_0$, $M(\theta) = M_0$, $V(\theta) = V_0$, $\theta \in [-\tau, 0]$. All the parameters are the same as in the systems (2.1) and (2.2) except for the positive constant τ which represents the length of the delay. As before, the total host population size $N_1(t)$ can be determined by $N_1(t) = S(t) + I(t) + R(t)$ or from the differential equation

$$N_1' = b_1 - \mu_1 N_1,$$

which is derived by adding the equations in (3.1). Similarly, the total number of vectors $N_2(t)$ can be determined by $N_2(t) = M(t) + V(t)$ or from the differential equation

$$N_2' = b_2 - \mu_2 N_2,$$

which is derived by adding the equations in (3.2). As before, the total population sizes of both host and vector populations are asymptotically constant, that is, $\lim_{t\to\infty} N_1(t) = \frac{b_1}{\mu_1}$ and $\lim_{t\to\infty} N_2(t) = \frac{b_2}{\mu_2}$. Then in our model we assume, without loss of generality, that $N_1(t) = \frac{b_1}{\mu_1}$, $N_2(t) = \frac{b_2}{\mu_2}$ for all $t \geqslant 0$ provided that $S_0 + I_0 + \bar{R}_0 = \frac{b_1}{\mu_1}$, $M_0 + V_0 = \frac{b_2}{\mu_2}$. The dynamics of systems (3.1) and (3.2) are qualitatively equivalent to the dynamics of the following system:

$$\begin{cases} \dfrac{dS(t)}{dt} = b_1 - \lambda_1 S(t)I(t) - \lambda_2 S(t)V(t) - \mu_1 S(t), \\ \dfrac{dI(t)}{dt} = \lambda_1 S(t)I(t) + \lambda_2 S(t)V(t) - \gamma I(t) - \mu_1 I(t), \\ \dfrac{dV(t)}{dt} = \lambda_3\left(\dfrac{b_2}{\mu_2} - V(t-\tau)\right)I(t-\tau) - \mu_2 V(t). \end{cases} \tag{3.3}$$

The values of R, M can be determined correspondingly by $R = \frac{b_1}{\mu_1} - S - I$ and $M = \frac{b_2}{\mu_2} - V$ or from $R' = \gamma I(t) - \mu_1 R(t)$ and $M' = b_2 - \lambda_3 M(t-\tau)I(t-\tau) - \mu_2 M(t)$.

With the same motivation as before, we study the system (3.3) in the same closed set Γ. It can be verified that Γ is positively invariant with respect to the system (3.3). As before, we denote by $\partial\Gamma$ and Γ^o the boundary and the interior of Γ in $\mathbf{R}^3$, respectively. Direct calculation shows that system (3.3) has the same disease-free equilibrium $E_0 = (\frac{b_1}{\mu_1}, 0, 0) \in \partial\Gamma$, as the system (2.3) which is also in the non-negative cone $\mathbf{R}^3_+$.

We introduce the reproduction number of the differential-delay model (3.3) which is given by a similar expression:

$$R_0 = \frac{b_1}{\mu_1}\left(\frac{\lambda_2}{(\gamma+\mu_1)}\frac{\lambda_3}{\mu_2}\frac{b_2}{\mu_2} + \frac{\lambda_1}{(\gamma+\mu_1)}\right).$$

The interpretation of the reproduction number is similar to the one before. The only difference consists in the observation that if one infectious host is introduced in a population of susceptible hosts and vectors, it will transmit the pathogen to $\frac{\lambda_3}{\mu_2}\frac{b_2}{\mu_2}$ susceptible vectors but they will survive the incubation period to infect other susceptible hosts. As in the ODE model, if $R_0 \leqslant 1$, the only equilibrium in Γ is the disease-free equilibrium E_0. If $R_0 > 1$, the disease-free equilibrium still exists and we will show in the next subsection that there is a unique endemic equilibrium E^* which exists in the interior of Γ.

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3.1. Existence and uniqueness of the endemic equilibrium

Assuming $R_0 > 1$, first we turn our attention to the existence and uniqueness of an endemic equilibrium. The derivation of this results largely parallels the one for the ODE model so we will only sketch it. An endemic equilibrium has components $E^* = (S^*, I^*, V^*)$ such that $I^* \neq 0$ and $V^* \neq 0$ and is a time-independent solution of the system (3.3). Since a time-independent solution has the same values at time t as at time $t - \tau$, the system satisfied by the endemic equilibrium of the system (3.3) is the same as the system (2.3). We apply the same techniques as before to obtain the unique positive endemic equilibrium $E^* = (S^*, I^*, V^*) \in \Gamma^o$ in the non-negative cone $\mathbf{R}^3_+$. Analogously to the ODE case we conclude that there exists a unique positive endemic equilibrium if and only if $R_0 > 1$.

3.2. Local and global stability of the disease-free equilibrium

In this section we turn to the study of the local and global stability of the disease-free equilibrium E_0 of the differential-delay model (3.3). We consider the local stability in two cases, namely when $R_0 < 1$ and when $R_0 > 1$.

