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GLOBAL ANALYSIS OF A DELAYED VECTOR-BIAS MODEL FOR MALARIA TRANSMISSION WITH INCUBATION PERIOD IN MOSQUITOES

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Abstract. A delayed vector-bias model for malaria transmission with incubation period in mosquitoes is studied. The delay τ corresponds to the time necessary for a latently infected vector to become an infectious vector. We prove that the global stability is completely determined by the threshold parameter, $R_0(\tau)$. If $R_0(\tau) \leq 1$, the disease-free equilibrium is globally asymptotically stable. If $R_0(\tau) > 1$ a unique endemic equilibrium exists and is globally asymptotically stable. We apply our results to Ross-MacDonald malaria models with an incubation period (extrinsic or intrinsic).

1. **Introduction.** Historically the mathematical model first for malaria transmission is introduced by Ronald Ross [17] and further extended by George MacDonald [14], it has influenced both the modelling and the application of control strategies to vector-transmitted diseases.

The classic Ross-MacDonald model of malaria consists of an autonomous nonlinear two-dimensional system describing changes in the proportion of infected humans and mosquitoes. Subsequent contributions have been made to extend the Ross–MacDonald malaria models, and of these extensions is the vector-bias model, first proposed by Kingsolver [6]. It investigate the greater attractiveness of infectious humans to mosquitoes. Empirical evidence suggests that mosquitoes show some bias for humans infected with malaria [11]. Hosack and coauthors [2] incorporate an implicit incubation period in mosquitoes, the vector-bias model to study the dynamics of the disease in terms of a basic reproductive number.

Following Kingsolver [6] and Hosack and coauthors [2] works, Chamchod and Britton [1] extended the vector-bias model and define the attractiveness in a different way. The model assumes that the human and vector populations are divided into classes or states containing susceptible, infectious individuals. At time t, there are $H(t)$ susceptible humans, $I(t)$ infectious humans, $S(t)$ susceptible mosquitoes and $V(t)$ infectious mosquitoes. In this model, it assumes a *Susceptible-Infectious-Susceptible* model for the human population, under the assumption that the disease

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does not confer immunity to infected humans after recovery. For the mosquito population, a *Susceptible-Infectious* model under the assumption that mosquitoes never recover from infection. The compartmental model is described by the following system of differential equations:

$$\begin{aligned}
&\tfrac{d}{dt}H(t) = \mu N - \tfrac{q\beta H(t)V(t)}{pI(t)+qH(t)} - \mu H(t) + \delta I(t),\\
&\tfrac{d}{dt}I(t) = \tfrac{q\beta H(t)V(t)}{pI(t)+qH(t)} - (\mu+\delta)I(t),\\
&\tfrac{d}{dt}S(t) = \eta M - \tfrac{p\kappa S(t)I(t)}{pI(t)+qH(t)} - \eta S(t),\\
&\tfrac{d}{dt}V(t) = \tfrac{p\kappa S(t)I(t)}{pI(t)+qH(t)} - \eta V(t).
\end{aligned} \tag{1}$$

The system assumed that the total human population has constant size with a birth and death rate constant equal to $\mu > 0$ and thus $N = H(t) + I(t)$ for all time t. Similarly, $M = S(t) + V(t)$ is total mosquito populations at time t, and the natural death rate and birth rate are assumed to be equal, denoted by $\eta > 0$. The infected humans recover at a constant rate $\delta > 0$. The parameter $\beta > 0$ is the transmission probability from vector to human, and $\kappa > 0$ is the transmission probability from human to vector. The incidence terms for human population and mosquitoes populations are given by

$$\frac{q\beta H(t)V(t)}{pI(t)+qH(t)} \text{ and } \frac{p\kappa S(t)I(t)}{pI(t)+qH(t)},$$

respectively. Where $0 < p < 1$ is the probability that a mosquito arrives at a human at random and picks that human if he is infectious, $0 < q < 1$ if the human is susceptible, where $p > q$. If $p = q$, the system is delayed Ross-MacDonald model with incubation time in the vector population.

