Analysis of Vector-Host Model with Latent Stage Having Partial Immunity

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Abstract

We describe a deterministic SEIRS epidemiological model with vital dynamics to determine the equilibria and their stability. The complete global analysis for the equilibria of the model is analyzed by constructing Lyapunov functions. The explicit formula for the reproductive number is obtained and it is shown that the disease-free equilibrium always exists and is globally asymptotically stable whenever $R_0$ is below unity. Furthermore, the disease persists at an endemic level when the reproductive number exceeds unity. The sensitivity analysis is also performed in order to determine the relative importance of model parameters to disease transmission and prevalence.

1 Introduction

Vector-borne diseases are infectious diseases caused by viruses, bacteria, protozoa or rickettsia which are primarily transmitted by disease transmitting biological agents, called vectors. Vector-borne diseases, in particular, mosquito-borne diseases such as malaria, dengue fever and West Nile Virus are transmitted to humans by blood-sucker mosquitoes. These have caused big problem for the public health in the world.
Mathematical modeling has proven to play an important role in gaining some insights into the transmission dynamics of infectious diseases and can play an important role in determining effective strategies in different transmission settings and also in quantifying the effects of control methods. Appropriate mathematical models can provide a qualitative assessment for the problem. Many mathematical models based on the transmission of infectious agents in human communities have been studied in order to detect trends of the diseases and to plan some programs for their control [1, 2].

Malaria was one of the first vector-borne diseases for which such modelling was applied. Sir Ronald Ross [3] developed this important technique and explained that malaria can be eradicated if the mosquito population can be reduced to below a certain threshold. Macdonald [4] modified the Ross’ work and introduced the theory of superinfection and latency into the dynamics of mosquito population. Anderson and May [5], in addition introduced latent class in human population dynamics models. The fundamental assumption of these models was that only the vector is capable of transmission of the disease from host to host. However, there is some evidence to show that direct transmission is possible through blood transfusion, vertically or through needlestick injury. Such models with direct transmission in addition to vector transmission have also been reported in malaria and Chagas diseases. For example, in the papers [6, 7, 8], epidemic models of a vector-host disease with direct transmission and the vector-mediated transmission have been investigated.

The dynamics of many communicable diseases have been extensively analyzed under the assumption that the duration of immunity is independent of exposure to infection [5]. However, the immunity to malaria appears to be sustained by continuing exposure [9]. Hence, the conventional definition of immunity as absolute refractoriness to infection may be too restrictive, as immunity may confer protection against severe illness without eliminating chronic, mild infections [10]. Incomplete immunity to malaria not only complicates the disease control strategies but also partially immune individuals having mild infections become the source of continuous transmission of the parasite in the community.

Following the ideas advanced in [11] and [12], we investigate the model by assuming that the persons who are partially immune to the disease may be infectious. The remaining part of the paper is organized as follows: In Section 2, we briefly describe the model and investigate the existence of steady states and also comment on the existence or non-existence of positive equilibria. Stability analysis close to the steady states is performed in Section 3 by constructing suitable Lyapunov functions, and we show that the disease-free and endemic equilibria are globally asymptotically stable. In Section 4, we discuss the sensitivity of the basic reproductive number and the infectious classes with respect to epidemiological and demographic parameters. Concluding remarks are presented in the last section.

2 Model Description

The mathematical formulation of our model consists of the following contact parameters:

\[ \beta_h = \text{The probability of direct transmission (possibly as a result of transfusion, transplantation,} \]
Analysis of vector-host model

and use of needle--stick) of the disease.

$\beta_{vh} = \text{The transmission probability as a result of biting by an infected mosquito to the susceptible human.}$

$\beta_{nv}(\beta) = \text{The transmission probability of transferring the infection from an infected nonimmune (partially immune) human to the susceptible mosquito.}$

The total human population denoted by $N_h(t)$ is sub-divided, into four mutually exclusive compartments according to the status of the disease: Susceptible individuals $S_h(t)$, individuals possessing latent stage $E_h(t)$, infectious individuals $I_h(t)$ and recovered individuals having protective immunity $R_h(t)$. Thus $N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$. Similarly the total mosquito population at any time $t$ is denoted by $N_v(t) = S_v(t) + E_v(t) + I_v(t)$ where $S_v(t)$, $E_v(t)$ and $I_v(t)$ denote Susceptible, Exposed and Infectious vectors, respectively. In contrast to the human population, the vectors once infected remain microparasite carriers throughout their life.

The model below is based on the following features:
1) Both humans and vectors are born susceptible.
2) Immunity in human population is temporary and lasts only for some time. Then they become susceptible to infection.
3) The class of persons who are partially immune to the disease may be infectious. We also assume that the infection acquired by a vector from an immune host is less infective than the infection acquired from a non-immune host.