Theorem 3.2.1. *The disease-free equilibrium E_0 of* (3.3) *is locally asymptotically stable in Γ if $R_0 < 1$. The disease-free equilibrium is unstable if $R_0 > 1$.*

Proof. Linearizing the system (3.3) around the disease-free equilibrium $E_0 = (\frac{b_1}{\mu_1}, 0, 0)$ we obtain one negative characteristic solution $\lambda = -\mu_1$ and the following transcendental characteristic equation for the disease-free equilibrium E_0 whose solutions (real and complex) give the remaining eigenvalues:

$$\lambda^2 + \left(\mu_1 + \gamma + \mu_2 - \frac{\lambda_1 b_1}{\mu_1}\right)\lambda + \mu_2\left(\mu_1 + \gamma - \frac{\lambda_1 b_1}{\mu_1}\right) - \frac{\lambda_2\lambda_3 b_1 b_2}{\mu_1\mu_2}e^{-\lambda\tau} = 0. \tag{3.4}$$

For $\tau = 0$, we obtain the same quadratic equation as in the ODE case. In that case we know from before that all eigenvalues of the characteristic equation (3.4) have negative real part. According to Hurwitz criterion, when $\tau = 0$, the disease-free equilibrium E_0 of (3.3) is locally asymptotically stable if $R_0 < 1$ and it is unstable if $R_0 > 1$.

To see the claim for the general non-zero delay $\tau \neq 0$, we first consider the case when $R_0 > 1$. We expect that in this case Eq. (3.4) has a positive root and the disease-free equilibrium is unstable. Indeed, to see this we rearrange Eq. (3.4) in the form

$$\lambda^2 + a_1\lambda = \mu_2(\mu_1 + \gamma)\left[\frac{\lambda_1 b_1}{(\gamma + \mu_1)\mu_1} + \frac{\lambda_2\lambda_3 b_1 b_2}{\mu_1\mu_2^2(\gamma + \mu_1)}e^{-\lambda\tau} - 1\right], \tag{3.5}$$

where a_1 is the coefficient of λ in (3.4). Suppose λ is real. Denote the left-hand side of Eq. (3.5) as $F(\lambda)$ and the right-hand side as $G(\lambda)$. We have that $F(0) = 0$ and $\lim_{\lambda\to\infty} F(\lambda) = \infty$. In contrast, the function $G(\lambda)$ is a decreasing function of λ and $G(0) = \mu_2(\gamma + \mu_1)[R_0 - 1] > 0$. Thus, the two functions must intersect for some $\lambda^* > 0$. Consequently, Eq. (3.4) has a positive real solution and the disease-free equilibrium is unstable.

Now, we turn to the case $R_0 < 1$. First, we notice that Eq. (3.5) does not have non-negative real roots since in this case $F(\lambda)$ is increasing for $\lambda \geqslant 0$ while $G(\lambda)$ is still decreasing function of λ but $G(0) = \mu_2(\gamma + \mu_1)[R_0 - 1] < 0$. Thus, if Eq. (3.4) has roots with non-negative real parts they must be complex and should have been obtained from a pair of complex conjugate roots which cross the imaginary axis. Consequently, Eq. (3.4) must have a pair of purely imaginary solutions for some $\tau > 0$. Assume that $\lambda = i\omega$, and without loss of generality we may assume that $\omega > 0$ is a root of Eq. (3.4). That is the case if and only if ω satisfies the equation

$$-\omega^2 + a_1\omega i + \mu_2\left(\gamma + \mu_1 - \frac{\lambda_1 b_1}{\mu_1}\right) - \frac{\lambda_2\lambda_3 b_1 b_2}{\mu_1\mu_2}\big(\cos(\omega\tau) - i\sin(\omega\tau)\big) = 0, \tag{3.6}$$

where a_1 again denotes the same coefficient in front λ. Separating the real and imaginary parts, we have the following system, satisfied by ω:

$$-\omega^2 + \mu_2\left(\gamma + \mu_1 - \frac{\lambda_1 b_1}{\mu_1}\right) = \frac{\lambda_2\lambda_3 b_1 b_2}{\mu_1\mu_2}\cos(\omega\tau), \tag{3.7}$$

$$\left(\gamma + \mu_1 + \mu_2 - \frac{\lambda_1 b_1}{\mu_1}\right)\omega = -\frac{\lambda_2\lambda_3 b_1 b_2}{\mu_1\mu_2}\sin(\omega\tau). \tag{3.8}$$

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To eliminate the trigonometric functions we square both sides of each equation above and we add the squared equations (3.7) and (3.8) to obtain the following forth order equation in ω:

$$\omega^4 + \mu_2^2\left(\gamma + \mu_1 - \frac{\lambda_1 b_1}{\mu_1}\right)^2 - 2\mu_2\left(\gamma + \mu_1 - \frac{\lambda_1 b_1}{\mu_1}\right)\omega^2 + \left(\gamma + \mu_1 + \mu_2 - \frac{\lambda_1 b_1}{\mu_1}\right)^2\omega^2 - \frac{\lambda_2^2\lambda_3^2 b_1^2 b_2^2}{\mu_1^2\mu_2^2} = 0. \tag{3.9}$$

To reduce this fourth order equation in ω to a quadratic equation we let $z = \omega^2$ and denote the coefficients as

$$a_{10} = \left(\gamma + \mu_1 + \mu_2 - \frac{\lambda_1 b_1}{\mu_1}\right)^2 - 2\mu_2\left(\gamma + \mu_1 - \frac{\lambda_1 b_1}{\mu_1}\right),$$
$$a_{20} = \mu_2^2\left(\gamma + \mu_1 - \frac{\lambda_1 b_1}{\mu_1}\right)^2 - \frac{\lambda_2^2\lambda_3^2 b_1^2 b_2^2}{\mu_1^2\mu_2^2}.$$

