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SENSITIVITY AND OPTIMIZING DENGUE CONTROL IN A TWO-TIME-DELAY TRANSMISSION MODEL

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Abstract. This study presents a detailed analysis of a dengue transmission model that incorporates two biologically motivated time delays, representing the intrinsic and extrinsic incubation periods in host-vector interactions. Based on a vector-host framework, the model examines the influence of key transmission parameters through sensitivity analysis and contour plots. The basic reproduction number R_0 is analytically derived and assessed for its dependence on epidemiologically significant factors. To curb disease spread, an optimal control problem is formulated by introducing three time-dependent control strategies: personal protection, vector control, and vector targeting efficiency. Employing Pontryagin's Maximum Principle, the necessary conditions for optimality are established. Numerical simulations under various delay scenarios reveal that optimal control strategies significantly reduce both human and vector infections. However, the presence and magnitude of delays affect the speed and efficacy of intervention. The findings emphasize the critical role of accounting for biological delays and implementing targeted controls in designing effective dengue prevention strategies.

Keywords: Vector-Host Model, Reproduction Number, Sensitivity Analysis, Delay, Optimal Control.

AMS Subject Classification: 34D23, 37B25, 34D20.

1. Introduction

Worldwide, dengue fever affects a large portion of the tropical and subtropical populations. This illness is spread by mosquitoes, namely Aedes aegypti and Aedes albopictus. The dengue fever virus has four distinct serotypes. Once a person recovers from an infection caused by one serotype, they will be completely protected against future infections of that serotype but will only have temporary protection against the other three.

Dengue fever may range from moderate to severe. Dengue hemorrhagic fever (DHF) and shock syndrome are the two categories of dengue that pose the greatest threat to human

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health. Hospitalization is often necessary for patients who have these more severe dengue fever complications. The mosquito is a key part of the dengue fever virus's life cycle, both as a transmitter (or vector) and as the primary host for the disease in humans. The only way to reduce or eliminate the transmission of dengue fever is to eradicate vectors or stop people from coming into touch with them [1].

With the use of mathematical modelling, researchers have been able to uncover and investigate the dynamics of disease transmission [2, 3]. Additionally, mathematical modelling has been utilized to evaluate the effectiveness of health treatments such as the treatment of Wolbachia bacteria [4, 5] and immunization [6, 7]. It is common practice to design the mathematical model of disease transmission as either a deterministic [8] or stochastic [9] mathematical model. The stability of a dengue transmission model that had two delays and looked for evidence of Hopf bifurcation, offering insights into disease spread [10]. Hasan et al. [11], analyzed SEIR-SEI dengue epidemiological models under vector–host frameworks, highlighting the role of compartmental extensions in better capturing dengue dynamics.

The goal of performing a sensitivity analysis in an epidemic model is to ascertain the influence of modifying the model's parameters on the projected outcomes. Policymakers and academics benefit from this study because it clarifies the model's robustness, reveals key elements, and names the parameters that matter most for the model's output. Adjusting the model's predictions is as simple as methodically changing these parameters within a certain range. When testing a model, researchers usually choose a range of reasonable values for each parameter and see how the results change depending on those values [12].

A sensitivity analysis takes into account several variables and how they affect the inherent uncertainty in a system's output [13, 14]. There are a number of scientific fields that make use of sensitivity and uncertainty analysis. Some examples of these tasks include finding important information while removing noise, improving the architecture of the system, and ranking the importance of various aspects affecting the system's behavior [15, 16]. The sensitivity analysis method might be used on a regional or international scale. One goal of local analysis is to look at how different paths or tiny changes to parameters affect the overall model [17]. Statistical and deterministic techniques are the two most used ways to do sensitivity analysis [14, 18]. Nevertheless, the aims and system under investigation dictate the methodology selection process. When dealing with complex systems or models, the computational price of sensitivity analysis might restrict its usefulness. Hasan et al. [20] investigated a dynamic vector–host dengue epidemic model incorporating vector control and sensitivity analysis, highlighting the role of intervention measures in reducing disease spread.

Optimal control theory is a rigorous mathematical methodology used to make decisions within the framework of intricate dynamical systems [33]. Optimal control approaches have been used to epidemiological models in several investigations, including those conducted by Blayneh et al. [34], Okosun and Makinde [35], Lashari and Zaman [36, 37], and others. Lashari and Zaman [37] used individual safety, blood testing, and vector-reduction strategies as very successful methods for managing the transmission of a vector-borne illness. A nonlinear epidemic model was examined by Kar and Batabyal [38], who used an optimum control tactic to mitigate the illness burden by the application of a vaccination program. Ozair et al. [39] performed a qualitative investigation and conducted a comprehensive analysis of the resulting model to investigate the factors and their influence on the propagation of vector-borne illnesses. Zizhen et al. [40] investigated the most effective method of controlling an epidemic model with a temporal delay using Pontryagin's maximal principle. This study contributed to the creation of a dengue control

method. The best control technique for dengue epidemics was presented by Chien-Hung et al. [41]. This strategy utilizes a compartment model with time delay, which facilitates timely intervention and preventative actions.