In the vector bias model (1) Chamchod and Britton [1] include explicitly the incubation period and adds a third class to the population of mosquitoes: Exposed class, $E(t)$. The model is formulated by the following system of delay differential equations:

$$\begin{aligned}
&\tfrac{d}{dt}H(t) = \mu N - \tfrac{q\beta H(t)V(t)}{pI(t)+qH(t)} - \mu H(t) + \delta I(t),\\
&\tfrac{d}{dt}I(t) = \tfrac{q\beta H(t)V(t)}{pI(t)+qH(t)} - (\mu+\delta)I(t),\\
&\tfrac{d}{dt}S(t) = \eta M - \tfrac{p\kappa S(t)I(t)}{pI(t)+qH(t)} - \eta S(t),\\
&\tfrac{d}{dt}E(t) = \tfrac{p\kappa S(t)I(t)}{pI(t)+qH(t)} - \tfrac{p\kappa S(t-\tau)I(t-\tau)}{pI(t-\tau)+qH(t-\tau)}e^{-\eta\tau} - \eta E(t),\\
&\tfrac{d}{dt}V(t) = \tfrac{p\kappa S(t-\tau)I(t-\tau)}{pI(t-\tau)+qH(t-\tau)}e^{-\eta\tau} - \eta V(t).
\end{aligned} \tag{2}$$

Here, the time delay τ is the extrinsic incubation period of parasites within the vector. The term $\frac{p\kappa S(t-\tau)I(t-\tau)}{pI(t-\tau)+qH(t-\tau)}e^{-\eta\tau}$ represents the mosquitoes who were exposed at time $t-\tau$ and survive to time t (with the death rate η), that is, represents the transformation of the exposed class, $E(t)$, to the infectious class, $V(t)$.

We now reduce system (2) to a three-dimensional system by eliminating $E(t)$, and $H(t)$ using the relation $I(t) = N - H(t)$. So in the rest of this paper, we will study the following retarded nonlinear system

$$\begin{aligned}
&\tfrac{d}{dt}S(t) = \eta M - \tfrac{\kappa p S(t)I(t)}{(p-q)I(t)+qN} - \eta S(t),\\
&\tfrac{d}{dt}V(t) = \tfrac{\kappa p S(t-\tau)I(t-\tau)}{(p-q)I(t-\tau)+qN}e^{-\eta\tau} - \eta V(t),\\
&\tfrac{d}{dt}I(t) = \beta\left(1 - \tfrac{pI(t)}{(p-q)I(t)+qN}\right)V(t) - (\mu+\delta)I(t).
\end{aligned} \tag{3}$$

The goal of this paper is study the global stability of delayed bias vector model (3) with incubation time in mosquitoes. We present the construction of Lyapunov functionals for this model.

The paper is organized as follows. In the next section, basic mathematical properties of the model are studied. The global asymptotic stability of the disease-free equilibrium is established in Section 3. The global asymptotic stability of the endemic equilibrium is obtained in Section 3. In Section 4, we apply our results to delayed Ross-MacDonald models.

2. **Basic properties.** We begin by presenting some notations that will be used throughout this paper. Let $\mathcal{C}([-\tau,0],\mathbb{R}^3_+)$ be the Banach space of continuous functions mapping the interval $[-\tau,0]$ into $\mathbb{R}^3_+$, where

$$\mathbb{R}^3_+ = \big\{(S,V,I)\in\mathbb{R}^3 : S\geq 0, V\geq 0, I\geq 0\big\}$$

It is biologically reasonable to consider the following initial conditions for (3):

$$S(\theta)=\varphi_1(\theta),\ V(\theta)=\varphi_2(\theta),\quad I(\theta)=\varphi_3(\theta),\ \theta\in[-\tau,0], \tag{4}$$

where $\varphi=(\varphi_1(0),\varphi_2(0),\varphi_3(0))\in\mathcal{C}$.

For model (3) to be mathematically tractable and biologically meaningful, it is important to prove that all the state variables (Susceptible mosquitoes, infectious mosquitoes, and infectious humans) are nonnegative for all time. We prove that all solutions of system (3) with positive initial data will remain positive for all time $t\geq 0$.