We can rewrite Eq. (3.9) as a quadratic equation in z:

$$z^2 + a_{10}z + a_{20} = 0. \tag{3.10}$$

Looking back at the coefficients of this quadratic equation, we see that we can expand the square in a_{10} and then completed again using the term outside it, while applying the formula for the difference of squares to a_{20} we obtain

$$a_{10} = \left(\gamma + \mu_1 - \mu_2 - \frac{\lambda_1 b_1}{\mu_1}\right)^2 + 2\mu_2\left(\gamma + \mu_1 - \frac{\lambda_1 b_1}{\mu_1}\right) > 0,$$
$$a_{20} = \mu_2(\gamma + \mu_1)(1 - R_0)\left[\mu_2\left(\gamma + \mu_1 - \frac{\lambda_1 b_1}{\mu_1}\right) + \frac{\lambda_2\lambda_3 b_1 b_2}{\mu_1\mu_2}\right] > 0.$$

Both inequalities above follow since $R_0 < 1$ which, in particular, implies that $\gamma + \mu_1 - \frac{\lambda_1 b_1}{\mu_1} > 0$. Thus, the two roots of Eq. (3.10) have positive product which means that they are complex or they are real but they have the same sign. In addition, they have negative sum which implies that they are either real and negative or complex conjugate with negative real parts. Consequently, Eq. (3.10) does not have positive real roots which lead to the conclusion that there is no ω such that $i\omega$ is a solution of Eq. (3.4). Therefore, it follows from Rouché's theorem [46, Theorem 9.17.4]) that the real parts of all the eigenvalues of the characteristic equation (3.4) are negative for all values of the delay $\tau \geqslant 0$. This implies that E_0 is locally asymptotically stable in Γ if $R_0 < 1$. This proves the theorem. □

In the next theorem we establish the global stability of the disease-free equilibrium.

Theorem 3.2.2. *If $R_0 \leqslant 1$, then the disease-free equilibrium E_0 of system* (3.3) *is globally asymptotically stable in Γ.*

Proof. We denote by x_t the translation of the solution of the system (3.3), that is, $x_t = (S(t+\theta), I(t+\theta), V(t+\theta))$ where $\theta \in [-\tau, 0]$. We introduce as before a new variable

$$L(x_t) = \mu_1 I(t) + \frac{\lambda_2 b_1}{\mu_2} V(t) + \frac{\lambda_2\lambda_3 b_1}{\mu_2}\int_{t-\tau}^{t}\left(\frac{b_2}{\mu_2} - V(\theta)\right) I(\theta)\, d\theta.$$

We note that $L \geqslant 0$ along the solutions of the system (3.3). This is because the solutions belong to Γ and therefore $V \leqslant b_2/\mu_2$. In addition $L = 0$ if and only if both I and V are equal to zero. The derivative of L along the solutions of (3.3) is given by

$$\begin{aligned} L'(x_t) &= \mu_1\lambda_1 S(t) I(t) + \mu_1\lambda_2 S(t) V(t) - \mu_1(\gamma + \mu_1) I(t) + \frac{\lambda_2\lambda_3 b_1}{\mu_2}\left(\frac{b_2}{\mu_2} - V(t-\tau)\right) I(t-\tau) - \lambda_2 b_1 V(t) \\ &\quad + \frac{\lambda_2\lambda_3 b_1}{\mu_2}\left(\frac{b_2}{\mu_2} - V(t)\right) I(t) - \frac{\lambda_2\lambda_3 b_1}{\mu_2}\left(\frac{b_2}{\mu_2} - V(t-\tau)\right) I(t-\tau) \\ &= \mu_1\lambda_1 S(t) I(t) + \mu_1\lambda_2 S(t) V(t) - \mu_1(\gamma + \mu_1) I(t) - \lambda_2 b_1 V(t) + \frac{\lambda_2\lambda_3 b_1 b_2}{\mu_2^2} I(t) - \frac{\lambda_2\lambda_3 b_1}{\mu_2} I(t) V(t) \end{aligned}$$

$$\leqslant \lambda_1 b_1 I(t) - \mu_1(\gamma+\mu_1)I(t) + \frac{\lambda_2\lambda_3 b_1 b_2}{\mu_2^2} I(t) - \frac{\lambda_2\lambda_3 b_1}{\mu_2} I(t)V(t)$$
$$= I(t)\left[\lambda_1 b_1 - \mu_1(\gamma+\mu_1) + \frac{\lambda_2\lambda_3 b_1 b_2}{\mu_2^2} - \frac{\lambda_2\lambda_3 b_1}{\mu_2} V(t)\right]$$
$$= I(t)\left[\mu_1(\gamma+\mu_1)(R_0-1) - \frac{\lambda_2\lambda_3 b_1}{\mu_2} V(t)\right] \leqslant 0,$$

where the first inequality follows from the same argument as in Theorem 2.1.2 and the last inequality follows from the assumption that $R_0 \leqslant 1$. Furthermore, in the case $R_0 < 1$ the derivative $L' = 0$ if and only if $I = 0$, while in the case $R_0 = 1$ the derivative $L' = 0$ if and only if $I = 0$ or $V = 0$. Therefore, the largest compact invariant set in $\{(S, I, V) \in \Gamma\colon\ L' = 0\}$, when $R_0 \leqslant 1$, is the singleton $\{E_0\}$. As before, LaSalle's invariance principle [45] implies that E_0 is globally asymptotically stable in Γ. This proves the theorem. □