The following model results from the features described above:

\[
\begin{align*}
\frac{dS_h(t)}{dt} &= b_h - \beta_h S_h I_h - \beta_{vh} S_h I_v - d_h S_h + \delta_h R_h, \\
\frac{dE_h(t)}{dt} &= \beta_h S_h I_h + \beta_{vh} S_h I_v - \eta E_h - d_h E_h, \\
\frac{dI_h(t)}{dt} &= \eta E_h - \alpha_h I_h - d_h I_h - \xi_h I_h, \\
\frac{dR_h(t)}{dt} &= \alpha_h I_h - \delta_h R_h - d_h R_h, \\
\frac{dS_v(t)}{dt} &= b_v - \beta_v S_v I_v - \beta R_v S_v - d_v S_v, \\
\frac{dE_v(t)}{dt} &= \beta_v S_v I_v + \beta R_v S_v - \gamma_v E_v - d_v E_v, \\
\frac{dI_v(t)}{dt} &= \gamma_v E_v - d_v I_v.
\end{align*}
\] (1)
The model also satisfies the initial conditions,
\[ S_h(0) \geq 0, E_h(0) \geq 0, I_h(0) \geq 0, R_h(0) \geq 0, S_v(0) \geq 0, E_v(0) \geq 0, I_v(0) \geq 0. \] (2)

In the above model \( b_h \) and \( b_v \) are the recruitment rates of humans and vectors respectively. Similarly \( d_h \) and \( d_v \) are the natural mortality rates of humans and vectors respectively. We assume that a disease may be fatal to some infectious host. As a result deaths due to the disease can be included in the model using the disease related death rate, \( \xi_h \) from infectious class. Exposed humans develop clinical symptoms of the disease and move to the infectious class at rate \( \eta \). The parameter \( \alpha_h \) is the recovery rate of humans. It is assumed that immune human individuals loose their immunity at a rate \( \delta_h \). The total human population is then governed by the following equation:
\[
\frac{dN_h}{dt} = b_h - d_h N_h - \xi_h I_h. \tag{3}
\]
The given initial conditions (2) make sure that \( N_h(0) \geq 0 \). Thus the total population \( N_h(t) \) remains positive and bounded for all finite time \( t > 0 \). Again the dynamics of the total vector population is governed by the equation:
\[
\frac{dN_v}{dt} = b_v - d_v N_v. \tag{4}
\]
It follows from eqs. (3) and (4) that \( \lim_{t \to \infty} \text{Sup} N_h \leq \frac{b_h}{d_h} \) and \( N_v = \frac{b_v}{d_v} \) provided that \( S_v(0) + E_v(0) + I_v(0) = N_v(0) = \frac{b_v}{d_v} \) for all \( t \geq 0 \). Thus the feasible region for the system (1) is
\[ \Omega = \{(S_h, E_h, I_h, R_h, S_v, E_v, I_v) \in R^7, S_h + E_h + I_h + R_h \leq \frac{b_h}{d_h}, S_v + E_v + I_v = \frac{b_v}{d_v}\}. \]

2.1 Disease-free equilibrium
Steady state solutions of the system when there is no disease are called disease-free equilibrium points. The diseased classes containing either exposed, infectious or recovered, the human or mosquito populations, are denoted by \( E_h, \ I_h, \ R_h, \ E_v, \ I_v \). Simple calculations shows that the system (1) has a disease-free equilibrium point given by \( E_0 = (\frac{b_h}{d_h}, 0, 0, 0, \frac{b_v}{d_v}, 0, 0) \), which exists for all positive values of the parameters. The dynamics of the disease is described by the basic reproduction number \( R_0 \), which is defined as the average number of secondary infections produced by an infected individual in a completely susceptible population. The basic reproduction number of model (1) is given by the expression
Analysis of vector-host model

\[ R_0 = \frac{\beta_h \eta b_h}{d_h Q_1 Q_2} + \frac{\beta_h \beta \alpha \eta b_h b_v}{d_h d_v d_h Q_1 Q_2 Q_3} + \frac{\beta_h \beta \eta \eta \gamma b_h b_v}{d_h d_v d_h Q_1 Q_2 Q_3 Q_4}, \] (5)

where \( Q_1 = \eta + d_h, Q_2 = d_h + \alpha_h + \xi_h, Q_3 = d_h + \delta_h, Q_4 = d_v + \gamma_v. \)

**Theorem 1** If \( R_0 < 1 \), then the disease-free equilibrium point \( E_0\left(\frac{b_h}{d_h}, 0, 0, \frac{b_v}{d_v}, 0, 0\right) \) of the model (1) is locally asymptotically stable, otherwise it is unstable.

