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DYNAMICAL ANALYSIS OF CHIKUNGUNYA AND DENGUE CO-INFECTION MODEL

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Abstract. The aim of this paper is to design and analyze a nonlinear mechanistic model for chikungunya (CHIKV) and dengue (DENV) co-endemicity. The model can assess the epidemiological consequences of the spread of each disease on the co-infection transmission dynamics. Although the two diseases are different, they exhibit similar dynamical features which show that to combat/control CHIKV virus (or co-infection with DENV virus) we can employ DENV control strategies and vice versa. Our analytical results show that each sub-model and the full model have two disease-free equilibria (i.e., trivial disease-free equilibrium (TDFE) and non-trivial disease-free equilibrium (NTDFE)). Further, qualitative analyses reveal that each of the sub-models exhibits the phenomenon of backward bifurcation (where a stable NTDFE co-exits with a stable endemic equilibrium (EE)). Epidemiologically, this implies that, in each case (CHIKV or DENV), the basic requirement of making the associated reproduction number to be less-than unity is no longer sufficient for the disease eradication. We further highlight that the full model, consisting of twenty-six (26) mutually exclusive compartments representing the human and mosquito dynamics, also exhibits the phenomenon of backward bifurcation. We fit the full model and its sub-models using realistic data from India. Sensitivity analysis using the partial rank correlation coefficient (PRCC) is used for ranking the importance of each parameter-output. The results suggested that the mosquito removal rates, the transmission rates, and the mosquito maturation rate are the top control parameters for combating CHIKV, DENV and CHIKV-DENV co-infection outbreaks.

Keywords: Co-infection, Chikungunya, Dengue, Stability, Sensitivity Analysis.

1. **Introduction.** Chikungunya virus (CHIKV), a vector-borne disease transmitted by an infected female mosquito, is an *arboviral* disease caused by a member of the genus *alphavirus* which belongs to the *Togaviridae* family [8, 26, 28, 30, 39, 49]. Whereas, Dengue virus (DENV) belongs to the genus *Flavivirus*, under the family of *Flaviviridae*, and there are four serotypes of DENV which causes a wide spectrum of illness from asymptomatic to symptomatic or severe fatal Dengue Hemorrhagic Fever or Dengue Shock Syndrome (DHF/DSS) (the DHF/DSS has

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a high mortality risk with death occurring within 24hrs after the onset of shock) [17, 25, 29, 33, 35, 46]. Infection with one serotype of DENV gives lifelong immunity to subsequent infection by the same serotype, but, it does not confer strong immunity against infection with other serotypes of the DENV virus, whereas CHIKV gives permanent immunity [2, 26, 39].

It has been reported that CHIKV virus is endemic in more than 60 countries in Asia, Africa, Europe, and America causing deaths and several clinical cases [10, 39, 47]. Unlike DENV, CHIKV has a low death rate, therefore it is not considered as a life-threatening disease [16]. Over 1.3 million suspected CHIKV cases and about 200 deaths have been reported in the Latin American countries, and Caribbean islands in April 2015 [47, 49]. While DENV is endemic in more than 100 countries in Africa, the Americas, the Eastern Mediterranean and subtropical regions of the world, populated by over 2.5 billion people [16, 26, 39, 48]. DENV is a deadly mosquito-borne disease, with an estimate of 390 million new infections of which about 96 million are clinical and more than 20 thousand deaths per year [2, 16, 30, 45, 49].

