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Dynamical Modelling for Epidemics of Vector Transmitted Diseases With Non-Monotone Incidence

Sujatha Janardhan[1,*]

[1]*Department of Mathematics, St. Francis De Sales College, Nagpur, Maharashtra, India*

Abstract

This paper presents a mathematical formulation for vector–host disease dynamics in which the transmission process is governed by incidence functions with non-monotone characteristics. The study investigates how changes in the behavior of susceptible hosts influence infection levels, alongside examining the role of vector deterrents in modifying vector infection dynamics. The long-term behavior of the system is analyzed by establishing the global asymptotic stability of equilibrium states using suitably constructed Lyapunov functionals. To support the analytical conclusions, numerical simulations are carried out. The proposed framework provides a clearer understanding of the mechanisms driving the spread of vector-borne infections.

Keywords: vector host epidemics; stability; disease-free equilibrium; endemic equilibrium.
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1. Introduction

Vectors such as mosquitoes and fleas act as carriers for a wide range of pathogenic agents, including bacteria, viruses, and parasites, enabling transmission between humans or from animals to humans. Diseases transmitted through such carriers are collectively known as vector-borne diseases and account for a significant proportion of global mortality each year [11]. Well-known examples include malaria, dengue, yellow fever, chikungunya, and Zika virus infection [2,3,12]. These illnesses impose severe consequences not only on public health systems but also on ecological balance and economic stability worldwide [4]. Once vectors acquire infection, they generally remain infectious for the duration of their lifespan, allowing sustained transmission. Consequently, controlling vector populations remains a crucial strategy for limiting disease spread. Alongside vector management, changes in human behaviour during epidemic situations-such as increased awareness and adoption of preventive practices-play a critical role in reducing transmission. Effective community engagement and precautionary interventions can substantially mitigate the risk of outbreaks. The persistent

*Corresponding author (sujata.sfscollege@gmail.com)

emergence and re-emergence of vector-borne infections have therefore motivated the development of mathematical models to better understand their transmission dynamics. During periods of rapid disease escalation, individuals and authorities often implement measures such as isolation of susceptible individuals, quarantine of infected cases, social distancing, and community-wide lockdowns [1]. These behavioural interventions can significantly reduce secondary transmission, even when the number of infectious individuals remains high. Simultaneously, strategies aimed at suppressing vector populations, such as the application of insecticides, use of personal repellents, and household vector control products, further contribute to lowering infection rates [15]. Given the current global importance of vector-borne epidemics, this study proposes a vector–host epidemic model incorporating non-monotonic incidence functions for both human hosts and vectors. The human incidence term accounts for the preventive impact of behavioural adaptations among susceptible individuals, while the vector incidence term reflects the influence of vector control measures. This modelling framework aims to provide a more realistic representation of disease transmission under behavioural and environmental interventions.

2. Model Construction and Analysis

The system of ordinary differential equations presented in (1) represents a vector–host epidemic model in which the transmission process for both hosts and vectors is governed by non-monotonic incidence functions. In this formulation, the parameter a_1 quantifies the reduction in disease transmission resulting from behavioral adaptations and preventive practices adopted by the susceptible host population, while a_2 characterizes the effectiveness of vector control interventions.

$$\begin{aligned}
\frac{dS(t)}{dt} &= \alpha_1 - \beta_1 S(t) - \frac{k_1\lambda_1 S(t)V(t)}{1+a_1V^2(t)} \\
\frac{dI(t)}{dt} &= \frac{k_1\lambda_1 S(t)V(t)}{1+a_1V^2(t)} - \gamma I(t) - \beta_1 I(t) \\
\frac{dR(t)}{dt} &= \gamma I(t) - \beta_1 R(t) \\
\frac{dM(t)}{dt} &= \alpha_2 - \beta_2 M(t) - \frac{k_2\lambda_2 M(t)I(t)}{1+a_2I(t)} \\
\frac{dV(t)}{dt} &= \frac{k_2\lambda_2 M(t)I(t)}{1+a_2I(t)} - \beta_2 V(t)
\end{aligned} \tag{1}$$

where, a_1 is the parameter which measures preventive effect from the behavioral changes of susceptible host population and a_2 is the parameter which measures the effect of vector control. Here,

- $S(t)$ = number of susceptible individuals in hosts
- $I(t)$ = number of infected individuals in hosts
- $R(t)$ = number of recovered individuals in hosts

and so total populations of hosts $P_H(t) = S(t) + I(t) + R(t)$

- $M(t)$ = number of susceptible vectors
- $V(t)$ = number of infective vectors

and so total populations of vectors $P_V(t) = M(t) + V(t)$. The parameter α_1 represents the recruitment rate of host population, λ_1 is the transmission rate from vector to host, β_1 is natural death rate of host population, γ is per capita recovery rate of host, α_2 is the recruitment rate of vector population, β_2 is natural death rate of vector population, λ_2 is the transmission rate from host to vector, the parameter a_1 represents the intensity of protective measures and behavioral adaptations adopted by the susceptible host population, while a_2 quantifies the effectiveness of vector control interventions. System (1) readily yields

$$\frac{d}{dt}P_H(t) = \alpha_1 - \beta_1 P_H(t) \tag{2}$$
$$\frac{d}{dt}P_V(t) = \alpha_2 - \beta_2 P_V(t) \tag{3}$$

As time progresses, the total host and vector populations stabilize to the following asymptotic values

$$\lim_{t\to\infty} P_H(t) = \frac{\alpha_1}{\beta_1}, \tag{4}$$
$$\lim_{t\to\infty} P_V(t) = \frac{\alpha_2}{\beta_2} \tag{5}$$

We can assume without loss of generality that

$$P_H(t) = \frac{\alpha_1}{\beta_1}, \quad P_V(t) = \frac{\alpha_2}{\beta_2}, \quad t \geq 0 \tag{6}$$