Theorem 2.1. *Let $(S(t),V(t),I(t))^T$ be any solution of system (3). Then under the initial conditions (4), all solutions $(S(t),V(t),I(t))^T$ are non-negative on $[0,+\infty)$ and ultimately bounded.*

Proof. If $S(t)$ were to lose its non-negativity on some local existence interval $[0,T)$ for some constant $T>0$, there would have to be a time at $t_1>0$ such that $S(t_1)=0$. By the first equation of (3) we have $\frac{d}{dt}S(t_1)=\eta M>0$. That means $S(t)<0$ for $t\in(t_1-\varepsilon,t_1)$, where ε is an arbitrarily small positive constant. This leads to a contradiction. It follows that $S(t)$ is always positive. Further, form the second and the third equations in (3), we have, respectively

$$V(t)=V(0)e^{-\eta t}+\int_0^t \frac{e^{-\eta\tau}\kappa pS(\phi-\tau)I(\phi-\tau)}{(p-q)I(\phi-\tau)+qN}e^{-\eta(t-\phi)}d\phi,$$
$$I(t)=I(0)e^{-(\mu+\delta)t}+\int_0^t \frac{\beta q(N-I(\phi))V(\phi)}{(p-q)I(\phi)+qN}e^{-(\mu+\delta)(t-\phi)}d\phi.$$

Then, it is easy to see that $V(t)$ and $I(t)$ are non-negative on $[0,T)$.

For $t\in[0,T)$, we have from (3) that $\frac{d}{dt}S(t)\leq \eta M-\eta S(t)$. Hence, well-known comparison principle implies that $S(t)$ is bounded on $[0,T)$, i.e., $M_1=\sup_{t\in[0,T)}S(t)<+\infty$. Therefore, we again have from (3) that on $[0,T)$,

- if $p=q$, then $\frac{dV(t)}{dt}\leq \frac{\kappa e^{-\eta\tau}M_1I(t-\tau)}{N}-\eta V(t)$, and $\frac{dI(t)}{dt}\leq \beta V(t)-(\mu+\delta)I(t)$,
- if $p>q$, then $\frac{dV(t)}{dt}\leq \frac{\kappa e^{-\eta\tau}pM_1}{(p-q)}-\eta V(t)$, and $\frac{dI(t)}{dt}\leq \beta V(t)-(\mu+\delta)I(t)$.

Hence, we also have from comparison principle that $V(t)$ and $I(t)$ are bounded on $[0,T)$. Boundedness of the solution $(S(t),V(t),I(t))^T$ implies that the local existence interval $[0,T)$ can be continued to $T=+\infty$. This proves that the solution $(S(t),V(t),I(t))^T$ is existent and non-negative on $[0,+\infty)$.

We define a function

$$U(t) = e^{-\eta\tau}S(t-\tau) + V(t) + \frac{\eta}{2\beta}I(t).$$

The time derivative of $U(t)$ computed along solutions of (3) is

$$\begin{aligned}\frac{dU(t)}{dt} &= e^{-\eta\tau}\eta M - e^{-\eta\tau}\eta S(t-\tau) - \frac{\eta}{2}V(t) - \frac{\eta(\mu+\delta)}{2\beta}I(t) - \frac{\eta p}{2}\frac{V(t)I(t)}{(p-q)I(t)+qN} \\ &\leq e^{-\eta\tau}\eta M - e^{-\eta\tau}\eta S(t-\tau) - \frac{\eta}{2}V(t) - \frac{\eta}{2\beta}\mu I(t).\end{aligned}$$

By non-negativity of the solution, it follows that

$$\frac{d}{dt}U(t) + \sigma U(t) \leq e^{-\eta\tau}\eta M.$$

where $\sigma = \min\{\frac{\eta}{2}, \mu\}$. This implies that $U(t)$ is ultimately bounded, and so are $S(t)$, $V(t)$ and $I(t)$. This completes the proof. □

The system (3) has two possible equilibria and they must satisfy the following algebraic equations:

$$\begin{aligned} 0 &= \eta M - \kappa\tfrac{pI}{(p-q)I+qN}S - \eta S, \\ 0 &= \kappa\tfrac{pI}{(p-q)I+qN}Se^{-\eta\tau} - \eta V, \\ 0 &= \beta\left(1 - \tfrac{pI}{(p-q)I+qN}\right)V - (\mu+\delta)I. \end{aligned} \tag{5}$$

We have the following result.

