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GLOBAL STABILITY ANALYSIS OF MALARIA TRANSMISSION DYNAMICS WITH VIGILANT COMPARTMENT

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Abstract. A deterministic compartmental model for the transmission dynamics of malaria incorporating vigilant human compartment is studied. The model is qualitatively analyzed to investigate its asymptotic behavior with respect to the equilibria. It is shown, using a linear Lyapunov function, that the disease-free equilibrium is globally asymptotically stable when the associated basic reproduction number is less than the unity. When the basic reproduction number is greater than the unity, under certain specifications on the model parameters, we prove the existence of a globally asymptotically stable endemic equilibrium with the aid of a suitable nonlinear Lyapunov function.

1. Introduction

Malaria is one of the most common infectious diseases that are posing great public health problem throughout the six World Health Organization regions today. It has been reported that an estimated 3.3 billion people across the globe are at risk of being infected with malaria while the burden is heaviest in the WHO African Region accounting for an estimated 90% of all malaria deaths [20]. The disease is caused by five species of parasites belonging to the genus *Plasmodium*, namely *P. falciparum*, *P. vivax*, *P. ovale*, *P. malariae* and *P. knowlesi*. Of these species, *P. falciparum* and *P. vivax* pose the greatest public health challenge [21].

Malaria is spread between humans via the bite of female *Anopheles* mosquitoes and it is characterized by symptoms which may include chills, illness, headaches, body aches, anemia, nausea and vomiting among others. However, the disease can be avoided and treated by adopting interventions such as vector control (which prevents mosquito from acquiring or passing on an infection through use of insecticide-treated mosquito nets (ITNs) or indoor residual spraying (IRS)); chemoprevention (which inhibits infections establishing themselves in humans); and case management (which includes prompt diagnosis and appropriate treatment) (see [11, 21]).

Deterministic compartmental models describing the transmission of malaria between human and mosquito populations have been developed with attempts to facilitate the understanding of the mechanisms involved in the transmission dynamics of the disease (see, [5, 14] and the references therein). The impact of some

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of the intervention strategies mentioned earlier are investigated as control functions using time dependent models (non-autonomous systems) (see, for instance, [1, 2, 11]). The choice of compartments used in mathematical models varies and largely depends on the observed features of the particular disease being modeled [6]. Recently, an autonomous discrete-age-structured model proposed in [15] incorporated vigilant human compartment into the malaria transmission dynamics. This vigilant compartment comprises individuals who adhere to the intervention strategies with a view to preventing further spread of the disease in the population.

The behavior of a dynamical system as its solution approaches a given equilibrium is an asymptotic stability property. In the literature of epidemic models, establishing global (unlike local) asymptotic stability results is usually a nontrivial and challenging mathematical problem [17]. Methods such as Dulac's criterion with Poincaré-Bendixson theorem (see [19, 22]); geometrical approach (see [3, 12]); comparison theorem (see [5, 13]); technique used in [4]; and Lyapunov method (see, for instance, [7, 16, 8, 9]) can be used to study global stability properties of disease models. Lyapunov method, whose role cannot be overemphasized in this direction, requires the construction of a suitable Lyapunov function which is positive definite and whose value never increases along the solution paths of the system. In this paper, the method of Lyapunov function is sought to extend the analysis in [15] beyond only a small region near the disease-free and endemic equilibria of the system.

This article is organized as follows. In section 2, the description of the formulated model is given. The global asymptotic stability of the disease-free equilibrium and endemic equilibrium are explored in Sections 3 and 4 respectively. Also we provide concluding remarks.

2. Model description

We consider the normalized form of the malaria transmission dynamics obtained in [15] with the concept that humans may not have equal likelihood of being infected with malaria parasites. The human population at discrete-age a_i (for $i = 0, 1, 2, \ldots, L$ and a_L being the maximum age) and at time t is subdivided into susceptible $S_h(t, a_i)$; exposed $E_h(t, a_i)$; infectious $I_h(t, a_i)$; and vigilant $V_h(t, a_i)$ individuals. On the other hand, the mosquito population is subdivided into susceptible $S_m(t)$; exposed $E_m(t)$; and infectious $I_m(t)$ mosquitoes.

It is assumed that susceptible humans are recruited into the population at a rate $\lambda_h(a_i)$ whose fraction $\tau\lambda_h(a_i)$ are recruited vigilant. Susceptible humans acquire malaria through contact with infectious mosquitoes and become exposed humans at rate $b\beta_h(a_i)\sigma$, where b is the biting rate, $\beta_h(a_i)$ is the probability that bite produces infection in human and σ is the contact rate of mosquito per human per unit time. The per capita rate of progression of exposed individuals is given by $\alpha(a_i)$ whose fraction θ can become vigilant upon treating malaria infection (e.g., *P. vivax*) usually at the dormant liver stage [21] while the remaining fraction $(1 - \theta)$ progresses to the infectious compartment following the development of the disease symptoms. Infectious humans become vigilant at per capita recovery rate $\gamma(a_i)$. It is further assumed that individuals in the vigilant compartment firmly adhere to the intervention strategies and cannot be re-infected.