In light of the aforementioned conversation, this research will provide a unique delayed model that incorporates artificial eradication of vectors. These mathematical frameworks will allow us to examine the effects of time delay and deliberate elimination of vectors on the transmission of illnesses. Concurrently, a problem of optimum control will be defined and resolved. Hence, our research diverges from the previous study [42] in terms of the deliberate elimination of vectors that are implicated in the examination of stability and optimum problem.

Murugadoss et al. [10], which is our earlier work, focused only on the stability analysis and Hopf bifurcation of a two-delay dengue model. The present study extends that framework by incorporating sensitivity analysis, contour plots, and a rigorous optimal control problem with multiple intervention strategies, thereby providing both theoretical and practical insights for disease control.

Prior research mostly concentrated on the stability and Hopf bifurcation within a two-delay dengue model, offering theoretical perspectives on the significance of delays but neglecting practical solutions. Their methodological contribution focused on stability and bifurcation analysis, excluding control optimization. This study enhances previous research by doing a comprehensive sensitivity analysis of the basic reproduction number, R_0, accompanied with contour visualizations to emphasize significant epidemiological factors. This approach incorporates sensitivity analysis, contour plots, and optimum control using Pontryagin's Maximum Principle to determine essential parameters and evaluate intervention efficacy. The practical implications of this research include providing actionable solutions such as personal protection, vector management, and exposure reduction, while also illustrating their efficacy across different delay circumstances.

2. Model Formulation

The dengue transmission dynamic model with two-time delay was presented in Murugadoss et al. [10]. Specifically, the model divides dengue transmission from vectors to hosts into three categories: susceptible hosts (S), infected hosts (I), and infected vectors (V). The governing delay differential equations of the model as follows,

$$\begin{cases} \frac{dS}{dt} = & \Omega_H - \alpha\beta_h S(t-\tau_1)V(t-\tau_2)e^{-d_h\tau_1} - d_h S + \theta I + \rho(\frac{\Omega_H}{d_h} - S - I) \\ \frac{dI}{dt} = & \alpha\beta_h S(t-\tau_1)V(t-\tau_2)e^{-d_h\tau_1} + \sigma\alpha\beta_h(\frac{\Omega_H}{d_h} - S - I)V - (\delta + d_h + \mu_h + \theta)I \\ \frac{dV}{dt} = & \alpha\beta_v(\frac{\Omega_V}{d_v} - V(t-\tau_2))I(t-\tau_2)e^{-d_v\tau_2} - (d_v + \mu_v)V \end{cases} \quad (1)$$

with the following initial conditions:

$$S(\psi) = S_0(\psi), I(\psi) = I_0(\psi), V(\psi) = V_0(\psi); \psi \in [-\tau, 0], \tau = max\{\tau_1, \tau_2\}. \quad (2)$$

At time t, the variables $S(t)$, $I(t)$ and $V(t)$ represent the number of susceptible hosts, infected host, and infected vector, and the governing equations are derived from a SIV model with two-time delays τ_1 and τ_2. The parameters Ω_H and Ω_V denote the rates at which the host and vector populations are recruited, respectively. The rate of illness transmission from hosts to vectors is described by the parameter β_h, whereas the rate of illness transmission from vectors to hosts is described by the value β_v. The intrinsic mortality rates of the host population and the vector population are denoted by d_h and d_v, respectively. The variables μ_h and μ_v represent the mortality rates caused by illness in the host and vector populations, respectively. The variables α, θ, δ, σ and ρ represent the daily

biting rate of the vector, group of susceptible individuals' recovery rate, host population's immune loss recovery rate, immunity loss rate overall, and partial immunization for hosts who recuperated from the initial illness, respectively. The extrinsic incubation period τ_1 and the intrinsic incubation period τ_2 are the time-based delays in host and vector populations, respectively, from the susceptible to infectious class.

The delay differential equations for the dengue transmission dynamic model with time delay described by system (1) has two equilibrium points: a contagion-free equilibrium C_0 given by $C_0 = \{\frac{\Omega_H}{d_h+\rho}, 0, 0\}$ and an endemic equilibrium $C_* = S_*, I_*, V_*$ where

$$S_* = \frac{((\frac{\Omega_H}{d_h})\rho+\Omega_H+(\theta-\rho)I_*)(\alpha\beta_v I_* e^{-d_v\tau_2}+(d_v+\mu_v))}{(d_h+\rho)(\alpha\beta_v I_* e^{-d_v\tau_2}+(d_v+\mu_v))+(\frac{\Omega_V}{d_v})\alpha^2\beta_h\beta_v I_* e^{-d_h\tau_1}e^{-d_v\tau_2}}$$

$$V_* = \frac{(\frac{\Omega_V}{d_v})\alpha\beta_v I_* e^{-d_v\tau_2}}{\alpha\beta_v I_* e^{-d_v\tau_2}+(d_v+\mu_v)}$$

I_* indicates the non-negative root of the following quadratic equation:

$$X_1 I_*^2 + X_2 I_* + X_3 = 0 \tag{3}$$

$I_1 = \frac{-X_2+\sqrt{X_2^2-4X_1X_3}}{2X_1}$ and $I_2 = \frac{-X_2-\sqrt{X_2^2-4X_1X_3}}{2X_1}$ be the roots of (3).