Theorem 2.2. *System (3) always has the disease-free equilibrium $E^\circ(M,0,0)$. If $p > q$ and $R_0(\tau) > 1$, there is a unique endemic equilibrium $E^*(S^*,V^*,I^*)$ where*

$$S^* = \frac{\eta M((p-q)I^* + qN)}{(\kappa p + (p-q)\eta)I^* + q\eta N},\ V^* = \frac{\kappa p M e^{-\eta\tau}I^*}{(\kappa p + (p-q)\eta)I^* + q\eta N},$$

$$I^* = \frac{-qNc + qN\sqrt{c^2 + 4\eta(p-q)(\kappa p + (p-q)\eta)\left(R_0(\tau)-1\right)}}{2(p-q)(\kappa p + (p-q)\eta)},$$

whit $c = \kappa p + q\eta R_0(\tau) + 2\eta(p-q)$.

For the case $p = q$, the endemic equilibrium state is shown in Section 4.

The threshold parameter for system (3) is

$$R_0(\tau) = \frac{\kappa\beta M p e^{-\eta\tau}}{qN\eta(\mu+\delta)}. \tag{6}$$

For vector-transmitted diseases, the basic reproductive number is more often reported as the square root of the threshold parameter, $\widehat{R_0}(\tau) = \sqrt{R_0(\tau)}$. The basic reproductive number of the disease, since it represents the average number of secondary infections caused by an infectious vector or infectious human.

Remark 1. Note that the basic reproductive number $\widehat{R_0}(\tau)$ is a decreasing function on time delay τ.

3. **Global asymptotic stability analysis.** In this section, we discuss the global stability of the disease-free equilibrium and the endemic equilibrium of system (3), respectively. The technique of proofs is the method of Lyapunov functionals. For simplicity, we will use the following notation in the proof $S = S(t)$, $V = V(t)$, $I = I(t)$, $g(I) = \frac{pI(t)}{(p-q)I(t)+qN}$, $S_i = S(t-i)$, $I_i = I(t-i)$ and $g(I_i) = \frac{pI(t-i)}{(p-q)I(t-i)+qN}$, where $i = \omega, \tau$.

The following result shows that if $R_0(\tau) \leq 1$ the disease-free equilibrium E° is stable globally.

Theorem 3.1. *If $p \geq q$ and $R_0(\tau) \leq 1$, then the disease-free equilibrium E° of (3) is globally asymptotically stable.*

Proof. Define the global Lyapunov functional

$$W(t) = e^{-\eta\tau}\int_M^S \left(1 - \frac{M}{\sigma}\right) d\sigma + V + \frac{\eta}{\beta} I + \kappa e^{-\eta\tau}\int_0^\tau g(I_\omega) S_\omega d\omega.$$

Computing the derivative of W along the solutions of system (3), we obtain

$$\begin{aligned}
\frac{dW}{dt} &= e^{-\eta\tau}\left(1 - \frac{M}{S}\right)\frac{dS}{dt} + \frac{dV}{dt} + \frac{\eta}{\beta}\frac{dI}{dt} + \kappa e^{-\eta\tau} g(I) S - \kappa e^{-\eta\tau} g(I_\tau) S_\tau, \\
&= \eta M e^{-\eta\tau}\left(2 - \frac{M}{S} - \frac{S}{M}\right) + \left(\frac{\kappa e^{-\eta\tau} M p}{(p-q)I + qN} - \frac{\eta(\mu+\delta)}{\beta}\right) I - \eta g(I) V, \\
&= -\eta M e^{-\eta\tau}\left(\sqrt{\frac{M}{S}} - \sqrt{\frac{S}{M}}\right)^2 - \frac{qN\eta(\mu+\delta)}{p\beta}\left(1 - \frac{\kappa\beta M p e^{-\eta\tau}}{qN\eta(\mu+\delta)}\right) g(I) \\
&- \eta\left(\frac{(\mu+\delta)(p-q)}{\beta p} I + V\right) g(I).
\end{aligned}$$