The mosquito population is increased at recruitment rate λ_m assumed to be susceptible. Following effective contact with infectious humans, susceptible mosquitoes

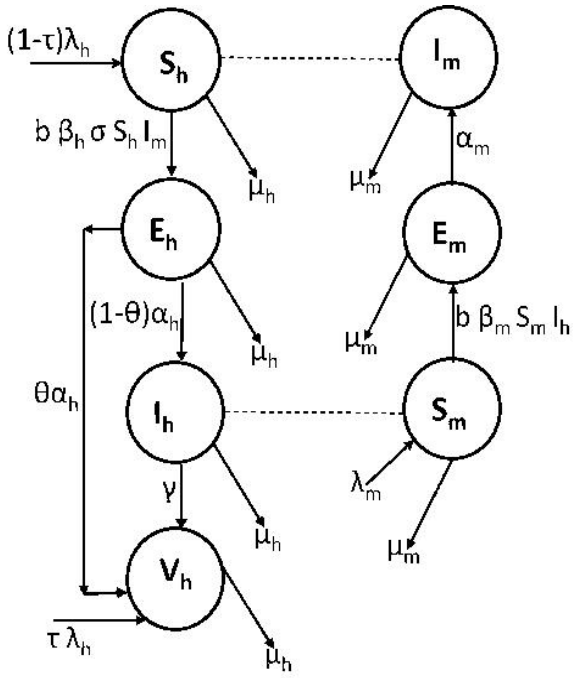

FIGURE 1. Schematic diagram of the malaria transmission dynamics regardless of the discrete-age a_i.

acquire infection and become exposed at rate $b\beta_m$, where β_m is the probability that bite produces infection in the mosquito. Exposed mosquitoes progress to become infectious at per capita rate α_m. The per capita natural death rates of humans and mosquitoes are, respectively, given by $\mu_h(a_i)$ and μ_m. The diagrammatic representation of the foregoing assumptions can be seen in Figure 1 and the corresponding model is governed by the following system of ordinary differential equations:

$$\begin{aligned}
\frac{dS_h(t,a_i)}{dt} &= (1-\tau)\lambda_h(a_i) - \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h(t,a_i)I_m - \mu_h(a_i)S_h(t,a_i),\\
\frac{dE_h(t,a_i)}{dt} &= \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h(t,a_i)I_m - (\alpha_h(a_i)+\mu_h(a_i))E_h(t,a_i),\\
\frac{dI_h(t,a_i)}{dt} &= \sum_{i=0}^{L}(1-\theta)\alpha_h(a_i)E_h(t,a_i) - (\gamma(a_i)+\mu_h(a_i)I_h(t,a_i),\\
\frac{dV_h(t,a_i)}{dt} &= \tau\lambda_h(a_i) + \theta\alpha_h(a_i)E_h(t,a_i) + \gamma(a_i)I_h(t,a_i) - \mu_h(a_i)V_h(t,a_i),\\
\frac{dS_m}{dt} &= \lambda_m - b\beta_m S_m(t)I_h(t,a_i) - \mu_m S_m(t),\\
\frac{dE_m}{dt} &= b\beta_m S_m(t)I_h(t,a_i) - (\alpha_m+\mu_m)E_m(t),\\
\frac{dI_m}{dt} &= \alpha_m E_m(t) - \mu_m I_m(t)
\end{aligned} \tag{2.1}$$

The parameters and variables of the formulated model (2.1) are nonnegative since the model monitors human and mosquito populations. Further, it is supposed that the recruitment terms for human and mosquito populations are balanced by the natural deaths $\mu_h(a_i)$ and μ_m respectively. So that system (2.1) can be analyzed in a positively invariant region $\mathfrak{D} = \mathfrak{D}_h \times \mathfrak{D}_m \subset \mathbb{R}^4_+ \times \mathbb{R}^3_+$ with

$$\mathfrak{D}_h = \big\{(S_h, E_h, I_h, V_h) \in \mathbb{R}^4_+ : S_h + E_h + I_h + V_h = 1\big\},$$
$$\mathfrak{D}_m = \big\{(S_m, E_m, I_m) \in \mathbb{R}^3_+ : S_m + E_m + I_m = 1\big\}.$$

A key notion in the analysis of infectious disease models is the basic reproduction number $\mathcal{R}_0$, an epidemiological threshold that determines whether disease dies out or persists in the population. Following [18], $\mathcal{R}_0$ for system (2.1) is given by

$$\mathcal{R}_0 = \Big(\sum_{i=0}^{L} \frac{b^2\beta_h(a_i)\sigma\alpha_h(a_i)\beta_m\alpha_m(1-\theta)(1-\tau)}{(\alpha_h(a_i)+\mu_h(a_i))(\gamma(a_i)+\mu_h(a_i))(\alpha_m+\mu_m)\mu_m}\Big)^{1/2} \tag{2.2}$$

The basic reproduction number (2.2) represents the average number of secondary cases (humans/mosquitoes) generated by one infectious case (mosquito/human) during the period of infectiousness in a completely susceptible (humans/mosquitoes) population.