3.3. Hopf bifurcation analysis

In this section we determine criteria for Hopf bifurcation to occur using the time delay τ as the bifurcation parameter. Throughout this subsection we will assume that $R_0 > 1$, that is, that the endemic equilibrium E^* exists. To study the stability of the endemic equilibrium E^*, we consider the linearization of system (3.3) at the point E^*. The following transcendental characteristic equation is obtained

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = e^{-\lambda\tau}\left(T_1\lambda^2 + T_2\lambda + T_3\right), \tag{3.11}$$

where the coefficients in this equation are expressed as follows:

$$a_1 = \lambda_1 I^* + \lambda_2 V^* - \lambda_1 S^* + 2\mu_1 + \mu_2 + \gamma,$$
$$a_2 = (\mu_1+\gamma)\left(\lambda_1 I^* + \lambda_2 V^*\right) + \mu_1\left(\mu_1 + \gamma - \lambda_1 S^*\right) + \mu_2\left(\mu_1 + \gamma - \lambda_1 S^* + \lambda_1 I^* + \lambda_2 V^* + \mu_1\right),$$
$$a_3 = \mu_2(\mu_1+\gamma)\left(\lambda_1 I^* + \lambda_2 V^*\right) + \mu_1\mu_2\left(\mu_1 + \gamma - \lambda_1 S^*\right),$$

$$T_1 = -\lambda_3 I^*,$$
$$T_2 = \mu_2\left(\mu_1 + \gamma - \lambda_1 S^*\right) - \left(\lambda_1 I^* + \lambda_2 V^* - \lambda_1 S^* + 2\mu_1 + \gamma\right)\lambda_3 I^*,$$
$$T_3 = \mu_1\mu_2\left(\mu_1 + \gamma - \lambda_1 S^*\right) - (\mu_1+\gamma)\left(\lambda_1 I^* + \lambda_2 V^*\right)\lambda_3 I^* - \lambda_3\mu_1 I^*\left(\gamma + \mu_1 - \lambda_1 S^*\right).$$

When $\tau = 0$, we obtain the same characteristic equation as in the ODE case. Consequently, all eigenvalues of the characteristic equation (3.11) have negative real parts as has been proved in Theorem 2.2.1. As a result of Hurwitz criterion, the endemic equilibrium E^* of (3.3) is locally asymptotically stable when $\tau = 0$. Furthermore, observe again that Eq. (3.11) does not have non-negative real solutions for any $\tau > 0$. First, we notice that once again we have $\gamma + \mu_1 - \lambda_1 S^* > 0$. This implies that $a_1 > 0$, $a_2 > 0$ and $a_3 > 0$. We rewrite Eq. (3.11) by moving the positive terms from the right-hand side to the left-hand side. The rewritten Eq. (3.11) takes the form

$$\lambda^3 + a_1\lambda^2 + \tilde{a}_2\lambda + \tilde{a}_3 = e^{-\lambda\tau}\left(T_1\lambda^2 + \tilde{T}_2\lambda + \tilde{T}_3\right), \tag{3.12}$$

where $\tilde{a}_2 = a_2 - e^{-\lambda\tau}\mu_2(\mu_1 + \gamma - \lambda_1 S^*)$ and $\tilde{a}_3 = a_3 - e^{-\lambda\tau}\mu_1\mu_2(\mu_1 + \gamma - \lambda_1 S^*)$. Therefore, $\tilde{a}_2 > 0$ and $\tilde{a}_3 > 0$ for all $\lambda \geqslant 0$ and $\tau > 0$. On the other hand $T_1 < 0$, $\tilde{T}_2 < 0$ and $\tilde{T}_3 < 0$. Consequently, the left-hand side in Eq. (3.12) is positive for all $\lambda \geqslant 0$ while the right-hand side is negative for all $\lambda \geqslant 0$ and the two cannot be equal for any $\lambda \geqslant 0$. We conclude that Eq. (3.11) cannot have real non-negative solutions. To rule out complex conjugate solutions with non-negative real parts we once again assume that $\lambda = i\omega$ with $\omega > 0$ is a root of Eq. (3.11). This is the case if and only if ω satisfies the following equation:

$$-i\omega^3 - a_1\omega^2 + a_2\omega i + a_3 = iT_2\omega\cos(\omega\tau) + \left(T_3 - T_1\omega^2\right)\cos(\omega\tau) + T_2\omega\sin(\omega\tau) - i\left(T_3 - T_1\omega^2\right)\sin(\omega\tau).$$

Separating again the real and imaginary parts, we have the following system that must be satisfied by ω:

$$a_2\omega - \omega^3 = T_2\omega\cos(\omega\tau) - \left(T_3 - T_1\omega^2\right)\sin(\omega\tau), \tag{3.13}$$
$$a_3 - a_1\omega^2 = \left(T_3 - T_1\omega^2\right)\cos(\omega\tau) + T_2\omega\sin(\omega\tau). \tag{3.14}$$

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We eliminate the trigonometric functions by squaring both sides of each equation above and adding the resulting equations. We obtain the following sixth degree equation for ω:

$$\omega^6 + \left(a_1^2 - 2a_2 - T_1^2\right)\omega^4 + \left(a_2^2 - 2a_1a_3 + 2T_1T_3 - T_2^2\right)\omega^2 + a_3^2 - T_3^2 = 0. \tag{3.15}$$

Since this equation contains only even powers of ω we can reduce the order by letting once again $z = \omega^2$. Then Eq. (3.15) becomes a third order equation in z:

$$z^3 + \alpha z^2 + \beta z + \vartheta = 0, \tag{3.16}$$

where we have used the following notation for the coefficients of Eq. (3.16):

$$\alpha = a_1^2 - 2a_2 - T_1^2,$$
$$\beta = a_2^2 - 2a_1a_3 + 2T_1T_3 - T_2^2,$$
$$\vartheta = a_3^2 - T_3^2.$$

In order to show that the endemic equilibrium E^* is locally stable we have to show that Eq. (3.16) does not have a positive real solution which might give the square of ω, that is, that Eq. (3.11) cannot have purely imaginary solutions. The lemma below establishes conditions leading to that result.