Rewritten $\frac{dW}{dt}$ in terms of threshold parameter (6), we have

$$\begin{aligned}
\frac{dW}{dt} &= -\eta M e^{-\eta\tau}\left(\sqrt{\frac{M}{S}} - \sqrt{\frac{S}{M}}\right)^2 - \frac{qN\eta(\mu+\delta)}{p\beta}\left(1 - R_0(\tau)\right) g(I) \\
&- \eta\left(\frac{(\mu+\delta)(p-q)}{\beta p} I + V\right) g(I).
\end{aligned}$$

If $p \geq q$ and $R_0(\tau) \leq 1$, then $\frac{dW}{dt} \leq 0$ any solution is also bounded on $[0, +\infty)$. If $R_0(\tau) < 1$, from Corollary 5.2 of [10], E° is globally asymptotically stable. Also, for $R_0(\tau) = 1$, $\frac{dW}{dt} = 0$ implies that $S(t) = M$ and $V(t) = I(t) = 0$. It is easy to show that $E^\circ(M, 0, 0)$ is the largest invariant set in $\left\{(S(t), V(t), I(t)) : \frac{dW}{dt} = 0\right\}$. By the classical Lyapunov-LaSalle invariance principle (Theorem 5.3 of [10]), E° is globally asymptotically stable. □

Remark 2. Korobeinikov constructed families of Lyapunov functions in [7],[8],[9] to prove global stability of the equilibrium states of infectious disease models and viral infection models. Recently, McCluskey and other authors studied the global stability of the equilibrium states of epidemiological models with delay [3], [5], [15], [16], [19], and in [4], [13], [18] analyzed virus dynamics models with intracellular delay, using a novel family of Lyapunov functionals.

In the following, we consider the global asymptotic stability of a unique endemic equilibrium E^*. Inspired by the works of Korobeinikov [7],[8],[9] and McCluskey [15], [16], in this paper, we construct a Lyapunov functional for endemic equilibrium.

Theorem 3.2. *If $p \geq q$ and $R_0(\tau) > 1$, then the endemic equilibrium E^* of (3) is globally asymptotically stable.*

Proof. Define the global Lyapunov functional for E^*,

$$L(t) = \widetilde{L}(t) + \kappa g(I^*) S^* e^{-\eta\tau} L_+(t)$$

where

$$\begin{aligned}\widetilde{L}(t) = e^{-\eta\tau} \int_{S^*}^{S} \left(1 - \frac{S^*}{\sigma}\right) d\sigma + \int_{V^*}^{V} \left(1 - \frac{V^*}{\sigma}\right) d\sigma \\ + \frac{\kappa g(I^*) S^* e^{-\eta\tau}}{(\mu+\delta) I^*} \int_{I^*}^{I} \left(1 - \frac{g(I^*)}{g(\sigma)}\right) d\sigma,\end{aligned}$$

and

$$L_+(t) = \int_0^\tau \left(\frac{g(I_\omega) S_\omega}{g(I^*) S^*} - 1 - \ln \frac{g(I_\omega) S_\omega}{g(I^*) S^*}\right) d\omega.$$

At endemic equilibrium, we have

$$\eta M = \eta S^* + \kappa g(I^*) S^*, \tag{7}$$

$$\eta = \kappa \frac{g(I^*) S^*}{V^*} e^{-\eta\tau}, \tag{8}$$

$$\beta = \beta g(I^*) + (\mu + \delta) \frac{I^*}{V^*}. \tag{9}$$

The time derivative of L computed along solutions of (3) is

$$\begin{aligned}\frac{d\widetilde{L}}{dt} = e^{-\eta\tau} \left(1 - \frac{S^*}{S}\right) (\eta M - \kappa g(I) S - \eta S) \\ + \left(1 - \frac{V^*}{V}\right) \left(\kappa g(I_\tau) S_\tau e^{-\eta\tau} - \eta V\right) \\ + \frac{\kappa g(I^*) S^* e^{-\eta\tau}}{(\mu+\delta) I^*} \left(1 - \frac{g(I^*)}{g(I)}\right) (\beta V - \beta g(I) V - (\mu + \delta) I),\end{aligned}$$