CHIKV and DENV are transmitted by the same vectors, that is *Aedes aegypti* and/or *Aedes albopictus* mosquitoes, and in each case, the viral infection has virtually the same signs and symptoms in the patients [16, 18, 26, 38, 39]. Therefore, it is very difficult to identify an individual infection or co-infection among the patients [39]. CHIKV-DENV co-infection cases were first reported in Thailand by Nimmannitya *et al.* [32], who detected many cases after diagnosing a number of patients. The fast-growing nature of the co-infection of diseases becomes a big threat to global public health and development [5, 13, 14, 26, 39]. In recent years the arboviral disease has been increased very rapidly, and in most of the cases CHIKV outbreaks are considered as a DENV due to the similarity of their primary signs and symptoms, this is especially in the DENV endemic regions. The co-infection of CHIKV and DENV and their co-circulation in the same region have made the problem more complicated and spreading very rapidly capturing many other countries [39]. Furthermore, the co-infection has been reported to occur in India, Indonesia, Sri Lanka, Malaysia, Gabon, Cameroon, Madagascar, Thailand, and Nigeria [5, 2, 13, 26, 32, 39]. Also, it has been reported that simultaneous/concurrent infection of CHIKV and DENV viruses in patients occurred in many countries such as India, Yemen, Singapore, Sri Lanka, Malaysia and so on [39]. Therefore it is very important to consider the individual infection and the co-infection cases in order to reduce or eliminate the problem of the CHIKV and DENV co-infection.

Currently, both CHIKV and DENV do not have a preventive vaccine (although dengvaxia, a vaccince for the DENV, produced by Sanofi Pasteur and approved in some countries [12]), but a number of candidate vaccines are still undergoing clinical trials [1, 12, 45]. There is no specific treatment for the two viruses, however, the treatment could be achieved by reducing the disease symptoms [1, 16, 47, 48].

Numerous mathematical modeling studies have been designed to show some insights into the transmission dynamics of CHIKV or DENV (see [1, 10, 16, 26, 30, 39, 49]). To the author's knowledge, the current study gives the first co-infection model for CHIKV and DENV with the aim to assess the impact of each disease on co-infection transmission dynamics. The model is an extension of some of the CHIKV and DENV transmission models (e.g., those in [1, 16, 26, 49, 51]) by

(i) incorporating CHIKV-DENV co-infection in both human and mosquito populations,

(ii) including the dynamics of aquatic (immature) stages of the mosquito life cycle (egg, larva and pupa stages) in each sub-model,
(iii) using a nonlinear biting rate (constant rate was used in [1, 16]), and
(iv) including a transovarial/vertical transmission of CHIKV in mosquito population [22].

The paper is organized as follows. A new model for the transmission dynamics of CHIKV-DENV co-infection is formulated in Section 2. Theoretical results for the associated sub-models are reported in Section 3. The full model is analyzed in Section 4. Numerical analyses are carried out in Section 5.

2. **Model formulation.** The total humans population at time t, denoted by $N_H(t)$, is divided into sixteen mutually exclusive compartments as follows: susceptible individuals, who are at risk of infection of both CHIKV and DENV ($S_H(t)$), individuals exposed to CHIKV ($E_C(t)$), individuals exposed to DENV ($E_D(t)$), individuals exposed (i.e., asymptomatic) to both CHIKV and DENV ($E_{CD}(t)$), CHIKV-infected (only) individuals with clinical symptoms of CHIKV ($I_C(t)$), DENV-infected (only) individuals with clinical symptoms of DENV ($I_D(t)$), CHIKV-infected individuals with clinical symptoms of CHIKV but exposed to DENV ($I_{CE}(t)$), DENV-infected individuals with clinical symptoms of DENV but exposed to CHIKV ($I_{DE}(t)$), dually-infected individuals with clinical symptoms of both CHIKV and DENV ($I_{CD}(t)$), individuals who recovered from CHIKV ($R_C(t)$), individuals who recovered from DENV ($R_D(t)$), individuals exposed to CHIKV but recovered from DENV with permanent immunity ($E_{CT}(t)$), individuals exposed to DENV but recovered from CHIKV with permanent immunity ($E_{DT}(t)$), CHIKV-infected individuals with clinical symptoms of CHIKV but recovered from DENV with permanent immunity ($I_{CT}(t)$), DENV-infected individuals with clinical symptoms of DENV but recovered from CHIKV with permanent immunity ($I_{DT}(t)$), individuals who recovered from both CHIKV and DENV with permanent immunity ($T(t)$), so that

$$N_H(t) = S_H(t) + E_C(t) + E_D(t) + E_{CD}(t) + E_{CT}(t) + E_{DT}(t) + I_C(t) + I_D(t) +$$

$$I_{CE}(t) + I_{DE}(t) + I_{CD}(t) + I_{CT}(t) + I_{DT}(t) + R_C(t) + R_D(t) + T(t).$$