**Proof** By linearizing the system (1) around \( E_0\left(\frac{b_h}{d_h}, 0, 0, \frac{b_v}{d_v}, 0, 0\right) \), the Jacobian matrix \( J \) is given by:

\[
J = \begin{bmatrix}
-\lambda - d_h & 0 & -\beta_h b_h & \delta_h & 0 & 0 & -\beta_v b_v \\
0 & -\lambda - Q_1 & \beta_h b_h & 0 & 0 & 0 & \beta_v b_v \\
0 & \eta & -\lambda - Q_2 & 0 & 0 & 0 & 0 \\
0 & 0 & \alpha_h & -\lambda - Q_3 & 0 & 0 & 0 \\
0 & 0 & -\beta_v b_v & -\beta_v b_v & -\lambda - d_v & 0 & 0 \\
0 & 0 & \beta_v b_v & \beta_v b_v & 0 & -\lambda - Q_4 & 0 \\
0 & 0 & 0 & 0 & 0 & \gamma_v & -\lambda - d_v
\end{bmatrix}
\]

The characteristic equation of the above matrix is

\[(\lambda + d_h)(\lambda + d_v)(\lambda^5 + m_1 \lambda^4 + m_2 \lambda^3 + m_3 \lambda^2 + m_4 \lambda + m_5) = 0\]

where

\[m_1 = d_v + Q_1 + Q_2 + Q_3 + Q_4\]
\[m_2 = Q_1 Q_2 (1 - \frac{\beta_h \eta b_h}{d_h Q_1 Q_2}) + Q_2 Q_3 + 2Q_3 Q_4 + (Q_1 + Q_2)Q_4 + d_v (Q_1 + Q_2 + Q_3 + Q_4)\]
\[m_3 = Q_1 Q_2 (d_v + Q_3 + Q_4) (1 - \frac{\beta_h \eta b_h}{d_h Q_1 Q_2}) + (Q_1 + Q_2)Q_3 Q_4 + d_v (Q_2 Q_3 + 2Q_3 Q_4 + Q_4) + Q_2 Q_4)\]
\[m_4 = Q_1 Q_2 Q_3 (d_v + Q_4) (1 - \frac{\beta_h \eta b_h}{d_h Q_1 Q_2}) + d_v Q_1 Q_2 Q_4 (1 - \frac{\beta_h \eta b_h}{d_h Q_1 Q_2}) - \frac{\beta_v b_v \eta \gamma b_h b_v}{d_h d_v d_v Q_1 Q_2 Q_3 Q_4}\]
\[m_5 = d_v Q_1 Q_2 Q_3 Q_4 (1 - R_0)\].

Two of the eigenvalues are \(-d_h\) and \(-d_v\), which are obviously negative. The remaining five eigenvalues are roots of the equation

\[g(\lambda) = \lambda^5 + m_1 \lambda^4 + m_2 \lambda^3 + m_3 \lambda^2 + m_4 \lambda + m_5 = 0.\] (6)
The necessary and sufficient condition for local asymptotic stability follows from the Routh-Hurwitz conditions applied to the above equation [13], i.e. \( m_i > 0 \) for \( i = 1, 2, 3, 4, 5 \) with \( m_1 m_2 m_3 > m_3^2 + m_1^2 m_4 \)
and
\[
(m_i m_4 - m_5)(m_1 m_2 m_3 - m_3^2 - m_1^2 m_4) > m_3 (m_1 m_2 - m_3)^2 + m_1 m_5^2.
\]
For \( R_0 < 1 \), we see that \( m_i > 0 \) for \( i = 1, 2, 3, 4, 5 \). The straightforward but rather lengthy calculations shows that
\[
m_1 m_2 m_3 - m_3^2 + m_1^2 m_4 > 0
\]
and
\[
(m_i m_4 - m_5)(m_1 m_2 m_3 - m_3^2 - m_1^2 m_4) - m_3 (m_1 m_2 - m_3)^2 - m_1 m_5^2 > 0.
\]
Hence all the eigenvalues of the characteristic equation (5) have negative real parts if and only if \( R_0 < 1 \), which shows that the disease-free equilibrium \( E_0 \) is locally asymptotically stable.

**Observation:** If \( R_0 > 1 \), we have \( g(0) < 0 \) and \( g(\lambda) = +\infty \) as \( \lambda \to +\infty \). Thus there exists at least one \( \lambda > 0 \) such that \( g(\lambda) = 0 \) which proves instability of disease-free equilibrium.