Lemma 3.3.1. *If $\alpha \geqslant 0$, $\vartheta \geqslant 0$ and $\beta > 0$, then Eq.* (3.16) *has no positive real roots.*

Proof. We denote the left-hand side of Eq. (3.16) as $h(z) = z^3 + \alpha z^2 + \beta z + \vartheta$. We take the derivative of $h(z)$ with respect to z, $h'(z) = 3z^2 + 2\alpha z + \beta$. We notice that for $z \geqslant 0$ the derivative $h'(z) > 0$, and therefore, the function $h(z)$ is an increasing function of $z \geqslant 0$. Since $h(0) = \vartheta \geqslant 0$, it follows that Eq. (3.16) has no positive real roots. This completes the proof of the lemma. □

Lemma 3.3.1 implies that there is no ω such that $i\omega$ is an eigenvalue of the characteristic equation (3.11). Therefore, by Rouché's theorem [46, Theorem 9.17.4], the real parts of all the eigenvalues of Eq. (3.11) are negative for all values of the delay $\tau \geqslant 0$. Summarizing the above analysis, we have the following theorem:

Theorem 3.3.1. *Assume that*

(i) $R_0 > 1$;
(ii) $\alpha \geqslant 0$, $\vartheta \geqslant 0$ *and* $\beta > 0$.

Then the endemic equilibrium E^* of (3.3) *is absolutely stable, that is, E^* is asymptotically stable for all values of the delay $\tau \geqslant 0$.*

Remark. Theorem 3.3.1 indicates that if the parameters satisfy conditions (i) and (ii), then the endemic equilibrium E^* of the system (3.3) is asymptotically stable for all values of the delay, that is, the endemic equilibrium E^* of the system (3.3) is asymptotically stable independently of the delay. However, we should point out that if the conditions in Theorem 3.3.1, particularly any of the inequalities in (ii), are not satisfied, then the stability of the endemic equilibrium depends on the delay value and as the delay varies the endemic equilibrium can lose stability which can lead to oscillations.

For example, if $\vartheta < 0$, then we have $h(0) < 0$ and $\lim_{z\to\infty} h(z) = \infty$. Thus Eq. (3.16) has at least one positive root, say z_0. Consequently, Eq. (3.15) has at least one positive root, denoted by $\omega_0 = \sqrt{z_0}$.

Now, we turn to the bifurcation analysis. We use the delay τ as bifurcation parameter. We view the solutions of Eq. (3.11) as functions of the bifurcation parameter τ. Let $\lambda(\tau) = \eta(\tau) + i\omega(\tau)$ be the eigenvalue of Eq. (3.19) such that for some initial value of the bifurcation parameter τ_0 we have $\eta(\tau_0) = 0$, and $\omega(\tau_0) = \omega_0$ (without loss of generality we may assume $\omega_0 > 0$). From Eqs. (3.13) and (3.14) we have

$$\tau_j = \frac{1}{\omega_0}\arccos\left(\frac{(a_1T_1 - T_2)\omega_0^4 + (a_2T_2 - a_3T_1 - a_1T_3)\omega_0^2 + a_3T_3}{T_2^2\omega_0^2 + \left(T_3 - T_1\omega_0^2\right)^2}\right) + \frac{2j\pi}{\omega_0}, \quad j = 0, 1, 2, \ldots.$$

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Also, we can verify that the following transversal condition

$$\frac{d\,\mathrm{Re}\,\lambda(\tau)}{d\tau}\Big|_{\tau=\tau_0} > 0$$

holds. By continuity, the real part of $\lambda(\tau)$ becomes positive when $\tau > \tau_0$ and the steady state becomes unstable. Moreover, a Hopf bifurcation occurs when τ passes through the critical value τ_0 (see [47]).

To apply the Hopf bifurcation theorem as stated in Marsden and McCracken [48] we state and prove the following theorem:

Theorem 3.3.2. *Suppose that ω_0 is the largest positive simple root of Eq.* (3.15). *Then $i\omega(\tau_0) = i\omega_0$ is a simple root of Eq.* (3.11) *and $\eta(\tau) + i\omega(\tau)$ is differentiable with respect to τ in a neighborhood of $\tau = \tau_0$.*

After computation we get that $i\omega_0$ is a simple root of Eq. (3.11), which is an analytic equation, and so, using the analytic version of the Implicit Function Theorem (Chow and Hale [49]), $\eta(\tau) + i\omega(\tau)$ is defined and analytic in a neighborhood of $\tau = \tau_0$.