The total mosquitoes population at time t, denoted by $N_v(t)$, is sub-divided into sub-populations of immature mosquitoes (eggs, larvae and pupae stages), denoted by $A(t)$, and adult mosquitoes (denoted by $N_I(t)$), so that:

$$N_v(t) = A(t) + N_I(t),$$

where $N_I(t)$ is further divided into nine compartments as follows: adult mosquitoes susceptible to both CHIKV and DENV viruses ($S_v(t)$), adult mosquitoes exposed to CHIKV ($E_{vC}(t)$), adult mosquitoes exposed to DENV ($E_{vC}(t)$), adult mosquitoes exposed to both CHIKV and DENV viruses ($E_M(t)$), CHIKV-infected (only) adult mosquitoes ($I_{vC}(t)$), DENV-infected (only) adult mosquitoes ($I_{vD}(t)$), CHIKV-infected adult mosquitoes that are exposed to DENV ($I_{vCE}(t)$), DENV-infected adult mosquitoes that are exposed to CHIKV ($I_{vDE}(t)$), adult mosquitoes infected to both CHIKV and DENV ($I_{vM}(t)$), so that

$$N_I(t) = S_v(t) + E_{vC}(t) + E_{vD}(t) + E_M(t) + I_{vC}(t) + I_{vD}(t) + I_{vCE}(t) + I_{vDE}(t) + I_{vM}(t).$$

The model for the CHIKV-DENV co-endemicity is given by the following deterministic system of nonlinear differential equations (a flow diagram of the model is

depicted in Figure 1 and 2 and the associated variables and parameters are tabulated in Tables 1 and 2, respectively):

$$
\begin{aligned}
\frac{dS_H}{dt} &= \Pi_H - (\lambda_C + \lambda_D)S_H - \mu_H S_H,\\
\frac{dE_C}{dt} &= \lambda_C S_H - \alpha_1\lambda_D E_C - (\sigma_C + \mu_H)E_C,\\
\frac{dE_D}{dt} &= \lambda_D S_H - \alpha_2\lambda_C E_D - (\sigma_D + \mu_H)E_D,\\
\frac{dE_{CD}}{dt} &= \alpha_1\lambda_D E_C + \alpha_2\lambda_C E_D - (\gamma_1 + \gamma_2 + \mu_H)E_{CD},\\
\frac{dI_C}{dt} &= \sigma_C E_C - \alpha_1\lambda_D I_C - (\tau_C + \delta_C + \mu_H)I_C,\\
\frac{dI_D}{dt} &= \sigma_D E_D - \alpha_2\lambda_C I_D - (\tau_D + \delta_D + \mu_H)I_D,\\
\frac{dI_{CE}}{dt} &= \alpha_1\lambda_D I_C + \gamma_1 E_{CD} - (\delta_{CE} + \gamma_3 + \mu_H)I_{CE},\\
\frac{dI_{DE}}{dt} &= \alpha_2\lambda_C I_D + \gamma_2 E_{CD} - (\delta_{DE} + \gamma_4 + \mu_H)I_{DE},\\
\frac{dI_{CD}}{dt} &= \gamma_3 I_{CE} + \gamma_4 I_{DE} - (\tau_C + \tau_D + \delta_{CD} + \mu_H)I_{CD},\\
\frac{dE_{CT}}{dt} &= \lambda_C R_D - (\sigma_{CT} + \mu_H)E_{CT},\\
\frac{dE_{DT}}{dt} &= \lambda_D R_C - (\sigma_{DT} + \mu_H)E_{DT},\\
\frac{dI_{CT}}{dt} &= \tau_D I_{CD} + \sigma_{CT}E_{CT} - (\delta_{CT} + \tau_C + \mu_H)I_{CT},\\
\frac{dI_{DT}}{dt} &= \tau_C I_{CD} + \sigma_{DT}E_{DT} - (\delta_{DT} + \tau_D + \mu_H)I_{DT},\\
\frac{dR_C}{dt} &= \tau_C I_C - \lambda_D R_C - \mu_H R_C,\\
\frac{dR_D}{dt} &= \tau_D I_D - \lambda_C R_D - \mu_H R_D,\\
\frac{dT}{dt} &= \tau_C I_{CT} + \tau_D I_{DT} - \mu_H T,
\end{aligned}
\tag{1}
$$

