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STABILITY ANALYSIS OF VECTOR-BORNE DISEASES MODEL WITH TREATMENT VIA FRACTIONAL-ORDER

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Abstract. In this article vector borne disease transmission model with treatment is analyzed via fractional-order. We analyzed, the global stability of equilibria of the proposed model under certain parametric conditions. A numerical simulations of this model is also conducted to investigate the effect of certain major parameters on the disease spread.

Keywords: fractional-order; stability; vector-borne.

2010 AMS Subject Classification: 92D25, 34C23, 34D23, 37B25

1. INTRODUCTION

Over recent days, the compartmental model has played a crucial role in the advancement of mathematical epidemiology, to capture disease dynamics in each compartment and to carry out effective medicine [1]. In this row, let us discuss few properties of Vector-borne diseases transmission as we propose to study in this paper. Vector-borne diseases are infectious diseases spread by arthropods such as mosquitoes, biting flies, ticks, etc. to humans and animals. Some

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of the well-known vector-borne diseases include Western dengue fever, Nile virus, malaria, viral encephalitis, and so on, transmitted by pathogens such as worms, bacteria, and viruses. When bearing these pathogens from an infected host, they are transmitted to the human host. In areas with hot weather, such as tropics and sub-saharan deserts, vector-borne diseases are more common. Diseases are some of the most relevant cause of global health illnesses and a lot of them are killer diseases. Based on the above evidence, the management of these diseases is crucial. It is therefore important to understand the complex behaviors of the diseases to achieve a thorough treatment of the infected hosts [2–17].

Real-life problems can also be modeled through ordinary and partial differential equations that do not depend on past history. However, the model investigated under classical derivatives and integrals suffer by the restriction for the use of various degrees of freedom. After noticing some limitations imposed by models with local classical derivatives, many authors converted to fractional calculus, a comparatively new and widely used field of mathematical analysis in which nonlocal differential operators possessing memory effects are used to model natural and physical phenomena showing anomalous behavior and nonlocal dynamics [18–24]. The use of fractional derivatives in the COVID-19 model under study is considered since memory effects significantly impact the evolution of an epidemiological process related to humans, and memory effects play a significant role in disease transmission. Furthermore, memory effects are appropriate to include in epidemiological investigations of real dynamical processes since such systems rely on memory strength, governed by order of fractional derivative [25, 26].

Since our ultimate goal is to examine the dynamic behavior of fractional-order vector-borne disease model, we can have a briefing on the impact of memory effect. Most infectious diseases are caused by infection, and some of them take a period of time to spread infection. This reveals that memory effect in epidemic models are worthwhile to observe their complicated dynamics with fractional-order system [27–29].

The rest of this paper is organized as follows. In Section 2, we present the description of the fractional-order system. In Section 3, we recall some preliminaries, then we compute equilibria and basic reproduction number and also we discuss the global stability of two equilibria. In Section 4, we present some numerical simulations of proposed system (1). Eventually, we end

with a conclusion in Section 5. We have discussed the future scope in Section 6. In next section, we brief on the nonlinear fractional-order vector borne system (1).

2. MODEL DESCRIPTION

The motivation of present work is from the article of Abdullah et al. [30], which deals with the dynamics of vector-borne diseases with vertical transmission and treatment. Let $N_1(t)$ and $N_2(t)$, denote the total population sizes of humans and vectors at time t respectively. The total human population $N_1(t)$ is partitioned into four different classes: the susceptible human population is denoted by $S(t)$, the infectious human population is denoted by $I(t)$, the human population under treatment is denoted by $T(t)$, and the recovered human population is denoted by $R(t)$. Thus, $N_1(t) = S(t) + I(t) + T(t) + R(t)$. The vector population $N_2(t)$ is divided into two classes: the susceptible vector population is denoted by $V(t)$ and the infected vector population is denoted by $W(t)$. Thus, $N_2(t) = V(t) + W(t)$. All six classes are mutually disjoint. The modified Abdullah et al. [30] model is governed by system of nonlinear fractional-order differential equation as follows,

$$
\begin{aligned}
D_t^\alpha S(t) =& \alpha_1 - \beta_1 S(t)I(t) - \beta_2 S(t)W(t) - \mu_1 S(t),\\
D_t^\alpha I(t) =& \beta_1 S(t)I(t) + \beta_2 S(t)W(t) - (\theta + \eta + d_1 + \mu_1)I(t),\\
D_t^\alpha T(t) =& \theta I(t) - (\gamma + d_1 + \mu_1)T(t),\\
D_t^\alpha R(t) =& \eta I(t) + \gamma T(t) - \mu_1 R(t),\\
D_t^\alpha V(t) =& \alpha_2 - \beta_3 V(t)I(t) - \mu_2 V(t),\\
D_t^\alpha W(t) =& \beta_3 V(t)I(t) - (d_2 + \mu_2)W(t).
\end{aligned} \tag{1}
$$