The basic reproduction number, determined through the next generation matrix, is

$$R_0 = \frac{\alpha^2\beta_h\beta_v\Omega_H\Omega_V e^{-d_h\tau_1}e^{-d_v\tau_2}}{(d_h+\rho)d_v(d_v+\mu_v)(\delta+d_h+\mu_h+\theta)} \tag{4}$$

3. Sensitivity Analysis

Through performing a sensitivity analysis, one may have a clear grasp of the virtual significance of each parameter in relation to its impact on disease transmission. The offered information is essential not just for experiment design but also for data integration and the simplification of complicated nonlinear frameworks [19, 21]. A sensitivity analysis is a common method for checking how well model predictions hold up when the parameters are changed. The importance of this problem is raised by the fact that mistakes are often made when collecting data and guessing parameter values. Using this statistic, we may identify the factors that significantly affect R_0 and determine which ones should be addressed via interventions. Regarding disease transmission, sensitivity analysis may provide insight on the most crucial factors.

A sensitivity index may be used to measure the proportional change in a variable resulting from an adjustment in a parameter. The normalized forward sensitivity index of a variable with regard to a parameter may be obtained by calculating the ratio of the parameter's change to the variable's change. The values of the model parameters are provided in Table 1.

The computation of the normalized sensitivity index entails using the differentiable variable R_0 and its related parameter p. It is formally defined as:

$$\Lambda_p^{R_0} = \frac{\partial R_0}{\partial p} X \frac{p}{R_0} \tag{5}$$

Using the value of R_0, the sensitivity index of every parameter is determined using System (5). Sensitivity index of model parameters associated to R_0 with time delay $\tau_1 = 0.5$ and $\tau_2 = 1$ is shown in Table 2 and the parameter values are presented in Table 1.

$$\Lambda_\alpha^{R_0} = \frac{\partial R_0}{\partial \alpha} X \frac{\alpha}{R_0} = 2 \qquad \Lambda_{d_h}^{R_0} = \frac{\partial R_0}{\partial d_h} X \frac{d_h}{R_0} = -0.373669$$

TABLE 1. Model Parameters and their Values

Parameters	**Value**	**Source**
Ω_H	Varies	-
Ω_V	Varies	-
β_h	0.002	[26]
β_v	0.375	[23]
d_h	0.01	[26]
d_v	0.06	[24]
μ_h	0.05	[25]
μ_v	0.1	[24]
α	0.3	[10]
θ	0.08	[25]
δ	0.143	[22]
σ	0.8	[26]
ρ	0.02	Assumed

TABLE 2. Model parameter index for R_0

Parameter	**Sensitivity index**
α	2
β_h	1
β_v	1
Ω_H	1
Ω_V	1
d_h	-0.373669
d_v	-1.435
ρ	-0.6667
μ_h	-0.176678
μ_v	-0.625
δ	-0.5053
θ	-0.282686

$$\Lambda_{\beta_h}^{R_0} = \frac{\partial R_0}{\partial \beta_h} X \frac{\beta_h}{R_0} = 1 \qquad \Lambda_{d_v}^{R_0} = \frac{\partial R_0}{\partial d_v} X \frac{d_v}{R_0} = -1.435$$

$$\Lambda_{\beta_v}^{R_0} = \frac{\partial R_0}{\partial \beta_v} X \frac{\beta_v}{R_0} = 1 \qquad \Lambda_{\rho}^{R_0} = \frac{\partial R_0}{\partial \rho} X \frac{\rho}{R_0} = -0.6667$$

$$\Lambda_{\Omega_H}^{R_0} = \frac{\partial R_0}{\partial \Omega_H} X \frac{\Omega_H}{R_0} = 1 \qquad \Lambda_{\mu_h}^{R_0} = \frac{\partial R_0}{\partial \mu_h} X \frac{\mu_h}{R_0} = -0.176678$$

$$\Lambda_{\Omega_V}^{R_0} = \frac{\partial R_0}{\partial \Omega_V} X \frac{\Omega_V}{R_0} = 1 \qquad \Lambda_{\mu_v}^{R_0} = \frac{\partial R_0}{\partial \mu_v} X \frac{\mu_v}{R_0} = -0.625$$

$$\Lambda_{\delta}^{R_0} = \frac{\partial R_0}{\partial \delta} X \frac{\delta}{R_0} = -0.5053 \qquad \Lambda_{\theta}^{R_0} = \frac{\partial R_0}{\partial \theta} X \frac{\theta}{R_0} = -0.282686$$

According to the sensitivity indices, the parameter with the greatest sensitivity and lowest sensitivity is displayed in Figure 1.