Hence, the dynamical system (1) is **qualitatively equivalent** to the following system

$$\begin{aligned}
\frac{dS(t)}{dt} &= \alpha_1 - \beta_1 S(t) - \frac{k_1\lambda_1 S(t)V(t)}{1 + a_1 V^2(t)} \\
\frac{dI(t)}{dt} &= \frac{k_1\lambda_1 S(t)V(t)}{1 + a_1 V^2(t)} - \gamma I(t) - \beta_1 I(t) \\
\frac{dV(t)}{dt} &= \frac{k_2\lambda_2}{1 + a_2 I(t)}\left(\frac{\alpha_2}{\beta_2} - V(t)\right) I(t) - \beta_2 V(t)
\end{aligned} \tag{7}$$

The values of R and M can be determined from $R = \frac{\alpha_1}{\beta_1} - S - I$ and $M = \frac{\alpha_2}{\beta_2} - V$, respectively. From a biological standpoint, all state variables are required to remain non-negative. Accordingly, our analysis is restricted to the dynamical behavior of the system within the biologically feasible region. Hence the system (7) is studied in the closed set

$$\Gamma = \left\{(S, I, V) \in \mathbb{R}^3_+ \mid 0 \leq S + I \leq \frac{\alpha_1}{\beta_1},\ 0 \leq V \leq \frac{\alpha_2}{\beta_2},\ S \geq 0,\ I \geq 0\right\} \tag{8}$$

System (7) has disease-free equilibrium $E_0\left(\frac{\alpha_1}{\beta_1}, 0, 0\right)$. To find the endemic equilibrium, we set

$$\alpha_1 - \frac{k_1\lambda_1 S(t)V(t)}{1+a_1V^2(t)} - \beta_1 S(t) = 0 \tag{9}$$

$$\frac{k_1\lambda_1 S(t)V(t)}{1+a_1V^2(t)} - \gamma I(t) - \beta_1 I(t) = 0 \tag{10}$$

$$\frac{k_2\lambda_2}{1+a_2I(t)}\left(\frac{\alpha_2}{\beta_2} - V(t)\right) I(t) - \beta_2 V(t) = 0 \tag{11}$$

This implies

$$a_1\beta_1\beta_2^2\left(\gamma+\beta_1\right)V^2 + \left[k_1k_2\alpha_1\lambda_1\lambda_2 + k_1\lambda_1\beta_2\left(\gamma+\beta_1\right) + a_2k_1\lambda_1\alpha_1\beta_2\right]V + \beta_1\beta_2\left(\gamma+\beta_1\right)\left[1-R_0\right] = 0 \tag{12}$$

The basic reproduction number is

$$R_0 = \frac{k_1k_2\alpha_1\alpha_2\lambda_1\lambda_2}{\beta_1\beta_2^2\left(\gamma+\beta_1\right)} \tag{13}$$

The endemic equilibrium $E^*(S^*, I^*, V^*)$ is given by the following equations

$$S^* = \frac{\alpha_1(1+a_1V^{*2})}{k_1\lambda_1V^* + \beta_1[1+a_1V^{*2}]} \tag{14}$$

$$I^* = \frac{k_1\alpha_1\lambda_1V^*}{\left(\gamma+\beta_1\right)\left[k_1\lambda_1V^* + \beta_1[1+a_1V^{*2}]\right]} \tag{15}$$

$$V^* = \frac{-\Delta + \sqrt{\Delta^2 - 4a_1\beta_1^2\beta_2^2\left(\gamma+\beta_1\right)^2\left(1-R_0\right)}}{2a_1\beta_1\beta_2\left(\gamma+\beta_1\right)} \tag{16}$$

where $\Delta = k_1k_2\alpha_1\lambda_1\lambda_2 + k_1\lambda_1\beta_2\left(\gamma+\beta_1\right) + a_2k_1\lambda_1\alpha_1\beta_2$. The disease-free and endemic steady states of system (7) are examined in detail, and the criteria governing their stability are systematically derived in the following results.

Theorem 2.1. *The disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable for $R_0 > 1$.*

Proof. At the disease-free equilibrium E_0, we have

$$J(E_0) = \begin{bmatrix} -\beta_1 & 0 & -\frac{k_1\lambda_1\alpha_1}{\beta_1} \\ 0 & -(\gamma+\beta_1) & \frac{k_1\lambda_1\alpha_1}{\beta_1} \\ 0 & \frac{k_2\lambda_2\alpha_2}{\beta_2} & -\beta_2 \end{bmatrix} \tag{17}$$

and the characteristic equation is given by

$$\left(\beta_1+l\right)\left[l^2 + \left(\gamma+\beta_1+\beta_2\right)l + \left(\gamma+\beta_1\right)\beta_2\left(1-R_0\right)\right] = 0 \tag{18}$$

When the basic reproduction number satisfies $R_0 < 1$, application of the Routh-Hurwitz stability criterion [8] shows that all eigenvalues of the Jacobian matrix possess negative real parts, ensuring local asymptotic stability of the disease-free equilibrium E_0. In contrast, for $R_0 > 1$, the eigenvalue spectrum consists of two eigenvalues with negative real parts and one with a positive real part, rendering the disease-free equilibrium E_0 unstable. □

Theorem 2.2. *If $R_0 \leq 1$ then the disease-free equilibrium E_0 is globally asymptotically stable and is unstable for $R_0 > 1$.*

Proof. Consider $L_1 = \dfrac{k_1\lambda_1\alpha_1}{\beta_1\beta_2}V + I$

$$\begin{aligned}
\frac{dL_1}{dt} &= \frac{k_1\lambda_1\alpha_1}{\beta_1\beta_2}\left[\frac{k_2\lambda_2 I}{1+a_2 I}\left(\frac{\alpha_2}{\beta_2} - V\right) - \beta_2 V\right] + \frac{k_1\lambda_1 SV}{1+a_1V^2} - (\gamma+\beta_1)I \\
&= -(\gamma+\beta_1)\left[1 - \frac{k_1k_2\alpha_1\alpha_2\lambda_1\lambda_2}{\beta_1\beta_2^2(\gamma+\beta_1)(1+a_2I)}\right]I - \frac{k_1k_2\alpha_1\alpha_2\lambda_1\lambda_2}{\beta_1\beta_2(1+a_2I)}VI - \frac{k_1\alpha_1\lambda_1}{\beta_1}V + \frac{k_1\lambda_1SV}{1+a_1V^2} \\
&\leq -(\gamma+\beta_1)[1-R_0]I - \frac{k_1k_2\alpha_1\lambda_1\lambda_2}{\beta_1\beta_2(1+a_2I)}VI - \frac{k_1\alpha_1\lambda_1}{\beta_1}V + \frac{k_1\lambda_1SV}{1+a_1V^2} \\
&\leq -(\gamma+\beta_1)[1-R_0]I - \frac{k_1k_2\alpha_1\lambda_1\lambda_2}{\beta_1\beta_2(1+a_2I)}VI - \frac{k_1\alpha_1\lambda_1}{\beta_1}V + k_1\lambda_1SV \\
&\leq -(\gamma+\beta_1)[1-R_0]I - \frac{k_1k_2\alpha_1\lambda_1\lambda_2}{\beta_1\beta_2(1+a_2I)}VI, \quad \text{as } S \leq \frac{\alpha_1}{\beta_1}
\end{aligned}$$