### 2.2 Endemic Equilibrium

Let \( E_1 = (S^*_h, E^*_h, I^*_h, R^*_h, S^*_v, E^*_v, I^*_v) \) represents any arbitrary endemic equilibrium of the model (1). Equating the right hand sides of all the equations in model (1) to zero, we have

\[
\begin{align*}
E^*_h &= \frac{Q_2}{\eta I^*_h} \\
R^*_h &= \frac{\alpha_s I^*_h}{Q_3} \\
S^*_v &= \frac{b_v Q_3}{(\beta_{hv} Q_3 + \beta \alpha_h)I^*_h + d_v Q_3} \\
E^*_v &= \frac{b_v (\beta_{hv} Q_3 + \beta \alpha_h)I^*_h}{Q_4[(\beta_{hv} Q_3 + \beta \alpha_h)I^*_h + d_v Q_3]} \\
I^*_v &= \frac{b_v \gamma_v (\beta_{hv} Q_3 + \beta \alpha_h)I^*_h}{(d_v Q_4[(\beta_{hv} Q_3 + \beta \alpha_h)I^*_h + d_v Q_3])}
\end{align*}
\]

(7)

In the above \( I^*_h \), is a positive solution of this equation

\[
A_1 I^*_{h}^2 + A_2 I^*_h + A_3 = 0,
\]

(8)
where
\[ A_1 = [\beta_h d_v Q_1 Q_2 Q_3 (\beta_{hv} Q_1 + \beta \alpha_h) + \delta_h \alpha_h \beta_h d_v Q_1 \eta (\beta_{hv} Q_3 + \beta \alpha_h)] \]
\[ A_2 = [\beta_h d_v Q_1 Q_2 Q_3 (\beta_{hv} Q_1 + \beta \alpha_h) + \delta_h \beta \alpha_h \beta_h d_v Q_1 \eta \eta d_v Q_3 + \delta_h \beta \alpha_h \beta_h d_v Q_1 \eta (\beta_{hv} Q_3 + \beta \alpha_h)] \]
\[ A_3 = d_h d_v Q_1 Q_2 Q_3 d_v Q_3 - b_h \beta_h d_v Q_1 Q_3 \eta d_v Q_3 = d_h d_v Q_1 Q_2 Q_3 (\beta_{hv} Q_3 + \beta \alpha_h) (1 - \frac{b_h \beta_h \eta}{d_h Q_1 Q_2}) \]
\[ (\eta) \quad (\xi) \]
From (9), we see that \( R_0 > 1 \) if and only if, \( A_3 < 0 \). Since \( A_1 > 0 \), Eq.(8) has a unique positive root in the feasible region \( \Omega \). If \( R_0 < 1 \), then \( A_3 > 0 \). Also, it can be easily seen that \( A_2 > 0 \) for \( R_0 < 1 \). Thus there will be no (positive) endemic equilibrium in this case. The above result is summarized below:

**Theorem 2** System (1) always has the infection-free equilibrium \( E_0 \). If \( R_0 > 1 \), system (1) has a unique endemic equilibrium \( E_1 = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*) \) defined in (7) and (8).

### 3 Analysis of Global Stability

#### 3.1 Global Stability of disease-free Equilibrium:
We analyze the global behavior of the equilibria for system (1). The following theorem provides the global property of the disease-free equilibrium \( E_0 \) of the system.

**Theorem 3** If \( R_0 < 1 \), then the infection−free equilibrium \( E_0 \) is globally asymptotically stable in the interior of \( \Omega \).

**Proof** To prove the global stability of the disease−free equilibrium, we construct the following Lyapunov function \( L \) and calculate its derivative \( L' \) and these are given below:
\[ L = \frac{\eta}{Q_1} E_h + \frac{\eta}{Q_1} I_h + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} R_h + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} E_v + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} I_v. \]
\[ L' = \frac{\eta}{Q_1} E_h' + \frac{\eta}{Q_1} I_h' + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} R_h' + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} E_v' + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} I_v'. \]
\[ L' = \frac{\eta}{Q_1} (\beta_h S_h I_h + \beta_{hv} S_h I_v - Q_1 E_h) + \frac{\eta}{Q_1} (\eta E_h - Q_2 I_h) + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} (\alpha_h I_h - Q_3 R_h) + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} (\beta_h S_h I_v + \beta_{hv} S_v I_v) + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} (\gamma_v E_v - d_v I_v) \]
\[ \leq \frac{\eta}{Q_1} (\beta_h S_h I_h + \beta_{hv} S_h I_v - Q_1 E_h) + \frac{\eta}{Q_1} (\eta E_h - Q_2 I_h) + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} (\alpha_h I_h - Q_3 R_h) + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} (\beta_h S_h I_v + \beta_{hv} S_v I_v) + \frac{b_h \beta_h \beta_h \eta}{d_h d_v d_v Q_1} (\gamma_v E_v - d_v I_v) \]
We see that $L'$ is negative if

\[
\frac{b_h \eta}{d_h} + \frac{b_h \beta_s \alpha}{d_h, d, d, d, d, d, d} < 1,
\]

which implies

\[
\frac{b_h \beta_s \beta}{d_h, d, d, d, d, d, d, d, d} + \frac{b_h \beta_s \beta \alpha}{d_h, d, d, d, d, d, d} < 1.
\]