The parameters α, β_h, β_v, Ω_H, Ω_V have a sensitivity index that is positive. On the other hand, the parameters d_h, d_v, ρ, μ_h, μ_v, δ, θ have a sensitivity index that is negative. The fact that the sensitivity indices are positive indicates that the population's reproductive capacity has increased significantly. Therefore, if the value of the parameter is raised (or lowered) while the value of the other parameters stays the same, this will result in an increase (or reduction) in the basic reproduction numbers. This is because the parameter's value is directly proportional to the other parameters. The occurrence of negative sensitivity indices makes it abundantly evident that the increase in the basic reproduction numbers carries with it a negative implication. To put it another way, if the

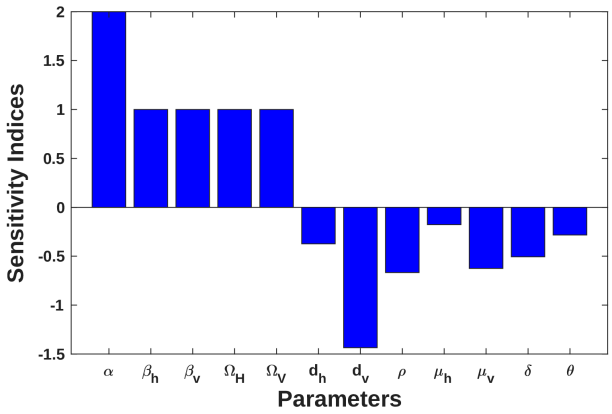


FIGURE 1. Forward normalized sensitivity indices of R_0 with time delay

value of the parameter is raised (or lowered) but the values of the other parameters remain the same, the basic reproduction numbers will fall (or rise) as a result of the change. This is due to the fact that the general parameters will stay unchanged.

Table 2 provides a descriptive account of sensitivity indices, these findings translate into practical interventions by highlighting that reducing parameters with positive sensitivity (such as biting rate and transmission coefficients) through personal protection and vector reduction, while enhancing those with negative sensitivity (such as host recovery and vector mortality) via improved treatment and vector control, can effectively suppress dengue transmission.

Eventually, the dengue sensitivity analysis identifies the key factors that control the way the illness is transmitted. The outcomes recommend that a control strategy that decreases the parameters α, β_h, β_v, Ω_H and Ω_V and control strategy that increases the disease transmission rate caused by vector (β_h) and biting rate of vector (α), will efficiently mitigate the transmission of the dengue virus among hosts.

4. Contour Plot Analysis

Contour plots are graphical representations that display the variation of a response variable with respect to two independent variables. In the context of an epidemic model, contour plots can be used to analyze how the dynamics of the epidemic vary with different parameter values.

The contour plots in Figure 2 show that the relationship between the biting rate (α) and the transmission probability from vector to host (β_h) is nonlinear, meaning that simultaneous increases amplify R_0 more strongly than individual changes. When resources are limited, prioritizing reduction in α (biting rate) is more impactful since it influences transmission multiplicatively with β_h. Hence, vector control measures that directly lower biting frequency should be emphasized over reducing β_h alone.

The control strategy for reducing disease transmission in dengue virus involves targeting both the vector and its biting rate. A reduction in vector abundance may be achieved by the use of vector control methods, such as the use of bed nets treated with pesticide and environmental management efforts to eliminate breeding habitats.

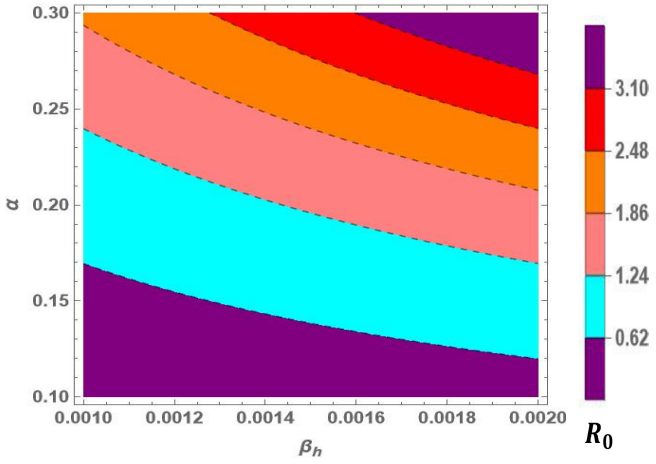


FIGURE 2. Contour plots of the value of R_0 with respect to α and β_h

5. Optimal Control Problem

5.1. **Formulation of the optimal control model.** In the system of delay differential equations (6), the functions $c_1(t)$, $c_2(t)$ and $c_3(t)$ represent time-dependent control strategies aimed at minimizing disease burden and intervention costs. Each control corresponds to a specific aspect of disease management in the human-vector transmission dynamics:

- $c_1(t)$: Represents the protection or personal preventive measures (e.g., use of mosquito nets, repellents, or behavior modification campaigns). It affects the transmission rate from infected vectors to susceptible humans by modifying the effective contact rate. The term $c_1(1-c_1)$ models the nonlinear impact of the control, capturing the diminishing returns or saturation effect when the control effort is very high or very low.
- $c_2(t)$: Denotes the vector control strategy (e.g., insecticide spraying or larvicidal treatment) that reduces the recruitment of infected vectors. It appears in the vector equation as a reduction factor on the transmission from infected humans to susceptible vectors.
- $c_3(t)$: Corresponds to the vector exposure reduction or targeting efficiency, reflecting efforts that affect the susceptibility of vectors to acquiring infection from humans (e.g., environmental management or spatial repellents). This control modifies the force of infection from humans to vectors.

To reduce or eliminate the illness from the human population, these management strategies are crucial. The vector-host model is then extended to include the aforementioned controls, yielding the following control issue:

$$\begin{cases} \frac{dS}{dt} = & \Omega_H - \alpha\beta_h c_1(1-c_1)S(t-\tau_1)V(t-\tau_2)e^{-d_h\tau_1} - d_hS + \theta I + \rho(\frac{\Omega_H}{d_h} - S - I) \\ \frac{dI}{dt} = & \alpha\beta_h c_1(1-c_1)S(t-\tau_1)V(t-\tau_2)e^{-d_h\tau_1} + \sigma\alpha\beta_h c_1(\frac{\Omega_H}{d_h} - S - I)V \\ & -(\delta + d_h + \mu_h + \theta)I \\ \frac{dV}{dt} = & (1-c_2)\alpha\beta_v c_3(\frac{\Omega_V}{d_v} - V(t-\tau_2))I(t-\tau_2)e^{-d_v\tau_2} - (d_v + \mu_v)V \end{cases} \tag{6}$$

With the model's stated non-negative initial conditions (2). To reduce the infection, the model's corresponding goal function is given below:

$$\mathcal{K}(c_1(t), c_2(t), c_3(t)) = \int_0^T [U_1 S + U_2 I + U_3 V + \frac{1}{2}(V_1 c_1^2(t) + V_2 c_2^2(t) + + V_3 c_3^2(t))]dt \quad (7)$$

The variables U_i and V_j, where i ranges from 1 to 3 and j ranges from 1 to 3, in equation (7) indicate the parameters that determine the cost of balancing. The weights U_1, U_2, U_3 correspond to the relative importance assigned to the susceptible, infected, and vector populations, while V_1, V_2, V_3 represent the relative economic costs of applying the three interventions: personal protection, vector control, and vector targeting efficiency. The variable T denotes the final value of the time level. The goal function examined in this study is quadratic in nature as a result of the nonlinearity of the intervention. This strategy is effectively used in the literature and the references included within [27, 28, 29, 30]. Moreover, the primary objective is to examine the optimum controls $c_1^*(t)$, $c_2^*(t)$ and $c_3^*(t)$ that result in

$$\mathcal{K}(c_1^*(t), c_2^*(t), c_3^*(t)) = \min_{\mathcal{C}}\{\mathcal{K}(c_1(t), c_2(t), c_3(t))\},$$

with the associated control set given as follows:
$\mathcal{C} = \{(c_1, c_2, c_3(t)) : [0, T] \to [0, 1], (c_1, c_2, c_3(t))$ is a Lebesgue measurable $\}$.

5.2. **Existence and characterization of optimum control.** This section aims to analyze the existence of the most efficient control pair within a certain time period for the system (6). In addition, we formulate the Hamiltonian of the optimal control problem in order to derive the necessary first-order conditions for the perfect control.

Theorem 5.1. *There is a control pair $c^*(t) = (c_1^*, c_2^*, c_3^*) \in \mathcal{C}$ that is optimum, along with associated optimal states S, I, V. These values satisfy the condition $\mathcal{K}(c_1^*(t), c_2^*(t), c_3^*(t)) = \min_{\mathcal{C}}\{\mathcal{K}(c_1(t), c_2(t), c_3(t))\}$, given the control system (6).*

Proof. We make use of the results that were reported in [31, 32] in order to establish that the most effective control for the problem that is being investigated is present. Both the state variable and the control variable have values that are either larger than or equal to zero, as can be seen and observed. Additionally, the control set U is closed and bounded in the sense that it is specified. The optimum system is constrained, which ensures that the compactness that is required for the presence of an optimal control is provided.