and

$$
\begin{aligned}
\frac{dA}{dt} &= \Pi_v - (1+\mu_A)A,\\
\frac{dS_v}{dt} &= \xi A - (\lambda_{vC}+\lambda_{vD})S_v - \mu_v S_v,\\
\frac{dE_{vC}}{dt} &= \lambda_{vC}S_v - \omega_1\lambda_{vD}E_{vC} - (\sigma_{vC}+\mu_v)E_{vC},\\
\frac{dE_{vD}}{dt} &= \lambda_{vD}S_v - \omega_2\lambda_{vC}E_{vD} - (\sigma_{vD}+\mu_v)E_{vD},\\
\frac{dE_M}{dt} &= \omega_1\lambda_{vD}E_{vC} + \omega_2\lambda_{vC}E_{vD} - (\theta_1+\theta_2+\mu_v)E_M,\\
\frac{dI_{vC}}{dt} &= (1-\xi)A + \sigma_{vC}E_{vC} - (\lambda_{vD}+\mu_v)I_{vC},\\
\frac{dI_{vD}}{dt} &= \sigma_{vD}E_{vD} - (\lambda_{vC}+\mu_v)I_{vD},\\
\frac{dI_{vCE}}{dt} &= \theta_1 E_M + \lambda_{vD}I_{vC} - (\rho_1+\mu_v)I_{vCE},\\
\frac{dI_{vDE}}{dt} &= \theta_2 E_M + \lambda_{vC}I_{vD} - (\rho_2+\mu_v)I_{vDE},\\
\frac{dI_{vM}}{dt} &= \rho_1 I_{vCE} + \rho_2 I_{vDE} - \mu_v I_{vM}.
\end{aligned}
\tag{2}
$$

where,

$$
\begin{aligned}
\lambda_C &= \frac{\beta_C b_1(N_H,N_v)}{N_v}[(\eta_{vC}E_{vC}+\eta_M E_M) + I_{vC} + I_{vCE} + I_{vM}],\\
\lambda_D &= \frac{\beta_D b_1(N_H,N_v)}{N_v}[(\eta_{vD}E_{vD}+\eta_M E_M) + I_{vD} + I_{vDE} + I_{vM}],\\
\lambda_{vC} &= \frac{\beta_v b_2(N_H,N_v)}{N_H}[(\eta_C(E_C+E_{CT})+\eta_{CD}E_{CD}) + I_C + I_{CT} + \eta(I_{CD}+I_{CE})],\\
\lambda_{vD} &= \frac{\beta_v b_2(N_H,N_v)}{N_H}[(\eta_D(E_D+E_{DT})+\eta_{CD}E_{CD}) + I_D + I_{DT} + \eta(I_{CD}+I_{DE})].
\end{aligned}
\tag{3}
$$