Again $L' = 0$ if and only if $I_h = 0 = I_v = E_h = E_v$. Therefore the largest compact invariant set in \{(E_h, I_h, E_v, I_v) \in \Omega, L' = 0\}, when $R_0 < 1$, consists of the singelton $\{E_0\}$. Hence, LaSalle's invariance principle [14] implies that $E_0$ is globally asymptotically stable in $\Omega$. This completes the proof.

### 3.2 Global Stability of Endemic Equilibrium

We shall prove Global stability of the endemic equilibrium $E_1 = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*)$ where $S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*$ and $I_v^*$ satisfy the following equations:

\[
\begin{align*}
b_h - \beta_h S_h^* I_v^* - \beta_{sh} S_h^* I_h^* - d_h S_h^* + \delta_h R_h^* &= 0, \\
\beta_h S_h^* I_h^* + \beta_{sh} S_h^* I_v^* - Q_h E_h^* &= 0, \\
\eta E_h^* - Q_h I_h^* &= 0, \\
\alpha_h I_h^* - Q_h R_h^* &= 0, \\
b_v - \beta_v S_v^* I_h^* - \beta R_h^* S_v^* - d_v S_v^* &= 0,
\end{align*}
\]

We have following theorem. [15]
Theorem 4 The unique endemic equilibrium $E_1$ is globally asymptotically stable in $\Omega / \Omega_0$ whenever $R_0 > 1$ and $1 + \frac{R_0S^*_h}{R^*_hS^*_h} - \frac{R_h}{R^*_h} - \frac{S^*_h}{S_h} \geq 0$.

Proof The proposed Lyapunov function is given by:

$$L = a_1(S_h - S^*_h - S^*_h \log \frac{S^*_h}{S_h}) + a_2\left(E_h - E^*_h - E^*_h \log \frac{E^*_h}{E_h}\right) + a_3\left(I_h - I^*_h - I^*_h \log \frac{I^*_h}{I_h}\right) + a_4\left(R_h - R^*_h - R^*_h \log \frac{R^*_h}{R_h}\right) + a_5\left(S_v - S^*_v - S^*_v \log \frac{S^*_v}{S_v}\right) + a_6\left(E_v - E^*_v - E^*_v \log \frac{E^*_v}{E_v}\right) + a_7\left(I_v - I^*_v - I^*_v \log \frac{I^*_v}{I_v}\right)$$

where $a_1, a_2, a_3, a_4, a_5, a_6, a_7$ will be chosen later. Differentiating $L$ with respect to $t$ along the solutions of (1), we have

$$L' = a_1(1 - \frac{S^*_h}{S_h})S_h' + a_2(1 - \frac{E^*_h}{E_h})E_h' + a_3(1 - \frac{I^*_h}{I_h})I_h' + a_4(1 - \frac{R^*_h}{R_h})R_h' + a_5(1 - \frac{S^*_v}{S_v})S_v' + a_6(1 - \frac{E^*_v}{E_v})E_v' + a_7(1 - \frac{I^*_v}{I_v})I_v'$$

Substituting the expressions from system (1) at the endemic steady state, we have

$$L' = a_1[\beta_1 S^*_h I^*_h (1 - \frac{S^*_h}{S_h} - \frac{S_d I_h}{S_h} + \frac{I_h}{I_h}) + \beta_2 S^*_h I^*_v (1 - \frac{S^*_h}{S_h} - \frac{S_d I_v}{S_v} + \frac{I_v}{I_v}) + \delta_1 S^*_h (2 - \frac{S^*_h}{S_h} - \frac{S_h}{S_h}) - \frac{E_h}{E_h} + 1)] + a_2[\beta_1 S^*_h I^*_h (\frac{S_d I_h}{S_h} - \frac{S_h I_v}{S_v} - \frac{E_h}{E_v} + 1)] + \beta_2 S^*_h I^*_v (\frac{S_d I_v}{S_v} - \frac{S_h I_v}{S_v}) + a_3[\eta E^*_h (\frac{E_h}{E_h} - \frac{E_v I^*_h}{I_h E_h} - \frac{I_h}{I_h} + 1)] + a_4[\alpha_1 I^*_h (\frac{I_h}{I_h} - \frac{I_h R_h}{I_h K_h} - \frac{R_h}{K_h} + 1)] + a_5[\beta_3 S^*_v I^*_h (1 - \frac{S^*_v}{S_v}) - \frac{I_h S_v}{I_h S_v} + \frac{I_h}{I_h}) + \beta R^*_h S^*_v (1 - \frac{S^*_v}{S_v} - \frac{R^*_h S_v}{K^*_h} + \frac{R_v}{K_v}) + d_s S^*_v (2 - \frac{S^*_v}{S_v} - \frac{S_v}{S_v})] + a_6[\beta_3 S^*_v I^*_h (\frac{I_h S_v}{I_h S_v} - \frac{I_h S_v}{I_h E_v} + \frac{I_h}{I_h}) + \beta R^*_h S^*_v (\frac{R_h S_v}{K^*_h S_v} - \frac{R_h S_v}{K^*_h} S_v - \frac{E_v}{E_v} + 1)] + a_7[\gamma E^*_v (\frac{E_v}{E_v} - \frac{E_v I^*_v}{I_v} - \frac{I_v}{I_v} + 1)]$$