When $R_0 \leq 1$, we get $\dfrac{dL_1}{dt} \leq 0$ for $t \geq 0$ and so L_1 is a Lyapunov function. The equality $\dfrac{dL_1}{dt} = 0$ holds at the disease-free equilibrium $E_0\left(\frac{\alpha_1}{\beta_1}, 0, 0\right)$. Thus, $\{E_0\}$ is the largest invariant set in the closed set Γ. Hence, E_0 is globally stable using LaSalle's invariance principle [8,10]. □

The Jacobian matrix at the endemic equilibrium becomes,

$$J(E^*) = \begin{bmatrix}
-\dfrac{k_1\lambda_1V^*}{1+a_1V^{*2}} - \beta_1 & 0 & -\dfrac{k_1\lambda_1S^*\left(1-a_1V^{*2}\right)}{\left(1+a_1V^{*2}\right)^2} \\
\dfrac{k_1\lambda_1V^*}{1+a_1V^{*2}} & -\gamma-\beta_1 & \dfrac{k_1\lambda_1S^*\left(1-a_1V^{*2}\right)}{\left(1+a_1V^{*2}\right)^2} \\
0 & \left(\dfrac{\alpha_2}{\beta_2} - V^*\right)\dfrac{k_2\lambda_2}{(1+a_2I^*)^2} & -\dfrac{k_2\lambda_2I^*}{1+a_2I^*} - \beta_2
\end{bmatrix} \tag{19}$$

Second additive compound matrix $J^{[2]}(E^*)$ is given by

$$J^{[2]}(E^*) = \begin{bmatrix} -\frac{k_1\lambda_1 V^*}{1+a_1V^{*2}} - 2\beta_1 - \gamma & \frac{k_1\lambda_1 S^*\left(1-a_1V^{*2}\right)}{\left(1+a_1V^{*2}\right)^2} & \frac{k_1\lambda_1 S^*\left(1-a_1V^{*2}\right)}{\left(1+a_1V^{*2}\right)^2} \\ \left(\frac{\alpha_2}{\beta_2} - V^*\right)\frac{k_2\lambda_2}{\left(1+a_2I^*\right)^2} & -\frac{k_1\lambda_1 V^*}{1+a_1V^{*2}} - \beta_1 - \frac{k_2\lambda_2 I^*}{1+a_2I^*} - \beta_2 & 0 \\ 0 & \frac{k_1\lambda_1 V^*}{1+a_1V^{*2}} & -\frac{k_2\lambda_2 I^*}{1+a_2I^*} - \gamma - \beta_1 - \beta_2 \end{bmatrix} \tag{20}$$

Theorem 2.3. *The endemic equilibrium E^* of the system (7) is locally asymptotically stable if $R_0 > 1$.*

Proof. From equation (19), we have

$$\operatorname{tr} J(E^*) = -\frac{k_1\lambda_1 V^*}{1+a_1V^{*2}} - \frac{k_2\lambda_2 I^*}{1+a_2I^*} - 2\beta_1 - \gamma - \beta_2 < 0 \tag{21}$$

and

$$\begin{aligned} \det J(E^*) &= \left(-\frac{k_1\lambda_1 V^*}{1+a_1V^{*2}} - \beta_1\right)\left[(-\gamma-\beta_1)\left(-\frac{k_2\lambda_2 I^*}{1+a_2I^*} - \beta_2\right)\right. \\ &\quad \left. -\frac{k_1\lambda_1 S^*\left(1-a_1V^{*2}\right)}{\left(1+a_1V^{*2}\right)^2}\left(\frac{\alpha_2}{\beta_2} - V^*\right)\frac{k_2\lambda_2}{\left(1+a_2I^*\right)^2}\right] \\ &\quad -\frac{k_1\lambda_1 S^*\left(1-a_1V^{*2}\right)}{\left(1+a_1V^{*2}\right)^2}\frac{k_1\lambda_1 V^*}{1+a_1V^{*2}}\left(\frac{\alpha_2}{\beta_2} - V^*\right)\frac{k_2\lambda_2}{\left(1+a_2I^*\right)^2} \\ &= -\left(\frac{k_1\lambda_1 V^*}{1+a_1V^{*2}} + \beta_1\right)(\gamma+\beta_1)\left(\frac{k_2\lambda_2 I^*}{1+a_2I^*} + \beta_2\right) \\ &\quad -\frac{k_1\lambda_1\beta_1 S^*\left(a_1V^{*2}-1\right)}{\left(1+a_1V^{*2}\right)^2}\left(\frac{\alpha_2}{\beta_2} - V^*\right)\frac{k_2\lambda_2}{\left(1+a_2I^*\right)^2} \\ \therefore \det J(E^*) &< 0, \quad \text{provided } V^{*2} > \frac{1}{a_1} \end{aligned}$$

