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Original articles

Dynamical behavior and bifurcation analysis for a theoretical model of dengue fever transmission with incubation period and delayed recovery

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ABSTRACT

As offered by the World Health Organisation (WHO), close to half of the population in the world's resides in dengue-risk zones. Dengue viruses are transmitted to individuals by Aedes mosquito species infected bite (Ae. Albopictus of Ae. aegypti). These mosquitoes can transmit other viruses, including Zika and Chikungunya. In this research, a mathematical model is formulated to reflect different time delays considered in both extrinsic and intrinsic incubation periods, as well as in the recovery periods of infectious individuals. Preliminary results for the non-delayed model including positivity and boundedness of solutions, non-dimensionalization and equalibria analysis are presented. The threshold parameter (reproduction number) of the model is obtained via next generation matrix schemes. The stability analysis of the model revealed that various dynamical behavior can be observed depending on delay parameters, where in particular the effect of delay in the recovery time of infectious individuals may lead to substantial changes in the dynamics. The ideas presented in this paper can be applied to other infectious diseases, providing qualitative evaluations for understanding time delays influencing the transmission dynamics.

1. Introduction

Dengue fever has recently been identified as the most rapidly transmitted arboviral disease [1,2]. The disease has been identified as a major public health concern in tropical and subtropical countries, with a significant impact on population health, as evidenced by several authoritative studies and reports [3–5]. Although, the dengue virus is common in the subtropical and tropical regions, it has been identified in approximately 128 countries, including those in North America and Europe, which are predominantly non-tropical countries. Reports indicate that approximately 390 million individuals are infected with dengue annually, resulting in 500,000 hospitalizations and 20,000 deaths from complications of the disease [4,5].

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The economic consequences of the fever epidemic have had a significant impact on several nations. From 2000 to 2007, the total annual cost in the United States, including both hospitalizations and ambulatory cases, was estimated at USD 2.1 billion [6]. Therefore, it is essential to ensure the implementation of health policy and the establishment of disease control schemes are needed [7].

The Aedes albopictus and Aedes aegypti are the two main species vectors [3]. Four serotypes of the virus can cause dengue fever: DEN-1, DEN-2, DEN-3, and DEN-4. Dengue fever is primarily transmitted by Aedes aegypti and Aedes albopictus mosquitoes, which become infected when they bite a person carrying the dengue virus. Following an incubation period of 8 to 12 days, the virus disseminates to the mosquito's salivary glands. This establishes the potential for transmission to other humans through subsequent bites. In humans, the virus incubates for 4 to 10 days before symptoms manifest. The symptoms of the disease include back pain, high fever, skin rash, muscular pains, nausea, eyes soreness, vomiting, face redness, red eyes, severe malaise, extreme weakness, and death. During this viremic phase, infected individuals can transmit the virus to other mosquitoes. Environmental factors, such as temperature and humidity, significantly influence the transmission dynamics, which affect mosquito populations and breeding sites [8,9]. In light of the increasing incidence of dengue virus disease in recent years, numerous studies have been undertaken to develop an optimally efficacious vaccine. Meanwhile, a satisfactory preventive vaccine remains unavailable; however, a partially active vaccine is available [10,11]. Thus, scientists rely on management policies and control mechanisms to eliminate viral transmission.

Epidemiological formulations are a fundamental tool in gaining deeper insight into the dynamics of infectious disease and the implementation of effective control techniques. The establishment of such mathematical formulations reveals an important area concerning the spreading and control [12,13]. Mathematical formulations for the dynamical interactions between the vector and the host were first proposed by Lotka [14] and Ross [15]. They were the first to examine the dynamic of host-vector malaria. The proposed variations in the framework can be adopted for the study of dengue fever [16–18]. Many scholars have previously adopted deterministic compartmental formulations to investigate the dengue disease dynamical propagation in specific areas or countries [16,17,19,20]. Recent mathematical models of dengue fever include different aspects of the disease, such as the effects of vector control, treatment and mass awareness on the transmission dynamics [21], specifically the birth-pulse mosquito population models with sex structures have been established [22], models with linear and nonlinear infection rate have been taken into account [23], individual immunological variability has also been considered [24].

While significant progress has been made in SIRS modeling, numerous open problems persist within the field. Recently, several authors have performed complex numerical experiments for various infectious disease models to study the sign of the eigenvalues obtained from the characteristic equation, evaluated at the endemic equilibrium [25–29]. This has facilitated the analysis of the impact of various parameter values on the dynamics of the system and proved that stability of the endemic equilibrium may lead to very complicated dynamics. In this paper, we focus on investigating the bifurcations of a dengue virus transmission model that considers the extrinsic and intrinsic incubation period, as well as the time delay in the recovery period. We demonstrate that the model is susceptible to various kinds of bifurcations, including transcritical bifurcation, period-doubling bifurcation, and Hopf bifurcation, as parameters change. In this context, we show that the delay in the recovery, τ_r, is particularly important for highly complex dynamics. Taking such terms into account in disease modeling may also prove instrumental in understanding different epidemic diseases, including the recent COVID-19 pandemic. For example, by studying period-doubling bifurcations, we can potentially identify parameter regions where complex dynamics, including deterministic chaos, may arise [28].

In this work, the authors investigate the impact of intrinsic and extrinsic incubation times on dengue fever infection dynamics by using a system of delay derivative equations. It is evident that a more comprehensive model of an epidemic should incorporate not only present-time information but also relevant data over past. Thus, the organization of the paper is as follows. In Section 2, we formulated a dynamical delayed-differential equation for effective dengue virus transmission between humans and vectors. In Section 3, we carried out preliminary results for a non-delayed model, such as positivity and boundedness of solution, non-dimensionalization, and equilibria points of the dimensionless model. Furthermore, in Section 4, we presented stability analysis for the non-delayed system using reproduction number as a threshold for the Dengue-free equilibrium point and Dengue-endemic equilibrium point, while in Section 5, we demonstrated stability analysis for the delayed system and performed the results for the numerical computation analysis (i.e. periodic oscillations). Finally, in Section 6, we presented the conclusion and future directions.

2. Model formulation

We are motivated by the work of [30], which stratified the two-interacting population into the human population ($N_h(t)$) and the vector population ($N_v(t)$) to study the dengue infection dynamics in a given locality at any time t. We shall make a slight modification to the model in [30] by incorporating delay terms τ_h and τ_v, which denote the meantime intrinsic and extrinsic incubations for humans and vectors, respectively while also modifying the forces of infection for both humans and vectors to reflect the delay terms. The total human population is divided into four categories based on disease status: susceptible $S_h(t)$, recovered $R_h(t)$, infectious $I_h(t)$ and exposed $E_h(t)$. The total human population at time t then defined as:

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$$

Likewise, the vector population is divided into three categories: susceptible vectors $S_v(t)$, infectious vectors $I_v(t)$ and exposed vectors $E_v(t)$, resulting in a total vector population N_v given as

$$N_v(t) = S_v(t) + E_v(t) + I_v(t)$$

Table 1
Description of the variables and parameters of the dengue fever model (1).

Variable	Description
S_h	Susceptible humans population
E_h	Exposed humans population
I_h	Infectious humans population
R_h	Recovered humans population
S_v	Susceptible vector population
E_v	Exposed vector population
I_v	Infectious vector population
Parameter	**Description**
π_h, π_v	Humans and vector recruitment rate respectively
β_{hv}	Probability of human-to-vector transmission
β_{vh}	Probability of vector-to-human transmission
b	Vector rate of biting
ν_h	Human antibody proportion in reaction to infection incidence induced by vector
ν_v	Vector antibody proportion in reaction to infection incidence induced by human
μ_v, μ_h	Vector and human natural death rate
δ_h	Disease induced death rate for humans
σ_h	Exposed to infectious module of disease rate of progression
σ_v	Exposed vector to infectious module of disease rate of progression
ξ_h	Recovery human infectious rate due to treatment
ω	Human immunity losing per capita rate
C_v	Control effect rate of vector
θ_c	Extrinsic incubation rate of vector

The mathematical formulation employed in the study of dengue fever dynamics is based on a nonlinear derivative deterministic system of equations:

$$\begin{aligned}
\frac{dS_h}{dt} &= \pi_h + \omega R_h - \lambda_h S_h - \mu_h S_h,\\
\frac{dE_h}{dt} &= \lambda_h S_h - (\mu_h + \sigma_h)E_h,\\
\frac{dI_h}{dt} &= \sigma_h E_h - (\xi_h + \mu_h + \delta_h)I_h,\\
\frac{dR_h}{dt} &= \xi_h I_h - (\mu_h + \omega)R_h,\\
\frac{dS_v}{dt} &= \pi_v - \lambda_v S_v - (\mu_v + C_v)S_v,\\
\frac{dE_v}{dt} &= \lambda_v S_v - (\theta_c + \sigma_v + \mu_v + C_v)E_v,\\
\frac{dI_v}{dt} &= (\theta_c + \sigma_v)E_v - (\mu_v + C_v)I_v,
\end{aligned} \tag{1}$$

where

$$\lambda_h = \frac{b\beta_{hv}I_v(t-\tau_h)}{1+\nu_h I_v(t-\tau_h)}, \qquad \lambda_v = \frac{b\beta_{vh}I_h(t-\tau_v)}{1+\nu_v I_h(t-\tau_v)}, \qquad I_h = I_h(t-\tau_r).$$

The emerging variables and parameters from the model are presented in Table 1.

The intrinsic (τ_h) and extrinsic (τ_v) delays refer to the periods associated with the virus's lifecycle in human hosts and mosquito vectors. These delays are critical for understanding how the disease spreads and predicting an outbreak's dynamics. The intrinsic delay refers to the time it takes for an infected person with dengue virus to become infectious to mosquitoes while the extrinsic delay refers to the time it takes for the dengue virus to become transmissible within a mosquito after the mosquito has bitten an infected human. Finally, the term $I_h(t-\tau_r)$ denotes the number of infectious individuals who commence the recovery process after a period of time τ_r [31].

It should be noted that the mean time extrinsic incubation, denoted by τ_v, and the mean time intrinsic incubation, denoted by τ_h, are to be understood as separate quantities.

The term $\lambda_h = \frac{b\beta_{hv}I_v(t-\tau_h)}{1+\nu_h I_v(t-\tau_h)}$ indicates that the infection time t is induced by an infectious class $I_v(t)$, and the earlier infection of τ_h units in time. Accordingly, τ_h denotes average incubation time of the produced human antibody in reaction to the vector infection. Similarly, the term $\lambda_v = \frac{b\beta_{vh}I_h(t-\tau_v)}{1+\nu_v I_h(t-\tau_v)}$ denotes the transmission time t prompted in infectious class $I_h(t)$, and τ_v represents the earlier infected time unit. Therefore, the term τ_v represents the average duration of the incubation period associated with vector-produced antibodies in response to human infection.