(11)

Setting the values of coefficients
\[ a_1 = a_2 = \frac{\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*}{\beta_2 S_h^* I_v^*} \]
\[ a_3 = \frac{(\beta_1 S_h^* I_h^* + \beta_2 S_h^* I_v^*)(\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*)}{\eta E_h^* \beta_2 S_h^* I_v^*} \]
\[ a_4 = \frac{\beta R_h^* S_v^*}{\alpha_h I_h^*} \]
\[ a_5 = a_6 = 1 \]
\[ a_7 = \frac{\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*}{\gamma_v E_v^*} \]

in (11), and after some calculation, we have

\[
L' = \frac{d_1 S_h^* (\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*)}{\beta_2 S_h^* I_v^*} (2 - \frac{S_h^*}{S_h^*} - \frac{S_h^*}{S_h^*}) + \frac{\beta_1 S_h^* I_h^* (\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*)}{\beta_2 S_h^* I_v^*} \\
(3 - \frac{S_h^* I_h^* E_h^*}{S_h^* E_h^* I_v^*} - \frac{S_h^* - E_h^* I_h^*}{S_h^*}) + d_2 S_v^* (2 - \frac{S_v^*}{S_v^*} - \frac{S_v^*}{S_v^*}) \\
+ \beta R_h^* S_v^* (7 - \frac{R_h^* S_v^* E_v^*}{R_h^* E_v^* I_v^* - E_v^* I_v^*} - \frac{S_h^* I_h^* E_h^*}{S_h^* E_h^* I_v^* - E_v^* I_v^*} - \frac{E_h^* I_h^*}{I_h^* E_h^* - R_h^* I_v^* - S_v^* - S_h^*}) \\
+ \beta_3 S_v^* I_h^* (6 - \frac{I_h^* S_v^* E_v^*}{I_h^* E_v^* S_v^* - E_v^* I_v^*} - \frac{S_h^* I_h^* E_h^*}{S_h^* I_v^* E_h^* - E_h^* I_v^*} - \frac{E_h^* I_h^*}{I_h^* E_h^* - S_h^* - S_v^*}) \\
- \frac{\delta_h R_h^* (\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*)}{\beta_2 S_h^* I_v^*} (1 + \frac{R_h^* S_h^*}{R_h^* S_h^*} - \frac{R_h^*}{R_h^*} - \frac{S_h^*}{S_h^*})
\]

The following inequalities hold:
Analysis of vector-host model

\[ 2 - \frac{S_h}{S^*_h} \frac{S^*_h}{S_h} \leq 0 \]

\[ 3 - \frac{S_h I_h E^*_h}{S^*_h E_h I^*_h} - \frac{S^*_h}{S_h} - \frac{E_h I^*_h}{I_h E^*_h} \leq 0 \]

\[ 2 - \frac{S_v}{S^*_v} \frac{S^*_v}{S_v} \leq 0 \]

\[ 7 - \frac{R_h S_v E^*_v}{R^*_h E_v S^*_v} - \frac{E_v I^*_v}{I_v E^*_v} - \frac{S_h I_v E^*_h}{S^*_h E_h I^*_v} - \frac{E_h I^*_h}{I_h E^*_h} - \frac{I_h R^*_h}{R_h I^*_h} - \frac{S^*_v}{S_v} - \frac{S^*_h}{S_h} \leq 0 \]

\[ 6 - \frac{I_h S_v E^*_v}{I^*_h E_v S^*_v} - \frac{E_v I^*_v}{I^*_v E^*_v} - \frac{S_h I_v E^*_h}{S^*_h I^*_v E^*_h} - \frac{E_h I^*_h}{I_h E^*_h} - \frac{S^*_v}{S_v} - \frac{S^*_h}{S_h} \leq 0 \]  \(12\)

Now, the condition \(1 + \frac{R_h S_v}{R^*_h S_h} - \frac{R_h}{R^*_h} - \frac{S_v}{S_h} \geq 0\) and \(12\) imply that \(L' \leq 0\). Hence, by Lyapunov’s first theorem the endemic equilibrium \(E_i = (S^*_h, E^*_h, I^*_h, R^*_h, S^*_v, E^*_v, I^*_v)\) is globally asymptotically stable.