From equation (20), we have

$$\begin{aligned} \det J^{[2]}(E^*) &= \left(-\frac{k_1\lambda_1 V^*}{1+a_1V^{*2}} - 2\beta_1 - \gamma\right)\left(-\frac{k_1\lambda_1 V^*}{1+a_1V^{*2}} - \beta_1 - \frac{k_2\lambda_2 I^*}{1+a_2I^*} - \beta_2\right) \\ &\quad \times\left(-\frac{k_2\lambda_2 I^*}{1+a_2I^*} - \gamma - \beta_1 - \beta_2\right) \\ &\quad -\frac{k_1\lambda_1 S^*\left(1-a_1V^{*2}\right)}{\left(1+a_1V^{*2}\right)^2}\left(\frac{\alpha_2}{\beta_2} - V^*\right)\frac{k_2\lambda_2}{\left(1+a_2I^*\right)^2}\left(-\frac{k_2\lambda_2 I^*}{1+a_2I^*} - \gamma - \beta_1 - \beta_2\right) \\ &\quad +\frac{k_1\lambda_1 S^*\left(1-a_1V^{*2}\right)}{\left(1+a_1V^{*2}\right)^2}\left(\frac{\alpha_2}{\beta_2} - V^*\right)\frac{k_2\lambda_2}{\left(1+a_2I^*\right)^2}\frac{k_1\lambda_1 V^*}{1+a_1V^{*2}} \\ &= -\left(\frac{k_1\lambda_1 V^*}{1+a_1V^{*2}} + 2\beta_1 + \gamma\right)\left(\frac{k_1\lambda_1 V^*}{1+a_1V^{*2}} + \frac{k_2\lambda_2 I^*}{1+a_2I^*} + \beta_2\right) \end{aligned}$$

$$\times \left(\frac{k_2\lambda_2 I^*}{1+a_2 I^*} + \gamma + \beta_1 + \beta_2 \right)$$
$$- \frac{k_1\lambda_1 S^* \left(a_1 V^{*2} - 1 \right)}{\left(1 + a_1 V^{*2}\right)^2} \left(\frac{\alpha_2}{\beta_2} - V^* \right) \frac{k_2\lambda_2}{\left(1+a_2 I^*\right)^2} \left(\frac{k_2\lambda_2 I^*}{1+a_2 I^*} + \gamma + \beta_1 + \beta_2 \right)$$
$$- \frac{k_1\lambda_1 S^* \left(a_1 V^{*2} - 1 \right)}{\left(1 + a_1 V^{*2}\right)^2} \left(\frac{\alpha_2}{\beta_2} - V^* \right) \frac{k_2\lambda_2}{\left(1+a_2 I^*\right)^2} \frac{k_1\lambda_1 V^*}{1+a_1 V^{*2}} < 0,$$
$$\text{as } \frac{\alpha_2}{\beta_2} - V^* > 0, \text{ provided } V^{*2} > \frac{1}{a_1}$$

We have $\operatorname{tr} J(E^*)$, $\det J(E^*)$, and $\det J^{[2]}(E^*)$ are all negative, and so $J(E^*)$ has all of the eigenvalues with negative real part [6]. Hence the theorem. □

Theorem 2.4. *If $R_0 > 1$, then system (7) is uniformly persistent, that is, there exists $\epsilon > 0$ (independent of initial conditions), such that $\liminf_{t\to\infty} S(t) > \epsilon$, $\liminf_{t\to\infty} I(t) > \epsilon$ and $\liminf_{t\to\infty} V(t) > \epsilon$.*

Proof. We begin by establishing the following assertions:

(i) The disease-free equilibrium E_0 is the unique omega-limit point on the boundary of the positively invariant set Γ.

(ii) For $R_0 > 1$, no trajectory originating in the interior of Γ admits E_0 as its omega-limit set.

To prove (i), we observe that the boundary of Γ is transverse to the system's vector field everywhere except along the S-axis. Moreover, the vector field associated with system (7) preserves invariance of the region Γ, ensuring that solutions remain within this biologically feasible domain. On the S-axis, we have

$$\frac{dS}{dt} = \alpha_1 - \beta_1 S \tag{22}$$

which gives $S \to \alpha_1/\beta_1$ as $t \to \infty$. Hence E_0 is only omega-limit point on the boundary of Γ for system (7).

(ii) We define the function L_2 in Γ such that

$$L_2(t) = \frac{\beta_1\beta_2\left(1+R_0\right)}{2k_1\lambda_1\alpha_1} I(t) + V(t) \tag{23}$$
$$\frac{dL_2(t)}{dt} = \frac{\beta_1\beta_2\left(1+R_0\right)}{2k_1\lambda_1\alpha_1} \left(\frac{k_1\lambda_1 S(t)V(t)}{1+a_1V^2(t)} - \left(\gamma+\beta_1\right) I(t) \right)$$
$$+ \frac{k_2\lambda_2}{1+a_2 I} \left(\frac{\alpha_2}{\beta_2} - V(t) \right) I(t) - \beta_2 V(t)$$
$$= \frac{\beta_1\beta_2\left(1+R_0\right) S(t)V(t)}{2\alpha_1(1+a_1V^2(t))} - \beta_2 V(t) + \frac{k_2\lambda_2}{1+a_2 I} \left(\frac{\alpha_2}{\beta_2} - V(t) \right) I(t) - \frac{\left(1+R_0\right)}{2R_0} \frac{k_2\lambda_2\alpha_2}{\beta_2} I(t)$$
$$\geq \frac{\beta_1\beta_2\left(1+R_0\right) S(t)V(t)}{2\alpha_1(1+a_1V^2(t))} - \beta_2 V(t) + k_2\lambda_2 \left(\frac{\alpha_2}{\beta_2} - V(t) - \frac{1}{2}\left(\frac{1}{R_0} + 1 \right) \frac{\alpha_2}{\beta_2} \right) I(t)$$
$$\geq \frac{\beta_1\beta_2\left(1+R_0\right)}{2\alpha_1} \left[S(t) - \frac{2\alpha_1}{\beta_1\left(1+R_0\right)} \right] V(t) + k_2\lambda_2 \left(\frac{\alpha_2}{\beta_2} - V(t) - \frac{1}{2}\left(\frac{1}{R_0} + 1 \right) \frac{\alpha_2}{\beta_2} \right) I(t)$$