3. Preliminary results for non-delayed model

3.1. Positivity and boundedness of solutions

Lemma 1. *In the absence of delay, let the initial data for the dengue fever model (1) be $D(0) \geq 0$, where $D(t) = \big(S_h(t), E_h(t), I_h(t), R_h(t), S_v(t), E_v(t), \quad I_v(t)\big)$. Then the solutions $D(t)$ of the model with non-negative initial data will remain non-negative for all time $t > 0$.*

Proof. Suppose the initial data set of the dengue model $D(0) \geq 0$ such that $D(t) = \big(S_h(t), E_h(t), I_h(t), R_h(t), S_v(t), E_v(t), \quad I_v(t)\big)$ is non-negative for all $t > 0$. We begin by showing the positivity of the solutions as follows:

Taking from the first model Eq. (1) that

$$\frac{\mathrm{d}S_h}{\mathrm{d}t} = \pi_h + \omega R_h - \lambda_h S_h - \mu_h S_h \geq \pi_h - \lambda_h S_h - \mu_h S_h, \tag{2}$$

Using the method of integrating factor, Eq. (2) takes the form

$$\frac{\mathrm{d}}{\mathrm{d}t}\left(S_h(t)\exp\left[\mu_h t + \int_0^t \lambda_h(\zeta)\mathrm{d}\zeta\right]\right) \geq \pi_h \exp\left[\mu_h t + \int_0^t \lambda_h(\zeta)\mathrm{d}\zeta\right],$$

Hence,

$$S_h(t_s)\exp\left[\mu_h t + \int_0^t \lambda_h(\zeta)\mathrm{d}\zeta\right] - S_h(0) \geq \int_0^t \pi_h\left(\exp\left[\mu_h x + \int_0^x \lambda_h(\zeta)\mathrm{d}\zeta\right]\right)\mathrm{d}x,$$

so that,

$$\begin{aligned} S_h(t) \geq\ & S_h(0)\exp\left[-\mu_h t - \int_0^t \lambda_h(\zeta)\mathrm{d}\zeta\right] \\ &+ \exp\left[-\mu_h t - \int_0^t \lambda_h(\zeta)\mathrm{d}\zeta\right] \times \int_0^t \pi_h\left(\exp\left[\mu_h x + \int_0^x \lambda_h(\zeta)\mathrm{d}\zeta\right]\right)\mathrm{d}x > 0. \end{aligned}$$

Similarly, the remaining state variable $\big\{S_h(t), E_h(t), I_h(t), R_h(t), S_v(t), E_v(t), I_v(t)\big\} > 0$ at time $t > 0$. Thus, the solutions to the $D(t)$ model (1) remain positive for all initial conditions non-negative.

3.2. Invariant region

Now, the dengue fever model (1) is biologically analyzed in the feasible region.

Given the feasible region $\mathcal{J} = \mathcal{J}_h \times \mathcal{J}_v \in \mathcal{R}_+^4 \times \mathcal{R}_+^3$ with

$$\mathcal{J}_h = \left\{(S_h, E_h, I_h, R_h) \in \mathcal{R}_+^4 : N_h \leq \frac{\pi_h}{\mu_h}\right\},$$

and

$$\mathcal{J}_v = \left\{(S_v, E_v, I_v) \in \mathcal{R}_+^3 : N_v \leq \frac{\pi_v}{\mu_v + C_v}\right\}.$$

Lemma 2. *The feasible region $\mathcal{J} \subset \mathcal{R}_+^7$ is positively invariant for the model (1) with non-negative initial conditions in $\mathcal{R}_+^7$.*

Proof. The rate of change of the human and vector total population are given respectively by

$$\frac{\mathrm{d}N_h}{\mathrm{d}t} = \pi_h - \mu_h N_h(t) - \delta_h I_h(t) \leq \pi_h - \mu_h N_h(t), \tag{3}$$

and

$$\frac{\mathrm{d}N_v}{\mathrm{d}t} = \pi_v - (\mu_v + C_v)N_v(t), \tag{4}$$

where $N_h = S_h + E_h + I_h + R_h$ and $N_v = S_v + E_v + I_v$. The above Eq. (3) and (4) yields $N_h(t) \leq N_h(0)e^{-\mu_h t} + \frac{\pi_h}{\mu_h}\left(1 - e^{-\mu_h t}\right)$ and $N_v(t) = N_v(0)e^{-(\mu_v + C_v)t} + \frac{\pi_v}{\mu_v + C_v}\left(1 - e^{-(\mu_v + C_v)t}\right)$. It follows that $N_h(t) \to \frac{\pi_h}{\mu_h}$ and $N_v(t) \to \frac{\pi_v}{\mu_v + C_v}$ as $t \to \infty$. In particular, $N_h(t) \leq \frac{\pi_h}{\mu_h}$ and $N_v(t) \leq \frac{\pi_v}{\mu_v + C_v}$ if the respective total human and vector population at the initial time $N_h(0) \leq \frac{\pi_h}{\mu_h}$ and $N_v(0) \leq \frac{\pi_v}{\mu_v + C_v}$. Hence, the feasible region $\mathcal{J}$ is positively invariant.

Thus, it is appropriate to dynamically examine the dengue infection formulation (1) in the bio-feasible region $\mathcal{J}$ in which the formulation is epidemiologically and mathematically well-defined [2,32].

3.3. Non-dimensionalization

Introducing dimensionless variables:

$$\tilde{S}_h = \frac{\mu_h}{\pi_h} S_h \qquad \tilde{E}_h = \frac{v_v \sigma_h}{\mu_h} E_h, \qquad \tilde{I}_h = v_v I_h, \qquad \tilde{R}_h = \frac{v_v \mu_h}{\xi_h} R_h,$$
$$\tilde{S}_v = \frac{\mu_h}{\pi_v} S_v, \qquad \tilde{E}_v = \frac{v_h(\theta_c + \sigma_v)}{\mu_h} E_v, \qquad \tilde{I}_v = v_h I_v, \qquad \tilde{t} = \mu_h t,$$

and new dimensionless parameters:

$$\tilde{\omega} = \frac{\omega \xi_h}{\mu_h \pi_h v_v}, \quad \tilde{b}_{1h} = \frac{b\beta_{hv}}{\mu_h v_h}, \quad \tilde{\alpha}_1 = \frac{\pi_h \sigma_h v_v}{\mu_h^2}, \quad \tilde{\sigma}_h = 1 + \frac{\sigma_h}{\mu_h},$$
$$\tilde{\xi}_n = 1 + \frac{\xi_h + \delta_h}{\mu_h}, \quad \tilde{\eta}_h = 1 + \frac{\omega}{\mu_h}, \quad \tilde{b}_{1v} = \frac{b\beta_{vh}}{v_v \mu_h}, \quad \tilde{c}_{1v} = 1 + \frac{c_v}{\mu_h},$$
$$\tilde{\alpha}_2 = \frac{\pi_h v_h(\theta_c + \sigma_v)}{\mu_h^2}, \quad \tilde{c}_{2v} = 1 + \frac{\theta_c + \sigma_v + c_v}{\mu_h}.$$

the dimensionless version of the non-delayed model is given in the following

$$\frac{dS_h}{dt} = 1 + \omega R_h - \lambda_h S_h - S_h, \tag{5}$$
$$\frac{dE_h}{dt} = \alpha_1 \lambda_h S_h - \sigma_h E_h, \qquad \sigma_h > 1 \tag{6}$$
$$\frac{dI_h}{dt} = E_h - \xi_h I_h, \qquad \xi_h > 1 \tag{7}$$
$$\frac{dR_h}{dt} = I_h - \eta_h R_h, \qquad \eta_h > 1 \tag{8}$$
$$\frac{dS_v}{dt} = 1 - \lambda_v S_v - c_{1v} S_v, \qquad c_{1v} > 1 \tag{9}$$
$$\frac{dE_v}{dt} = \alpha_2 \lambda_v S_v - c_{2v} E_v, \qquad c_{2v} > 1 \tag{10}$$
$$\frac{dI_v}{dt} = E_v - c_{1v} I_v, \tag{11}$$

where

$$\lambda_h = \frac{b_{1h} I_v(t - \tau_h)}{1 + I_v(t - \tau_h)}, \qquad \lambda_v = \frac{b_{1v} I_h(t - \tau_v)}{1 + I_h(t - \tau_v)}, \qquad I_h = I_h(t - \tau_r). \tag{12}$$

Here $(\tilde{\cdot})$ is omitted for simplicity.

3.4. Equilibria of the dimensionless model

The system (5)–(11) always has a disease-free equilibrium that is

$$D_0 = (S_{ho}, E_{h0}, I_{h0}, R_{h0}, S_{v0}, E_{v0}, I_{v0}) = \left(1, 0, 0, 0, \frac{1}{c_{1v}}, 0, 0\right)$$

which corresponds to the disappearance of dengue disease. One possible way to understand more about the other roots of the equations for equilibria points of the system (5)–(11) is to express all the variables in terms of S_h. First, we will derive the expressions for these equations in this section. Using (5) and (6) gives

$$\frac{\sigma_h}{\alpha_1} E_h = 1 + \omega R_h - S_h, \tag{13}$$

Note that Eqs. (7) and (8) lead to $E_h = \xi_h I_h = \xi_h \eta_h R_h$. Substituting this in (13) gives the relation

$$R_h = \frac{\alpha_1(1 - S_h)}{(\sigma_h \xi_h \eta_h - \alpha_1 \omega)}. \tag{14}$$

with $\sigma_h \xi_h \eta_h > \alpha_1 \omega$. It is now straightforward to find the expressions for I_h and E_h in terms of S_h as

$$I_h = \frac{\eta_h \alpha_1(1 - S_h)}{(\sigma_h \xi_h \eta_h - \alpha_1 \omega)} \quad \text{and} \quad E_h = \frac{\xi_h \eta_h \alpha_1(1 - S_h)}{(\sigma_h \xi_h \eta_h - \alpha_1 \omega)}. \tag{15}$$

Similarly substituting E_h given in Eq. (15) into (6):

$$I_v = \frac{\sigma_h \xi_h \eta_h (1 - S_h)}{b_{1h} S_h (\sigma_h \xi_h \eta_h - \alpha_1 \omega) - \sigma_h \xi_h \eta_h (1 - S_h)}. \tag{16}$$

Since $E_v = c_{1v} I_v$ in Eq. (11), it is straightforward to obtain E_v with respect to S_h. Besides, using Eq. (9) and the first expression of Eq. (15), susceptible vector population S_v takes the form of susceptible human population as

$$S_v = \frac{\sigma_h \xi_h \eta_h - \alpha_1 \omega + \eta_h \alpha_1 (1 - S_h)}{c_{1v}(\sigma_h \xi_h \eta_h - \alpha_1 \omega) + \eta_h \alpha_1 (c_{1v} + b_{1v})(1 - S_h)}. \tag{17}$$

Lastly, substituting the expressions for S_v and E_v in Eq. (9), the analytical expression for S_h can be found in terms of system parameters using

$$S_h = 1 - \left(\frac{u_2 u_4 - u_1 u_5}{u_1 u_6 - u_3 u_4} \right), \tag{18}$$

where

$$\begin{aligned}
u_1 &= \eta_h \alpha_1 b_{1v}, \\
u_2 &= c_{1v}(\sigma_h \xi_h \eta_h - \alpha_1 \omega), \\
u_3 &= \eta_h \alpha_1 (c_{1v} + b_{1v}), \\
u_4 &= c_{1v} c_{2v} \sigma_h \xi_h \eta_h, \\
u_5 &= \alpha_2 b_{1h}(\sigma_h \xi_h \eta_h - \alpha_1 \omega), \\
u_6 &= -\alpha_2 (b_{1h}(\sigma_h \xi_h \eta_h - \alpha_1 \omega) + \sigma_h \xi_h \eta_h),
\end{aligned}$$

One should note that all entrenched terms from the model are positive for their biological meaning, and hence Eq. (18) has a positive root if $u_4(u_2 + u_3) < u_1(u_5 + u_6)$.