The fractional derivative of model (1) is in the sense of Caputo. Here $\alpha \in (0,1]$ is the order of the fractional derivative and D_t^α denotes $\dfrac{d^\alpha}{dt^\alpha}$. The classical version of the proposed system (1) is retained when $\alpha = 1$. The description of parameters used in system (1) is provided in Table 1. The system (1), with the initial conditions

$$
S(0) = S_2, I(0) = I_2, T(0) = T_2, R(0) = R_2, V(0) = V_2 \text{ and } W(0) = W_2. \tag{2}
$$

Furthermore, we assume that

$$S(t) > 0, I(t) \geq 0, T(t) \geq 0, R(t) \geq 0, V(t) \geq 0, W(t) \geq 0, \ for\ all\ t > 0. \tag{3}$$

TABLE 1. Parameters description

Parameters	Description	Values as taken in [30]	Units [30]
α_1	The recruitment rate of human population	50	day^{-1}
α_2	The recruitment rate of vector population	100	day^{-1}
θ	The rate constant at which the infectious human are treated	0.4	day^{-1}
η	The natural recovery rate	0.0001	day^{-1}
γ	The rate at which treated humans recover	0.004	day^{-1}
μ_1	Natural death rate of a human population	0.00039	day^{-1}
μ_2	Natural death rate of a vector population	0.1	day^{-1}
d_1	The death rate of human due to disease	0.01	day^{-1}
d_2	The death rate of vector population due to disease	0.21	day^{-1}
β_1	The rate of direct transmission of the disease	0.0000001	day^{-1}
β_2	The rate of vector mediated transmission of the disease	0.00000012	day^{-1}
β_3	The rate at which Susceptible mosquitoes become infected by biting infected human	0.0000991	day^{-1}

3. MODEL ANALYSIS

The nonlinear fractional-order vector borne disease model (1) is studied in this section for its analytical properties.

3.1. Preliminaries. In this section, we recall some basic definitions of fractional-order derivatives. Consider the system

$$D^{\alpha} x(t) = f(x), \alpha \in (0,1], x \in R^n, \tag{4}$$

where D^{α} is the Caputo fractional derivative which is given in the following definition.

Definition 1. *[21] The Caputo fractional derivative of order α of a function $f(t) \in C^n([t_1,\infty),\mathbb{R})$ is defined as*

$$D_t^{\alpha} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_{t_1}^{t} \frac{f^{(n)}(\xi)}{(t-\xi)^{\alpha+1-n}} d\xi,$$

where $t \geq t_1$, $\Gamma(.)$ is the Gamma function, and n is the positive integer such that $\alpha \in (n-1,n)$. When $\alpha \in (0,1)$, one has

$$D_t^{\alpha} f(t) = \frac{1}{\Gamma(1-\alpha)} \int_{t_1}^{t} \frac{f^{'}(\xi)}{(t-\xi)^{\alpha}} d\xi.$$

3.2. The basic reproduction number and equilibrium points. The biological meaning of the basic reproduction number (R_0) is defined as the average number of secondary infections caused by a single infectious individual during the course of its infectious period. A disease dies out if $R_0 < 1$ and spreads if $R_0 > 1$.

The basic reproduction number for the nonlinear system (1) has been derived using the method of the next-generation matrix [31].

$R_0 = \dfrac{\alpha_1\beta_1\mu_2 m + \alpha_1\alpha_2\beta_2\beta_3}{\mu_1\mu_2 km}$.

