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New mathematical model of vertical transmission and cure of vector-borne diseases and its numerical simulation

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Abstract

In this research article, a new mathematical model for the transmission dynamics of vector-borne diseases with vertical transmission and cure is developed. The non-negative solutions of the model are shown. To understand the dynamical behavior of the epidemic model, the theory of basic reproduction number is used. As this number increases, the disease invades the population and vice versa. The effect of vertical transmission and cure rate on the basic reproduction number is shown. The disease-free and endemic equilibria of the model are found and both their local and global stabilities are presented. Finally, numerical simulations are carried out graphically to show the dynamical behaviors. These results show that vertical transmission and cure have a valuable effect on the transmission dynamics of the disease.

Keywords: Vector-borne disease; Vertical transmission; Cure; Stability; Numerical simulation

1 Introduction

Vector-borne diseases are infectious diseases transmitted to humans and animals by blood-feeding arthropods. Some common vector-borne diseases are West Nile virus, dengue fever, Rift Valley fever, malaria, and viral encephalitis caused by pathogens such as bacteria, viruses, and parasites. The arthropods are blood sucking insects and arachnids such as ticks, mosquitoes, biting flies, and lice called vectors [1]. The vectors receive pathogens from an infected host and transmit them to a human host, as humans are the major host, or animals. However, direct transmissions, such as transplantation related transmission, transfusion related transmission, and needle-stick-related transmission, are also possible [2]. In case of some diseases such as AIDS and Hepatitis B, it is possible for the offspring of infected parents to be born infected. This type of transmission is called vertical transmission. Now it is found that vector-borne diseases can also be transmitted vertically [3, 4]. Also new research shows that virus is transmitted from female mosquitos to their eggs at a high rate [5], which causes vertical transmission of the disease.

Vector-borne diseases are prevalent in hot areas, such as tropics and subtropics, and are relatively rare in temperate zones. Vector-borne infectious diseases remain amongst the

most important cause of global health illness and are major killers, particularly of children. The World Health Organization reports the numbers of deaths in different regions of the world annually. Nearly 700 million people get mosquito-borne illnesses that cause about one million deaths each year. Worldwide, malaria is the leading cause of premature mortality, particularly in children under the age of five. Nearly half of the world's population is at risk of malaria, and every year 198 million cases (uncertainty range: 124–283 million) and 584,000 deaths (range: 367,000–755,000) occur according to the World Malaria Report 2014 [6]. According to WHO, an estimated of 3.3 billion people in 97 countries are at risk of malaria. Currently, dengue threatens up to 40% of the world's population, and there may be 50–100 million infections annually [7]. More than 2.5 billion people over 40% of the world's population are now at risk of dengue.

From the above discussion it is clear that it is necessary to control such epidemic diseases. Control measures for vector-borne diseases are important because most are zoonoses. For the control measure, it is necessary to understand the dynamical features of diseases and treat the infected hosts. Therefore, deciphering the mechanisms and modeling of such diseases are of great interest. Our paper involves such an epidemic model for the transmission dynamics of vector-borne diseases that incorporates both horizontal and vertical transmission in the vector–host population.

Up to date, many mathematical models have been investigated to understand the mechanism of real world phenomena. Researchers investigate different methods to solve these models both analytically and numerically (e.g., see [8–21]). Several models of infectious diseases have been developed in the literature [22–27]. The model first proposed by Ross [28] and subsequently modified by Macdonald [29] has influenced both the modeling and the application of control strategies to a vector-borne disease. The model presented in [30] studied the analysis of a simple vector–host epidemic model with horizontal transmission. We extend their model by including vertical transmission in both vector and host populations, and treatment class in the host population with different interaction rates.

The structure of this paper is as follows: Section 1 represents the introductory remarks with a brief history. Section 2 is about the derivation of SITR epidemic model and shows the non-negative solutions of the proposed model. In Section 3, we find the disease-free and endemic equilibria and prove their local stability. In Section 4, we use mathematical analysis to establish global stability results for the proposed model. We use Lyapunov function theory to show global stability of both disease-free and endemic equilibria. Parameter estimation and numerical results are discussed in Section 5. Finally, we give conclusion.

2 Model framework

The total population sizes at time t for human hosts and vectors are denoted by $N_1(t)$ and $N_2(t)$, respectively. The population of size $N_1(t)$ is divided into four distinct classes: the susceptible population of size $S(t)$, the infectious population of size $I(t)$, the population under treatment of size $T(t)$, and the recovered population of size $R(t)$. Thus $N_1(t) = S(t) + I(t) + T(t) + R(t)$. The vector population $N_2(t)$ has the subclasses denoted by $V(t)$ and $W(t)$ for the susceptible and infected classes, respectively. Thus, $N_2(t) = V(t) + W(t)$. The mathematical model can be represented by the following nonlinear system of ordinary