4. Stability analysis for the non-delayed system using reproduction number threshold

The disease threshold R_0 can be defined as the average number of dengue disease caused by a unitary infected vector or human when introduced into a population containing only susceptible individuals. The reproduction number can only be valid for dimensional models and real model parameters obtained from various statistical methods. By applying non-dimensionalization, we remove the dimensions and focus only on dynamics. Therefore, the reproduction number R_0 can be called differently.

Using the dimensionless model given by (5)–(11), a relation between λ_h and λ_v can be obtained with

$$\lambda_v = \frac{A_1 \lambda_h}{A_2 \lambda_h + \eta_h \sigma_h \xi_h}, \quad \text{or} \quad \lambda_h = \frac{A_3 \lambda_v}{A_4 \lambda_v + A_5}, \tag{19}$$

where $A_1 = b_{1v}\alpha_1\eta_h$, $A_2 = \eta_h\sigma_h\xi_h + \eta_h\alpha_1 - \omega\alpha_1$, $A_3 = b_{1h}\alpha_2$, $A_4 = c_{1v}c_{2v} + \alpha_2$, $A_5 = c_{2v}c_{1v}^2$. This leads to

$$(A_1 A_4 + A_2 A_5)\lambda_h^2 + (\eta_h \sigma_h \xi_h A_5 - A_1 A_3)\lambda_h = 0,$$

resulting in

$$\lambda_h = 0 \quad or \quad \lambda_h = \frac{A_1 A_3 - \eta_h \sigma_h \xi_h A_5}{A_1 A_4 + A_2 A_5} > 0 \quad if \quad R_0 > 1.$$

Namely, one can conclude

$$\frac{A_1 A_3 - \eta_h \sigma_h \xi_h A_5}{A_1 A_4 + A_2 A_5} = \frac{\eta_h \sigma_h \xi_h A_5}{(A_1 A_4 + A_2 A_5)} \left(\frac{A_1 A_3}{\eta_h \sigma_h \xi_h A_5} - 1 \right), \tag{20}$$

with

$$\frac{\eta_h \sigma_h \xi_h A_5}{(A_1 A_4 + A_2 A_5)} \left(R_0^2 - 1 \right) > 0, \quad if \quad R_0 > 1.$$

Thus,

$$\lambda_h = 0, \quad or \quad \lambda_h = \frac{\eta_h \sigma_h \xi_h A_5}{(A_1 A_4 + A_2 A_5)} (R_0^2 - 1).$$

Note that,

$$\begin{aligned}
&A_1 A_4 + A_2 A_5 = A_1 A_4 + A_5(\eta_h \sigma_h \xi_h + \eta_h \alpha_1 - \omega \alpha_1), \\
&= A_1 A_4 + A_5(\eta_h \sigma_h \xi_h + (\eta_h - \omega)\alpha_1) > 0, \quad if \quad \eta_h \geq \omega.
\end{aligned}$$

The matrix $\mathcal{F}$ associated with new infections and the matrix $\mathcal{V}$ containing the remaining expressions are given by

$$\mathcal{F} = \begin{bmatrix} \alpha_1 \lambda_h S_h \\ 0 \\ \alpha_2 \lambda_v S_v \\ 0 \end{bmatrix} \quad \text{and} \quad -\mathcal{V} = \begin{bmatrix} \sigma_h E_h \\ -E_h + \xi_h I_h \\ c_{2v} E_v \\ -E_v + c_{1v} I_v \end{bmatrix}.$$

Here, using the next-generation matrix technique, the transmission (F) and transition (V) matrices of the system (5)–(11) at dengue-free equilibrium are:

$$F|_{D0} = \begin{bmatrix} 0 & 0 & 0 & \alpha_1 b_{1h} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\alpha_2 b_{1v}}{c_{1v}} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V|_{D0} = \begin{bmatrix} \sigma_h & 0 & 0 & 0 \\ -1 & \xi_h & 0 & 0 \\ 0 & 0 & c_{2v} & 0 \\ 0 & 0 & -1 & c_{1v} \end{bmatrix}.$$

The basic reproduction number R_0 is described to be the spectral radius of the matrix FV^{-1}. Here the dominant eigenvalue is associated with the basic reproduction ratio that is

$$R_0 = \sqrt{\frac{A_1 A_3}{\eta_h \sigma_h \xi_h A_5}}. \tag{21}$$

The Eq. (21) is described as the number of secondary infections caused by an infectious host and vector in the infection stage. It is important to note that infected vectors are produced by infected hosts, and vice-versa as a result of cross-infections between humans and vectors. The square root in Eq. (21) rises because it describes the number of expected geometric mean in human and vector secondary cases.

Case 1: $\lambda_h = 0$ (Dengue-free equilibrium point that is no infection transmission in the population)

By substituting $\lambda_h = 0$ in Eq. (19) gives $\lambda_v = 0$. Thus,

$$(\lambda_{h0}, \lambda_{v0}) = (0, 0), \quad \text{corresponding to the DFE.}$$

Hence, the components of the DFE denoted by D_0 is

$$D_0 = \left(S_{ho}, E_{h0}, I_{h0}, R_{h0}, S_{v0}, E_{v0}, I_{v0}\right),$$

where

$$S_{ho} = 1,\ E_{h0} = 0,\ I_{h0} = 0,\ R_{h0} = 0,\ S_{v0} = \frac{1}{c_{1v}},\ E_{v0} = 0,\ I_{v0} = 0.$$

Case 2: $\lambda_h = \dfrac{\eta_h \sigma_h \xi_h A_5}{\left(A_1 A_4 + A_2 A_5\right)}\left(R_0^2 - 1\right)$ (Dengue-endemic equilibrium point that is a state where there is dengue transmission in both population)

By substituting $\lambda_h = \dfrac{\eta_h \sigma_h \xi_h A_5}{\left(A_1 A_4 + A_2 A_5\right)}\left(R_0^2 - 1\right)$ in (19), we have

$$\lambda_v = \frac{A_1 \dfrac{\eta_h \sigma_h \xi_h A_5}{A_1 A_4 + A_2 A_5}\left(R_0^2 - 1\right)}{A_2 \dfrac{\eta_h \sigma_h \xi_h A_5}{A_1 A_4 + A_2 A_5}\left(R_0^2 - 1\right) + \eta_h \sigma_h \xi_h} > 0, \quad if \quad R_0 > 1.$$

Thus,

$$(\lambda_h^*, \lambda_v^*) = \left(\frac{\eta_h \sigma_h \xi_h A_5}{A_1 A_4 + A_2 A_5}\left(R_0^2 - 1\right), \frac{A_1 \dfrac{\eta_h \sigma_h \xi_h A_5}{A_1 A_4 + A_2 A_5}\left(R_0^2 - 1\right)}{A_2 \dfrac{\eta_h \sigma_h \xi_h A_5}{A_1 A_4 + A_2 A_5}\left(R_0^2 - 1\right) + \eta_h \sigma_h \xi_h} \right),$$

corresponds to the Dengue-endemic equilibrium, D^* with the following components:

$$D^* = \left(S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*\right),$$

where

$$S_h^* = \frac{\eta_h \xi_h \sigma_h}{\left(\eta_h \sigma_h \xi_h - \omega \alpha_1\right)\lambda^\star + \eta_h \sigma_h \xi_h}, \quad E_h^* = \frac{\eta_h \xi_h \alpha_1 \lambda_h^\star}{\left(\eta_h \sigma_h \xi_h - \omega \alpha_1\right)\lambda^\star + \eta_h \sigma_h \xi_h},$$
$$I_h^* = \frac{\eta_h \alpha_1 \lambda_h^\star}{\left(\eta_h \sigma_h \xi_h - \omega \alpha_1\right)\lambda^\star + \eta_h \sigma_h \xi_h}, \quad R_h^* = \frac{\alpha_1 \lambda_h^\star}{\left(\eta_h \sigma_h \xi_h - \omega \alpha_1\right)\lambda^\star + \eta_h \sigma_h \xi_h},$$
$$S_v^* = \frac{1}{\lambda_v^* + c_{1v}}, \quad E_v^* = \frac{\alpha_2 \lambda_v^*}{c_{2v}(\lambda_v^* + c_{1v})}, \quad I_v^* = \frac{\alpha_2 \lambda_v^*}{c_{2v} c_{1v}(\lambda_v^* + c_{1v})}.$$

Theorem 1. *Consider the nondimensional system (5)–(11), there exists a unique positive endemic equilibrium if and only if $R_0 > 1$, otherwise. The result follows that the DFE, D_0 is locally stable if $R_0 < 1$ otherwise unstable.*

Suppose that the stability of D_0 is independent of the initial size of the infected population, thus there is need to consider the global stability of D_0. Consider the following Lyapunov function with carefully chosen scalar quantities

$$L(E_h, I_h, E_v, I_v) = \alpha_2 b_{1v} E_h + \sigma_h \alpha_2 b_{1v} I_h + \sigma_h \xi_h c_{1v} E_v + \sigma_h \xi_h c_{1v} c_{2v} I_v. \tag{22}$$

The differentiation of (22) with respect to t along the system solutions (12), yields

$$L' = \alpha_2 b_{1v} E_h' + \sigma_h \alpha_2 b_{1v} I_h' + \sigma_h \xi_h c_{1v} E_v' + \sigma_h \xi_h c_{1v} c_{2v} I_v'$$

$$L' = \alpha_2 b_{1v}\left(\alpha_1 \lambda_h S_h - \sigma_h E_h\right) + \sigma_h \alpha_2 b_{1v}\left(E_h - \xi_h I_h\right)$$
$$+ \sigma_h \xi_h c_{1v}\left(\alpha_2 \lambda_v S_v - c_{2v} E_v\right) + \sigma_h \xi_h c_{1v} c_{2v}\left(E_v - c_{1v} I_v\right),$$

leading to

$$L' = \frac{\alpha_2 b_{1v}\alpha_1 b_{1h} I_v}{1+I_v} S_h + \frac{\alpha_2 b_{1v}\xi_h\sigma_h c_{1v} I_h}{1+I_h} S_v - \sigma_h\alpha_2\xi_h b_{1v} I_h - \xi_h\sigma_h c_{2v} c_{1v}^2 I_v.$$

Further simplification yields,

$$L' = \left(\alpha_2 b_{1v}\alpha_1 b_{1h} S_h - \xi_h\sigma_h c_{2v} c_{1v}^2\right) I_v - \frac{\alpha_2 b_{1v}\alpha_1 b_{1h} I_v^2}{(1+I_v)} S_h - \frac{\xi_h\sigma_h\alpha_2 b_{1v} c_{1v} I_h^2}{(1+I_h)} S_v.$$

At the disease free equilibrium, we have

$$S_h = 1, \quad S_v = \frac{1}{c_{1v}}.$$

Then L' reduced to

$$L' = \xi_h\sigma_h c_{2v} c_{1v}^2 \left(\frac{\alpha_1\alpha_2 b_{1v} b_{1h}}{\xi_h\sigma_h c_{2v} c_{1v}^2} - 1\right) I_v - \frac{\alpha_1\alpha_2 b_{1v} b_{1h} I_v^2}{(1+I_v)} - \frac{\xi_h\sigma_h\alpha_2 b_{1v} I_h^2}{(1+I_h)}.$$

Therefore,

$$L' = \xi_h\sigma_h c_{2v} c_{1v}^2 \left(R_0^2 - 1\right) I_v - \frac{\alpha_1\alpha_2 b_{1v} b_{1h} I_v^2}{(1+I_v)} - \frac{\xi_h\sigma_h\alpha_2 b_{1v} I_h^2}{(1+I_h)} < 0,$$

when $R_0 \le 1$.