Using (7)–(9), we obtain

$$\begin{aligned}
\frac{d\widetilde{L}}{dt} &= e^{-\eta\tau}\left(1-\frac{S^*}{S}\right)\left(\eta\left(S^*-S\right)+\kappa g(I^*)S^*\left(1-\frac{g(I)S}{g(I^*)S^*}\right)\right)\\
&+\kappa g(I^*)S^*e^{-\eta\tau}\left(1-\frac{V^*}{V}\right)\left(\frac{g(I_\tau)S_\tau}{g(I^*)S^*}-\frac{V}{V^*}\right)\\
&+\frac{\kappa g(I^*)S^*e^{-\eta\tau}}{(\mu+\delta)I^*}\left(1-\frac{g(I^*)}{g(I)}\right)\left(\beta g(I^*)\left(1-\frac{g(I)}{g(I^*)}\right)V\right)\\
&+\kappa g(I^*)S^*e^{-\eta\tau}\left(1-\frac{g(I^*)}{g(I)}\right)\left(\frac{V}{V^*}-\frac{I}{I^*}\right),\\
&=\eta S^*e^{-\eta\tau}\left(2-\frac{S^*}{S}-\frac{S}{S^*}\right)+\kappa e^{-\eta\tau}\left(g(I_\tau)S_\tau-g(I)S\right)\\
&+\kappa g(I^*)S^*e^{-\eta\tau}\left(\frac{g(I)}{g(I^*)}-\frac{I}{I^*}\right)\left(1-\frac{g(I^*)}{g(I)}\right)\\
&+\frac{\kappa\beta g^2(I^*)S^*e^{-\eta\tau}}{(\mu+\delta)I^*}\left(2-\frac{g(I^*)}{g(I)}-\frac{g(I)}{g(I^*)}\right)V\\
&+\kappa g(I^*)S^*e^{-\eta\tau}\left(3-\frac{g(I_\tau)S_\tau V^*}{g(I^*)S^*V}-\frac{g(I^*)V}{g(I)V^*}-\frac{S^*}{S}\right).
\end{aligned}$$

It is easy to see that

$$\begin{aligned}
\frac{dL_+}{dt} &= \frac{d}{dt}\int_0^\tau\left(\frac{g(I_\omega)S_\omega}{g(I^*)S^*}-1-\ln\frac{g(I_\omega)S_\omega}{g(I^*)S^*}\right)d\omega,\\
&=\int_0^\tau\frac{d}{dt}\left(\frac{g(I_\omega)S_\omega}{g(I^*)S^*}-1-\ln\frac{g(I_\omega)S_\omega}{g(I^*)S^*}\right)d\omega,\\
&=-\int_0^\tau\frac{d}{d\omega}\left(\frac{g(I_\omega)S_\omega}{g(I^*)S^*}-1-\ln\frac{g(I_\omega)S_\omega}{g(I^*)S^*}\right)d\omega,\\
&=-\left[\frac{g(I_\omega)S_\omega}{g(I^*)S^*}-1-\ln\frac{g(I_\omega)S_\omega}{g(I^*)S^*}\right]_{\omega=0}^{\tau},\\
&=-\frac{g(I_\tau)S_\tau}{g(I^*)S^*}+\frac{g(I)S}{g(I^*)S^*}+\ln\frac{g(I_\tau)S_\tau}{g(I^*)S^*}-\ln\frac{g(I)S}{g(I^*)S^*},\\
&=-\frac{g(I_\tau)S_\tau}{g(I^*)S^*}+\frac{g(I)S}{g(I^*)S^*}+\ln\frac{g(I_\tau)S_\tau V^*}{g(I^*)S^*V}+\ln\frac{g(I^*)V}{g(I)V^*}+\ln\frac{S^*}{S}.
\end{aligned}$$