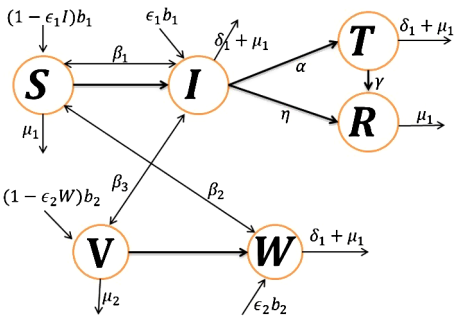

Figure 1 The flow chart represents the interactions and transfer of a vector-borne disease in both human and vector populations

differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= (1-\epsilon_1 I)b_1 - \beta_1 SI - \beta_2 SW - \mu_1 S,\\
\frac{dI}{dt} &= \epsilon_1 b_1 I + \beta_1 SI + \beta_2 SW - \alpha I - \eta I - \delta_1 I - \mu_1 I,\\
\frac{dT}{dt} &= \alpha I - \gamma T - \delta_1 T - \mu_1 T,\\
\frac{dR}{dt} &= \eta I + \gamma T - \mu_1 R,\\
\frac{dV}{dt} &= (1-\epsilon_2 W)b_2 - \beta_3 VI - \mu_2 V,\\
\frac{dW}{dt} &= \epsilon_2 b_2 W + \beta_3 VI - \delta_2 W - \mu_2 W,
\end{aligned} \tag{1}$$

with the initial conditions

$$S(0)\geq 0, \qquad I(0)\geq 0, \qquad T(0)\geq 0, \qquad R(0)\geq 0, \qquad V(0)\geq 0, \qquad W(0)\geq 0. \tag{2}$$

The human host population is recruited at a constant birth rate b_1 in which a fraction ϵ_1 were born infected from their infected parents. β_1 is the rate of direct transmission of the disease, β_2 is the vector mediated transmission rate, μ_1 is the natural mortality rate of a human. Infectious humans are treated at a rate α, recover naturally at a rate η, and suffer disease-induced death at a rate δ_1. Treated humans recover at a rate γ. It is assumed that recovered individuals acquire lifelong immunity against re-infection. Similarly, b_3 is the constant recruitment rate of vector population in which the ratio ϵ_2 are infected by birth from their infected parents. Susceptible mosquitoes become infected by biting infected human at a rate β_3, μ_2 is the natural mortality rate of vector population. Infectious vectors die due to disease at a rate δ_2. The complete dynamics of the proposed model is represented by the flow chart in Figure 1.

2.1 Properties of solutions

The proposed model (1) is a system of nonlinear ordinary differential equations with the initial conditions (2). To be epidemiologically and mathematically meaningful, it is important to prove that all the solutions with the given initial conditions will remain nonnegative and bounded for all finite time. The model shall be analyzed in a biologically meaningful feasible region governed by a positive invariant set.

Theorem 2.1 *There exists a unique and bounded solution of the system of equations* (1), *in a positively invariant set, that remains for all finite time* $t \geq 0$.

Proof The right-hand side of each equation is continuous in the convex domain $E = (t, S(t), I(t), T(t), R(t), V(t), W(t))$ of $(6+1)$-dimensional space R_+^{6+1} with continuous partial derivatives. So problem (1) has a unique solution in R_+^6 which exists for a given finite time $t \in [0, \infty)$ and initial conditions (2).

As the total population sizes are $N_1 = S + I + T + R$ and $N_2 = V + W$, so from (1) we get

$$\frac{dN_1}{dt} = b_1 - \mu_1 N_1 - \delta_1(I + T) \quad \text{and} \quad \frac{dN_2}{dt} = b_2 - \mu_2 N_v - \delta_2 I_v. \tag{3}$$

Then

$$\begin{aligned} &\frac{dN_1}{dt} \leq b_1 - \mu_1 N_1 \quad \text{and} \quad \frac{dN_2}{dt} \leq b_2 - \mu_2 N_v. \\ &\Rightarrow \quad N_1 \leq N_1(0)e^{-\mu_1(t)} + \frac{b_1}{\mu_1}\left(1 - e^{-\mu_1(t)}\right) \quad \text{and} \\ &\qquad N_2 \leq N_2(0)e^{-\mu_2(t)} + \frac{b_2}{\mu_2}\left(1 - e^{-\mu_2(t)}\right), \end{aligned}$$

which shows that

$$\lim_{t\to\infty} \sup N_1 \leq \frac{b_1}{\mu_1} \quad \text{and} \quad \lim_{t\to\infty} \sup N_2 \leq \frac{b_2}{\mu_2}. \tag{4}$$

The given initial conditions (2) make sure that $N_1(0) \geq 0$ and $N_2(0) \geq 0$. Thus the feasible region for system (1) is

$$\Phi = \left\{(S, I, T, R, V, W) \in R_+^6, N_1 \leq \frac{b_1}{\mu_1}, N_2 \leq \frac{b_2}{\mu_2}\right\}.$$

Thus the total populations and each population class remain bounded for all finite time $t \geq 0$. □

The above theorem shows that model (1) is well posed epidemiologically and mathematically in a positively invariant set Φ. We shall study the dynamics of this basic model in Φ, so, all the solutions of system (1) start and remain in Φ for all $t \geq 0$. All the parameters and state variables for the model should be non-negative for all time because they represent the number of the population sizes of humans and vectors.

3 Equilibrium points

3.1 Disease-free equilibrium

The ability to invade a population is an important concern of an infectious disease. The steady state solutions of an epidemiological model at which the population remains in the absence of disease is called disease-free equilibrium point. In order to find the disease-free equilibrium of the proposed model (1), we set the right-hand side of all equations equal to zero and set $I = T = 0$ and $W = 0$. Also there is no infected recruitment in the populations, so we put the parameters $\epsilon_1 = \epsilon_2 = 0$, which implies that $(1 - \epsilon_1)b_1 = b_1$ and $(1 - \epsilon_2)b_2 = b_2$

mean that the total recruited population is only susceptible. By direct calculations, we get the disease-free equilibrium point E_1 in the feasible region Φ, which is given by

$$E_1 = (S_1, I_1, T_1, R_1, V_1, W_1) = \left(\frac{b_1}{\mu_1}, 0, 0, 0, \frac{b_2}{\mu_2}, 0\right).$$