Since $R_0 > 1$, then $\frac{1}{2}\left(\frac{1}{R_0}+1\right)$ and $\frac{2}{(1+R_0)}$ are less than one, therefore, there exists a neighborhood U of E_0 such that for $(S,I,V) \in U \cap \operatorname{Int}\Gamma$, the expression $\left[S(t) - \frac{2\alpha_1}{\beta_1(1+R_0)}\right]$ is positive. In this neighborhood $U(E_0)$, we have that $\frac{dL_2(t)}{dt} > 0$ in $U(E_0) - \{E_0\}$. Observe that the level surfaces of the Lyapunov function L_2 are defined by the plane $\frac{\beta_1\beta_2(1+R_0)}{2k_1\lambda_1\alpha_1}I(t) + V(t) = C$, which recede from the S-axis as the constant C increases. Since L_2 is strictly increasing along trajectories initiating in $U \cap \operatorname{Int}\Gamma$, it follows that every solution of system (7) evolves away from the disease-free equilibrium E_0. □

Theorem 2.5. *If $R_0 > 1$, then the endemic equilibrium E^* is globally asymptotically stable.*

Proof. For $R_0 > 1$, the system (7) is uniformly persistent and E^* is locally asymptotically stable. To establish the proposition, we demonstrate that system (7) satisfies the criterion of stability with respect to periodic solutions [9]. Let $P(t) = (S(t), I(t), V(t))$ be a periodic solution of system (7). To prove the stability of periodic orbits, it is sufficient to prove that the following linear non-autonomous system,

$$W'(t) = \left(J^{[2]}(P(t))\right) W(t) \tag{24}$$

is asymptotically stable. The second additive compound matrix is given by

$$J^{[2]}(S,I,V) = \begin{bmatrix} -\frac{k_1\lambda_1 V}{1+a_1V^2} - 2\beta_1 - \gamma & \frac{k_1\lambda_1 S\left(1-a_1V^2\right)}{\left(1+a_1V^2\right)^2} & \frac{k_1\lambda_1 S\left(1-a_1V^2\right)}{\left(1+a_1V^2\right)^2} \\ \left(\frac{\alpha_2}{\beta_2} - V\right)\frac{k_2\lambda_2}{(1+a_2I)^2} & -\frac{k_1\lambda_1 V}{1+a_1V^2} - \beta_1 - \frac{k_2\lambda_2 I}{1+a_2I} - \beta_2 & 0 \\ 0 & \frac{k_1\lambda_1 V}{1+a_1V^2} & -\frac{k_2\lambda_2 I}{1+a_2I} - \gamma - \beta_1 - \beta_2 \end{bmatrix} \tag{25}$$

For the values of $P(t)$, equation (24) becomes,

$$\begin{aligned} W_1'(t) &= -\left(\frac{k_1\lambda_1 V}{1+a_1V^2} + 2\beta_1 + \gamma\right) W_1(t) + \frac{k_1\lambda_1 S\left(1-a_1V^2\right)}{\left(1+a_1V^2\right)^2} W_2(t) + \frac{k_1\lambda_1 S\left(1-a_1V^2\right)}{\left(1+a_1V^2\right)^2} W_3(t) \\ W_2'(t) &= \left(\frac{\alpha_2}{\beta_2} - V\right)\frac{k_2\lambda_2}{(1+a_2I)^2} W_1(t) - \left(\frac{k_1\lambda_1 V}{1+a_1V^2} + \beta_1 + \frac{k_2\lambda_2 I}{1+a_2I} + \beta_2\right) W_2(t) \\ W_3'(t) &= \frac{k_1\lambda_1 V}{1+a_1V^2} W_2(t) - \left(\frac{k_2\lambda_2 I}{1+a_2I} + \gamma + \beta_1 + \beta_2\right) W_3(t) \end{aligned} \tag{26}$$

To prove that system is asymptotically stable, we consider the following function

$$L_3\left(W_1(t), W_2(t), W_3(t), S(t), I(t), V(t)\right) = \left\| W_1(t), \frac{I(t)}{V(t)} W_2(t), \frac{I(t)}{V(t)} W_3(t) \right\| \tag{27}$$

where $\|\cdot\|$ is the norm in $\mathbb{R}^3$ defined by

$$\|W_1(t), W_2(t), W_3(t)\| = \sup\{|W_1|, |W_2 + W_3|\} \tag{28}$$

We obtain that the orbit of $P(t)$ remains at a positive distance from the boundary of Γ. There exists

constant $c > 0$ such that

$$L_3\left(W_1(t), W_2(t), W_3(t), S(t), I(t), V(t)\right) \geq c \left\| W_1(t), W_2(t), W_3(t) \right\| \tag{29}$$

Let $(W_1(t), W_2(t), W_3(t))$ be a solution of the system and

$$L_3(t) = \sup\left\{ \left| W_1(t) \right|, \frac{I(t)}{V(t)} \left| W_2(t) + W_3(t) \right| \right\} \tag{30}$$

Thus, we obtain the following inequalities

$$\begin{aligned}
D_+ \left| W_1(t) \right| &\leq -\left(\frac{k_1\lambda_1 V}{1+a_1V^2} + 2\beta_1 + \gamma \right) \left| W_1(t) \right| + \frac{k_1\lambda_1 S\left(1-a_1V^2\right)}{\left(1+a_1V^2\right)^2} \left(\left| W_2(t) + W_3(t) \right| \right) \\
&\leq -\left(\frac{k_1\lambda_1 V}{1+a_1V^2} + 2\beta_1 + \gamma \right) \left| W_1(t) \right| + \frac{k_1\lambda_1 S\left(1-a_1V^2\right)}{\left(1+a_1V^2\right)^2} \frac{V}{I} \left(\frac{I}{V} \left| W_2(t) + W_3(t) \right| \right) \\
D_+ \left| W_2(t) \right| &\leq \left(\frac{\alpha_2}{\beta_2} - V \right) \frac{k_2\lambda_2}{\left(1+a_2I\right)^2} \left| W_1(t) \right| - \left(\frac{k_1\lambda_1 V}{1+a_1V^2} + \beta_1 + \frac{k_2\lambda_2 I}{1+a_2I} + \beta_2 \right) \left| W_2(t) \right| \\
D_+ \left| W_3(t) \right| &\leq \frac{k_1\lambda_1 V}{1+a_1V^2} \left| W_2(t) \right| - \left(\frac{k_2\lambda_2 I}{1+a_2I} + \gamma + \beta_1 + \beta_2 \right) \left| W_3(t) \right|
\end{aligned}$$