The biquadratic and quadratic properties of the control variables c_1, c_2 and c_3 ensure that the function $\int_0^T [U_1 S + U_2 I + U_3 V + \frac{1}{2}(V_1 c_1^2(t) + V_2 c_2^2(t) + V_3 c_3^2(t))]dt$ is convex over the control set $\mathcal{C}$. Furthermore,

$$U_1 S + U_2 I + U_3 V + \frac{1}{2}(V_1 c_1^2(t) + V_2 c_2^2(t) + V_3 c_3^2(t)) \geq \omega_1(|c_1|^2 + |c_2|^2 + |c_3|^2)^{\frac{\vartheta}{2}} - \omega_2$$

is satisfied by positive integers ω_1, ω_2, and a constant $\vartheta > 1$.

We establish the optimum control by considering the boundedness of the state variables. □

5.3. **Derivation of the Optimality System.** Let the adjoint (costate) variables be $\lambda_1(t)$ corresponding to $S(t)$, $\lambda_2(t)$ corresponding to $I(t)$, $\lambda_3(t)$ corresponding to $V(t)$.

We define Hamiltonian Function as

$$\begin{cases} \mathcal{H} = & A_1 I + A_2 V + \frac{B_1}{2} c_1^2 + \frac{B_2}{2} c_2^2 + \frac{B_3}{2} c_3^2 \\ & +\lambda_1[\Omega_H - \alpha\beta_h c_1(1-c_1)S(t-\tau_1)V(t-\tau_2)e^{-d_h\tau_1} - d_h S + \theta I + \rho(\frac{\Omega_H}{d_h} - S - I)] \\ & +\lambda_2[\alpha\beta_h c_1(1-c_1)S(t-\tau_1)V(t-\tau_2)e^{-d_h\tau_1} + \sigma\alpha\beta_h c_1(\frac{\Omega_H}{d_h} - S - I)V - (\delta + d_h \\ & +\mu_h + \theta)I] \\ & +\lambda_3[(1-c_2)\alpha\beta_v c_3 \left(\frac{\Omega_V}{d_v} - V(t-\tau_2)\right) I(t-\tau_2)e^{-d_v\tau_2} - (d_v + \mu_v)V] \end{cases}$$

The adjoint equations are derived by:

$$\frac{d\lambda_i}{dt} = -\frac{\partial \mathcal{H}}{\partial x_i}, \quad \text{with terminal conditions } \lambda_i(T) = 0, \quad i = 1, 2, 3$$

Due to the delay, this system is advanced backward in time and requires the delayed state and adjoint terms. Also a transversality conditions at final time T:

$$\lambda_i(T) = 0.$$

To find the optimal controls, we use the maximum principle which requires:

$$\frac{\partial \mathcal{H}}{\partial c_i} = 0, \quad \text{for } i = 1, 2, 3$$

For each control, derive: For c_1:

$$\frac{\partial \mathcal{H}}{\partial c_1} = B_1 c_1 - \lambda_1 \cdot \alpha\beta_h(1-2c_1)S(t-\tau_1)V(t-\tau_2)e^{-d_h\tau_1}$$
$$+ \lambda_2 \cdot \left[\alpha\beta_h(1-2c_1)S(t-\tau_1)V(t-\tau_2)e^{-d_h\tau_1} + \sigma\alpha\beta_h\left(\frac{\Omega_H}{d_h} - S - I\right)V\right] = 0$$

Solve for c_1^*, then apply projection onto control set:

$$c_1^*(t) = \min\left(1, \max\left(0, \text{respective expression}\right)\right)$$

Similarly, derive $c_2^*(t)$ and $c_3^*(t)$ from:

$$\frac{\partial \mathcal{H}}{\partial c_2} = B_2 c_2 - \lambda_3 \cdot \alpha\beta_v c_3 \left(\frac{\Omega_V}{d_v} - V(t-\tau_2)\right) I(t-\tau_2)e^{-d_v\tau_2} = 0$$