Since

$$\frac{dL}{dt}=\frac{d\widetilde{L}}{dt}+\kappa g(I^*)S^*e^{-\eta\tau}\frac{dL_+}{dt},$$

we obtain

$$\begin{aligned}\frac{dL}{dt} &= \eta S^* e^{-\eta\tau}\left(2-\frac{S^*}{S}-\frac{S}{S^*}\right)\\ &+\kappa g(I^*)S^* e^{-\eta\tau}\left(\frac{g(I)}{g(I^*)}-\frac{I}{I^*}\right)\left(1-\frac{g(I^*)}{g(I)}\right)\\ &+\frac{\kappa\beta g^2(I^*)S^* e^{-\eta\tau}}{(\mu+\delta)I^*}\left(2-\frac{g(I^*)}{g(I)}-\frac{g(I)}{g(I^*)}\right)V\\ &-\kappa g(I^*)S^* e^{-\eta\tau}\left(\frac{g(I_\tau)S_\tau V^*}{g(I^*)S^*V}-1-\ln\frac{g(I_\tau)S_\tau V^*}{g(I^*)S^*V}\right)\\ &-\kappa g(I^*)S^* e^{-\eta\tau}\left(\frac{g(I^*)V}{g(I)V^*}-1-\ln\frac{g(I^*)V}{g(I)V^*}\right)\\ &-\kappa g(I^*)S^* e^{-\eta\tau}\left(\frac{S^*}{S}-1-\ln\frac{S^*}{S}\right).\end{aligned}$$

Notice that

$$\left(\frac{g(I)}{g(I^*)}-\frac{I}{I^*}\right)\left(1-\frac{g(I^*)}{g(I)}\right)=-\frac{(p-q)qN(I-I^*)^2}{I^*((p-q)I^*+qN)((p-q)I+qN)}.$$

Thus,

$$\begin{aligned}\frac{dL}{dt} &= -\eta S^* e^{-\eta\tau}\left(\sqrt{\frac{S^*}{S}}-\sqrt{\frac{S}{S^*}}\right)^2-\kappa g(I^*)S^* e^{-\eta\tau}\left(\frac{S^*}{S}-1-\ln\frac{S^*}{S}\right)\\ &-\kappa g(I^*)S^* e^{-\eta\tau}\frac{(p-q)qN(I-I^*)^2}{I^*((p-q)I^*+qN)((p-q)I+qN)}\\ &-\frac{\kappa\beta g^2(I^*)S^* e^{-\eta\tau}}{(\mu+\delta)I^*}\left(\sqrt{\frac{g(I^*)}{g(I)}}-\sqrt{\frac{g(I)}{g(I^*)}}\right)^2 V\\ &-\kappa g(I^*)S^* e^{-\eta\tau}\left(\frac{g(I_\tau)S_\tau V^*}{g(I^*)S^*V}-1-\ln\frac{g(I_\tau)S_\tau V^*}{g(I^*)S^*V}\right)\\ &-\kappa g(I^*)S^* e^{-\eta\tau}\left(\frac{g(I^*)V}{g(I)V^*}-1-\ln\frac{g(I^*)V}{g(I)V^*}\right).\end{aligned}$$

Then $\frac{dL}{dt}\leq 0$ for all $S,V,I>0$. By Corollary 5.2 of [10], solutions limit to $\mathbb{M}$, the largest invariant subset of $\left\{\frac{dL}{dt}=0\right\}$. Furthermore, $\frac{dL}{dt}=0$ if and only if $S(t)=S(t-\tau)=S^*$, $V(t)=V^*$ and $I(t)=I(t-\tau)=I^*$. Therefore the largest compact invariant set in $\mathbb{M}$ is the singleton $\{E^*\}$, where E^* is the endemic equilibrium. This shows that $\lim_{t\to\infty}(S(t),V(t),I(t))=(S^*,V^*,I^*)$. By the classical Lyapunov-LaSalle invariance principle (Theorem 5.3 of [10]), then E^* is globally asymptotically stable. □

Remark 3. Huang and coauthors, in [4] and [5] constructed Lyapunov functionals and analyzed a class of viral infection and epidemic models in three dimensional with a discrete delay, that incorporate a generalized nonlinear incidence rate. The model (3) does not correspond to the structures of the equations of the study systems in [4] and [5].