4 Sensitivity Analysis

By analyzing different factors that are responsible for the disease transmission and prevalence, we can try to reduce human mortality and morbidity due to disease. Initial disease transmission depends upon the reproductive number whereas disease prevalence is directly related to the endemic equilibrium point. We examine that

\[ \frac{\partial R_0}{\partial d_v} = -\eta \gamma \frac{b_h}{d_v} \frac{b_v}{d_v} \frac{\beta_h (2\gamma_h + 3d_h)}{(\eta + d_h)(\alpha_h + \delta_h + d_h)(\gamma_v + d_v)^2} \left( \frac{\beta \alpha_h + \beta \delta_h (\delta_h + d_h)}{\delta_h + d_h} \right) \]

So \(R_0\) is a decreasing function of \(d_v\). Also we observe that \(R_0\) is inversely related to the parameters \(\alpha_h, d_h, \delta_h\) and \(\xi_h\). We want to determine the most crucial parameter in order to decrease the reproductive number less than unity. We can also estimate that this parameter is how much reducing the reproductive number.

Definition The normalized forward sensitivity index of a variable, \(h\), that depends differentiably on a parameter, \(l\), is defined as \(\Gamma^h_l = \frac{\partial h}{\partial l} \times \frac{l}{h}\).

We will calculate the sensitivity indices of the reproductive number, \(R_0\), with respect to the parameter values given in Table 1 for the model. These values are given in Table 2.
Table (1): Values of the parameters used for sensitivity analysis

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>$b_h$</td>
<td>0.00011</td>
<td>[7]</td>
</tr>
<tr>
<td>$b_v$</td>
<td>0.13</td>
<td>[7]</td>
</tr>
<tr>
<td>$\delta_h$</td>
<td>0.7</td>
<td>[7]</td>
</tr>
<tr>
<td>$\beta_{vh}$</td>
<td>0.022</td>
<td>[7]</td>
</tr>
<tr>
<td>$\beta_{hv}$</td>
<td>0.48</td>
<td>[7]</td>
</tr>
<tr>
<td>$\beta_h$</td>
<td>0.004</td>
<td>[16]</td>
</tr>
<tr>
<td>$d_h$</td>
<td>0.000016</td>
<td>[7]</td>
</tr>
<tr>
<td>$d_v$</td>
<td>0.03</td>
<td>[7]</td>
</tr>
<tr>
<td>$\eta$</td>
<td>0.10</td>
<td>[16]</td>
</tr>
<tr>
<td>$\beta$</td>
<td>0.048</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\gamma_v$</td>
<td>0.091</td>
<td>[16]</td>
</tr>
<tr>
<td>$\alpha_h$</td>
<td>0.0035</td>
<td>[16]</td>
</tr>
<tr>
<td>$\xi_h$</td>
<td>0.00009</td>
<td>[16]</td>
</tr>
</tbody>
</table>

By analyzing the sensitivity indices we observe that the most sensitive parameter for the reproductive number is the death rate of mosquitoes $d_v$. We can say that an increase or decrease in death rate of mosquitoes by 10% decreases or increases $R_0$ by 20%. But it is difficult to make $R_0 < 1$ by increasing the death rate of mosquitoes $d_v$ or other parameters dramatically in practice. Although all these measures given above are very effective to control and eradicate the disease but these measures require more cost and labor.
**Table (2):** Sensitivity indices of $R_0$ to parameters for the model, evaluated at the parameter values given in Table 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Sensitivity index</th>
</tr>
</thead>
<tbody>
<tr>
<td>$b_h$</td>
<td>Recruitment rate of humans</td>
<td>1</td>
</tr>
<tr>
<td>$b_v$</td>
<td>Recruitment rate of vectors</td>
<td>0.999973</td>
</tr>
<tr>
<td>$\delta_h$</td>
<td>Rate of loss of immunity</td>
<td>−0.000499714</td>
</tr>
<tr>
<td>$\beta_{vh}$</td>
<td>The transmission probability as a result of biting by an infected mosquito to the susceptible human.</td>
<td>0.999973</td>
</tr>
<tr>
<td>$\beta_{hv}$</td>
<td>The transmission probability of transferring the infection from an infected human to the susceptible mosquito.</td>
<td>0.999473</td>
</tr>
<tr>
<td>$\beta_h$</td>
<td>The probability of direct transmission of the disease.</td>
<td>0.000027074</td>
</tr>
<tr>
<td>$d_h$</td>
<td>Death rate of humans</td>
<td>−1.0046</td>
</tr>
<tr>
<td>$d_v$</td>
<td>Death rate of vectors</td>
<td>−2.03186</td>
</tr>
<tr>
<td>$\eta$</td>
<td>Rate of progression of humans from exposed class to infectious class</td>
<td>0.000159974</td>
</tr>
<tr>
<td>$\beta$</td>
<td>The transmission probability of transferring the infection from a partially immune human to the susceptible mosquito.</td>
<td>0.000499725</td>
</tr>
<tr>
<td>$\gamma_v$</td>
<td>Rate of progression of vectors from exposed class to infectious class</td>
<td>0.031914</td>
</tr>
<tr>
<td>$\alpha_h$</td>
<td>Recovery rate of humans</td>
<td>−0.970105</td>
</tr>
<tr>
<td>$\xi_h$</td>
<td>Disease related death rate of humans</td>
<td>−0.0249584</td>
</tr>
</tbody>
</table>