Thus, the DFE is globally asymptotically stable. In the case where conditions that favor. Dengue disease to thrive, then the DFE is no longer globally stable, leading to an endemic state. It is desirable to study the stability analysis of the endemic equilibrium. The eigenvalues using the upper triangular matrix of the Jacobian matrix of the system (5)–(11) at the endemic equilibrium D^* are given by

$$\lambda_1 = -\left(1 + \frac{b_{1h} I_v^*}{1+I_v^*}\right), \quad \lambda_2 = -\sigma_h, \quad \lambda_3 = -\xi_h, \quad \lambda_4 = -\eta_h,$$
$$\lambda_5 = -A = -\left(c_{1v} + \frac{b_{1v} I_h^*}{1+I_h^*}\right), \quad \lambda_6 = -c_{1v}, \quad \lambda_7 = -c_{1v}.$$

Clearly, both λ_1 and λ_5 are negative if $R_0 > 1$. Therefore, the Dengue-endemic equilibrium is locally asymptotically stable.

5. Stability analysis for the delayed system

The model (5)–(11) can be rewritten in a closed form:

$$\frac{\mathrm{d}}{\mathrm{d}t}\mathcal{V}(t) = H(\mathcal{V}(t), \mathcal{V}(t-\tau_h), \mathcal{V}(t-\tau_v), \mathcal{V}(t-\tau_r), P), \tag{23}$$

where $\mathcal{V} = (S_h, E_h, I_h, R_h, S_v, E_v, I_v)^T$ and $H : \mathbb{R}^{7\times 7} \times \mathbb{R}^p \to \mathbb{R}^7$ is a nonlinear function with a number of parameters $P \in \mathbb{R}^p$ and constant time delays τ_h, τ_v and τ_r. Using the linearization argument, the corresponding system at an equilibrium $\Sigma^* = \left(S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*\right)$ can be given as

$$\frac{\mathrm{d}}{\mathrm{d}t}\mathcal{Z}(t) = J_1^0\mathcal{Z}(t) + \sum_i J_{\tau_i}^D\mathcal{Z}(t-\tau_i), \qquad i = \{h, v, r\}. \tag{24}$$

Here, $\mathcal{Z} = (S_h', E_h', I_h', R_h', S_v', E_v', I_v')^T$ where the variables with primes represent perturbed variables for the linearization, e.g. for the susceptible human population we consider $S_h = S_h^* + S_h'$. The matrices are given as

$$J_0^D = \left(\frac{\partial H}{\partial \mathcal{V}}\right)\Big|_\Sigma^*, \quad J_{\tau_h}^D = \left(\frac{\partial H}{\partial \mathcal{V}(t-\tau_h)}\right)\Big|_\Sigma^*,$$

$$J_{\tau_v}^D = \left(\frac{\partial H}{\partial \mathcal{V}(t-\tau_v)}\right)\Big|_\Sigma^*, \quad J_{\tau_r}^D = \left(\frac{\partial H}{\partial \mathcal{V}(t-\tau_r)}\right)\Big|_\Sigma^*.$$

Defining a 7×7 characteristic matrix

$$J_\tau = J_0^D + \sum_i J_{\tau_i}^D \mathrm{e}^{-\lambda\tau_i}, \qquad i = \{h, v, r\}. \tag{25}$$

Then, the corresponding characteristic equation is found by using

$$\Psi(\lambda) = \mathrm{Det}\left(\lambda I_7 - J_\tau\right) = 0. \tag{26}$$

Solution of Eq. (26) gives an infinite number of eigenvalues which determine the stability of the equilibrium. Since eigenvalues sufficiently close to the imaginary axis are enough to analyze stability, a finite number of solutions, where $\mathrm{Real}(\lambda) > r$, $r \in \mathbb{R}^+$ can be taken into consideration.

5.1. Endemic equilibrium

We now focus on the endemic equilibrium given by $\Sigma_1^* = \left(S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*\right)$. Using the linearization argument the explicit form of the matrix given in Eq. (25) is given as follows

$$J_\tau = \begin{pmatrix} -m-1 & 0 & 0 & \omega & 0 & 0 & -f\mathrm{e}^{-\lambda\tau_h} \\ \alpha_1\ m & -\sigma_h & 0 & 0 & 0 & 0 & \alpha_1 f\mathrm{e}^{-\lambda\tau_h} \\ 0 & 1 & -\xi_h\mathrm{e}^{-\lambda\tau_r} & 0 & 0 & 0 & 0 \\ 0 & 0 & \mathrm{e}^{-\lambda\tau_r} & -\eta_h & 0 & 0 & 0 \\ 0 & 0 & -h\mathrm{e}^{-\lambda\tau_v} & 0 & -g-c_{1v} & 0 & 0 \\ 0 & 0 & \alpha_2 h\mathrm{e}^{-\lambda\tau_v} & 0 & \alpha_2 g & -c_{2v} & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 & -c_{1v} \end{pmatrix}.$$

Therefore, Eq. (26) becomes

$$\Psi_{\Sigma_1^*}(\lambda) = (\lambda + c_{1v})\Omega_{\Sigma_1^*}(\lambda, \tau_h, \tau_v, \tau_r), \tag{27}$$

where

$$\begin{aligned} \Omega_{\Sigma_1^*}(\lambda, \tau_h, \tau_v, \tau_r) =& \lambda(\lambda+m+1)(\lambda+c_{1v}+g)(\lambda+c_{2v})(\lambda+\sigma_h)(\lambda+\eta_h) \\ & \mathrm{e}^{-\lambda\tau_r}(\lambda+c_{2v})(\lambda+c_{1v}+g)\left[\xi_h(\lambda+\eta_h)(\lambda+m+1)(\lambda+\sigma_h)\right. \\ & \left.-m\alpha_1\omega\right] - \alpha_1\alpha_2 f h\mathrm{e}^{-\lambda(\tau_h+\tau_v)}(\lambda+1)(\lambda+\eta_h), \end{aligned} \tag{28}$$

by which Eq. (27) leads to a seventh order equation of

$$\begin{aligned} & \lambda\left[\lambda^6 + a_1\lambda^5 + a_2\lambda^4 + a_3\lambda^3 + a_4\lambda^2 + a_5\lambda + a_6\right] + \mathrm{e}^{-\lambda\tau_r}\left[\xi_h\left(\lambda^6 + a_1\lambda^5\right.\right. \\ & \left.\left. + a_2\lambda^4 + a_3\lambda^3 + a_4\lambda^2 + a_5\lambda + a_6\right) - m\alpha_1\omega\left(\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3\right)\right] \\ & + \mathrm{e}^{-\lambda(\tau_h+\tau_v)}\left[-\alpha_1\alpha_2 f h\left(\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3\right)\right] = 0, \end{aligned} \tag{29}$$

where

$$\begin{aligned} a_1 =& 2c_{1v} + c_{2v} + \eta_h + g + m + \sigma_h + 1, \\ a_2 =& c_{1v}c_{2v} + (m+1)(c_{1v} + c_{2v} + \eta_h + \sigma_h) + \sigma_h(c_{1v} + c_{2v} + \eta_h) \\ & + \eta_h(c_{1v} + c_{2v}) + (c_{1v} + g)(c_{1v} + c_{2v} + \eta_h + m + \sigma_h + 1), \\ a_3 =& \sigma_h(c_{1v}c_{2v} + \eta_h(c_{1v} + c_{2v})) + (c_{1v} + g)(c_{1v}c_{2v} + (m+1)(c_{1v} \\ & + c_{2v} + \eta_h + \sigma_h) + \sigma_h(c_{1v} + c_{2v} + \eta_h) + \eta_h(c_{1v} + c_{2v})) \\ & + (m+1)(c_{1v}c_{2v} + \sigma_h(c1v + c_{2v} + \eta_h) + \eta_h(c_{1v} + c_{2v})) + c_{1v}c_{2v}\eta_h, \\ a_4 =& (\sigma_h(c_{1v}c_{2v} + \eta_h(c_{1v} + c_{2v})) + c_{1v}c_{2v}\eta_h)(m+1) + (c_{1v} + g)(\sigma_h(c_{1v}c_{2v} \\ & + \eta_h(c_{1v} + c_{2v})) + (m+1)(c_{1v}c_{2v} + \sigma_h(c_{1v} + c_{2v} + \eta_h) \\ & + \eta_h(c_{1v} + c_{2v})) + c_{1v}c_{2v}\eta_h) + c_{1v}c_{2v}\eta_h\sigma_h, \\ a_5 =& (c_{1v} + g)((\sigma_h(c_{1v}c_{2v} + \eta_h(c_{1v} + c_{2v})) + c_{1v}c_{2v}\eta_h)(m+1) \\ & + c_{1v}c_{2v}\eta_h\sigma_h) + c_{1v}c_{2v}\eta_h\sigma_h(m+1), \\ a_6 =& c_{1v}c_{2v}\eta_h\sigma_h(c_{1v} + g)(m+1), \\ b_1 =& 2c_{1v} + c_{2v} + g, \quad b_2 = c_{1v}c_{2v} + (c_{1v} + c_{2v})(c_{1v} + g), \quad b_3 = c_{1v}c_{2v}(c_{1v} + g), \\ c_1 =& c_{1v} + \eta_h + 1, \quad c_2 = c_{1v} + \eta_h + c_{1v}\eta_h, \quad c_3 = c_{1v}\eta_h, \end{aligned} \tag{30}$$

with

$$m = \frac{b_{1h}I_v^*}{1+I_v^*}, \quad f = \frac{b_{1h}S_h^*}{(1+I_v^*)^2}, \quad h = \frac{b_{1v}S_v^*}{(1+I_h^*)^2}, \quad g = \frac{b_{1v}I_h^*}{1+I_h^*}. \tag{31}$$

It can be easily observed from Eq. (27) that there is one negative eigenvalue, that is $\lambda_1 = -c_{1v}$. Note that the values of τ_h and τ_v given in Eq. (29) do not independently affect the model. In fact, the sum of the delay parameters has a role in the system dynamics. For simplicity we consider $\tau_h = \tau_v = \tau$.