Lemma 3.3.2. *Supposed that x_1, x_2, x_3 are the roots of equation*

$$g(x) = x^3 + \alpha x^2 + \beta x + \vartheta = 0 \quad (\beta < 0),$$

and x_3 is the largest positive simple root, then

$$\frac{dg(x)}{dx}\Big|_{x=x_3} > 0.$$

This proof is omitted.

To establish the Hopf bifurcation at $\tau = \tau_0$, we need to show that $\frac{d\,\mathrm{Re}\,\lambda(\tau)}{d\tau}|_{\tau=\tau_0} > 0$. From (3.11) differentiating with respect to τ, we get

$$\big(3\lambda^2 + 2a_1\lambda + a_2\big)\frac{d\lambda}{d\tau} = \big[-\tau e^{-\lambda\tau}\big(T_1\lambda^2 + T_2\lambda + T_3\big) + e^{-\lambda\tau}(2T_1\lambda + T_2)\big]\frac{d\lambda}{d\tau} - \lambda e^{-\lambda\tau}\big(T_1\lambda^2 + T_2\lambda + T_3\big).$$

This gives

$$\begin{aligned}\left(\frac{d\lambda}{d\tau}\right)^{-1} &= \frac{3\lambda^2 + 2a_1\lambda + a_2 + \tau e^{-\lambda\tau}(T_1\lambda^2 + T_2\lambda + T_3) - e^{-\lambda\tau}(2T_1\lambda + T_2)}{-\lambda e^{-\lambda\tau}(T_1\lambda^2 + T_2\lambda + T_3)}\\ &= \frac{3\lambda^2 + 2a_1\lambda + a_2}{-\lambda e^{-\lambda\tau}(T_1\lambda^2 + T_2\lambda + T_3)} + \frac{2T_1\lambda + T_2}{\lambda(T_1\lambda^2 + T_2\lambda + T_3)} - \frac{\tau}{\lambda}\\ &= \frac{2\lambda^3 + a_1\lambda^2 - a_3}{-\lambda^2(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3)} + \frac{T_1\lambda^2 - T_3}{\lambda^2(T_1\lambda^2 + T_2\lambda + T_3)} - \frac{\tau}{\lambda}.\end{aligned}$$

Thus,

$$\begin{aligned}\mathrm{Sign}\left\{\frac{d(\mathrm{Re}\,\lambda)}{d\tau}\right\}_{\lambda=i\omega_0} &= \mathrm{Sign}\left\{\mathrm{Re}\left(\frac{d\lambda}{d\tau}\right)^{-1}\right\}_{\lambda=i\omega_0}\\ &= \mathrm{Sign}\left\{\mathrm{Re}\left[\frac{2\lambda^3 + a_1\lambda^2 - a_3}{-\lambda^2(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3)}\right]_{\lambda=i\omega_0} + \mathrm{Re}\left[\frac{T_1\lambda^2 - T_3}{\lambda^2(T_1\lambda^2 + T_2\lambda + T_3)}\right]_{\lambda=i\omega_0}\right\}\\ &= \mathrm{Sign}\left\{\mathrm{Re}\left[\frac{-2\omega_0^3 i - a_1\omega_0^2 - a_3}{\omega_0^2(-\omega_0^3 i - a_1\omega_0^2 + a_2\omega_0 i + a_3)}\right] + \mathrm{Re}\left[\frac{-T_1\omega_0^2 - T_3}{-\omega_0^2(-T_1\omega_0^2 + T_2\omega_0 i + T_3)}\right]\right\}\\ &= \mathrm{Sign}\left\{\frac{2\omega_0^6 + (a_1^2 - 2a_2)\omega_0^4 - a_3^2}{\omega_0^2[(a_1\omega_0^2 - a_3)^2 + (\omega_0^3 - a_2\omega_0)^2]} + \frac{T_3^2 - T_1^2\omega_0^4}{\omega_0^2[(T_3 - T_1\omega_0^2)^2 + T_2^2\omega_0^2]}\right\}\end{aligned}$$

$$= \operatorname{Sign}\left\{ \frac{2\omega_0^6 + (a_1^2 - 2a_2 - T_1^2)\omega_0^4 + T_3^2 - a_3^2}{\omega_0^2[(a_1\omega_0^2 - a_3)^2 + (\omega_0^3 - a_2\omega_0)^2]} \right\}$$
$$= \operatorname{Sign}\left\{ \frac{3\omega_0^4 + 2(a_1^2 - 2a_2 - T_1^2)\omega_0^2 + (a_2^2 - 2a_1a_3 + 2T_1T_3 - T_2^2)}{(a_1\omega_0^2 - a_3)^2 + (\omega_0^3 - a_2\omega_0)^2} \right\}.$$

Since

$$h(z) = z^3 + \alpha z^2 + \beta z + \vartheta.$$

Thus,

$$\frac{dh(z)}{dz} = 3z^2 + 2\alpha z + \beta = 3z^2 + 2\big(a_1^2 - 2a_2 - T_1^2\big)z + \big(a_2^2 - 2a_1a_3 + 2T_1T_3 - T_2^2\big).$$

As ω_0 is the largest positive simple root of Eq. (3.15), from Lemma 3.3.2 we have

$$\frac{dh(z)}{dz}\Big|_{z=\omega_0^2} > 0.$$

Hence

$$\frac{d\,\mathrm{Re}\,\lambda}{d\tau}\Big|_{\omega=\omega_0,\tau=\tau_0} = \frac{\frac{dh(\omega_0^2)}{dz}}{(a_1\omega_0^2 - a_3)^2 + \big(\omega_0^3 - a_2\omega_0\big)^2} > 0.$$

The above analysis can be summarized into the following theorem:

Theorem 3.3.3. *Suppose that*

(i) $R_0 > 1$.