The dynamics of model (1) is analyzed by a dimensionless number called basic reproduction number denoted by R_0, defined as "The expected number of secondary cases produced by a typical infected individual during its entire period of infectiousness in a completely susceptible population" [31]. Mathematically, R_0 is defined as

$$R_0 \propto \left(\frac{\text{infection}}{\text{contact}}\right) \cdot \left(\frac{\text{contact}}{\text{time}}\right) \cdot \left(\frac{\text{time}}{\text{infection}}\right).$$

More precisely,

$$R_0 \propto T \cdot C \cdot D,$$

where T is the transmissibility (i.e., probability of infection given contact between a susceptible individual and an infected one), C is the average rate of contact between susceptible and infected individuals, and D is the duration of infectiousness. This quantity serves as a threshold parameter that predicts whether a disease will spread in a community or will simply die out. It can be calculated by the method of next generation matrix given in [32]. In the vector–host model (1), infected states are I, T, and W and uninfected states are S, R, and V. The matrices $\mathcal{F}$ and $\mathcal{V}$ are the rate of production of new infections and the transition rates between states, respectively, which are given by

$$\mathcal{F} = \begin{pmatrix} \epsilon_1 b_1 I + \beta_1 SI + \beta_2 SW \\ 0 \\ 0 \end{pmatrix}, \qquad \mathcal{V} = \begin{pmatrix} (\alpha + \eta + \delta_1 + \mu_1)I \\ -\alpha I + (\gamma + \delta_1 + \mu_1)T \\ -\epsilon_2 b_2 W - \beta_3 VI + (\delta_2 + \mu_2)W \end{pmatrix}.$$

At the disease-free equilibrium $S = N_1 = \frac{b_1}{\mu_1}$, $I = T = 0$, $V = N_2 = \frac{b_2}{\mu_2}$, and $W = 0$. The Jacobian matrices at the disease-free equilibrium of $\mathcal{F}$ and $\mathcal{V}$ are F and V, respectively, where

$$F = \begin{pmatrix} \epsilon_1 b_1 + \beta_1 N_1 & 0 & \beta_2 N_1 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} \alpha + \eta + \delta_1 + \mu_1 & 0 & 0 \\ -\alpha & \gamma + \delta_1 + \mu_1 & 0 \\ -\beta_3 N_2 & 0 & -\epsilon_2 b_2 + \delta_2 + \mu_2 \end{pmatrix}.$$

F and V are the rates for new infections and transitions near the equilibrium. We used MATLAB(R2010A) to find V^{-1} and FV^{-1}, which gives the times spent in each state and the total production of new infections over the course of an infection, respectively. The

largest eigenvalue of FV^{-1} is the basic reproduction number R_0, given by

$$R_0 = \frac{\epsilon_1 b_1 + \beta_1 N_1}{k} + \frac{\beta_2 \beta_3 N_1 N_2}{mk},$$

where $k = \alpha + \delta_1 + \mu_1 + \eta$ and $m = \delta_2 + \mu_2 - \epsilon_2 b_2$. When there is no vertical transmission, $\epsilon_1 = \epsilon_2 = 0$, then R_0 is the basic reproductive number for the model with only horizontal transmission. Geometrically it means that the number of new infections comes from both direct and indirect transmission. In the presence of vertical transmission, $\epsilon_1, \epsilon_2 > 0$, R_0 increases as these vertical transmission parameters increase, because vertical transmission directly increases the number of infectious populations. Also we can see the inverse relation of treatment strategies with R_0 and the direct relation with new infections and total population.

The basic reproduction number R_0 has a significant effect on the dynamics of infection. As we can see from the first and second equations of model (1),

$$\frac{dS}{dt} = b_1 - kR_0 I - \mu_1 S, \qquad \frac{dI}{dt} = k(R_0 - 1)I. \tag{5}$$

When $R_0 < 1$, it means that each infected individual infects less than one other individual averagely by ever kind of transmission, then the change in the number of infected population is negative, so the disease simply dies out. On the other hand, when $R_0 > 1$, it means that each infected individual infects more than one other individual, then the change is positive and invasion is always possible (see the survey paper by Hethcote [33]). For $R_0 = 1$, it means that each infectious individual infects one other individual as a whole, then there is no change in the infected population, so the infection constantly remains in the population. Also the effect of R_0 on the susceptible population is shown in the first equation of (5). All these facts are shown in Figures 2 and 3.

Theorem 3.1 *The disease-free equilibrium point E_1 is locally asymptotically stable if $R_0 < 1$, otherwise unstable.*

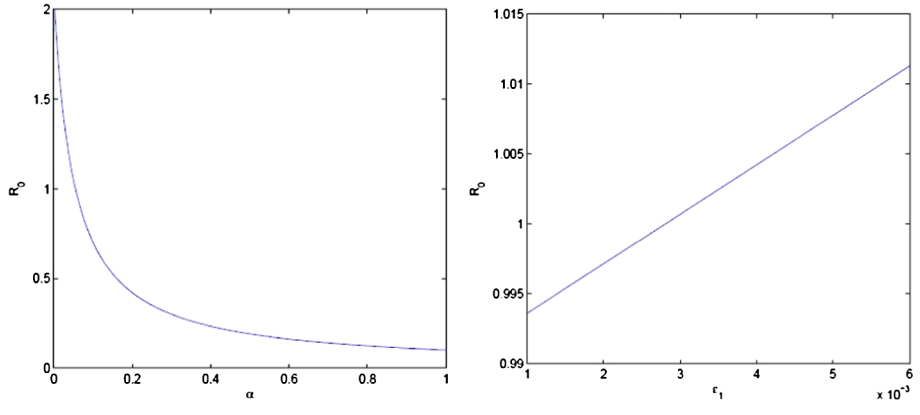

Figure 2 The first figure shows that R_0 decreases with increasing cure rate. The second figure shows that R_0 increases as vertical transmission increases