3. Global stability of disease-free equilibrium

The steady-state solution of the model (2.1), the disease-free equilibrium, is given by

$$\mathcal{E}_0 = (1-\tau, 0, 0, \tau, 1, 0, 0) \tag{3.1}$$

The following result establishes the global asymptotic behavior of system (2.1) around (3.1) determined by the basic reproduction number (2.2).

Theorem 3.1. *The disease-free equilibrium* (3.1) *of model* (2.1) *is globally asymptotically stable in* $\mathfrak{D}$ *whenever* $\mathcal{R}_0 \le 1$.

Proof. Consider the linear Lyapunov function of the form

$$\mathfrak{F} = kE_h + \frac{I_h}{\gamma(a_i)+\mu_h(a_i)} + \frac{E_m}{b\beta_m} + \Big(\frac{\alpha_m+\mu_m}{b\beta_m\alpha_m}\Big)I_m, \tag{3.2}$$

where

$$k = \frac{\alpha_h(a_i)(1-\theta)}{(\alpha_h(a_i)+\mu_h(a_i))(\gamma(a_i)+\mu_h(a_i))}.$$

The time derivative of (3.2) along the solutions of the system (2.1) is

$$\begin{aligned}
\dot{\mathfrak{F}} = &\sum_{i=0}^{L}\Big(\frac{\alpha_h(a_i)(1-\theta)}{(\alpha_h(a_i)+\mu_h(a_i))(\gamma(a_i)+\mu_h(a_i))}\Big)\\
&\times [b\beta_h(a_i)\sigma S_h(t,a_i)I_m - (\alpha_h(a_i)+\mu_h(a_i))E_h(t,a_i)]\\
&+\sum_{i=0}^{L}\Big(\frac{1}{\gamma(a_i)+\mu_h(a_i)}\Big)\\
&\times [(1-\theta)\alpha_h(a_i)E_h(t,a_i) - (\gamma(a_i)+\mu_h(a_i))I_h(t,a_i)]\\
&+\big(\frac{1}{b\beta_m}\big)[b\beta_m S_m I_h(t,a_i) - (\alpha_m+\mu_m)E_m]\\
&+\big(\frac{\alpha_m+\mu_m}{b\beta_m\alpha_m}\big)[\alpha_m E_m - \mu_m I_m]
\end{aligned} \tag{3.3}$$

Algebraic expansion of (3.3) and further simplification yield

$$\begin{aligned}
\dot{\mathfrak{F}} &= \sum_{i=0}^{L}\Big(\frac{b\beta_h(a_i)\sigma\alpha_h(a_i)(1-\theta)}{(\alpha_h(a_i)+\mu_h(a_i))(\gamma(a_i)+\mu_h(a_i))}\Big)S_h(t,a_i)I_m \\
&\quad -\sum_{i=0}^{L}\Big(\frac{\alpha_h(a_i)(1-\theta)}{(\gamma(a_i)+\mu_h(a_i))}\Big)E_h(t,a_i) - I_h(t,a_i) + S_m I_h(t,a_i) \\
&\quad +\sum_{i=0}^{L}\Big(\frac{\alpha_h(a_i)(1-\theta)}{(\gamma(a_i)+\mu_h(a_i))}\Big)E_h(t,a_i) - \Big(\frac{(\alpha_m+\mu_m)\mu_m}{b\beta_m\alpha_m}\Big)I_m \\
&\le \sum_{i=0}^{L}\Big(\frac{b\beta_h(a_i)\sigma\alpha_h(a_i)(1-\theta)(1-\tau)}{(\alpha_h(a_i)+\mu_h(a_i))(\gamma(a_i)+\mu_h(a_i))}\Big)I_m - \Big(\frac{(\alpha_m+\mu_m)\mu_m}{b\beta_m\alpha_m}\Big)I_m \\
&= \Big(\sum_{i=0}^{L}\frac{b\beta_h(a_i)\sigma\alpha_h(a_i)(1-\theta)(1-\tau)}{(\alpha_h(a_i)+\mu_h(a_i))(\gamma(a_i)+\mu_h(a_i))} - \frac{(\alpha_m+\mu_m)\mu_m}{b\beta_m\alpha_m}\Big)I_m \\
&= \Big(\frac{(\alpha_m+\mu_m)\mu_m}{b\beta_m\alpha_m}\Big)(\mathcal{R}_0^2-1)I_m
\end{aligned}$$