We now show some numerical results complementing the theoretical formulas. In the rest of the paper, unless stated otherwise, parameters are fixed to $\omega = 2, b_{1h} = 4, \alpha_1 = 1, \sigma_h = 1.1, \xi_h = 1.2, \eta_h = 2, b_{1v} = 1.2, c_{1v} = 1.2, \alpha_2 = 1.2, c_{2v} = 1.4$.

In Fig. 1, the temporal dynamics of all seven populations is presented for two different delay cases. In the first case, the effect of the change in the extrinsic and intrinsic incubation delay is shown in Fig. 1(a) and (b), where it is assumed that infectious individuals begin to recover without delay, e.g. $\tau_r = 0$. Fig. 1(a) represents the stable dynamics of the system (5)–(11) in the absence of delay. Increasing incubation delays from $\tau = 0$ to $\tau = 5$ ($\tau_h = \tau_v = \tau$) in Fig. 1(b), all variables stabilize after some initial oscillatory dynamics ended at around $t = 100$. For mathematical convenience, in Fig. 1(c) and (d), intrinsic and extrinsic incubation time is chosen to be half of the recovery delay, e.g. ($\tau_h = \tau_v = \tau$) and $\tau_r = 2\tau$. Considering $\tau = 0.55$ ($\tau_r = 1.1$) in Fig. 1(c) damping oscillations are observed leading to stability and high frequency periodic oscillations with a Hopf bifurcation is observed with $\tau = 0.6$ ($\tau_r = 1.2$).

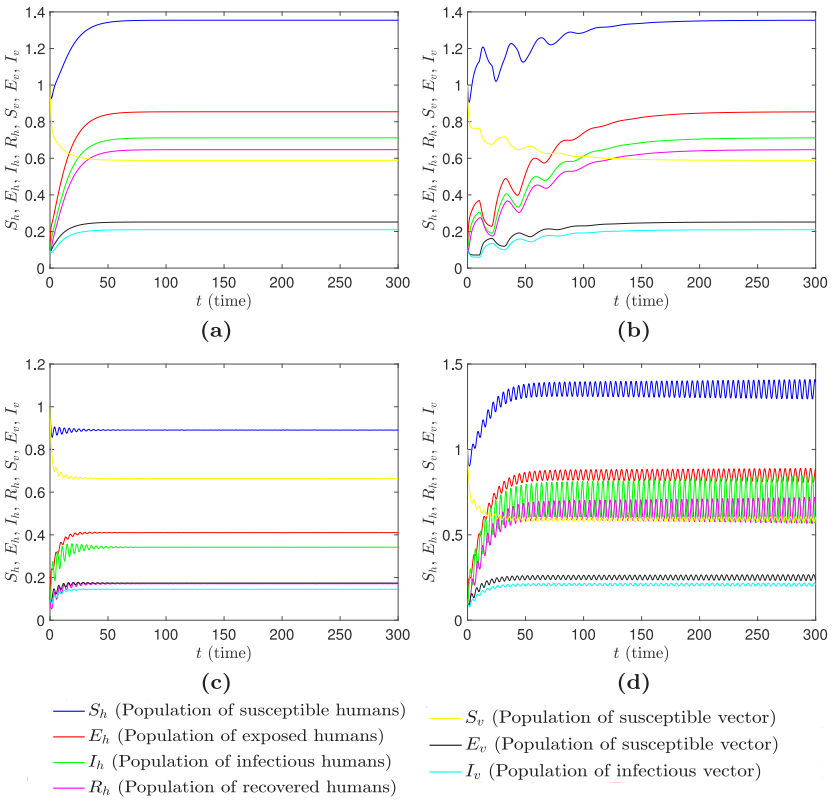

Fig. 1. Time evolution of each variable in the model (5)–(11) in the absence of delay terms, e.g. $\tau_r = \tau_h = \tau_v = 0$ (a) in the presence of average extrinsic and intrinsic incubation time with $\tau_r = 0$ and $\tau_h = \tau_v = 5$ (b), in the presence of delay terms with $\tau_r = 1.1$, $\tau_h = \tau_v = 0.55$ (c) and $\tau_r = 1.2$, $\tau_h = \tau_v = 0.6$ (d). In the presence of all three delay terms it is assumed that $\tau_r = 2\tau_h = 2\tau_v$.

Fig. 2 shows the system dynamics in the absence of intrinsic and extrinsic incubation time ($\tau_h = \tau_v = 0$) and it is assumed that the infectious individuals start to recover after some delay $\tau_r \neq 0$. If the recovery delay is lower than some threshold, all variables saturates to their steady states with damping oscillations. Then a Hopf bifurcation at a critical value of delay, e.g. $\tau_r^c \approx 1.4472$, is encountered leading to a limit cycle surrounding the positive coexistence state. In Fig. 2(b) dynamics with small amplitude oscillations is shown with $\tau_r = 1.7$. Further away from the Hopf point, e.g. $\tau_r = 2.2$, dynamics for all variables exhibits sustainable periodic oscillations and the size of the limit cycle is substantially increased. If there is an excessive increase in the recovery delay with $\tau_r = 2.4$, periodic oscillations with a stable limit cycle translate into chaos after some time, see Fig. 2(d).

In Fig. 3, stability of the susceptible human population along the equilibrium branches is shown with respect to α_1 and b_{1h} for a non-delayed case. The number of unstable eigenvalues, denoted as n_e, is associated with blue ($n_e = 0$) and red colors ($n_e = 1$). Here only transcritical bifurcation where endemic equilibrium and disease free equilibrium intersect is detected. Bifurcation of the other components with respect to other parameters can be similarly performed, yet similar dynamics is obtained.

5.2. Case I : $\tau_r = 2\tau$

Let $\lambda(\tau) = i\mu$ be a root of Eq. (29). Substituting this into (29), and considering the relation $e^{-}ix = \cos(x) - i\sin(x)$ gives the imaginary part as

$$M_1(\mu)\cos 2\mu\tau - M_2(\mu)\sin 2\mu\tau = M_3(\mu) \tag{32}$$

and real part as

$$M_2(\mu)\cos 2\mu\tau + M_1(\mu)\sin 2\mu\tau = M_4(\mu) \tag{33}$$

where

$$M_1(\mu) = \xi_h a_1\mu^5 + k_1\mu^3 + k_2\mu, \quad M_2(\mu) = -\xi_h\mu^6 + \xi_h a_2\mu^4 + k_3\mu^2 + k_4,$$
$$M_3(\mu) = \mu^7 - a_2\mu^5 + a_4\mu^3 - a_6\mu, \quad M_4(\mu) = a_1\mu^6 - a_3\mu^4 + a_5\mu^2.$$

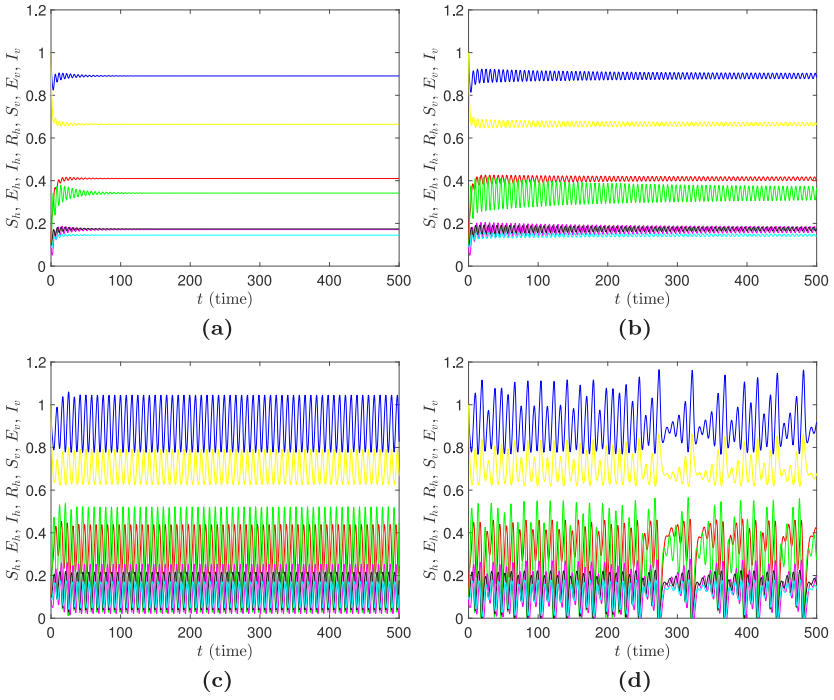

Fig. 2. Time evolution of the system given by (5)–(11) in the presence of the delay in recovery of the individuals with $\tau_h = \tau_v = 0$ and $\tau_r = 1.4$ (a), $\tau_r = 1.7$ (b), $\tau_r = 2.2$ (c) and $\tau_r = 2.4$ (d). The initial conditions are set to $(1, 0.1, 0.1, 0.1, 1, 0.1, 0.1)$ and other parameters are given in the text.

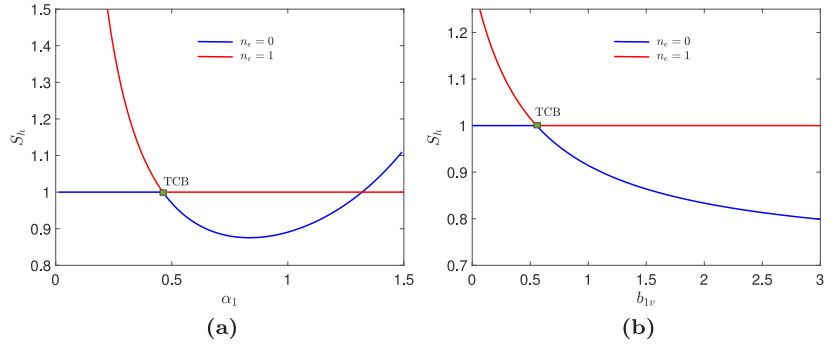

Fig. 3. Bifurcation diagram of the variable for susceptible human population with respect to two parameters: α_1 (a) and β_{1h} (b) in the absence of delay constants, e.g. $\tau_h = \tau_v = \tau_r = 0$. The green square represents a transcritical bifurcation where endemic equilibrium and disease free equilibrium intersect. The initial conditions are set to $(1, 0.1, 0.1, 0.1, 1, 0.1, 0.1)$ and other parameters are given in the text. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

with $k_1 = -xi_h a_3 + d_1$, $k_2 = \xi_h a_5 - d_3$, $k_3 = -\xi_h a_4 + d_2$ and $\xi_h a_6 - d_4$ where

$$d_j = \begin{bmatrix} m\alpha_1\omega \\ \alpha_1\alpha_2 f h \end{bmatrix} \cdot \begin{bmatrix} b_{j-1} \\ c_{j-1} \end{bmatrix}, \quad j = 1, 2, 3, 4, \tag{34}$$

for which $b_0 = c_0 = 1$. With a simple calculation, it can be shown from Eqs. (32)–(33) that $M_1^2 + M_2^2 = M_3^2 + M_4^2$, leading to a fourteenth order equation:

$$\mu^{14} + B_1\mu^{12} + B_2\mu^{10} + B_3\mu^8 + B_4\mu^6 + B_5\mu^4 + B_6\mu^2 + B_7 = 0, \tag{35}$$

where

$$\begin{aligned} B_1 =& a_1^2 - 2a_2 - \xi_h^2, \\ B_2 =& -a_1^2\xi_h^2 - 2a_3a_1 + a_2^2 + 2a_2\xi_h^2 + 2a_4, \\ B_3 =& 2a_1a_5 - 2a_6 - 2a_2a_4 + 2k_3\xi_h + a_3^2 - a_2^2\xi_h^2 - 2a_1k_1\xi_h, \end{aligned}$$

$$
\begin{aligned}
B_4 =& a_4^2 - k_1^2 + 2a_2a_6 - 2a_3a_5 + 2k_4\xi_h - 2a_1k_2\xi_h - 2a_2k_3\xi_h,\\
B_5 =& a_5^2 - k_3^2 - 2a_4a_6 - 2k_1k_2 - 2a_2k_4\xi_h,\\
B_6 =& a_6^2 - k_2^2 - 2k_3k_4,\\
B_7 =& -k_4^2.
\end{aligned}
$$