The given non-linear dynamical system (1) possesses two equilibrium points which are described below.

i) The disease free equilibrium point is $E_0(S_0,0,0,0,V_0,0) = E_0\left(\dfrac{\alpha_1}{\mu_1},0,0,0,\dfrac{\alpha_2}{\mu_2},0\right)$,

ii) The endemic equilibrium point is $E_1(S_1,I_1,T_1,R_1,V_1,W_1)$,

where,

$$S_1 = \frac{\alpha_1 - kI_1}{\mu_1}, I_1 = \frac{lT_1}{\theta}, T_1 = \frac{\theta I_1}{l}, R_1 = \frac{(l\eta + \gamma\theta)I_1}{\mu_1 l},$$
$$V_1 = \frac{mW_1}{\beta_3 I_1} \text{ and } W_1 = \frac{\alpha_2\beta_3 I_1}{\beta_3(d_2+\mu_2)I_1 + \mu_2 m}.$$

Where,

$k = \theta + d_1 + \mu_1 + \eta$, $m = d_2 + \mu_2$ and $l = \gamma + d_1 + \mu_1$.

In the following section, by constructing a Lyapunov functional, we can actually obtain globally asymptotic stability of the system (1) under certain conditions.

3.3. Global behavior at equilibrium points. To establish global stability, we construct suitable Lyapunov functionals and use LaSalle's invariance principle theory.

Lemma 1. *[32] let $y(t) \in \mathbb{R}^+$ be derivable and continuous function. Then, for any time $t \geq t_0$,*

$$ {}_{t_0}D^{\alpha}\left[y(t) - y^* - y^* ln\frac{y(t)}{y^*}\right] \leq \left(1 - \frac{y^*}{y(t)}\right){}_{t_0}D^{\alpha}y(t), \tag{5}$$

$\forall \alpha \in (0,1)$, $y^* \in \mathbb{R}^+$.

Note that for $\alpha = 1$, the inequalities in (5) *becomes equalities.*

Let us denote

$$\left[y(t) - y^* - y^* ln\frac{y(t)}{y^*}\right] = g\left(\frac{y(t)}{y^*}\right)$$

in upcoming results.

In this section, we analyze the global stability of the system (1) at the equilibrium points. To establish global stability, we construct suitable Lyapunov functionals and use the theory of LaSalle's invariance principle. We define a function $g : \mathbb{R}^+ \longrightarrow \mathbb{R}^+ \cup \{0\}$ as $g(u) = u - 1 - \ln u$. Note that $g(u) \geq 0$, for any $u > 0$ and attains a global minimum 0 at $u = 1$.

Theorem 3.3.1. *If $R_0 < 1$, the disease free equilibrium E_0 of the system* (1) *is globally asymptotically stable.*

Proof. Let $(S(t), I(t), T(t), R(t), V(t), W(t))$ be any positive solution of the system (1), define a Lyapunov functional $W_1(t)$ as follows,

$$W_1(t) = I(t) + P_1 W(t). \tag{6}$$

where, $P_1 = \dfrac{\beta_1 \alpha_1}{m\mu_1}$.

Differentiating $W_1(t)$ along the solution of system (1), we obtain

$$\begin{aligned} \frac{dW_1(t)}{dt} &\leq \beta_1 I(t)S(t) + \beta_2 W(t)S(t) - (\theta + \eta + d_1 + \mu_1)I(t) \\ &\quad + P_1\left(\beta_3 V(t)I(t) - (d_2 + \mu_2)W(t)\right). \end{aligned} \tag{7}$$

$$\begin{aligned} \frac{dW_1(t)}{dt} &\leq -(\theta + \eta + d_1 + \mu_1)I(t) + P_1\left(\beta_3 V(t)I(t) - (d_2 + \mu_2)W(t)\right) \\ &\quad + \beta_1 I(t)S(t) + \beta_2 W(t)S(t). \end{aligned} \tag{8}$$

By using the values of P_1 we have,

$$\begin{aligned}\frac{dW_1(t)}{dt} \leq & -kI(t)+\frac{\beta_1\alpha_1}{m\mu_1}\Big(\beta_3V(t)I(t)-(d_2+\mu_2)W(t)\Big)\\ & +\beta_1\frac{\alpha_1}{\mu_1}I(t)+\beta_2\frac{\alpha_1}{\mu_1}S(t).\end{aligned} \tag{9}$$

$$\frac{dW_1(t)}{dt} \leq (R_0-1)kI(t). \tag{10}$$

It follows from Eq.(14) that $\frac{dW_1(t)}{dt} \leq 0$ with equality holding at $S(t)=S_0, I(t)=T(t)=R(t)=0, V(t)=V_0, W(t)=0$. By the LaSalle invariance principle, the disease free equilibrium of the model (1) is globally asymptotically stable. □