It follows that $\dot{\mathfrak{F}} \le 0$ whenever $\mathcal{R}_0 \le 1$ with $\dot{\mathfrak{F}} = 0$ if and only if $I_m = 0$. Further, one sees that $(S_h(t,a_i), E_h(t,a_i), I_h(t,a_i), V_h(t,a_i), S_m(t), E_m(t))$ tends to $((1-\tau),0,0,0,1,0)$ as $t \to \infty$ since $I_m \to 0$ as $t \to \infty$. By LaSalle's invariance principle [10], one concludes that every solution of the model (2.1) in $\mathfrak{D}$ approaches the disease-free equilibrium (3.1) as $t \to \infty$. Hence $\mathcal{E}_0$ is globally asymptotically stable. □

The epidemiological implication of Theorem 3.1 shows that malaria can be controlled or eliminated from the community if the associated basic reproduction number of the model (2.1) is less than the unity.

4. Global stability of endemic equilibrium

The disease-present (endemic) equilibrium of the model (2.1) is referred to the steady-state solution where at least one of the infected compartments is nonzero. Let the arbitrary endemic equilibrium of the model (2.1) be represented by

$$\mathcal{E}_1 = (S_h^{**}(a_i), E_h^{**}(a_i), I_h^{**}(a_i), V_h^{**}(a_i), S_m^{**}, E_m^{**}, I_m^{**}),$$

considering the fact that the method of Lyapunov function requires no knowledge of solutions in establishing the global stability [19]. However, see [15], for possible existence of the endemic equilibrium $\mathcal{E}_1$ of the model (2.1) at $\mathcal{R}_0 > 1$.

Here, the global asymptotic stability of the endemic equilibrium $\mathcal{E}_1$ is explored for a special case of the model (2.1) where $\tau = \theta = 0$. Let

$$\mathfrak{D}_0 = \big\{(S_h, E_h, I_h, V_h, S_m, E_m, I_m) \in \mathfrak{D} : E_h = I_h = V_h = E_m = I_m = 0\big\}$$

be the stable manifold of the disease-free equilibrium $\mathcal{E}_0$. The following result is claimed.

Theorem 4.1. *The endemic equilibrium of the model* (2.1) *is globally asymptotically stable in* $\mathfrak{D}\backslash\mathfrak{D}_0$ *whenever* $\mathcal{R}_0|_{\tau=\theta=0} > 1$

Proof. Consider the Goh-Volterra nonlinear Lyapunov function

$$\begin{aligned}
\mathcal{V} &= S_h(t,a_i) - S_h^*(a_i) - S_h^*(a_i)\ln\Big(\frac{S_h(t,a_i)}{S_h^*(a_i)}\Big)\\
&\quad + E_h(t,a_i) - E_h^*(a_i) - E_h^*(t,a_i)\ln\Big(\frac{E_h(t,a_i)}{E_h^*(a_i)}\Big)\\
&\quad + \sum_{i=0}^{L}\frac{\alpha_h(a_i)+\mu_h(a_i)}{\alpha_h(a_i)}\Big[I_h(t,a_i) - I_h^*(a_i) - I_h^*(a_i)\ln\Big(\frac{I_h(t,a_i)}{I_h^*(a_i)}\Big)\Big]\\
&\quad + S_m - S_m^* - S_m^*\ln\Big(\frac{S_m}{S_m^*}\Big) + E_m - E_m^* - E_m^*\ln\big(\frac{E_m}{E_m^*}\big)\\
&\quad + \frac{\alpha_m+\mu_m}{\alpha_m}\Big[I_m - I_m^* - I_m^*\ln\Big(\frac{I_m}{I_m^*}\Big)\Big],
\end{aligned} \tag{4.1}$$

The time derivative of (4.1) along the solution of (2.1) gives

$$\begin{aligned}
\dot{\mathcal{V}} &= \dot{S}_h(t,a_i) - \frac{S_h^*(a_i)}{S_h(t,a_i)}\dot{S}_h(t,a_i) + \dot{E}_h(t,a_i) - \frac{E_h^*(a_i)}{E_h(t,a_i)}\dot{E}_h(t,a_i)\\
&\quad + \sum_{i=0}^{L}\frac{\alpha_h(a_i)+\mu_h(a_i)}{\alpha_h(a_i)}\Big(\dot{I}_h(t,a_i) - \frac{I_h^*(a_i)}{I_h(t,a_i)}\dot{I}_h(t,a_i)\Big)\\
&\quad + \dot{S}_m - \frac{S_m^*}{S_m}\dot{S}_m + \dot{E}_m - \frac{E_m^*}{E_m}\dot{E}_m + \frac{\alpha_m+\mu_m}{\alpha_m}\big(\dot{I}_m - \frac{I_m^*}{I_m}\dot{I}_m\big).
\end{aligned} \tag{4.2}$$