Taking $v = \mu^2$, Eq. (35) equation can be rewritten as

$$v^7 + B_1v^6 + B_2v^5 + B_3v^4 + B_4v^3 + B_5v^2 + B_6v + B_7 = 0. \tag{36}$$

The default parameter space given in Section 3.3 guarantee at least one positive root for Eq. (36). Assuming μ_c is the square root of positive root of (36) and using Eqs. (32)–(33) we find the critical threshold for time delay

$$\tau_j^c = \frac{1}{2\mu_c}\cos^{-1}\left(\frac{M_1M_3 + M_2M_4}{M_1^2 + M_2^2}\right) + \frac{\pi j}{\mu_c}, \quad j = 1, 2, \dots, \tag{37}$$

where μ_c is a positive root. Differentiating both sides of Eq. (27) with respect to τ yields

$$\mathrm{Re}\left[\frac{\mathrm{d}\lambda}{\mathrm{d}\tau}\right]\Big|_{\tau=\tau_c} = \frac{\Psi_E'(\nu_c)}{M_1^2 + M_2^2}, \qquad \nu_c = \mu_c^2. \tag{38}$$

Thus if $\Psi_E(\mu_c^2) \neq 0$ holds, then $\mathrm{Re}\left[\left(\frac{\mathrm{d}\lambda}{\mathrm{d}\tau}\right)^{-1}\right] \neq 0$ for the existence of Hopf bifurcation theorem for a system with time delay [33].

In Fig. 4, the effect of time delays is investigated using a numerical continuation and the stability analyses of both endemic and disease free states are determined under parameter variation. For example, we study the case where the dynamics is determined parameters α_1, b_{1h}, b_{1v} and c_{1v} are varied with $\tau_h = \tau_v = \tau = 0.62$ and $\tau_r = 1.24$. In this case, the system may exhibits Hopf bifurcation (purple square) in addition to transcritical bifurcation (green square). The blue line corresponds to stable state, the red dashed line corresponds to unstable periodic orbits emanating from Hopf bifurcations with ($n_e = 1$), yellow and gray lines stand for the unstable branches, for which the number of eigenvalues with positive real parts is respectively $n_e = 2$ and $n_e = 3$. In all cases, instability occurs through Hopf points where unstable equilibrium is surrounded by unstable periodic orbits. Note that there many be countably infinitely many roots of the characteristic equation Eq. (25) of the system (5)–(11) at the endemic equilibrium $\Sigma_1^* = (0.8906, 0.4102, 0.3418, 0.1709, 0.6642, 0.1740, 0.1450)$. However, it may be enough to consider the eigenvalues sufficiently close to imaginary axis. Therefore, the minimal real parts of the eigenvalues are set to real(λ) = −3 for stability.

5.3. Case II : $\tau_r \neq 0$ and $\tau = 0$

Now it is assumed that there is no average extrinsic and intrinsic incubation time, e.g. $\tau_h = \tau_v = \tau = 0$ and the infectious people start recovering after a period of time τ_r. Hence the characteristic equation given by (29) becomes

$$
\begin{aligned}
&\lambda\left[\lambda^6 + a_1\lambda^5 + a_2\lambda^4 + a_3\lambda^3 + a_4\lambda^2 + a_5\lambda + a_6\right] - \xi_h e^{-\lambda\tau_r}\left[\lambda^6 + a_1\lambda^5\right.\\
&\left. + \ a_2\lambda^4 + a_3\lambda^3 + a_4\lambda^2 + a_5\lambda + a_6\right] - m\alpha_1\omega e^{-\lambda\tau_r}(\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3)\\
&- \alpha_1\alpha_2 fh(\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3) = 0.
\end{aligned}
$$

Similarly, substituting $\lambda = i\mu$ leads to coefficients for real and imaginary parts given in (32) and (33) as

$$
\begin{aligned}
\hat{M}_1(\mu) &= \xi_h a_1\mu^5 + k_1\mu^3 + k_2\mu,\\
\hat{M}_2(\mu) &= -\xi_h\mu^6 + \xi_h a_2\mu^4 + k_3\mu^2 + k_4,\\
\hat{M}_3(\mu) &= \mu^7 - a_2\mu^5 + k_5\mu^3 + k_6\mu,\\
\hat{M}_4(\mu) &= a_1\mu^6 - a_3\mu^4 + k_7\mu^2 + k_8,
\end{aligned}
$$

with $k_1 = -\xi_h a_3 + m\alpha_1\omega$, $k_2 = \xi_h a_5 - m\alpha_1\omega b_2$, $k_3 = -\xi_h a_4 + m\alpha_1\omega b_1$ and $k_4 = \xi_h a_6 - m\alpha_1\omega b_3$, $k_5 = a_4 - \alpha_1\alpha_2 fh$, $k_6 = -a_6 + \alpha_1\alpha_2 fhc_2$, $k_7 = a_5 - \alpha_1\alpha_2 fhc_1$, $k_8 = \alpha_1\alpha_2 fhc_3$. Besides, $\hat{M}_1^2 + \hat{M}_2^2 = \hat{M}_3^2 + \hat{M}_4^2$ leads to a fourteenth order equation

$$\mu^{14} + \hat{B}_1\mu^{12} + \hat{B}_2\mu^{10} + \hat{B}_3\mu^8 + \hat{B}_4\mu^6 + \hat{B}_5\mu^4 + \hat{B}_6\mu^2 + \hat{B}_7 = 0, \tag{39}$$

where

$$
\begin{aligned}
\hat{B}_1 =& A_1,\\
\hat{B}_2 =& -a_1^2\xi_h^2 - 2a_3a_1 + a_2^2 + 2a_2\xi_h^2 + 2 * k_5,\\
\hat{B}_3 =& 2 * k_6 - 2a_2k_5 + 2a_1k_7 + 2k_3\xi_h + a_3^2 - a_2^2\xi_h^2 - 2a_1k_1\xi_h,\\
\hat{B}_4 =& -k_1^2 + k_5^2 - 2a_2k_6 + 2a_1k_8 - 2a_3k_7 + 2k_4\xi_h - 2a_1k_2\xi_h - 2a_2k_3\xi_h,\\
\hat{B}_5 =& -k_3^2 + k_7^2 - 2a_3k_8 - 2k_1k_2 + 2k_5k_6 - 2a_2k_4\xi_h,\\
\hat{B}_6 =& -k_2^2 + k_6^2 - 2k_3k_4 + 2k_7k_8,\\
\hat{B}_7 =& k_8^2 - k_4^2.
\end{aligned}
$$

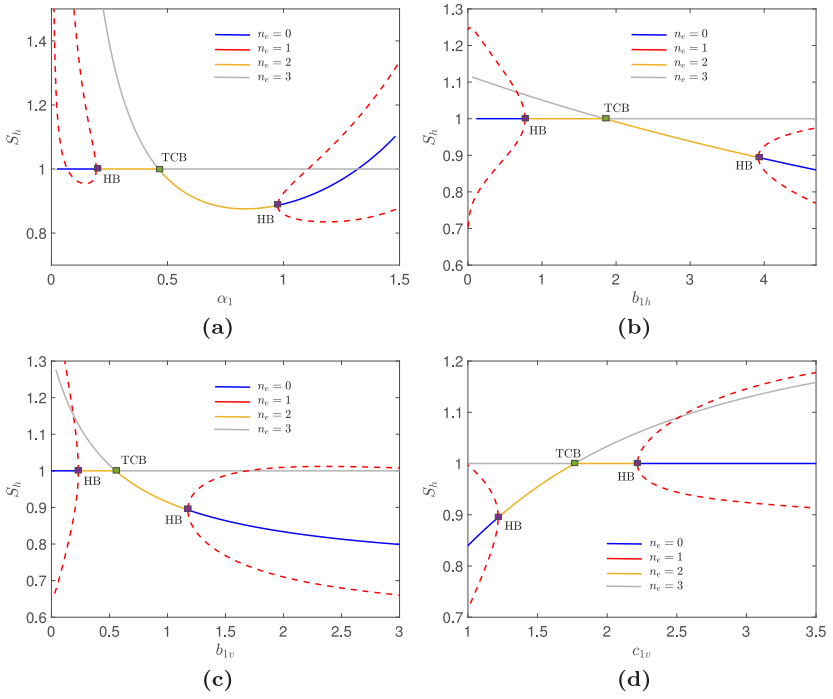

Fig. 4. Single parameter numerical continuation of susceptible human population with respect to parameters α_1 (a), b_{1h} (b), b_{1v} (c) and c_{1v} (d) with $\tau_h = \tau_v = \tau = 0.62$ and $\tau_r = 1.24$. The colored lines represent the number of eigenvalues with positive real parts. The dashed lines stand for the branches emanating from Hopf bifurcation and represent the maximum of the periodic orbits. Purple and green squares respectively represent Hopf and transcritical bifurcations. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Following the ideas presented for Case I, we find the critical threshold for time delay as

$$\tau_{r_j}^c = \frac{1}{\mu_c}\cos^{-1}\left(\frac{\hat{M}_1\hat{M}_3 + \hat{M}_2\hat{M}_4}{\hat{M}_1^2 + \hat{M}_1^2}\right) + \frac{2\pi j}{\mu_c}, \quad j = 1, 2, \dots, \tag{40}$$

where μ_c is a positive root.

In Fig. 5, stability of the susceptible human population is shown with respect to the variations of parameters α_1 (a), b_{1h} (b), b_{1v} (c) and c_{1v} (d) in the absence of intrinsic and extrinsic incubation times ($\tau_h = \tau_v = 0$) and in the presence of delay in the recovery period ($\tau_r = 2.4$). As seen, period doubling bifurcations (cyan square), where stability of the dynamics is switched from stable to unstable, arise in the periodic orbits emanating from Hopf bifurcations. Besides, interestingly the unstable branches emanating from Hopf point for disease free equilibrium exhibits spiral like trajectories.