Theorem 3.3.2. *Let $R_0>1$, if endemic equilibrium E_1 of the system (1) exist, then it is is globally asymptotically stable, provided that $k>\frac{\beta_2\alpha_2}{\mu_2}+\frac{\beta_1\alpha_1}{\mu_1}$ and $m>\frac{\beta_2\alpha_1}{\mu_1}$.*

Proof. Let us consider a Lyapunov functional $W_2(t)$ as follows,

$$W_2(t) = g\left(\frac{I(t)}{I_1}\right)+g\left(\frac{W(t)}{W_1}\right). \tag{11}$$

Differentiating the Eq. (11), with respect to time yields

$$\begin{aligned}\frac{dW_2(t)}{dt} \leq & \left(1-\frac{I_1}{I(t)}\right)\Big(\beta_1S(t)I(t)+\beta_2S(t)W(t)-(\theta+\eta+d_1+\mu_1)I(t)\Big)\\ & +\left(1-\frac{W_1}{W(t)}\right)\Big(\beta_3V(t)I(t)-(d_2+\mu_2)W(t)\Big)\\ & +\beta_1I(t)S(t)-\beta_1I(t)S(t)+\beta_2W(t)S(t)-\beta_2W(t)S(t).\end{aligned} \tag{12}$$

$$\begin{aligned}\frac{dW_2(t)}{dt} \leq & -kI(t)-\frac{I_1}{I(t)}\beta_1I(t)S(t)-\frac{I_1}{I(t)}\beta_2I(t)S(t)\\ & +I_1K+\beta_3V(t)I(t)-mW(t)-\frac{W_1}{W(t)}\beta_3V(t)I(t)+mW_1\\ & +\beta_1S(t)I(t)+\beta_2S(t)W(t).\end{aligned} \tag{13}$$

$$\frac{dW_2(t)}{dt} \leq \left(-k+\frac{\beta_2\alpha_2}{\mu_2}+\frac{\beta_1\alpha_1}{\mu_1}\right)I(t)+\left(-m+\frac{\beta_2\alpha_1}{\mu_1}\right)W(t). \tag{14}$$

It follows from Eq.(14) that by the LaSalle invariance principle, the endemic equilibrium of the model (1) is globally asymptotically stable. □

4. NUMERICAL SIMULATION AND DISCUSSION

Here, we numerically solve the system of nonlinear fractional-order vector borne disease transmission model to observe the dynamics of the system (1). A nonlinear fractional-order vector borne disease transmission model (1) has been solved numerically by adopting predictor-corrector algorithm [33–35]. The solution trajectories are plotted in following figures. Using the values from Table 1 we can demonstrate how the dynamics of the system depends on the parameter values using *MATLAB(R2015)*.

Fig.(1) has been plotted using the values from Table 1. Here, Fig.(1), depicts the graphical representation of the system (1) when $R_0 < 1$. It can be observed that the disease dies out after some time and only the susceptible human population and the susceptible vector population attains a constant value, hereby, indicating that when $R_0 < 1$, the disease cannot persist for longer duration of time, biologically. Fig.(2) has been plotted using the values from Table 1. except for $\beta_1 = 0.0000004$, $\beta_2 = 0.00001$, $\beta_3 = 0.0191$, $\theta = 0.6$ and $\eta = 0.01$. Also Fig.(2), depicts the graphical representation of the system (1) when $R_0 > 1$. It can be observed that the infected human population and the infected vector population approaches a constant value. This biologically means that the infected population exist as $R_0 > 1$.

It can be seen from Fig.(1)-Fig.(2), that fractional-order solution is the trace of its integer order. The findings indicate that the order of the fractional derivative has a significant impact on the dynamic process. In addition, the results show that the memory effect is zero for $\alpha = 1$. In case of fractional-order system memory effect is indirectly proportional to the value of α.