Substituting the appropriate equations of the model (2.1) into (4.2) gives

$$\begin{aligned}
\dot{\mathcal{V}} &= (1-\tau)\mu_h(a_i) - \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h(t,a_i)I_m - \mu_h(a_i)S_h(ta_i)\\
&\quad - \sum_{i=0}^{L}\frac{S_h^*(a_i)}{S_h(t,a_i)}\left((1-\tau)\mu_h(a_i) - b\beta_h(a_i)\sigma S_h(t,a_i)I_m - \mu_h(a_i)S_h(t,a_i)\right)\\
&\quad + \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h(t,a_i)I_m - (\alpha_h(a_i)+\mu_h(a_i))E_h(t,a_i)\\
&\quad - \sum_{i=0}^{L}\frac{E_h^*(a_i)}{E_h(t,a_i)}\left(b\beta_h(a_i)\sigma S_h(t,a_i)I_m - [\alpha_h(a_i)+\mu_h(a_i)]\,E_h(t,a_i)\right)\\
&\quad + \sum_{i=0}^{L}\frac{\alpha_h(a_i)+\mu_h(a_i)}{\alpha_h(a_i)}\left[(1-\theta)\alpha_h(a_i)E_h(t,a_i) - (\gamma(a_i)+\mu_h(a_i))I_h(t,a_i)\right]\\
&\quad - \sum_{i=0}^{L}\frac{\alpha_h(a_i)+\mu_h(a_i)}{\alpha_h(a_i)}\Big(\frac{I_h^*(a_i)}{I_h(t,a_i)}\Big)\\
&\quad \times \left[(1-\theta)\alpha_h(a_i)E_h(t,a_i) - (\gamma(a_i)+\mu_h(a_i))I_h(t,a_i)\right]\\
&\quad + \mu_m - b\beta_h(a_i)S_mI_h(t,a_i) - \mu_mS_m - \frac{S_m^*}{S_m}\left(\mu_m - b\beta_h(a_i)S_mI_h(t,a_i) - \mu_mS_m\right)\\
&\quad + b\beta_mS_mI_h(t,a_i) - [\alpha_m+\mu_m]\,E_m - \frac{E_m^*}{E_m}\left(b\beta_mS_mI_h - [\alpha_m+\mu_m]\,E_m\right)\\
&\quad + \frac{\alpha_m+\mu_m}{\alpha_m}\Big[\alpha_mE_m - \mu_mI_m - \frac{I_m^*}{I_m}\left(\alpha_mE_m - \mu_mI_m\right)\Big].
\end{aligned}$$

Further simplification yields

$$\begin{aligned}
\dot{\mathcal{V}} &= \sum_{i=0}^{L} \mu_h(a_i)\Big(1 - \frac{S_h^*(a_i)}{S_h(t,a_i)}\Big) - \mu_h(a_i)S_h(t,a_i)\Big(1 - \frac{S_h^*(a_i)}{S_h(t,a_i)}\Big) \\
&+ \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h^*(a_i)I_m - \frac{E_h^*(a_i)b\beta_h(a_i)\sigma S_h(t,a_i)I_m}{E_h(t,a_i)} \\
&+ \sum_{i=0}^{L} (\alpha_h(a_i) + \mu_h(a_i))E_h^*(a_i) - \frac{(\alpha_h(a_i) + \mu_h(a_i))(\gamma(a_i) + \mu_h(a_i))I_h(t,a_i)}{\alpha_h(a_i)} \\
&- \sum_{i=0}^{L} \frac{(\alpha_h(a_i) + \mu_h(a_i))I_h^*(a_i)E_h(t,a_i)}{I_h(t,a_i)} + \mu_m\Big(1 - \frac{S_m^*}{S_m}\Big) \\
&+ \frac{(\alpha_h(a_i) + \mu_h(a_i))(\gamma(a_i) + \mu_h(a_i))I_h^*(a_i)}{\alpha_h(a_i)} - \mu_m S_m\Big(1 - \frac{S_m^*}{S_m}\Big) \\
&+ b\beta_m S_m^* I_h(t,a_i) - \frac{E_m^* b\beta_m S_m I_h(t,a_i)}{E_m} + (\alpha_m + \mu_m)E_m^* - \frac{(\alpha_m + \mu_m)\mu_m I_m}{\alpha_m} \\
&- \frac{(\alpha_m + \mu_m)I_m^* E_m}{I_m} + \frac{(\alpha_m + \mu_m)\mu_m I_m}{\alpha_m}.
\end{aligned} \tag{4.3}$$