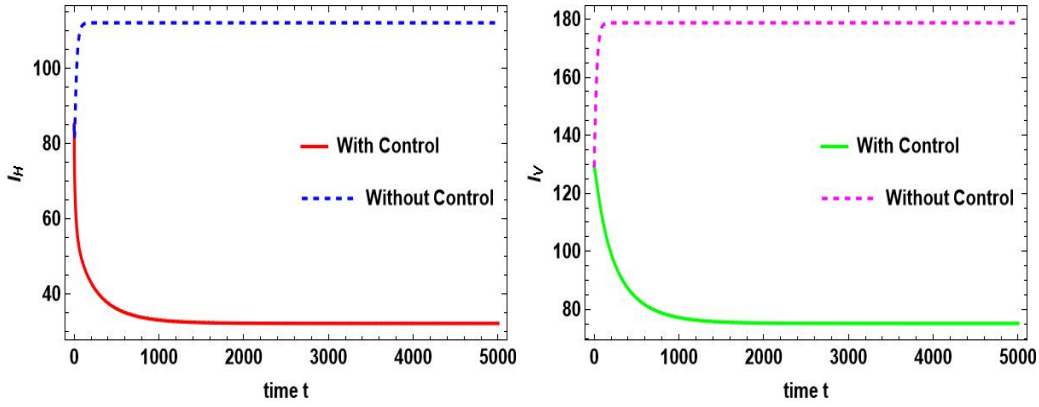
$$\frac{\partial \mathcal{H}}{\partial c_3} = B_3 c_3 + \lambda_3 \cdot (1-c_2)\alpha\beta_v \left(\frac{\Omega_V}{d_v} - V(t-\tau_2)\right) I(t-\tau_2)e^{-d_v\tau_2} = 0$$

Then project each control to lie in $[0, 1]$.

5.4. **Results and Discussion.** The effect of optimal control strategies on the dynamics of infected humans and infected vectors is illustrated under various delay scenarios. The simulations are conducted for four distinct combinations of time delays τ_1 and τ_2, representing delays in human-to-vector and vector-to-human transmission respectively. The objective functional's weight constants are as follows: The assumed values are as follows: $U_1 = 1, U_2 = 11.7, U_3 = 30, V_1 = 70, V_2 = 120, V_3 = 400$.

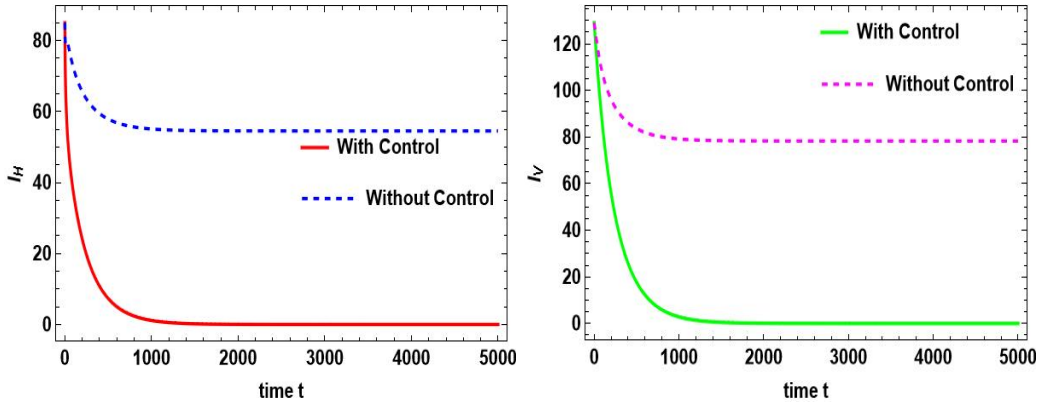
In Figure 3(a-b) the absence of any delays, the application of optimal controls significantly reduces both the infected human and vector populations over time. The controlled trajectories exhibit a faster decline in infections compared to the uncontrolled case, highlighting the efficacy of immediate intervention measures.

In Figure 4(a-b) introducing a delay in the vector-to-human transmission pathway ($\tau_2 = 37$) while keeping $\tau_1 = 0$ leads to a moderate increase in infections compared to the no-delay scenario. Nonetheless, the optimal control remains effective in reducing the burden



(a) (b)

FIGURE 3. Variation for with and without control in (a) Infected Human and (b) Infected Vector for time delay $\tau_1 = \tau_2 = 0$



(a) (b)

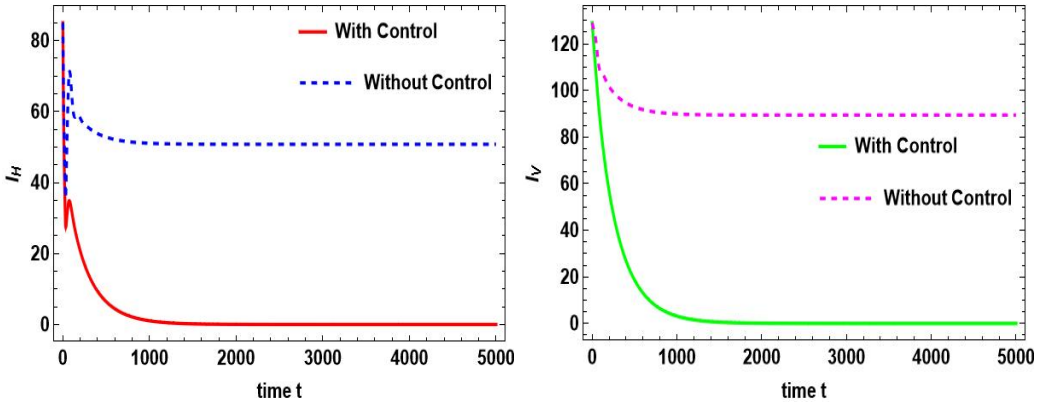
FIGURE 4. Variation for with and without control in (a) Infected Human and (b) Infected Vector for time delay $\tau_1 = 0$ and $\tau_2 = 37$

of infection, though the presence of delay slightly dampens the rate of decline in infected individuals.