In Eqns (3), β_C and β_D are the transmission probabilities of CHIKV and DENV respectively, and $\eta_C, \eta_D, \eta_{CD}, \eta_{vC}, \eta_{vD}, \eta_M, \eta < 1$ (and is positive) are the modification parameters accounting for the assumption that infected humans and mosquitoes are more infectious than exposed humans and mosquitoes, respectively [11], other parameters are defined in Table 2. Furthermore, $b_1(N_H, N_v)$ is the per capita biting rate of female adult mosquito on the human host per unit time. Similarly, $b_2(N_H, N_v)$ is the number of bites per adult mosquito per unit time. Following [6], the biting rates $b_1(N_H, N_v)$ and $b_2(N_H, N_v)$ are respectively given by,

$$
b_1(N_H,N_v) = \frac{\sigma_m\sigma_H N_v}{\sigma_m N_v + \sigma_H N_H}, \quad \text{and} \quad b_2(N_H,N_v) = \frac{\sigma_m\sigma_H N_H}{\sigma_m N_v + \sigma_H N_H}.
$$

2.1. **Basic properties.** The basic properties of the model Eqns (1)-(2) will now be explored. Let $\mu = \min\{\mu_A, \mu_v\}$. Consider the following equations for the rate of change of the total human and mosquito populations at time t.

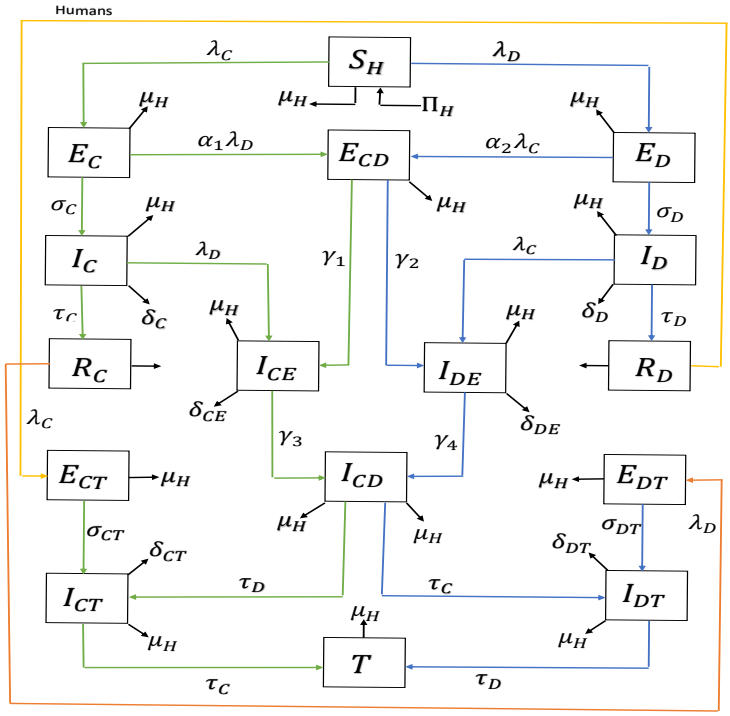

FIGURE 1. Schematic diagram of the model Eqn (1). The yellow line represent infection of CHIKV from the DENV recovered class, while the orange line represent infection of DENV from the CHIKV recovered class. The green and the blue lines represent CHIKV and DENV new infection as well as the recovery of individuals, all the parameters are defined in Table 2.

$$\begin{aligned}\frac{dN_H}{dt} =& \Pi_H - \mu_H N_H - \delta_C I_C - \delta_D I_D - \delta_{CD} I_{CD} - \delta_{CE} I_{CE} \\ & - \delta_{DE} I_{DE} - \delta_{CT} I_{CT} - \delta_{DT} I_{DT} \leq \Pi_H - \mu_H N_H,\end{aligned} \tag{4}$$

and

$$\frac{dN_v}{dt} = \Pi_v - \mu_A A - \mu_v N_I \leq \Pi_v - \mu N_v. \tag{5}$$

Furthermore, consider the region

$$\Omega = \Big\{(S_H, E_C, E_D, E_{CD}, I_C, I_D, I_{CE}, I_{DE}, I_{CD}, E_{CT}, E_{DT}, I_{CT}, I_{DT}, R_C, R_D, T,$$

$$A, S_v, E_{vC}, E_{vD}, E_M, I_{vC}, I_{vD}, I_{vCE}, I_{vDE}, I_{vM}) \in \mathbb{R}^{26}_+ : N_H \leq \tfrac{\Pi_H}{\mu_H} : N_v \leq \tfrac{\Pi_v}{\mu}\Big\}.$$

It can be shown, by solving for N_H and N_v in Eqns (4)-(5), that all solutions of the system starting in the region Ω will remain in Ω for all time t (≥ 0). Thus, the region Ω is positive-invariant, and it is sufficient to consider solutions restricted in Ω. In this region, the usual existence, uniqueness and continuation results hold for the model Eqns (1)-(2) [21, 43].