One sees that the following equilibrium relations hold from model (2.1),

$$\begin{aligned}
\mu_h(a_i) &= \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h^*(a_i)I_m^* + \mu_h(a_i)S_h^*(a_i), \\
\alpha_h(a_i) + \mu_h(a_i) &= \sum_{i=0}^{L} \frac{b\beta_h(a_i)\sigma S_h^*(a_i)I_m^*}{E_h^*(a_i)}, \\
\gamma(a_i) + \mu_h(a_i) &= \sum_{i=0}^{L} \frac{\alpha_h(a_i)E_h^*(a_i)}{I_h^*(a_i)}, \\
\mu_m &= b\beta_m S_m^* I_h^*(t,a_i) + \mu_m S_m^*, \\
\alpha_m + \mu_m &= \frac{b\beta_m S_m^* I_h^*(t,a_i)}{E_m^*}, \quad \mu_m = \frac{\alpha_m E_m^*}{I_m^*}
\end{aligned} \tag{4.4}$$

Consequently, using (4.4) in (4.3) gives

$$\begin{aligned}
\dot{\mathcal{V}} &= \sum_{i=0}^{L} \mu_h(a_i)S_h^*(a_i)\Big(2 - \frac{S_h^*(a_i)}{S_h(t,a_i)} - \frac{S_h(t,a_i)}{S_h^*(a_i)}\Big) \\
&+ \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h^*(a_i)I_m^* - \frac{b\beta_h(a_i)\sigma(S_h^*(a_i))^2 I_m^*}{S_h(t,a_i)} \\
&+ \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h^*(a_i)I_m - \frac{E_h^*(a_i)b\beta_h(a_i)\sigma S_h(t,a_i)I_m}{E_h(t,a_i)} \\
&+ \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h^*(a_i)I_m^* - \frac{b\beta_h(a_i)\sigma S_h^*(a_i)I_h(t,a_i)I_m^*}{I_h^*(a_i)}
\end{aligned}$$

$$
\begin{aligned}
&- \sum_{i=0}^{L} \frac{b\beta_h(a_i)\sigma S_h^*(a_i) I_h^*(a_i) E_h(t,a_i) I_m^*}{E_h^*(a_i) I_h(t,a_i)} + b\beta_h(a_i)\sigma S_h^*(a_i) I_m^* \\
&+ \mu_m S_m^* \Big(2 - \frac{S_m^*}{S_m} - \frac{S_m}{S_m^*}\Big) + b\beta_m S_m^* I_h^* - \frac{b\beta_m (S_m^*)^2 I_h^*}{S_m} \\
&+ b\beta_m S_m^* I_h(t,a_i) - \frac{E_m^* b\beta_m S_m I_h(t,a_i)}{E_m} + b\beta_m S_m^* I_h^*(a_i) \\
&- \frac{b\beta_m S_m^* I_m I_h^*(a_i)}{I_m^*} - \frac{b\beta_m S_m^* I_h^* E_m I_h^*}{E_m^* I_m} + b\beta_m S_m^* I_h^*(a_i). \qquad (4.5)
\end{aligned}
$$

Adding and subtracting $b\beta_h(a_i)\sigma S_h^*(a_i) I_m^*$,

$$
\sum_{i=0}^{L} \frac{b\beta_h(a_i)\sigma S_h^*(a_i) I_h(t,a_i)(I_m^*)^2}{I_h^*(a_i) I_m},
$$

$b\beta_m S_m^* I_h^*(a_i)$ and

$$
\frac{b\beta_m S_m^* I_m (I_h^*(a_i))^2}{I_m^* I_h(t,a_i)}
$$

in (4.5) systematically, gives

$$
\begin{aligned}
\dot{\mathcal{V}} &= \sum_{i=0}^{L} \mu_h(a_i) S_h^*(a_i) \Big(2 - \frac{S_h^*(a_i)}{S_h(t,a_i)} - \frac{S_h(t,a_i)}{S_h^*(a_i)}\Big) + \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h^*(a_i) I_m^* \\
&\times \Big[4 - \frac{S_h^*(a_i)}{S_h(t,a_i)} - \frac{E_h^*(a_i) S_h(t,a_i) I_m}{E_h(t,a_i) S_h^*(a_i) I_m^*} - \frac{I_h^*(a_i) E_h(t,a_i)}{I_h(t,a_i) E_h^*(a_i)} - \frac{I_h(t,a_i) I_m^*}{I_h^*(a_i) I_m}\Big] \\
&+ \sum_{i=0}^{L} b\beta_h(a_I)\sigma S_h^*(a_I) I_m - \frac{b\beta_h(a_I)\sigma S_h^*(a_I) I_h(t,a_I) I_m^*}{I_h^*(t,a_I)} \\
&+ \sum_{i=0}^{L} \frac{b\beta_h(a_i)\sigma S_h^*(a_i) I_h(t,a_i)(I_m^*)^2}{I_h^*(a_i) I_m} - b\beta_h(a_i)\sigma S_h^*(a_i) I_m^* \\
&+ \mu_m S_m^* \left(2 - \frac{S_m^*}{S_m} - \frac{S_m}{S_m^*}\right) + b\beta_m S_m^* I_h^*(a_i) \\
&\times \left[4 - \frac{S_m^*}{S_m} - \frac{E_m^* S_m I_h(t,a_i)}{E_m S_m^* I_h^*(a_i)} - \frac{I_m^* E_m}{I_m E_m^*} - \frac{I_m I_h^*(a_i)}{I_m^* I_h(t,a_i)}\right] \\
&+ b\beta_m S_m^* I_h(t,a_i) - \frac{b\beta_m S_m^* I_m I_h^*(a_i)}{I_m^*} + \frac{b\beta_m S_m^* I_m (I_h^*(a_i))^2}{I_m^* I_h(t,a_i)} \\
&- b\beta_m S_m^* I_h^*(a_i)
\end{aligned}
$$