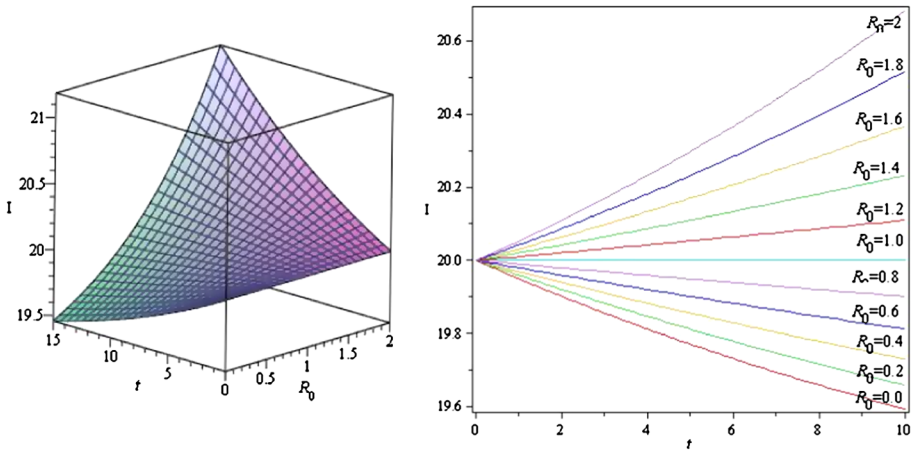

Figure 3 The figures show the threshold behavior of R_0 and its critical value $R_0 = 1$

Proof This can be proved by linearizing system (1) around E_1, which gives the following Jacobian matrix:

$$J_1 = \begin{bmatrix} -\mu_1 & -\epsilon_1 b_1 - \beta_1 \frac{b_1}{\mu_1} & 0 & 0 & 0 & -\beta_2 \frac{b_1}{\mu_1} \\ 0 & \epsilon_1 b_1 + \beta_1 \frac{b_1}{\mu_1} - k & 0 & 0 & 0 & \beta_2 \frac{b_1}{\mu_1} \\ 0 & \alpha & -l & 0 & 0 & 0 \\ 0 & \eta & \gamma & -\mu_1 & 0 & 0 \\ 0 & -\beta_3 \frac{b_2}{\mu_2} & 0 & 0 & -\mu_2 & -\epsilon_2 b_2 \\ 0 & \beta_3 \frac{b_2}{\mu_2} & 0 & 0 & 0 & -m \end{bmatrix},$$

where $l = \gamma + \delta_1 + \mu_1$.

The characteristic equation of J_1 is

$$(x + \mu_1)(x + \mu_1)(x + \mu_2)(x + l)\left(c_0 x^2 + c_1 x + c_2\right) = 0, \tag{6}$$

where

$$c_0 = \mu_1 \mu_2,$$
$$c_1 = k\mu_1\mu_2 + m\mu_1\mu_2 - \beta_1 b_1 \mu_2 - b_1 \epsilon_1 \mu_1 \mu_2,$$
$$c_2 = km\mu_1\mu_2(1 - R_0).$$

Four eigenvalues $-\mu_1$, $-\mu_1$, $-\mu_2$, and $-l$ out of six have a negative real part. The remaining two eigenvalues are the roots of the equation $c_0 x^2 + c_1 x + c_2 = 0$. For $R_0 < 1$ and $k + m > \beta_1 N_1 + b_1 \epsilon_1$, we have $c_1 > 0$ and $c_1 c_2 > 0$. So, according to the Routh–Hurwitz criteria [34], these two eigenvalues have a negative real part.

Since each eigenvalue of the characteristic equation (6) has a negative real part when $R_0 < 1$, according to the Routh–Hurwitz method [34], system (1) is locally asymptotically stable at the disease-free equilibrium point E_2 and unstable when $R_0 > 1$. The dynamical behaviors of the model at disease-free equilibrium are shown in Figure 4. □

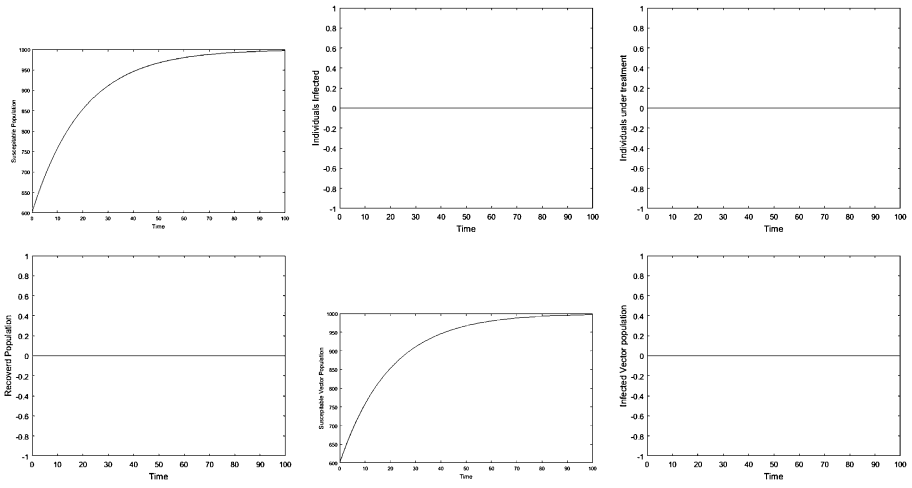

Figure 4 The plots show the dynamical behavior of the model at disease-free equilibrium