If either

(ii) $\vartheta < 0$

or

(iii) $\vartheta \geqslant 0$ *and* $\beta < 0$

is satisfied, and ω_0 *is the largest positive simple of Eq.* (3.15)*, then the endemic equilibrium* E^* *of the delay model* (3.3) *is asymptotically stable when* $\tau < \tau_0$ *and unstable when* $\tau > \tau_0$*, where*

$$\tau_0 = \frac{1}{\omega_0}\arccos\left(\frac{(a_1T_1 - T_2)\omega_0^4 + (a_2T_2 - a_3T_1 - a_1T_3)\omega_0^2 + a_3T_3}{T_2^2\omega_0^2 + (T_3 - T_1\omega_0^2)^2} \right),$$

when $\tau = \tau_0$*, a Hopf bifurcation occurs*; *that is a family of periodic solutions bifurcates from* E^* *as* τ *passes through the critical value* τ_0.

In this way, using time delay as a bifurcation parameter, Theorem 3.3.3 indicates that the delay model could exhibit Hopf bifurcation at a certain value τ_0 of the delay if the parameters satisfy the conditions (ii) or (iii). They show that the introduction of a time delay in the host-to-vector transmission term can destabilize the system and periodic solutions can arise through Hopf bifurcation.

4. Conclusion

We consider in this article a Ross–MacDonald type model where the population of the vector is described by a system for the susceptible and infected vector while the dynamics of the host is described by an SIR model. Mathematical analyzes of the model equations with regard to invariance of non-negativity, boundedness of solutions, nature

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of equilibria, permanence and global stability are analyzed. The basic reproduction number is obtained and it completely determines the dynamics of the ODE model. If $R_0 \leqslant 1$, the disease-free equilibrium is globally stable and the disease dies out. If $R_0 > 1$, a unique endemic equilibrium exists and is locally asymptotically stable in the interior of the feasible region. We determine criteria for Hopf bifurcation using the time delay as the bifurcation parameter based on the differential-delay model. They show that positive equilibrium is locally asymptotically stable when time delay is suitably small, while a loss of stability by a Hopf bifurcation can occur as the delay increases. Hopf bifurcation has helped us in finding the existence of a region of instability in the neighborhood of a non-zero endemic equilibrium where the population will survive undergoing regular fluctuations.