Further algebraic simplification yields

$$
\begin{aligned}
\dot{\mathcal{V}} &= -\mathcal{V}_1 - \mathcal{V}_2 - \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h^*(a_i) I_m^* \Big(\frac{I_m}{I_h(t,a_i)} - \frac{I_m^*}{I_h^*(a_i)}\Big)\Big(1 - \frac{I_m}{I_m^*}\Big) \\
&\quad - \mathcal{V}_3 - \mathcal{V}_4 - \sum_{i=0}^{L} b\beta_m S_m^* I_h^*(a_i) \Big(\frac{I_h(t,a_i)}{I_m} - \frac{I_h^*(a_i)}{I_m^*}\Big)\Big(1 - \frac{I_h(t,a_i)}{I_h^*(a_i)}\Big),
\end{aligned} \qquad (4.6)
$$

where

$$
\mathcal{V}_1 = \sum_{i=0}^{L} \mu_h(a_i) S_h^*(a_i) \Big(\frac{S_h^*(a_i)}{S_h(t,a_i)} + \frac{S_h(t,a_i)}{S_h^*(a_i)} - 2\Big),
$$

$$\mathcal{V}_2 = \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h^*(a_i)I_m^*\Big[\frac{S_h^*(a_i)}{S_h(t,a_i)} + \frac{E_h^*(a_i)S_h(t,a_i)I_m}{E_h(t,a_i)S_h^*(a_i)I_m^*} + \frac{I_h^*(a_i)E_h(t,a_i)}{I_h(t,a_i)E_h^*(a_i)} + \frac{I_h(t,a_i)I_m^*}{I_h^*(a_i)I_m} - 4\Big],$$

$$\mathcal{V}_3 = \mu_m S_m^*\Big(\frac{S_m^*}{S_m} + \frac{S_m}{S_m^*} - 2\Big),$$

$$\mathcal{V}_4 = b\beta_m S_m^* I_h^*(a_i)\Big[\frac{S_m^*}{S_m} + \frac{E_m^* S_m I_h(t,a_i)}{E_m S_m^* I_h^*(a_i)} + \frac{I_m^* E_m}{I_m E_m^*} + \frac{I_m I_h^*(a_i)}{I_m^* I_h(t,a_i)} - 4\Big].$$

Since arithmetic mean is greater than or equal to the geometric mean (AM–GM inequality), one sees that $\mathcal{V}_1 \geq 0$, $\mathcal{V}_2 \geq 0$, $\mathcal{V}_3 \geq 0$, $\mathcal{V}_4 \geq 0$ and whenever the sign conditions

$$\Big(\frac{I_m}{I_h(t,a_i)} - \frac{I_m^*}{I_h^*(a_i)}\Big)\Big(1 - \frac{I_m}{I_m^*}\Big) \geq 0$$

with

$$\Big(\frac{I_h(t,a_i)}{I_m} - \frac{I_h^*(a_i)}{I_m^*}\Big)\Big(1 - \frac{I_h(t,a_i)}{I_h^*(a_i)}\Big) \geq 0$$

hold, it follows from (4.6) that $\dot{\mathcal{V}} \leq 0$ with $\dot{\mathcal{V}} = 0$ if and only if $S_h(t,a_i) = S_h^{**}(a_i)$, $E_h(t,a_i) = E_h^{**}(a_i)$, $I_h(t,a_i) = I_h^{**}(a_i)$, $S_m = S_m^{**}$, $E_m = E_m^{**}$, $I_m = I_m^{**}$. This further implies that $V_h(t,a_i) \to \gamma(a_i)I_h^*(a_i)/\mu_h(a_i) = V_h^*(a_i)$ as $t \to \infty$ since $(S_h, E_h, I_h, S_m, E_m, I_m) \to (S_h^{**}(a_i), E_h^{**}(a_i), I_h^{**}(a_i), S_m^{**}, E_m^{**}, I_m^{**})$. Therefore, by LaSalle's principle [10], it follows that every solution of the model (2.1) starting in $\mathfrak{D}\backslash\mathfrak{D}_0$ approaches the endemic equilibrium $\mathcal{E}_1$ as $t \to \infty$. □

The epidemiological implication of Theorem 4.1 is that malaria can persist in the population whenever the intervention strategies are not adhered to and the associated basic reproduction is greater than one.