In Theorem (4) it has been proved that the endemic equilibrium $E^*$ is globally asymptotically stable whenever $R_0 > 1$. We can decrease the endemic level of the diseased classes besides in making the reproductive number less than unity. The sensitivity indices corresponding to all the parameter values given in Table (1) for the infectious vectors and infectious humans are given in Table (3). We analyze that the endemic level of infectious vectors is most sensitive to the mortality rate of vectors and also to the recruitment rate of infectious vectors. The endemic level of infectious humans is most sensitive to the rate of loss of immunity. This suggest that the strategies
that can be applied in controlling the disease are to target the mosquito biting rate to the partially
immuned persons and death rate of the mosquitoes such as the use of insecticide-treated bed nets
and indoor residual spray.

Table (3): The sensitivity indices of the state variables at the endemic equilibrium, $x_i$, to the
parameters, $p_j$, for parameter values given in Table 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>$I_h^*$</th>
<th>$I_v^*$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$b_h$</td>
<td>0.999632</td>
<td>0.125391</td>
</tr>
<tr>
<td>$b_v$</td>
<td>0.000376465</td>
<td>1.00005</td>
</tr>
<tr>
<td>$\delta_h$</td>
<td>$-0.999605$</td>
<td>$-0.125451$</td>
</tr>
<tr>
<td>$\beta_{vh}$</td>
<td>0.000376465</td>
<td>0.0000472229</td>
</tr>
<tr>
<td>$\beta_{hv}$</td>
<td>$4.797 \times 10^{-6}$</td>
<td>0.125376</td>
</tr>
<tr>
<td>$\beta_h$</td>
<td>$-0.000372916$</td>
<td>$-0.0000467777$</td>
</tr>
<tr>
<td>$d_h$</td>
<td>$-0.00462088$</td>
<td>$-0.000579633$</td>
</tr>
<tr>
<td>$d_v$</td>
<td>$-0.00039328$</td>
<td>$-1.1574$</td>
</tr>
<tr>
<td>$\eta$</td>
<td>0.000159886</td>
<td>0.0000200557</td>
</tr>
<tr>
<td>$\beta$</td>
<td>$-1.25778 \times 10^{-6}$</td>
<td>0.0000625283</td>
</tr>
<tr>
<td>$\gamma_v$</td>
<td>0.0000120148</td>
<td>0.0319164</td>
</tr>
<tr>
<td>$\alpha_h$</td>
<td>$-0.970253$</td>
<td>$-0.121643$</td>
</tr>
<tr>
<td>$\xi_h$</td>
<td>$-0.0249446$</td>
<td>$-0.00312899$</td>
</tr>
</tbody>
</table>

Conclusions

In this paper, we work with the assumption that recovered individuals may loose their immunity
and may be infectious. The model has two steady states, one the disease disease -free and two
endemically infected steady state. We have proved by applying Routh-Hurwitz conditions to the
polynomial of degree (5) that the disease -free equilibrium is locally asymptotically stable. Global
asymptotic stability of disease -free and endemic equilibria is proved by applying the theory of
Analysis of vector-host model

Lyapunov functions. We observe that the condition $1 + R_S S_h - R_h - S_h^* S_h \geq 0$ is not necessary if the disease confers permanent immunity against re-infection. We perform sensitivity analysis of reproductive number as well as endemic level of infectious vectors and humans and analyze that the disease can be eradicated by increasing the mortality rate of mosquitoes. In practice it is not possible to remove all the infectious vectors so we determine which parameters are responsible to reduce the endemic level of infectious vectors and humans.

References


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