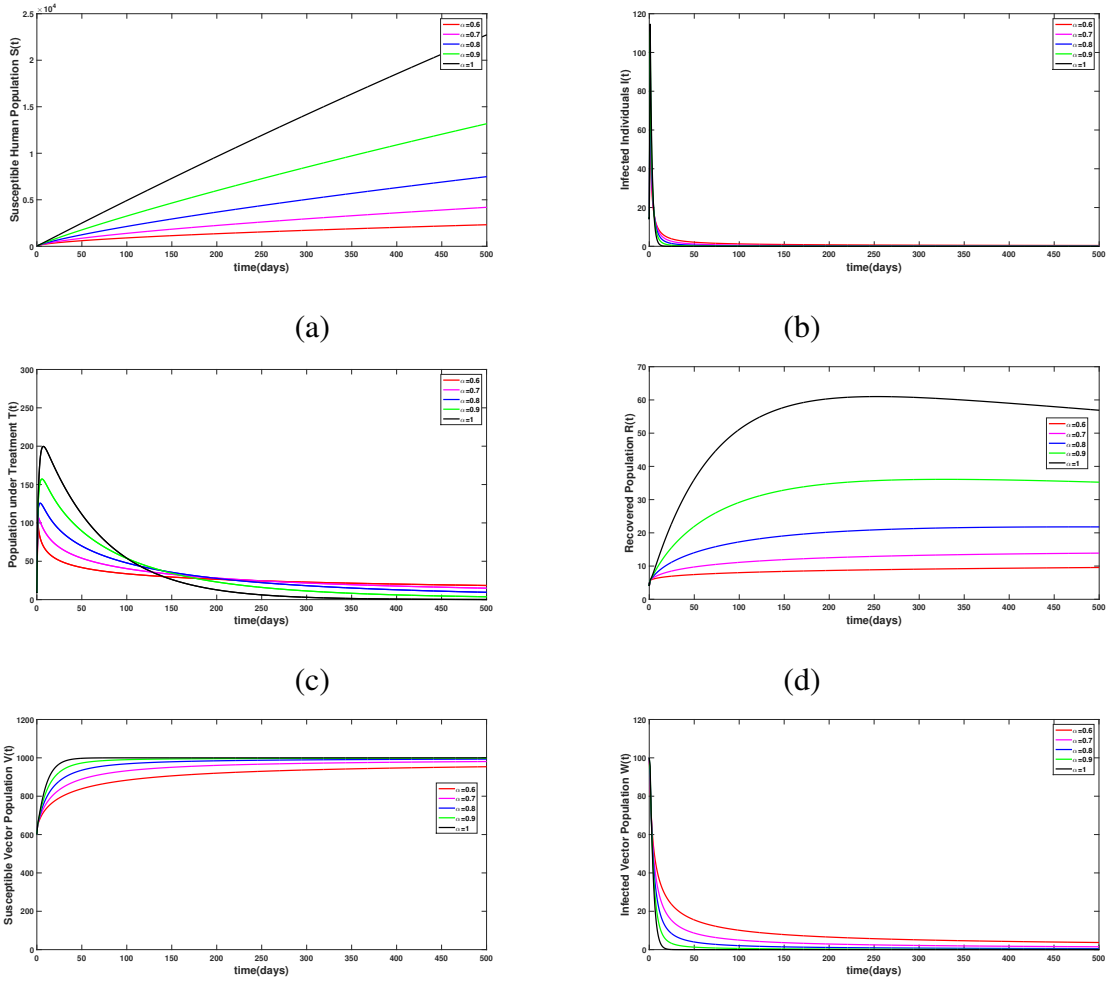

FIGURE 1. The above figure denotes graph trajectories of $S(t), I(t), T(t), R(t), V(t)$ and $W(t)$ versus time t of system (1) choosing the initial conditions as $S(0) = 20, I(0) = 15, T(0) = 10, R(0) = 5, V(0) = 600$ and $W(0) = 100$. Where $R_0 = 0.8913 < 1$.