Conclusion. In this article, a malaria transmission dynamics with vigilant compartment governed by system of differential equations has been theoretically analyzed. The analysis is centered on the global asymptotic behavior of solutions of the system (2.1) around the disease-free and endemic (malaria-present) equilibria using Lyapunov functions. The system has a globally asymptotically stable disease-free equilibrium whenever the basic reproduction is less than the unity. Moreover, the endemic equilibrium of the system, when it exists in the absence of the vigilant fractions of susceptible and treated exposed human populations, is shown to be globally asymptotically stable whenever the associated basic reproduction number is greater than the unity.

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References

[1] F. B. Agusto, N. Marcus, K. O. Okosun; *Application of optimal control to the epidemiology of malaria*, Electronic Journal of Differential Equations, 2012 (2012), no. 81, 1–22.

[2] K. Blayneh, Y. Cao, H-D. Kwon; *Optimal control of vector-borne diseases: treatment and prevention*, Discrete and continuous dynamical systems series B, **11** (2009), no. 3, 587–611.

[3] B. Buonomo, D. Lacitignola; *On the use of the geometric approach to global stability for three dimensional ODE systems: a bilinear case*, J. Math. Anal. Appl., **348** (2008), 255–266.

[4] C. Castillo-Chavez, Z. Feng, W. Huang; *On the computation of R_0 and its role on global stability*, Mathematical Approaches for Emerging and Reemerging Infectious Diseases: An Introduction. The IMA Volumes in Mathematics and its Applications **125**, 229–250, Springer, New York, 2002. C. Castillo-Chavez, S. Blower, P. van den Driessche, D. Kirschner, A. A. Yakubu (Eds).

[5] F. Forouzannia, A. Gumel; *Dynamics of an age-structured two strain model for malaria transmission*, Applied Mathematics and Computation, **250** (2015), 860–886.

[6] H. W. Hethcote; *The mathematics of infectious diseases*, SIAM Review, **42** (2000), no. 4, 599–653.

[7] E. Iboi, D. Okuonghae; *Population dynamics of a mathematical model for syphilis*, Applied Mathematical Modelling, **40** (2016), 3573–3590.

[8] A. Korobeinikov; *Global properties of SIR and SEIR epidemic models with multiple parallel infectious stages*, Bulletin of Mathematical Biology **71** (2009), no. 1, 75–83.

[9] A. Korobeinikov, G. C. Wake; *Lyapunov functions and global stability for SIR, SIRS, and SIS epidemiological models*, Appl. Math. Lett., **15** (2002), no. 8, 955–960.

[10] J. P. LaSalle; *The stability of dynamical systems*, SIAM, Philadelphia, 1976.

[11] A. A. Lashari, S. Aly, K. Hattaf, G. Zaman, I. H. Jung, X. Li; *Presentation of malaria epidemics using multiple optimal controls*, Journal of Applied Mathematics, Article ID 946504, (2012).

[12] M. Y. Li, J. S. Muldowney; *A geometric approach to global-stability problems*, SIAM J. Math. Anal. **27** (1996), 1070–1083.

[13] S. Mushayabasa, J. M. Tchuenche, C. P. Bhunu, E. Ngarakana-Gwasira; *Modeling gonorrhea and HIV co-infection*, BioSystems, **103** (2011), 27–37.

[14] E. T. Ngarakana-Gwasira, C. P. Bhunu, M. Masocha, E. Mashonjowa; *Assessing the role of climate change in malaria transmission in Africa*, Malaria Research and Treatment, Article ID 7104291, (2016).

[15] O. S. Obabiyi, S. Olaniyi; *Asymptotic stability of malaria dynamics with vigilant human compartment*, International Journal of Applied Mathematics, **29** (2016), no. 1, 127–144.

[16] S. Olaniyi, O. S. Obabiyi; *Qualitative analysis of malaria dynamics with nonlinear incidence function*, Applied Mathematical Sciences, **8** (2014), no. 78, 3889–3904.

[17] Z. Shuai, P. van den Driessche; *Global stability of infectious disease models using lyapunov functions*, SIAM J. Appl. Math., **73** (2013), no. 4, 1513–1532.

[18] P. van den Driessche, J. Watmough; *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Mathematical Biosciences, **180** (2002), 29–48.

[19] C. Vargas-De-León; *Stability analysis of a SIS epidemic model with standard incidence*, Foro-Red-Mat: Revista Electrónica de Contenido Matemático, **28** (2011), no. 4, 1-11.

[20] WHO - World Health Organization; *World malaria report*, WHO Press, Geneva, 2014.

[21] WHO - World Health Organization; *World malaria report*, WHO Press, Geneva, 2015.

[22] D. Xiao, S. Ruan; *Global analysis of an epidemic model with nonmonotone incidence rate*, Math. Biosci., **208** (2007), 419–429.

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