3.2 Endemic equilibrium

The constant presence of a disease or an infectious agent within a given geographic area is called endemic. The endemic equilibrium state is the state where the disease cannot be totally eradicated but remains in the population. In order to find positive solutions of system (1), let $E_2 = (S_2, I_2, T_2, R_2, V_2, W_2)$ represent any arbitrary endemic equilibrium. Setting left-hand side equal to zero and solving the equations simultaneously at steady state, we obtain

$$S_2 = \frac{b_1 - kI_2}{\mu_1}, \qquad T_2 = \frac{\alpha I_2}{l}, \qquad R_2 = \frac{(l\eta + \gamma\alpha)I_2}{\mu_1 l},$$
$$V_2 = \frac{mW_2}{\beta_3 I_2}, \qquad W_2 = \frac{\mu_2\beta_3 N_2 I_2}{\beta_3(\delta_2 + \mu_2)I_2 + \mu_2 m}.$$

Theorem 3.2 *The endemic equilibrium point E_2 is locally asymptotically stable if $R_0 > 1$, otherwise unstable.*

Proof To show these results, we linearize system (1) around E_2, which gives the following Jacobian matrix:

$$J_2 = \begin{bmatrix} -Q & -T & 0 & 0 & 0 & -\beta_2 S_2 \\ Q - \mu_1 & T - K & 0 & 0 & 0 & \beta_2 S_2 \\ 0 & \alpha & -l & 0 & 0 & 0 \\ 0 & \eta & \gamma & -\mu_1 & 0 & 0 \\ 0 & -\beta_3 V_2 & 0 & 0 & -\beta_3 I_2 - \mu_2 & -\epsilon_2 b_2 \\ 0 & \beta_3 V_2 & 0 & 0 & \beta_3 I_2 & -m \end{bmatrix},$$

where

$$Q = \beta_1 I_2 + \beta_2 W_2 + \mu_1, \qquad T = \epsilon_1 b_1 + \beta_1 S_2.$$

Two of the eigenvalues are $-\mu_1$ and $-l$. The remaining eigenvalues are the eigenvalues of the following matrix:

$$J_2^* = \begin{bmatrix} -Q & -T & 0 & -\beta_2 S_2 \\ Q-\mu_1 & T-K & 0 & \beta_2 S_2 \\ 0 & -\beta_3 V_2 & -\beta_3 I_2 - \mu_2 & -\epsilon_2 b_2 \\ 0 & \beta_3 V_2 & \beta_3 I_2 & -m \end{bmatrix}.$$

We make an elementary row operation for the Jacobian matrix J_2^* to obtain the following matrix:

$$J_2^* = \begin{bmatrix} -Q & -T & 0 & -\beta_2 S_2 \\ 0 & \frac{\mu_1 T}{Q} - K & 0 & \frac{\mu_1}{Q}\beta_2 S_2 \\ 0 & 0 & -\mu_2 & -\epsilon_2 b_2 - m \\ 0 & 0 & 0 & -M \end{bmatrix},$$

where

$$M = m + L + \frac{\beta_3 I_2}{\mu_2}(m + \epsilon_2 b_2) \quad \text{and} \quad L = m + \frac{\mu_1 \beta_2 \beta_3 S_2 V_2}{\mu_1 T - KQ}.$$

J_2^* is a lower triangular matrix and its eigenvalues are the elements of the main diagonal which are given by $-Q$, $\frac{\mu_1 T}{Q} - K$, $-\mu_2$, and $-M$. Three of the eigenvalues have a negative real part. The second eigenvalue $\frac{\mu_1 T}{Q} - K$ has a negative real part if and only if $\frac{\mu_1 T}{Q} - K < 0$. Using the value of Q and T, we can rewrite this equation by rearranging it as follows:

$$-2\beta_1\beta_3 K(\delta_2 + \mu_2) I_2^2 + \left[\beta_3(\delta_2 + \mu_2)\mu_1 K(1 - R_0)\right] I_2 + \mu_2 m \mu_1 K(1 - R_0). \tag{7}$$

All the coefficients of this equation are negative if $R_0 > 1$. Thus all the eigenvalues have negative real parts, which shows that the endemic equilibrium point E_2 is locally asymptotically stable iff $R_0 > 1$. □

4 Global stability analysis

In this section, we study the global analysis of the disease-free and endemic equilibria using the direct Lyapunov method which requires the construction of a function with specific properties. In order to do this, we derive the following results.

Theorem 4.1 *When $R_0 < 1$, then the disease-free equilibrium E_1 of system* (1) *is globally asymptotically stable on* Φ.