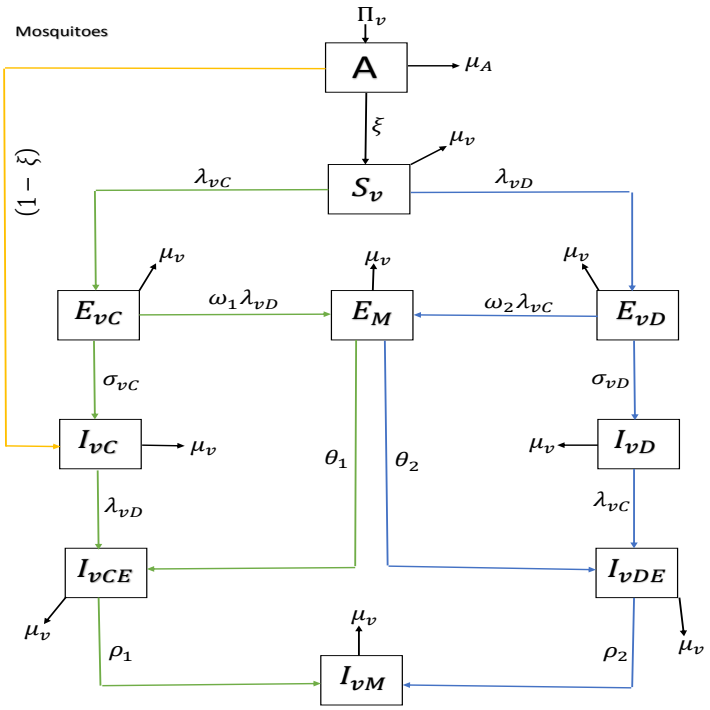

FIGURE 2. Schematic diagram of the model Eqns (2). The yellow line represent the vertical transmission of CHIKV virus from aquatic stage. The green and blue lines represent new infection of CHIKV and DENV, all the parameters are defined in Table 2.

3. **Analysis of sub-models.** It is instructive, however to analyse the sub-models (CHIKV only model and DENV only model) from the full model Eqns (1)-(2) first of all. This is done below.

3.1. **The CHIKV only sub-model.** The CHIKV only sub-model (obtained by setting $E_D = E_{CD} = I_D = I_{CE} = I_{DE} = I_{CD} = E_{CT} = E_{DT} = I_{CT} = I_{DT} = R_D = T = E_{vD} = E_M = I_{vD} = I_{vCE} = I_{vDE} = I_{vM} = 0$ in Eqns (1)-(2)) is given

TABLE 1. Interpretation of the compartmental variables of the model Eqns (1)-(2).