5.4. Disease free equilibrium (DFE)

Disease free equilibrium is found as $\Sigma_2^* = \left(1, 0, 0, 0, \frac{1}{c_{1v}}, 0, 0\right)$. Using the linearization argument the explicit form of the matrix given in Eq. (25) is given as follows

$$J_\tau = \begin{pmatrix} -1 & 0 & 0 & \omega & 0 & 0 & -f\mathrm{e}^{-\lambda\tau_h} \\ 0 & -\sigma_h & 0 & 0 & 0 & 0 & \alpha_1 f\mathrm{e}^{-\lambda\tau_h} \\ 0 & 1 & -\xi_h\mathrm{e}^{-\lambda\tau_r} & 0 & 0 & 0 & 0 \\ 0 & 0 & \mathrm{e}^{-\lambda\tau_r} & -\eta_h & 0 & 0 & 0 \\ 0 & 0 & -h\mathrm{e}^{-\lambda\tau_v} & 0 & -c_{1v} & 0 & 0 \\ 0 & 0 & \alpha_2 h\mathrm{e}^{-\lambda\tau_v} & 0 & 0 & -c_{2v} & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 & -c_{1v} \end{pmatrix}.$$

The calculations performed in Section 5.1 are valid here with the exception of Eq. (31). Since only susceptible human and susceptible vector population exist for disease free state, the coefficients in Eq. (31) becomes

$$d = 0, \quad f = b_{1h}, \quad h = \frac{b_{1v}}{c_{1v}}, \quad g = 0. \tag{41}$$

As an example let us concentrate on the case where $\tau_r \neq 0$ and $\tau_h = \tau_v = 0$, leading to

$$\Psi_{\Sigma_2^*}(\lambda) = (\lambda + 1)(\lambda + \eta_h)(\lambda + c_{1v})\Omega_{\Sigma_2^*}(\lambda, 0, 0, \tau_r) = 0, \tag{42}$$

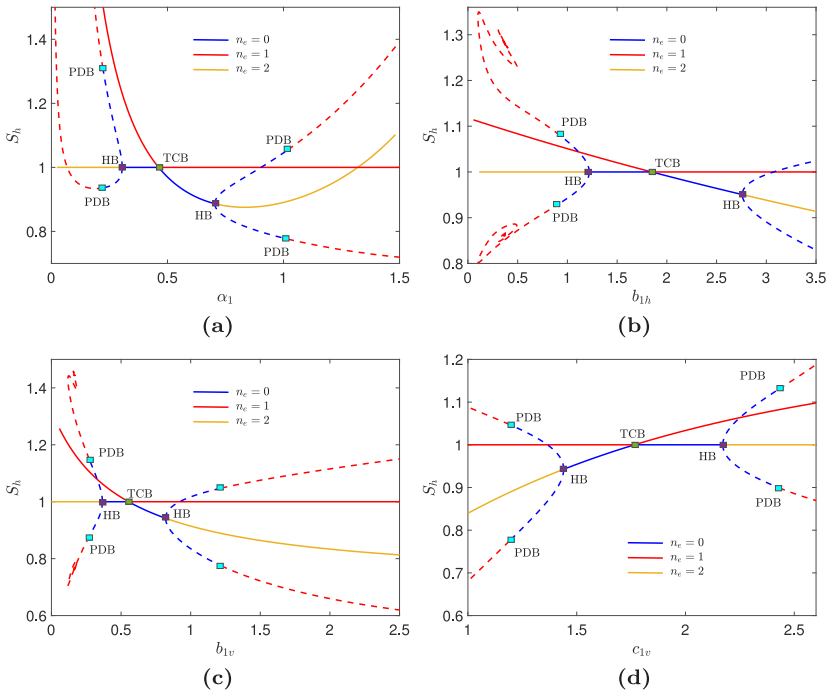

Fig. 5. Single parameter numerical continuation of susceptible human population with respect to parameters α_1 (a), b_{1h} (b), b_{1v} (c) and c_{1v} (d) with $\tau_h = \tau_v = \tau = 0$ and $\tau_r = 2.4$. The colored lines represent the number of eigenvalues with positive real parts. The dashed lines stand for the branches emanating from Hopf bifurcation and represent the maximum of the periodic orbits. Purple, cyan and green squares respectively represent Hopf, Period doubling and transcritical bifurcations. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

where

$$\Omega_{\Sigma_2^*}(\lambda, 0, 0, \tau_r) = (\lambda + c_{1v})(\lambda + c_{2v})(\lambda + \sigma_h)(\lambda + \xi_h e^{-\lambda\tau_r}) - fh\alpha_1\alpha_2 fh. \tag{43}$$

It is straightforward to see from Eq. (42) that three negative real eigenvalues arising from the disease-free system are $\lambda_1 = -c_{1v}$, $\lambda_2 = -\eta_h$ and $\lambda_3 = -1$, and the other four eigenvalues can be determined depending on the delay parameter τ_r, for which Eq. (43) can be rewritten as

$$\Omega_{\Sigma_2^*}(\lambda, 0, 0, \tau_r) = (\lambda^3 + d_1\lambda^2 + d_2\lambda + d_3)(\lambda + \xi e^{-\lambda\tau_r}) - fh\alpha_1\alpha_2 = 0, \tag{44}$$

where

$$\begin{aligned} d_1 &= c_{1v} + c_{2v} + \sigma_h, \\ d_2 &= c_{1v}c_{2v} + c_{1v}\sigma_h + c_{2v}\sigma_h, \\ d_3 &= c_{1v}c_{2v}\sigma_h. \end{aligned}$$

Let $i\mu_0$ is a solution of Eq. (44). Thus an eight order equation can be written as

$$\begin{aligned} &\mu_0^8 + (d_1^2 - 2d_2 - \xi^2)\mu^6 + (-2d_1d_3 + d_2^2 + 2\xi^2 d_2 - \xi^2 d_1^2 - 2fh\alpha_1\alpha_2)\mu^4 \\ &+ (d_3^2 - \xi^2 d_2^2 + 2\xi^2 d_1 d_3 + 2d_2 fh\alpha_1\alpha_2)\mu^2 + (fh\alpha_1\alpha_2)^2 - d_3^2\xi^2 = 0. \end{aligned} \tag{45}$$

Setting $v_0 = \mu_0^2$, a fourth order polynomial is found as in the following

$$f(v_0) = v_0^4 + D_1 v_0^3 + D_2 v_0^2 + D_3 v_0 + D_4 = 0, \tag{46}$$

with $D_1 = d_1^2 - 2d_2 - \xi^2$, $D_2 = -2d_1d_3 + d_2^2 + 2\xi^2 d_2 - \xi^2 d_1^2 - 2fh\alpha_1\alpha_2$, $D_3 = d_3^2 - \xi^2 d_2^2 + 2\xi^2 d_1 d_3 + 2d_2 fh\alpha_1\alpha_2$ and $D_4 = (fh\alpha_1\alpha_2)^2 - d_3^2\xi^2$, and therefore

$$f'(v_0) = 4v_0^3 + 3D_1 v_0^2 + 2D_2 v_0 + D_3 = 0. \tag{47}$$

Denoting $y_D = v_0 + \frac{3D_1}{4}$, Eq. (47) becomes

$$y_0^3 + e_1 y_0 + e_2 = 0, \tag{48}$$

where

$$e_1 = \frac{8D_2 - 3D_1^2}{16} \quad \text{and} \quad e_2 = \frac{D_1^3 - 4D_2D_1 + 32D_3}{32}.$$

The solutions of Eq. (46) can be obtained based on the argument given in [34]:

$$y_{01} = \sqrt[3]{-\frac{e_2}{2} + \sqrt{\hat{\Delta}}} + \sqrt[3]{-\frac{e_2}{2} - \sqrt{\hat{\Delta}}},$$
$$y_{02} = \zeta\sqrt[3]{-\frac{e_2}{2} + \sqrt{\hat{\Delta}}} + \zeta^2\sqrt[3]{-\frac{e_2}{2} - \sqrt{\hat{\Delta}}},$$
$$y_{03} = \zeta^2\sqrt[3]{-\frac{e_2}{2} + \sqrt{\hat{\Delta}}} + \zeta\sqrt[3]{-\frac{e_2}{2} - \sqrt{\hat{\Delta}}},$$

where

$$\hat{\Delta} = \left(\frac{e_1}{3}\right)^3 + \left(\frac{e_2}{2}\right)^2, \qquad \zeta = \frac{-1 + \sqrt{3}i}{2}, \quad \text{and} \quad \nu_{0j} = y_{0j} - \frac{3D_1}{4}, \quad j = 1, 2, 3.$$

Lemma 3. *One has the following results for Eq. (46):*
$\boldsymbol{C_1}$ **:** *If $fh\alpha_1\alpha_2 < d_3\xi$ $(D_4 < 0)$ Eq. (46) has at least one positive root,*
$\boldsymbol{C_2}$ **:** *If $fh\alpha_1\alpha_2 \geq d_3\xi$ and $\hat{\Delta} \geq 0$ then Eq. (46) has positive roots if and only if $\nu_{01} > 0$ and $f(\nu_{01}) < 0$,*
$\boldsymbol{C_3}$ **:** *If $fh\alpha_1\alpha_2 \geq d_3\xi$ and $\hat{\Delta} < 0$ then Eq. (46) has positive roots if and only if there exists at least one $\nu_0{}^* > 0$ for which $f(\nu_0{}^*) < 0$. Here $\nu_0{}^* \in \{\nu_{01}, \nu_{02}, \nu_{03}\}$.*

If the condition $\boldsymbol{C_1}$ holds, Eq. (46) has positive root ν_c such that Eq. (44) has a pair of imaginary roots: $\pm i\mu_c = \pm i\sqrt{\nu_c}$. Besides using the similar argument given in Eq. (40) where the components in Eq. (30) are computed for $d = 0$, $g = 0$, $f = b_{1h}$ and $h = \frac{b_{1v}}{c_{1v}}$. Furthermore, differentiating both side of Eq. (42) with respect to λ, it can be similarly shown that $\text{Re}\left[\frac{d\lambda}{d\tau}\right]^{-1} \neq 0$.