Proof To show the global stability of the disease-free equilibrium E_1, we construct the following Lyapunov function, following the method used in [35]:

$$U(t) = I + \frac{\beta_2 b_1}{m\mu_1} W, \quad \text{with time derivative } U'(t) = \dot{I} + \frac{\beta_2 b_1}{m\mu_1}\dot{W}. \tag{8}$$

Then U is C^1 on the interior of Φ, E_1 is the global minimum of U on Φ, and $U(t) = 0$ at E_1. Putting the values from model (1), we obtain

$$\begin{aligned} U'(t) &= \epsilon_1 b_1 I + \beta_1 SI + \beta_2 SW - \alpha I - \eta I - \delta_1 I - \mu_1 I \\ &\quad + \frac{\beta_2 b_1}{m\mu_1}(\epsilon_2 b_2 W + \beta_3 VI - \delta_2 W - \mu_2 W), \\ &\leq \epsilon_1 b_1 I + \beta_1 N_1 I + \beta_2 N_1 W - kI \\ &\quad + \frac{\beta_2 b_1}{m\mu_1}(\beta_3 N_2 I - mW), \quad \text{since } S \leq N_1, \text{ and } V \leq N_2 \\ &= (R_0 - 1)I. \end{aligned} \tag{9}$$

Equation (9) shows that $U'(t)$ is negative if $R_0 < 1$. Also $U'(t) = 0$ at E_1. Substituting $I = T = R = W = 0$ in the equations for $S(t)$ and $V(t)$ of model (1) shows that $S(t) \to \frac{b_1}{\mu_1}$ and $V(t) \to \frac{b_2}{\mu_2}$ as $t \to \infty$. Similarly, substituting in the equations for $T(t)$ and $R(t)$ shows that $(T(t), R(t)) \to (0, 0)$ as $t \to \infty$. Therefore the largest compact invariant set in $\{(S_h, E_h, I_h, N_h, S_v, E_v, I_v) \in \Phi : U'(t) = 0\}$ is the singleton disease-free equilibrium point $\{E_f\}$. Therefore, from LaSalle's principle [36], the disease-free equilibrium E_f is globally asymptotically stable in Φ. □

Theorem 4.2 *For $R_0 > 1$, the endemic equilibrium E_2 is globally asymptotically stable.*

Proof For the global stability of the endemic equilibria, we construct the following Lyapunov function:

$$Y(t) = \frac{1}{\beta_1 S_2}(S - S_2 \log S) + \frac{1}{\beta_3 V_2}(V - V_2 \log V) + \frac{1}{\beta_1 S_2} I + \frac{1}{\beta_3 V_2} W. \tag{10}$$

Taking the time derivative of W, we get

$$\begin{aligned} Y'(t) &= \frac{1}{\beta_1 S_2}(S - S_2)\left[\frac{b_1}{S} - \frac{\epsilon_1 b_1 I}{S} - \beta_1 I - \beta_2 W - \mu_1\right] \\ &\quad + \frac{1}{\beta_3 V_2}(V - V_2)\left[\frac{b_2}{V} - \frac{\epsilon_2 b_2 W}{V} - \beta_3 I - \mu_2\right] \\ &\quad + \frac{1}{\beta_1 S_2}[\beta_1 SI + \beta_2 SW - K_1 I], \end{aligned} \tag{11}$$

where $K_1 = \alpha + \delta_1 + \mu_1 + \eta - \epsilon_1 b_1$. Let us consider

$$\begin{aligned} &\mu_1 = \frac{b_1}{S_2} \quad \Rightarrow \quad b_1 = \mu_1 S_2, \qquad \mu_2 = \frac{b_2}{V_2} \quad \Rightarrow \quad b_2 = \mu_2 V_2, \\ &K_1 = 2\beta_1 S_2, \quad \text{and} \quad m = \frac{\beta_2 \beta_3 V_2}{\beta_1}. \end{aligned} \tag{12}$$

Rearranging equation (11), we get

$$Y'(t) = -\frac{\mu_1}{\beta_1}\left(\frac{S}{S_2} + \frac{S_2}{S} - 2\right) - \frac{\mu_2}{\beta_3}\left(\frac{V}{V_2} + \frac{V_2}{V} - 2\right). \tag{13}$$

Since

$$\frac{S}{S_2} + \frac{S_2}{S} \geq 2 \quad \text{and} \quad \frac{V}{V_2} + \frac{V_2}{V} \geq 2, \tag{14}$$

because the arithmetic mean is greater than or equal to the geometric mean. Thus $Y'(t) \leq 0$ for all $(S, I, T, R, V, W) \in \Phi$ and the equality $(Y'(t) = 0)$ holds for E_2. The proof is completed as in the proof of Theorem (4.1). □

5 Numerical simulation and graphs

We collect data from different sources and use the Runge–Kutta fourth order scheme to solve the model. Some of the parameter values are based on reality, for example, the death rate of humans by nature, corresponding to life expectancy of a 70-year-old human, is $\mu_1 = 0.000039$ per day, and the death rate of mosquitoes is $\mu_2 = 0.1$ per day corresponding