From system (26), we have

$$\begin{aligned}
D_+ \left(\left| W_2(t) + W_3(t) \right| \right) &\leq \left(\frac{\alpha_2}{\beta_2} - V \right) \frac{k_2\lambda_2}{\left(1+a_2I\right)^2} \left| W_1(t) \right| - \left(\beta_1 + \frac{k_2\lambda_2 I}{1+a_2I} + \beta_2 \right) \left| W_2(t) \right| \\
&\quad - \left(\frac{k_2\lambda_2 I}{1+a_2I} + \gamma + \beta_1 + \beta_2 \right) \left| W_3(t) \right| \\
&\leq \left(\frac{\alpha_2}{\beta_2} - V \right) \frac{k_2\lambda_2}{\left(1+a_2I\right)^2} \left| W_1(t) \right| - \left(\beta_1 + \frac{k_2\lambda_2 I}{1+a_2I} + \beta_2 \right) \left| W_2(t) + W_3(t) \right|
\end{aligned}$$

Thus, we obtain

$$\begin{aligned}
D_+ \left(\frac{I}{V} \left| W_2(t) + W_3(t) \right| \right) &= \left(\frac{I'}{I} - \frac{V'}{V} \right) \frac{I}{V} \left| W_2(t) + W_3(t) \right| + \frac{I}{V} D_+ \left| W_2(t) + W_3(t) \right| \\
&\leq \left(\frac{I'}{I} - \frac{V'}{V} \right) \frac{I}{V} \left| W_2(t) + W_3(t) \right| + \left(\frac{\alpha_2}{\beta_2} - V \right) \frac{k_2\lambda_2}{\left(1+a_2I\right)^2} \frac{I}{V} \left| W_1(t) \right| \\
&\quad - \frac{I}{V} \left(\beta_1 + \frac{k_2\lambda_2 I}{1+a_2I} + \beta_2 \right) \left| W_2(t) + W_3(t) \right| \\
&\leq \left(\frac{\alpha_2}{\beta_2} - V \right) \frac{k_2\lambda_2}{\left(1+a_2I\right)^2} \frac{I}{V} \left| W_1(t) \right| \\
&\quad + \left[\frac{I'}{I} - \frac{V'}{V} - \beta_1 - \frac{k_2\lambda_2 I}{1+a_2I} - \beta_2 \right] \frac{I}{V} \left| W_2(t) + W_3(t) \right|
\end{aligned}$$

From the first equation of system (26) and the above inequality, we get

$$D_+ L_3(t) \leq \sup\left\{ g_1(t), g_2(t) \right\} W(t) \tag{31}$$

where

$$g_1(t) = -\left(\frac{k_1\lambda_1 V}{1+a_1V^2} + 2\beta_1 + \gamma\right) + \frac{k_1\lambda_1 S\left(1-a_1V^2\right)}{\left(1+a_1V^2\right)^2}\frac{V}{I} \tag{32}$$

$$g_2(t) = \frac{k_2\lambda_2}{\left(1+a_2I\right)^2}\left(\frac{\alpha_2}{\beta_2} - V\right)\frac{I}{V} + \frac{I'}{I} - \frac{V'}{V} - \beta_1 - \frac{k_2\lambda_2 I}{1+a_2I} - \beta_2 \tag{33}$$

Rewriting the second and third equation of system,

$$\frac{I'}{I} = \frac{k_1\lambda_1 SV}{\left(1+a_1V^2\right)I} - (\gamma+\beta_1) \tag{34}$$

$$\frac{V'}{V} = \frac{k_2\lambda_2}{1+a_2I}\left(\frac{\alpha_2}{\beta_2} - V\right)\frac{I}{V} - \beta_2 \tag{35}$$

We have

$$\begin{aligned} g_1(t) &\leq -\left(\frac{k_1\lambda_1 V}{1+a_1V^2} + 2\beta_1 + \gamma\right) + \frac{k_1\lambda_1 S}{\left(1+a_1V^2\right)^2}\frac{V}{I} \\ &= -\frac{k_1\lambda_1 V}{1+a_1V^2} - \beta_1 - \beta_1 - \gamma + \frac{k_1\lambda_1 S}{\left(1+a_1V^2\right)^2}\frac{V}{I} \\ &= -\frac{k_1\lambda_1 V}{1+a_1V^2} - \beta_1 + \frac{I'}{I}, \\ g_1(t) &\leq -\beta_1 + \frac{I'}{I} \end{aligned} \tag{36}$$

$$\begin{aligned} g_2(t) &= \frac{V'}{V} + \frac{I'}{I} - \frac{V'}{V} - \beta_1 - \frac{k_2\lambda_2 I}{1+a_2I} \\ g_2(t) &= \frac{I'}{I} - \beta_1 - \frac{k_2\lambda_2 I}{1+a_2I} \\ g_2(t) &\leq -\beta_1 + \frac{I'}{I} \end{aligned} \tag{37}$$

Hence

$$\sup\left\{g_1(t), g_2(t)\right\} \leq -\beta_1 + \frac{I'}{I} \tag{38}$$

From (31) and Gronwall's inequality, we obtain

$$L_3(t) \leq L_3(0)I(t)e^{-\beta_1 t} < L_3(0)\frac{\alpha_1}{\beta_1}e^{-\beta_1 t} \tag{39}$$

which implies that $L_3(t) \to 0$ as $t \to \infty$. By (29), we obtain $(W_1(t), W_2(t), W_3(t)) \to 0$ as $t \to \infty$. These observations establish that the linearized system given in (26) is asymptotically stable, which in turn implies asymptotic orbital stability of the associated periodic solution. Consequently, the endemic equilibrium E^* is globally asymptotically stable. □

3. Numerical Results

The theoretical conclusions of the model are corroborated through numerical experiments carried out using MATLAB. For values of the basic reproduction number satisfying $R_0 < 1$, the disease-free equilibrium remains stable, leading to extinction of the infection, as depicted in Fig. 3.1. Conversely, Fig. 3.2 illustrates sustained disease prevalence when $R_0 > 1$. Furthermore, Fig. 3.3 shows that an increase in preventive actions and behavioral adaptations among hosts results in a reduction of infected individuals. Similarly, Fig. 3.4 demonstrates that effective implementation of vector control measures leads to a decrease in the infected vector population.