When delays are present in both transmission pathways, the overall infection burden increases in the absence of control in Figure 5. However, with control implementation, both infected human and vector populations experience a substantial reduction, albeit at a slower rate than the no-delay case. This suggests that control strategies remain beneficial even when implementation or biological factors introduce delays.

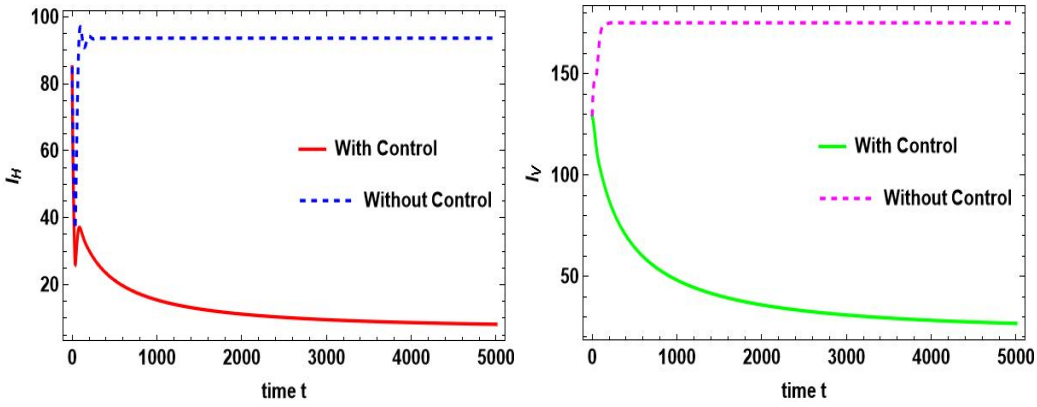
In Figure 6, with delay only in the host population, control strategies remain somewhat effective, but again the infection persists for a longer duration before stabilizing. Compared to the vector-delay-only case, the delay in the host response leads to slower control impact in reducing the infected human population.

The weights assigned to the cost terms directly influence the balance between reducing infection levels and minimizing intervention expenses. If higher weight is placed on the



(a) (b)

FIGURE 5. Variation for with and without control in (a) Infected Human and (b) Infected Vector for time delay $\tau_1 = 30$ and $\tau_2 = 31$



(a) (b)

FIGURE 6. Variation for with and without control in (a) Infected Human and (b) Infected Vector for time delay $\tau_1 = 33$ and $\tau_2 = 30$

infection-related terms (U_i), the strategy will prioritize aggressive disease suppression, often leading to sustained and intensive use of controls regardless of their cost.

Conversely, if greater emphasis is given to the intervention cost terms (V_j), the optimal strategy will favor more conservative and selective use of controls, potentially allowing higher infection levels but reducing economic or resource burden. Intermediate weightings create trade-offs, where controls may be applied more dynamically intensified during peak transmission but relaxed when infections are lower. Thus, altering cost weightings reshapes the optimal trajectory of interventions, shifting the balance between public health outcomes and economic feasibility.

Our findings highlight that delays hinder the speed of intervention impact; thus, actionable strategies such as strengthening early case detection, ensuring rapid diagnostic confirmation, and expediting vector control mobilization are recommended to minimize delay effects and enhance intervention effectiveness.

6. Conclusion

In this study, we developed and investigated a dengue transmission model incorporating both extrinsic and intrinsic incubation delays, reflecting realistic biological processes in vector-host dynamics. The basic reproduction number R_0 was derived analytically and analyzed through a detailed sensitivity analysis, which identified key parameters—such as the biting rate and transmission coefficients between humans and vectors—as major contributors to disease spread. Contour plots further illustrated the synergistic effects of these parameters on transmission potential, offering valuable insights into threshold behaviors.To mitigate the epidemic impact, an optimal control problem was formulated, integrating practical intervention strategies such as personal protection, vector control, and targeted exposure reduction. The associated Hamiltonian system, derived via Pontryagin's Maximum Principle, provided the necessary conditions for characterizing the optimal control trajectories. Numerical simulations across multiple delay scenarios revealed that the proposed control strategies are effective in significantly reducing both human and vector infections. Nonetheless, the presence of time delays particularly in vector or host response affects the rate at which control measures take effect. These findings emphasize the importance of timely intervention, and confirm the robustness and applicability of the optimal control strategies under biologically realistic, delay-affected conditions.

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