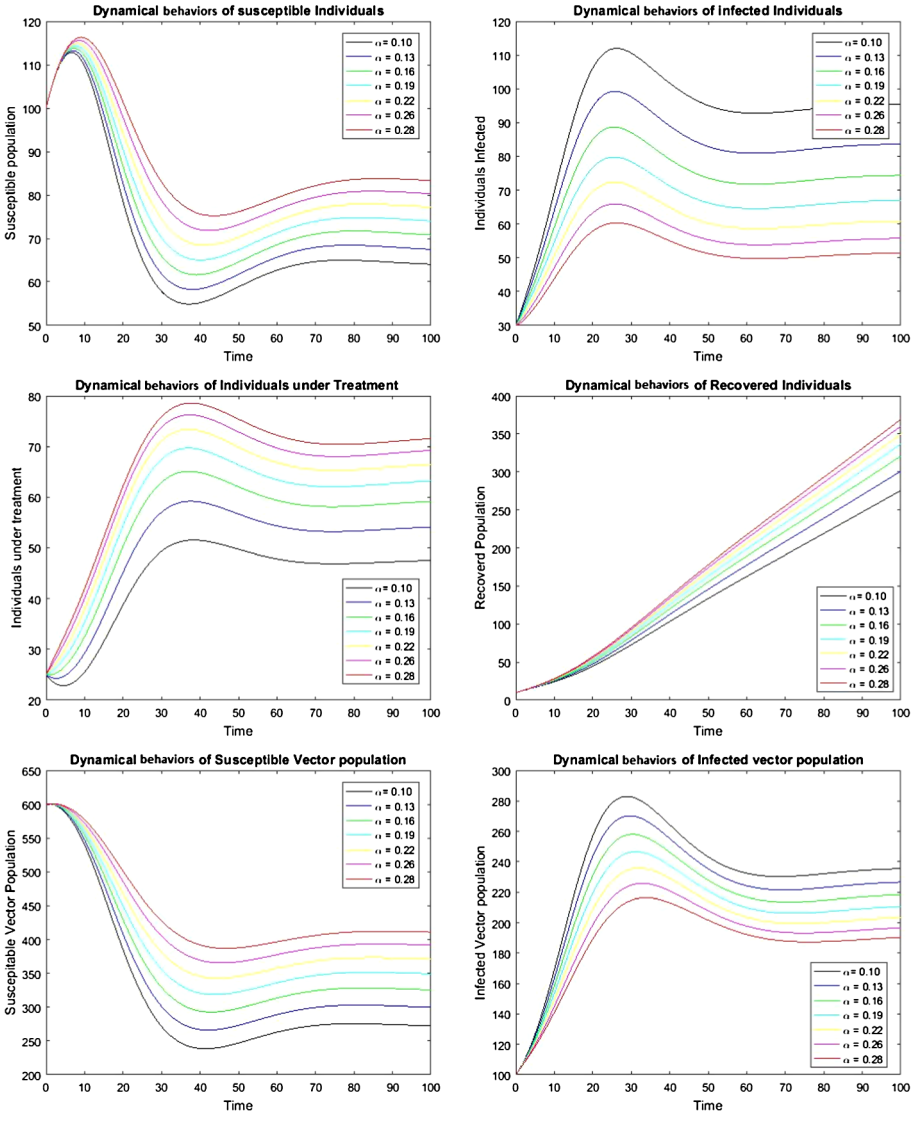

Figure 5 The plots show the dynamical behaviors of population sizes with increasing cure rate

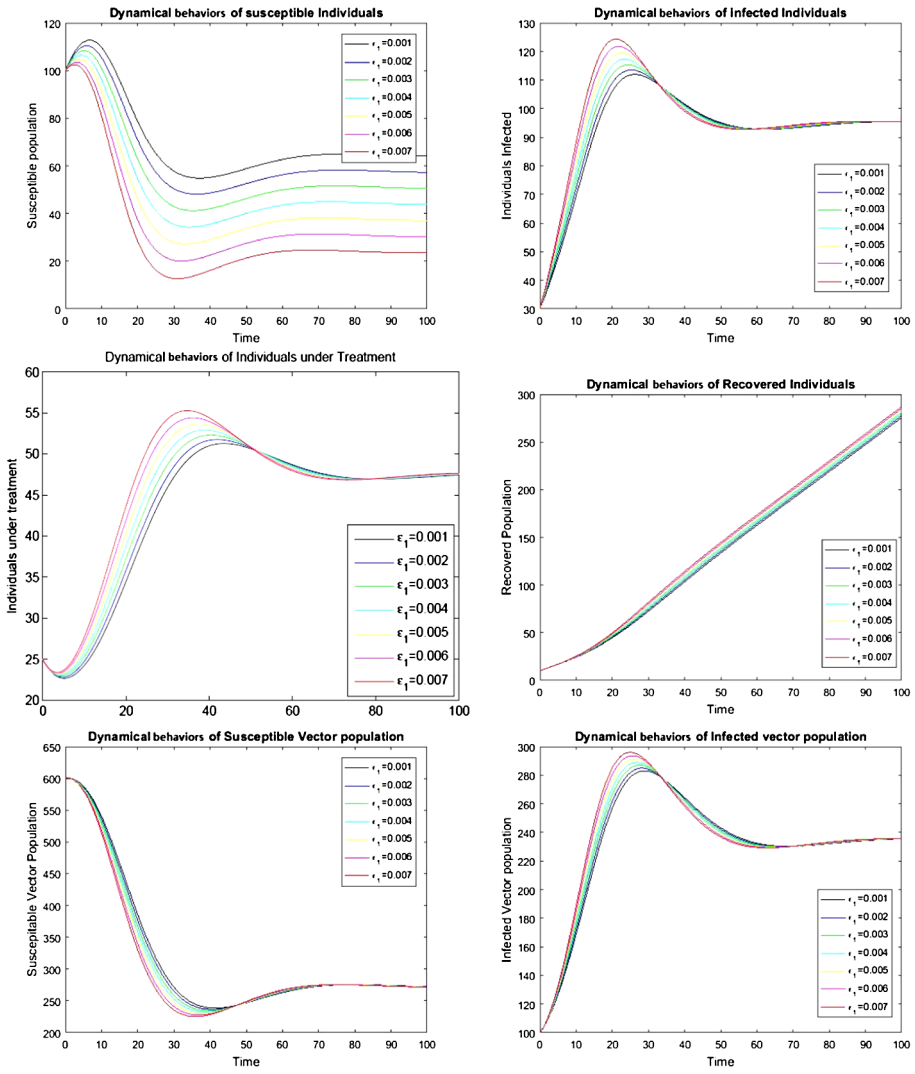

Figure 6 The plots show the dynamical behavior of population sizes with increasing vertical transmission rate