References

[1] S. Harrus, G. Baneth, Drivers for the emergence and re-emergence of vector-borne protozoal and bacterial diseases, Int. J. Parasitol. 35 (2005) 1309.
[2] A.A. Marfin, D.J. Gubler, West Nile Encephalitis: An emerging disease in the United States, Clin. Infect. Dis. 33 (2001) 1713.
[3] D.H. Molyneux, Patterns of change in vector-borne diseases, Ann. Trop. Med. Paras. 91 (1997) 827.
[4] D.J. Gubler, Resurgent vector-borne diseases as a global health problem, Emerg. Inf. Dis. 4 (1998) 442.
[5] R.T. Watson, et al., Environmental health implications of global climate change, J. Environ. Monit. 7 (2005) 834.
[6] A.A. Khasnis, M.D. Nettleman, Global warming and infectious disease, Arch. Med. Res. 36 (2005) 689.
[7] R.W. Sutherst, Global change and human vulnerability to vector-borne diseases, Microbiol. Rev. 17 (2004) 136.
[8] D.J. Rogers, The dynamics of vector-transmitted diseases in human communities, Philos. Trans. R. Soc. Lond. Ser. B 321 (1988) 513.
[9] F.E. McKenzie, Why model malaria?, Parasitol. Today 16 (2000) 511.
[10] D.J. Rodríguez, L. Torres-Sorando, Models of infectious diseases in spatially heterogeneous environments, Bull. Math. Biol. 63 (2001) 547.
[11] H. Ishikawa, A. Ishii, N. Nagai, H. Ohmae, M. Harada, S. Suguri, J. Leafasia, A mathematical model for the transmission of plasmodium vivax malaria, Parasitol. Int. 52 (2003) 81.
[12] J.C. Koella, R. Antia, Epidemiological models for the spread of anti-malarial resistance, Malar. J. 2 (2003).
[13] M.J. Mackinnon, Drug resistance models for malaria, Acta Trop. 94 (2005) 207.
[14] J.C. Koella, C. Boete, A model for the convolution of immunity and immune evasion in vector-borne diseases with implications for the epidemiology of malaria, Am. Nat. 161 (2003) 698.
[15] Z. Feng, D.L. Smith, F.E. McKenzie, S.A. Levin, Coupling ecology and evolution: Malaria and the S-gene across time scales, Math. Biosci. 189 (2004) 1.
[16] C.H. Calisher, Persistent emergence of dengue, Emerg. Inf. Dis. 11 (2005) 738.
[17] R. Cologna, P.M. Armstrong, R. Rico-Hesse, Selection for virulent dengue viruses occurs in human and mosquitoes, J. Virol. 79 (2005) 853.
[18] E.A. Newton, P. Reiter, A model of the transmission of dengue fever with an evaluation of the impact of ultra-low volume (ULV) insecticide applications on dengue epidemics, Am. J. Trop. Med. Hyg. 47 (1992) 709.
[19] L. Esteva, C. Vargas, Analysis of a dengue disease transmission model, Math. Biosci. 150 (1998) 131.
[20] Z. Feng, J.X. Velasco-Hernandez, Competitive exclusion in a vector-host model for the dengue virus, J. Math. Biol. 35 (1997) 523.
[21] L. Esteva, C. Vargas, A model for dengue disease with variable human population, J. Math. Biol. 38 (1999) 220.
[22] L. Esteva, C. Vargas, Coexistence of different serotype of dengue virus, J. Math. Biol. 46 (2003) 31.
[23] N.M. Ferguson, C.A. Donnelli, R.M. Anderson, Transmission dynamics and epidemiology of dengue: Insights from age-stratified servo-prevalence surveys, Philos. Trans. R. Soc. Lond. Ser. B 354 (1999) 757.
[24] N.M. Ferguson, R.M. Anderson, S. Gupta, The effect of antibody-dependent enhancement on the transmission dynamics and persistence of multiple-strain pathogens, Proc. Natl. Acad. Sci. USA 96 (1996) 790.
[25] I. Kawaguchi, A. Sasaki, M. Boots, Why are dengue virus serotype so distantly related? Enhancement and limiting serotype similarity between dengue virus strains, Proc. Biol. Sci. 270 (2003) 2241.
[26] L. Esteva, C. Vargas, Influence of vertical and mechanical transmission on the dynamics of dengue disease, Math. Biosci. 167 (2000) 51.
[27] P. Pongsumpun, I.M. Tang, A realistic age structured transmission model for dengue hemorrhagic fever in Thailand, Southeast. Asian J. Trop. Med. Public Health 32 (2001) 336.
[28] U.C. Chaturvedi, R. Shrivastava, R. Nigar, Dengue vaccines: Problems and prospects, Indian J. Med. Res. 121 (2005) 639.
[29] M. Derouich, A. Boutayeg, E.H. Twizell, A model of dengue fever, Biomed. Eng. Online 2 (2003) 4.
[30] L.T. Takahashi, N.A. Maidana, W.C. Ferreira Jr., P. Pulino, H.M. Yang, Mathematical models for the *Aedes aegypti* dispersal dynamics: Traveling waves by wing and wind, Bull. Math. Biol. 67 (2005) 509.
[31] L. Esteva, H. Mo Yang, Mathematical model to assess the control of Aedes aegypti mosquitoes by the sterile insect technique, Math. Biosci. 198 (2005) 132.
[32] G. Cruz-Pacheco, L. Esteva, J.A. Montaño-Hirose, C. Vargas, Modelling the dynamics of West Nile virus, Bull. Math. Biol. 67 (2005) 1157.
[33] C. Bowman, A.B. Gumel, P. van den Driessche, J. Wu, H. Zhu, A mathematical model for assessing control strategies against West Nile virus, Bull. Math. Biol. 67 (2005) 1107.
[34] T.C. Porco, A mathematical model of the ecology of Lyme disease, IMA J. Math. Appl. Med. Biol. 16 (1999) 261.
[35] T. Caraco, G. Gardner, W. Maniatty, E. Deelman, B.K. Szymanski, Lyme disease: Self-regulation and pathogen invasion, J. Theoret. Biol. 193 (1998) 561.
[36] M. Gosh, A. Pugliese, Seasonal population dynamics of ticks, and its influence on infection transmission: A semi-discrete approach, Bull. Math. Biol. 66 (2004) 1659.

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[37] R. Rosa, A. Pugliese, R. Norman, P.J. Hudson, Thresholds for disease persistence in models for tick-borne infections including non-viraemia transmission, extended feeding and tick aggregation, J. Theoret. Biol. 224 (2003) 359.
[38] H.G. Mwambi, Ticks and tick-borne diseases in Africa: A disease transmission model, IMA J. Math. Appl. Med. Biol. 19 (2002) 275.
[39] J.S. Brownstein, T.R. Holford, D. Fish, A climate-based model predicts the spatial distribution of the Lyme disease vector Ixodes scapularies in the United States, Environ. Health Perspect. 111 (2003) 1152.
[40] Y. Takeuchi, W. Ma, E. Beretta, Global asymptotic properties of a delay *SIR* epidemic model with finite incubation times, Nonlinear Anal. 42 (2000) 931.
[41] H. Inaba, H. Sekine, A mathematical model for Chagas disease with infection-age-dependent infectivity, Math. Biosci. 190 (2004) 39.
[42] H.R. Thieme, Convergence results and a Poincar Bendixson trichotomy for asymptotically autonomous differential equations, J. Math. Biol. 30 (1992) 755.
[43] R.M. Aderson, R.M. May, Population biology of infectious diseases: Part 1, Nature 280 (1979) 361.
[44] H.W. Hethcote, Qualitative analysis of communicable disease models, Math. Biosci. 28 (1976) 335.
[45] J. Diendonné, Foundations of Modern Analysis, Academic Press, New York, 1960.
[46] J.P. LaSalle, The Stability of Dynamical Systems, SIAM, Philadelphia, PA, 1976.
[47] B.D. Hassard, N.D.K. Azarinoff, Y.H. Wan, Theory and Applications of Hopf Bifurcation, Cambridge University, Cambridge, 1981.
[48] J.E. Marsden, M. McCracken, The Hopf Bifurcation and Its Applications, Springer-Verlag, New York, 1976.
[49] S.N. Chow, J.K. Hale, Methods of Bifurcation Theory, Springer-Verlag, New York, 1982.

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