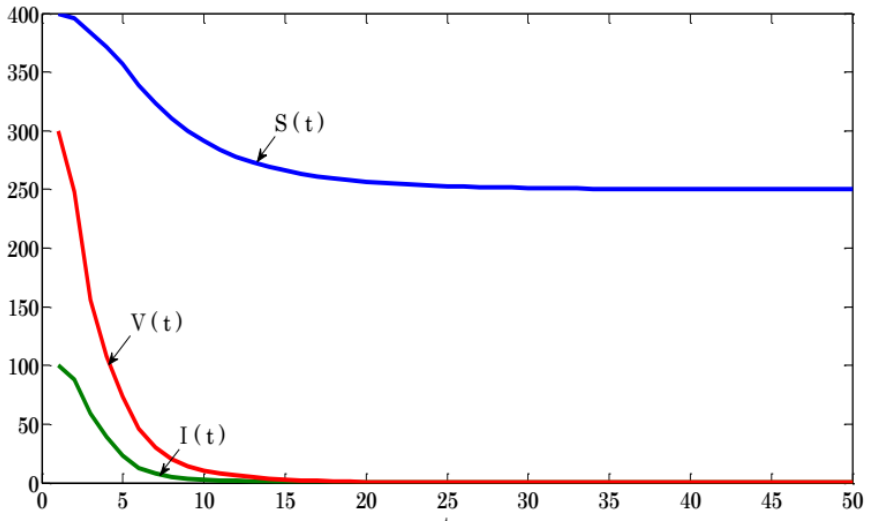

Figure 1: Here $S(0) = 400$, $I(0) = 100$, $V(0) = 300$, $k_1 = 0.02$, $k_2 = 0.3$, $\alpha_1 = 10$, $\alpha_2 = 110$, $\lambda_1 = 0.006$, $\lambda_2 = 0.009$, $\beta_1 = 0.04$, $\beta_2 = 0.31$, $\gamma = 0.15$, $a_1 = 0.001$, $a_2 = 0.012$, $R_0 = 0.488$.

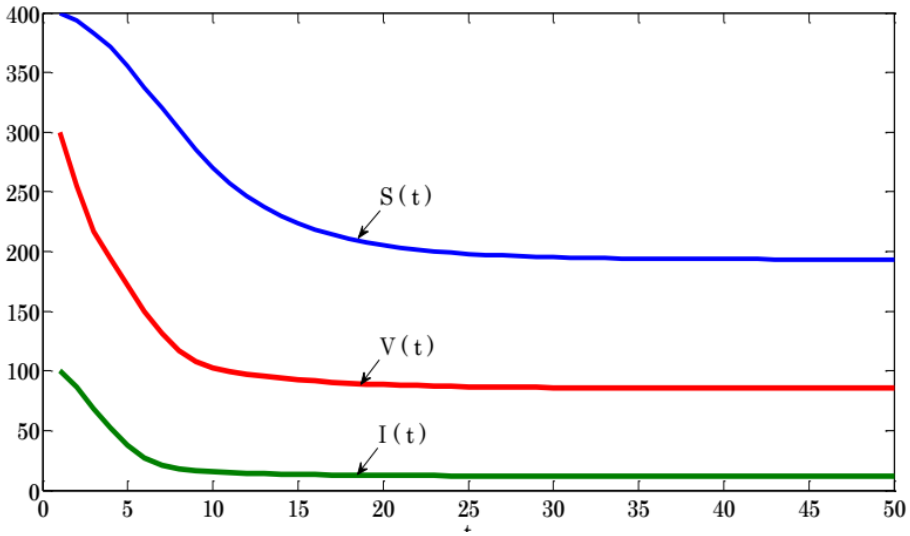

Figure 2: Here $S(0) = 400$, $I(0) = 100$, $V(0) = 300$, $k_1 = 0.19$, $k_2 = 1.05$, $\alpha_1 = 10$, $\alpha_2 = 110$, $\lambda_1 = 0.006$, $\lambda_2 = 0.009$, $\beta_1 = 0.04$, $\beta_2 = 0.31$, $\gamma = 0.15$, $a_1 = 0.001$, $a_2 = .012$, $R_0 = 16.225$.

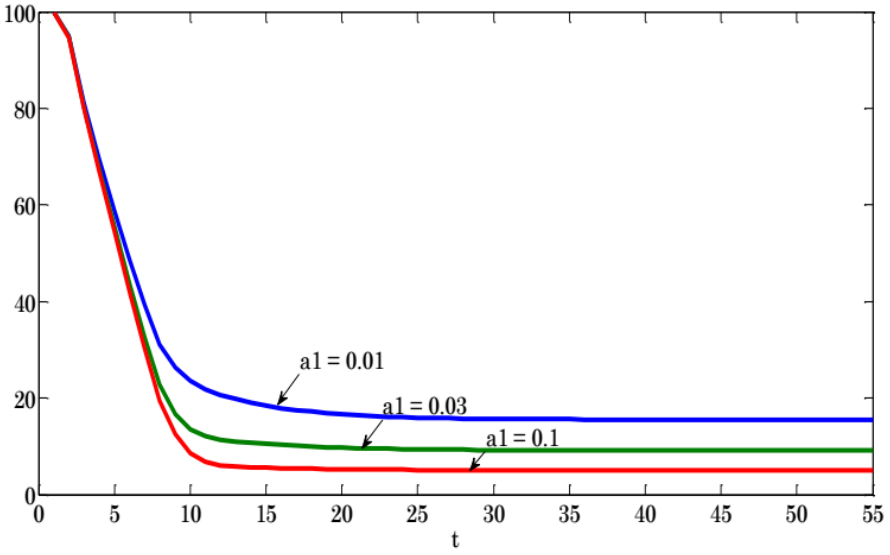

Figure 3: Dependence of I^* on a_1.