to mosquito's average life span of 10 days. Some of the parameter values are chosen from [25, 35]. The human's and vector's recruitment rates are $b_1 = 20$ and $b_2 = 100$ per day, respectively. The disease-induced death rates of humans and mosquitoes are $\delta_1 = 0.01$ and $\delta_2 = 0.21$, respectively. $\beta_1 = 0.00001$ and $\beta_2 = 0.0012$ are the transmission probabilities of dengue from human to human and vector to human population, respectively, $\beta_3 = 0.001$ is the transmission probability of dengue from human to vector population. Given different values to the treatment parameter $0 \leq \alpha \leq 1$ to check the treatment effects. The natural recovery rate is $\eta = 0.01$, and the recovery rate due to treatment is $\gamma = 0.4$. We suppose the values of ϵ_1, ϵ_2 and the initial population sizes. In rare cases the new offspring of infected parents are infected so take $\epsilon_1 = 0.001$ and the vertical transmission rate for mosquitos is $\epsilon_2 = 0.002$. For initial values, let $S(0) = 100$, $I(0) = 30$, $T(0) = 25$, $R(0) = 10$, $V(0) = 600$, and $W(0) = 100$. After solving we draw the results graphically and show the effect of cure rate and vertical transmission. Figure 5 shows the effect of cure rate on each population class, and Figure 6 shows the effect of vertical transmission. Figures 7 and 8 show the

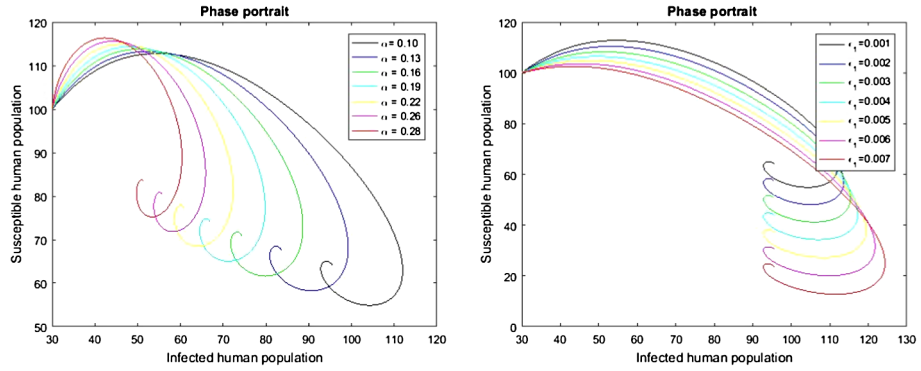

Figure 7 The plots show the phase portrait of the susceptible human population versus the infected human population

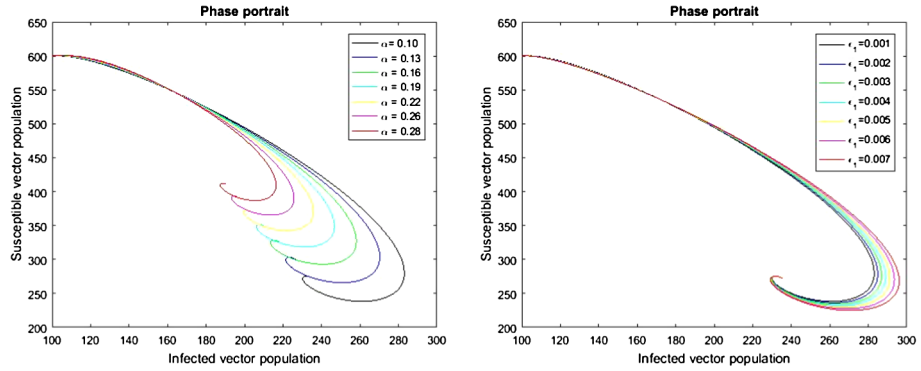

Figure 8 The plots show the phase portrait of the susceptible vector population versus the infected vector population

phase portraits of susceptible population versus infected population of human and vector populations, respectively.

6 Conclusion

The spread of different infectious diseases causes very high mortality rates in a population. Vector-borne diseases are infectious diseases transmitted to humans and animals through vectors. These diseases propagate from the infected to the susceptible population in different ways. This paper formulated an epidemic model for the transmission dynamics of vector-borne diseases with both vertical and horizontal transmissions with treatment strategy. The equilibrium points and the basic reproduction of the model are found. The basic reproduction number, which is a threshold quantity, has an important role in the epidemiology of the disease. As this number increases the disease invades the population, and as it decreases the disease simply dies out. Figure 2 shows that R_0 decreases as treatment strategies increase and increases as vertical transmission increases. Figure 3 shows the threshold behavior of R_0 and the critical value $R_0 = 1$. As R_0 increases, the infected population increases with time. For $R_0 < 1$, the number of infected population decreases; for $R_0 = 1$, the infected population remains constant; and for $R_0 > 1$, the number of infected population increases. It is also shown that when $R_0 < 1$ the disease-free equilibrium is lo-

cally and globally asymptotically stable; and for $R_0 > 1$, the positive endemic equilibrium is locally and globally asymptotically stable.

Numerical simulations are carried out graphically to show the dynamical behavior of the diseases. Figure 5 shows the effect of cure rate on the transmission dynamics of the disease. As treatment strategy increases, the susceptible population and the recovered human population increase while the infected population decreases. Figure 6 shows the effect of vertical transmission. As vertical transmission increases, the susceptible population decreases and the infected population increases. Finally, Figures 7 and 8 show the phase portraits of the susceptible populations versus the infected populations which move towards the stable points.

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Competing interests
The authors declare that they have no competing interests.

Authors' contributions
All authors read and approved the final manuscript.

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