4. **Delayed Ross-MacDonald malaria models.** Recently, Lou and Zhao [12] proposed the following malaria model with incubation period in the vector population:

$$\begin{aligned} \tfrac{d}{dt}S(t) &= \eta M - \tfrac{\kappa}{N}S(t)I(t) - \eta S(t),\\ \tfrac{d}{dt}V(t) &= \tfrac{\kappa}{N}S(t-\tau)I(t-\tau)e^{-\eta\tau} - \eta V(t),\\ \tfrac{d}{dt}I(t) &= \tfrac{\beta}{N}\left(N - I(t)\right)V(t) - (\mu+\delta)I(t). \end{aligned} \tag{10}$$

When $p = q$ the system (3) becomes a Ross-MacDonald model (10) with incubation period in mosquitoes. Hence, from Theorems 3.1 and 3.2, we obtain the global dynamic behavior for system (10):

Theorem 4.1. *Let the threshold parameter $R_0^v(\tau)$ be defined by*

$$R_0^v(\tau) = \frac{\kappa\beta M e^{-\eta\tau}}{N\eta(\mu+\delta)}.$$

If $R_0^v(\tau) \le 1$, then the disease-free equilibrium $E_v^\circ(M,0,0)$ of (10) is globally asymptotically stable. If $R_0^v(\tau) > 1$, then the endemic equilibrium

$$E_v^*\left(\eta MN/(\kappa I^* + \eta N), \kappa M e^{-\eta\tau} I^*/(\kappa I^* + \eta N), \eta N(R_0^v(\tau)-1)/(\eta R_0^v(\tau) + \kappa)\right)$$

of (10) is globally asymptotically stable.

We proposed the Ross-MacDonald model with incubation period in the human population:

$$\begin{aligned} \tfrac{d}{dt}H(t) &= \mu N - \tfrac{\beta}{N}H(t)V(t) - \mu H(t),\\ \tfrac{d}{dt}I(t) &= \tfrac{\beta}{N}H(t-\nu)V(t-\nu)e^{-\mu\nu} - \mu I(t),\\ \tfrac{d}{dt}V(t) &= \tfrac{\kappa}{N}\left(M - V(t)\right)I(t) - \eta V(t). \end{aligned} \tag{11}$$

Here, the time delay ν is the intrinsic incubation period of malaria parasites in humans, the system (11) leads to the threshold parameter into the following form:

$$R_0^h(\nu) = \frac{\kappa\beta M e^{-\mu\nu}}{N\eta\mu}.$$

We exploit the "isomorphism" between system (11) and the delayed model (10), and we prove the global properties of model (11). From Theorem 4.1, we obtain the following corollary.

Corollary 1. *If $R_0^h(\nu) \le 1$, then the disease-free equilibrium $E_h^\circ(N,0,0)$ of (11) is globally asymptotically stable. If $R_0^h(\nu) > 1$, then the endemic equilibrium*

$$E_h^*\left(\mu N^2/(\beta V^* + \mu N), \beta N V^*/e^{\mu\nu}(\beta V^* + \mu N), \mu\eta N(R_0^h(\nu)-1)/\beta(\kappa e^{-\mu\nu} + \eta)\right)$$

of (11) is globally asymptotically stable.

5. **Conclusion.** Our main goal was to investigate the qualitative behavior of the vector-bias model, this model incorporates the effect that infectious humans may be more attractive to mosquitoes than susceptible humans.

It is known that the method of Lyapunov functionals plays a central role in the study of the global stability of retarded nonlinear systems. However, it is generally difficult to construct Lyapunov functionals that satisfy the condition required in the stability theory.

In this paper, by constructing two suitable Lyapunov functionals, we found the sufficient and necessary conditions of the global stability for the disease-free equilibrium and endemic equilibrium of vector-bias model. The results show that, for the vector-bias malaria model (3), the time delay has no effect on both global asymptotic properties of the disease-free equilibrium and global asymptotic properties of the endemic equilibrium.

Our results solve the open problem of global stability of system (3) developed in [1], and extended our result to delayed models with extrinsic incubation period (10) or intrinsic incubation period (11).

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