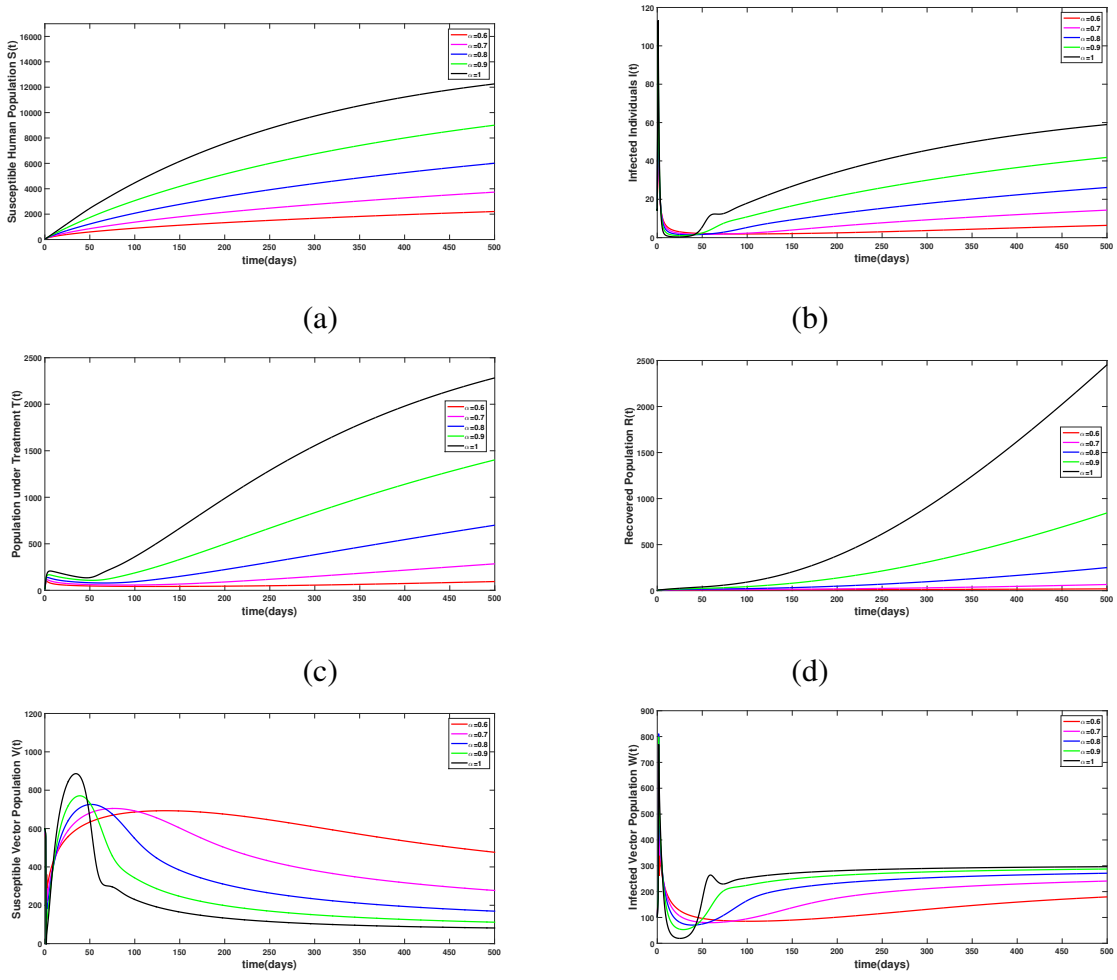

FIGURE 2. The above figure denotes graph trajectories of $S(t), I(t), T(t), R(t), V(t)$ and $W(t)$ versus time t of system (1) choosing the initial conditions as $S(0) = 20, I(0) = 15, T(0) = 10, R(0) = 5, V(0) = 600$ and $W(0) = 100$. Where $R_0 = 3.6787 > 1$.

5. CONCLUSION

This paper has been concerned with modeling the fractional-order vector borne disease transmission with treatment. Fractional-order system provides better dynamics than the classical system. We have shown through the mathematical analysis of the model that the basic reproduction number R_0 acts as a threshold parameter. The disease dies out when the basic reproduction number of diseases is less than unity and persists when the basic reproduction number of the disease

is greater than unity.

The main contribution of the study have been enlisted below:

(1) We compute basic reproduction number R_0, disease free and endemic equilibrium point.
(2) We have analyzed global stability of the equilibria.
(3) Numerical simulation has been performed to observe the dynamics of all six compartments.

From the model analysis, we observed that the disease could be controlled by maintaining the value of R_0 less than unity. It can be achieved only by proper treatment.

6. FUTURE SCOPE

In this section, we discuss the limitation of the current study, to full fill the constraints we need some support from various research departments. Some challenges of the research have enlisted below:

i. An appropriate data will surely full fill one of the constrains, but such data collection requires enormous investment.
ii. The current vector-borne model can also be extended to a delayed fractional-order vector-borne model to capture its dynamics.
iii. The discussed vector-borne disease model can also be studied for its stochastic version to capture its dynamics.

The effectiveness of this study therefore depends on how well the above-mentioned problems were responded to. We may conclude that this research needs people from different backgrounds to fulfill the intended function.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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