Lemma 4. *When $d_1^2 = 2d_2 + \xi^2$ and $d_2^2\xi^2 = d_3^2 + 2d_1d_3\xi^2 + 2d_2fh\alpha_1\alpha_2$. Eq. (46) becomes*

$$\mu_0^8 + D_2\mu_0^4 + D_4 = 0. \tag{49}$$

Taking $x_D = \upsilon_0^2$ and $\Delta_D = D_2^2 - 4D_4$ then we have the following conclusions:

- *When $D_2^2 > 4D_4$, then Eq. (49) has two unequal real roots: $x_D^{(1,2)} = \frac{D_2 \pm \sqrt{\Delta_D}}{2}$ then Eq. (46) has eight unequal real roots which are $\lambda_{1,2,3,4} = \pm\sqrt{x_D^{(1)}}$ and $\lambda_{5,6,7,8} = \pm\sqrt{x_D^{(2)}}$.*
- *When $\Delta_D = 0$, Eq. (49) has two equal real roots and when $\Delta_D < 0$, Eq. (49) has two unequal and conjugate imaginary roots $x_D^{(1,2)} = \frac{D_2 \pm i\sqrt{\Delta_D}}{2}$ then Eq. (46) has eight unequal and conjugate imaginary roots.*

6. Conclusion

In this research, a theoretical dengue fever propagation analysis for the extrinsic and intrinsic incubation period is modeled via a system of delay differential equations. As our focus is on the dynamics, we perform non-dimensionalization for model Eq. (1). Here, the dengue fever non-dimensionalized model exhibits existence of dual equilibrium points: the Dengue-free equilibrium and the Dengue-endemic equilibrium. The Dengue-free equilibrium in the absence of delay is locally and globally stable at $R_0 \leq 1$. Otherwise, the Dengue-endemic equilibrium exists for $R_0 > 1$. In this context, R_0 represents a threshold that determines whether the dengue disease is eradicated or persists in the population of interest.

Since time delays appear in infectious groups I_v and I_h, the dengue disease model presented in system (1) relies on the system of solutions at earlier periods. In fact, like in many real-life applications, time lags occur in many disease models and thus give much more reasonable insights into population dynamics. As illustrated in the time simulations given in Section 5, compared to the cases where there is no delay, a substantial change in the dynamics is observed, particularly with the increase in delay term τ_r, representing the time after that infectious individuals begin to recover. Then the results are extended to include single-parameter bifurcation diagrams of the system and the effect of delay parameters on the system dynamics is explicitly presented. In this context, only transcritical bifurcation (TCB) has been detected for the case without delays. However, more complicated dynamics with period-doubling bifurcation and Hopf bifurcation have been observed in the occurrence of time lags. In fact, several authors have performed complex numerical experiments for bifurcation analysis for various infectious disease models [25,28,29]. This may also play an important role in understanding different epidemic diseases. For instance, by studying period-doubling bifurcations, we can potentially identify parameter regions where complex dynamics, such as chaos, arise [28].

The numerical simulations in this paper have been carried out via MATLAB's dde23 solver. The results for numerical continuation have been obtained through DDE-BIFTOOL software, which is a collection of MATLAB programs. In this regard, the number of unstable eigenvalues is associated with specific line types, as illustrated in Fig. 3. Here, one should note that the countable indefinite

eigenvalues number can be gotten via the characteristic equation exponential terms. Meanwhile, only the eigenvalues which are close sufficient to the imaginary axis matter for stability. Therefore, we only consider the eigenvalues which satisfy $\mathrm{Re}(\mu) > -3$.

The ideas presented in this paper support the existence of more complex dynamics, e.g. periodic and non-periodic oscillations. Our analytical and numerical experiments have shown that delay term τ_r has an especially great impact on the model dynamics. Therefore, further analysis in terms of the biological and analytical meaning of τ_r should be performed. Besides, the backward bifurcation phenomenon in infectious dispersion formulations is often studied for the purpose of epidemic control. However, as seen in this paper, some of these models also support other types of bifurcations, including transcritical, period doubling, and Hopf bifurcations, under certain conditions. Therefore, it is essential to have insight into the role of these bifurcations from an epidemiological point of view. Further analysis on this topic is reserved for future papers. Dengue infection is an endemic health disease, commonly in the sub-tropical and tropical areas of the globe. Thus, it is significant to consider the possible re-occurrence of the outbreak by examining various strategies of optimal control and effective cost analysis.

CRediT authorship contribution statement

Burcu Gürbüz: Conceptualization, Data curation, Funding acquisition, Investigation, Methodology, Project administration, Resources, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing. **Aytül Gökçe:** Conceptualization, Data curation, Resources, Software, Validation, Visualization, Writing – original draft, Writing – review & editing, Investigation. **Segun I. Oke:** Formal analysis, Writing – original draft, Writing – review & editing, Validation. **Michael O. Adeniyi:** Conceptualization, Formal analysis, Investigation, Writing – original draft, Writing – review & editing, Resources. **Mayowa M. Ojo:** Writing – original draft, Resources.

References

[1] M.G. Guzman, E. Harris, Dengue, Lancet 385 (9966) (2015) 453–465.
[2] M. Ojo, F. Akinpelu, Lyapunov functions and global properties of SEIR epidemic model, Int. J. Chem. Math. Phys. 1 (1) (2017).
[3] W.H. Organization, et al., Comprehensive guideline for prevention and control of dengue and dengue haemorrhagic fever, 2011.
[4] W.H. Organization, et al., Report of the Meeting of the WHO/VMI Workshop on Dengue Modeling: 25-26 August 2010, Geneva, Switzerland, Tech. Rep., World Health Organization, 2011.
[5] WHO Expert Committee on the Selection and Use of Essential Medicines and World Health Organization, The Selection and Use of Essential Medicines: Report of the WHO Expert Committee, 2013 (including the 18th WHO Model List of Essential Medicines and the 4th WHO Model List of Essential Medicines for Children), vol. 985, World Health Organization, 2014.
[6] D.S. Shepard, L. Coudeville, Y.A. Halasa, B. Zambrano, G.H. Dayan, Economic impact of dengue illness in the Americas, Am. J. Trop. Med. Hyg. 84 (2) (2011) 200.
[7] D.-L. Luh, C.-C. Liu, Y.-R. Luo, S.-C. Chen, Economic cost and burden of dengue during epidemics and non-epidemic years in Taiwan, J. Infect. Public Heal. 11 (2) (2018) 215–223.
[8] D.J. Gubler, Dengue and dengue hemorrhagic fever, Clin. Microbiol. Rev. 11 (3) (1998) 480–496.
[9] M.G. Guzmán, G. Kouri, J. Bravo, L. Valdes, V. Susana, S.B. Halstead, Effect of age on outcome of secondary dengue 2 infections, Int. J. Infect. Dis. 6 (2) (2002) 118–124.
[10] S. East, World's First Dengue Fever Vaccine Launched in the Philippines, CNN, 2016, Archived from the original on 18.
[11] K.K. Tan, S. Nellis, N.I. Zulkifle, S. Sulaiman, S. AbuBakar, Autochthonous spread of DENV-3 genotype III in Malaysia mitigated by pre-existing homotypic and heterotypic immunity, Epidemiol. Infect. 146 (13) (2018) 1635–1641.
[12] Y. Xiao, T. Zhao, S. Tang, Dynamics of an infectious diseases with media/psychology induced non-smooth incidence, Math. Biosci. Eng. 10 (2) (2013) 445.
[13] F. Ndaïrou, I. Area, J.J. Nieto, C.J. Silva, D.F. Torres, Fractional model of COVID-19 applied to galicia, Spain and Portugal, Chaos Solitons Fractals 144 (2021) 110652.
[14] R. Ross, History of Mosquito-Borne Disease Modeling, 1897-1969.
[15] R. Ross, The Prevention of Malaria, John Murray, 1911.
[16] F. Agusto, M. Khan, Optimal control strategies for dengue transmission in Pakistan, Math. Biosci. 305 (2018) 102–121.
[17] L. Esteva, C. Vargas, Analysis of a dengue disease transmission model, Math. Biosci. 150 (2) (1998) 131–151.
[18] N. Hamdan, A. Kilicman, Analysis of the fractional order dengue transmission model: a case study in Malaysia, Adv. Difference Equ. 2019 (1) (2019) 1–13.
[19] A. Abidemi, N.A.B. Aziz, Optimal control strategies for dengue fever spread in Johor, Malaysia, Comput. Methods Programs Biomed. 196 (2020) 105585.
[20] S. Syafruddin, M.S.M. Noorani, Lyapunov function of SIR and SEIR model for transmission of dengue fever disease, Int. J. Simul. Process. Model. 8 (2–3) (2013) 177–184.
[21] B.Z. Naaly, T. Marijani, A. Isdory, J.Z. Ndendya, Mathematical modeling of the effects of vector control, treatment and mass awareness on the transmission dynamics of dengue fever, Comput. Methods Programs Biomed. Updat. 6 (2024) 100159.
[22] X. Zhang, J. Li, X. Liu, Birth-pulse models to assess the effects of wolbachia-carrying mosquito releases on the control of dengue fever, Appl. Math. Model. 134 (2024) 175–197.
[23] P. Muthu, B. Modak, Stability of in-host models of dengue virus transmission with linear and nonlinear infection rate, Differ. Equ. Dyn. Syst. (2024) 1–27.
[24] V. Anam, B.V. Guerrero, A.K. Srivastav, N. Stollenwerk, M. Aguiar, Within-host models unravelling the dynamics of dengue reinfections, Infect. Dis. Model. 9 (2) (2024) 458–473.
[25] B.W. Kooi, M. Aguiar, N. Stollenwerk, Bifurcation analysis of a family of multi-strain epidemiology models, J. Comput. Appl. Math. 252 (2013) 148–158.
[26] V. Steindorf, S. Oliva, N. Stollenwerk, M. Aguiar, Symmetry in a multi-strain epidemiological model with distributed delay as a general cross-protection period and disease enhancement factor, Commun. Nonlinear Sci. Numer. Simul. 128 (2024) 107663.
[27] V. Steindorf, A.K. Srivastav, N. Stollenwerk, B.W. Kooi, M. Aguiar, Modeling secondary infections with temporary immunity and disease enhancement factor: Mechanisms for complex dynamics in simple epidemiological models, Chaos Solitons Fractals 164 (2022) 112709.
[28] N. Stollenwerk, S. Spaziani, J. Mar, I.E. Arrizabalaga, D. Knopoff, N. Cusimano, V. Anam, A. Shrivastava, M. Aguiar, et al., Seasonally forced SIR systems applied to respiratory infectious diseases, bifurcations, and chaos, Comput. Math. Methods 2022 (2022).
[29] F. Zhang, W. Cui, Y. Dai, Y. Zhao, Bifurcations of an SIRS epidemic model with a general saturated incidence rate, Math. Biosci. Eng. 19 (11) (2022) 10710–10730.

[30] S.M. Garba, A.B. Gumel, M.A. Bakar, Backward bifurcations in dengue transmission dynamics, Math. Biosci. 215 (1) (2008) 11–25.
[31] S. Gonçalves, G. Abramson, M.F. Gomes, Oscillations in SIRS model with distributed delays, Eur. Phys. J. B 81 (2011) 363–371.
[32] M.M. Ojo, B. Gbadamosi, T.O. Benson, O. Adebimpe, A. Georgina, Modeling the dynamics of lassa fever in Nigeria, J. Egyptian Math. Soc. 29 (1) (2021) 1–19.
[33] B.D. Hassard, B. Hassard, N.D. Kazarinoff, Y.-H. Wan, Y.W. Wan, Theory and Applications of Hopf Bifurcation, vol. 41, CUP Archive, 1981.
[34] X. Li, J. Wei, On the zeros of a fourth degree exponential polynomial with applications to a neural network model with delays, Chaos Solitons Fractals 26 (2) (2005) 519–526.