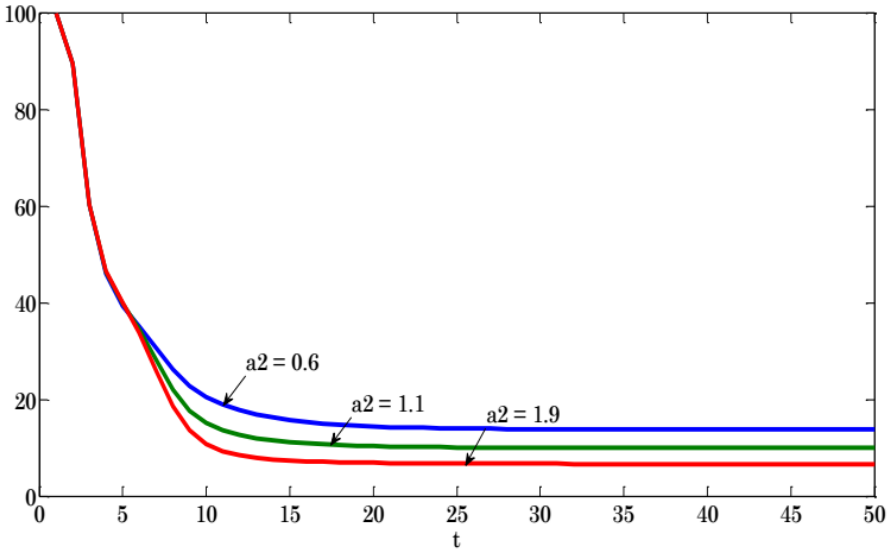

Figure 4: Dependence of I^* on a_2.

4. Conclusion

In this study, a vector-host epidemic framework incorporating non-monotonic transmission functions for both human hosts and vectors have been developed. The theoretical results are supported through numerical experiments conducted using MATLAB and Simulink. The analysis demonstrates that when the basic reproduction number satisfies $R_0 \leq 1$, the disease-free equilibrium is globally asymptotically stable, leading to eventual elimination of the infection from the population. Conversely, for $R_0 > 1$, the disease persists and the endemic equilibrium becomes globally asymptotically stable. Although the expression for the basic reproduction number given in (13) does not explicitly involve the parameters a_1 and a_2, numerical investigations reveal that the equilibrium level of infected hosts I^* declines as these parameters increase. Furthermore, equation (13) indicates that the implementation of stronger preventive strategies-such as quarantine and isolation in regions

affected by vector-borne diseases-can significantly reduce infection prevalence, transmission intensity, and the environmental carrying capacity for vectors. From the endemic equilibrium expression in (16), it is observed that the infected vector population V^* approaches zero as the vector control parameter a_2 increases without bound. This highlights the importance of maintaining hygienic conditions and adopting effective vector management practices, including indoor and outdoor insecticide spraying and the use of repellents, to minimize disease burden. Similarly, the steady-state solution for infected hosts in (15) shows that I^* tends toward zero as both preventive and vector control measures become increasingly effective. The proposed model also captures the influence of social awareness among susceptible individuals. Increased public knowledge of protective measures-such as vaccination programs, use of bed nets, and personal hygiene-plays a significant role in suppressing disease transmission within the host population. In parallel, awareness regarding environmental cleanliness and vector control contributes to reducing vector density, ultimately interrupting disease spread. Additionally, improvements in sanitation, access to clean drinking water, and availability of quality healthcare services further enhance disease control. Numerical simulations suggest that early detection, timely treatment, and reduction of vector breeding sites can substantially lower transmission levels. Notably, the computed values of the basic reproduction number in the present model are considerably smaller than those reported in earlier studies [2,3,6,7,10,13], where behavioral and psychosocial factors were not explicitly incorporated. Overall, the model offers a valuable tool for policymakers to design effective intervention strategies, anticipate outbreak dynamics, and support future research and educational initiatives in mathematical epidemiology.

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References

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

[2] F. Brauer, C. Castillo-Chavez, A. Mubayi and S. Towers, *Some models for epidemics of vector-transmitted diseases*, Infectious Disease Modelling, 1(1)(2016), 79-87.

[3] H.-M. Wei, X.-Z. Li and M. Martcheva, *An epidemic model of a vector-borne disease with direct transmission and time delay*, J. Math. Anal. Appl., 342(2008), 895-908.

[4] J. F. Lindahl and D. Grace, *The consequences of human actions on risks for infectious diseases: a review*, Infection Ecology & Epidemiology, 5(2015), 30048.

[5] K. O. Okosun and O. D. Makinde, *Optimal control analysis of malaria in the presence of non-linear incidence rate*, Appl. Comput. Math., 12(2013), 20-32.

[6] L. Cai, S. Guo, X. Li and M. Ghosh, *Global dynamics of a dengue epidemic mathematical model*, Chaos, Solitons & Fractals, 42(2009), 2297-2304.

[7] L. Cai and X. Li, *Global analysis of a vector-host epidemic model with nonlinear incidences*, Appl. Math. Comput., 217(2010), 3531-3541.

[8] L. Perko, *Differential Equations and Dynamical Systems*, 7th ed., Springer-Verlag, New York, (1996).

[9] M. Y. Li and J. S. Muldowney, *Global stability for the SEIR model in epidemiology*, Math. Biosci., 125(1995), 155-164.

[10] M. Ozair, A. A. Lashari, I. H. Jung and K. O. Okosun, *Stability analysis and optimal control of a vector-borne disease with nonlinear incidence*, Discrete Dyn. Nat. Soc., 2012(2012), 1-21.

[11] N. G. Gratz, *Emerging and resurging vector-borne diseases*, Annu. Rev. Entomol., 44(1999), 51-75.

[12] R. Ross, *The Prevention of Malaria*, Murray, London, (1911).

[13] S. Guo, X. Li and M. Ghosh, *Analysis of a dengue disease model with nonlinear incidence*, Discrete Dyn. Nat. Soc., 2013(2013), 1-10.

[14] Z. Qiu, *Dynamical behavior of a vector–host epidemic model with demographic structure*, Comput. Math. Appl., 56(12)(2008), 3118-3129.

[15] Z. Ma and J. Li, *Dynamical Modelling and Analysis of Epidemics*, World Scientific, Singapore, (2009).