Variable	Interpretation/Description
N_H	Total population of humans
S_H	Population of susceptible humans
E_C	Population of asymptomatic CHIKV individuals
E_D	Population of asymptomatic DENV individuals
E_{CD}	Population of humans exposed to both CHIKV and DENV parasite
I_C	Population of CHIKV-infected (only) humans with clinical symptoms of CHIKV
I_D	Population of DENV-infected (only) humans with clinical symptoms of DENV
I_{CD}	Population of dually-infected humans with symptoms of both CHIKV and DENV
I_{CE}	Population of CHIKV-infected humans with clinical symptoms of CHIKV but exposed to DENV
I_{DE}	Population of DENV-infected humans with clinical symptoms of DENV but exposed to CHIKV
R_C	Recovered CHIKV-infected humans
R_D	Recovered DENV-infected humans
E_{CT}	Population of individuals exposed to CHIKV but recovered from DENV with permanent immunity
E_{DT}	Population of individuals exposed to DENV but recovered from CHIKV with permanent immunity
I_{CT}	Population of CHIKV-infected individuals with clinical symptoms of CHIKV but recovered from DENV with permanent immunity
I_{DT}	Population of DENV-infected individuals with clinical symptoms of DENV but recovered from CHIKV with permanent immunity
T	Population of individuals who recovered from both CHIKV and DENV with permanent immunity
N_V	Total population of mosquitoes
A	population of immature mosquitoes (egg, lava and pupa stages)
N_I	Total population of adult mosquitoes
S_v	population of adult mosquitoes susceptible to both CHIKV and DENV
E_{vC}	population of adult mosquitoes exposed to CHIKV
E_{vD}	population of adult mosquitoes exposed to DENV
E_M	population of adult mosquitoes exposed to both CHIKV and DENV viruses
I_{vC}	Population of CHIKV-infected (only) adult mosquitoes
I_{vD}	Population of DENV-infected (only) adult mosquitoes
I_{vCE}	Population of CHIKV-infected adult mosquitoes that are exposed to DENV
I_{vDE}	Population of DENV-infected adult mosquitoes that are exposed to CHIKV
I_{vM}	Population of adult mosquitoes infected to both CHIKV and DENV

by

$$\begin{aligned}
\frac{dS_H}{dt} &= \Pi_H - \lambda_C S_H - \mu_H S_H,\\
\frac{dE_C}{dt} &= \lambda_C S_H - (\sigma_C + \mu_H)E_C,\\
\frac{dI_C}{dt} &= \sigma_C E_C - (\tau_C + \delta_C + \mu_H)I_C,\\
\frac{dR_C}{dt} &= \tau_C I_C - \mu_H R_C,\\
\frac{dA}{dt} &= \Pi_v - (1+\mu_A)A,\\
\frac{dS_v}{dt} &= \xi A - \lambda_{vC} S_v - \mu_v S_v,\\
\frac{dE_{vC}}{dt} &= \lambda_{vC} S_v - (\sigma_{vC} + \mu_v)E_{vC},\\
\frac{dI_{vC}}{dt} &= (1-\xi)A + \sigma_{vC} E_{vC} - \mu_v I_{vC},
\end{aligned} \tag{6}$$

where,

$$\begin{aligned}
\lambda_C &= \frac{\beta_C \sigma_m \sigma_H}{\sigma_m N_v + \sigma_H N_H}(\eta_{vC} E_{vC} + I_{vC}),\\
\lambda_{vC} &= \frac{\beta_v \sigma_m \sigma_H}{\sigma_m N_v + \sigma_H N_H}(\eta_C E_C + I_C),
\end{aligned} \tag{7}$$

with $N_H = S_H + E_C + I_C + R_C$, and $N_v = A + S_v + E_{vC} + I_{vC}$.
Consider the region

$$\Omega_C = \left\{(S_H, E_C, I_C, R_C, A, S_v, E_{vC}, I_{vC}) \in \mathbb{R}^8_+ : N_H \leq \tfrac{\Pi_H}{\mu_H}, N_v \leq \tfrac{\Pi_v}{\mu}\right\},$$

it can be shown, as in section 2, that the region Ω_C is positively-invariant.

Let $R_{NC} = \frac{\Pi_v}{\mu_v(1+\mu_A)}$ be the threshold quantity which accounts for eggs production by female adult mosquito.

The CHIKV only sub-model (6) has two disease-free equilibria, namely the trivial disease-free equilibrium (denoted by TDFE) and a non-trivial disease-free equilibrium (denoted by NDFE) as described below.

(i) $R_{NC} \leq 1$, the CHIKV only sub-model (6) has a TDFE (where no mosquito exist) given by: $\Upsilon_{0C} = (S_H^*, E_C^*, I_C^*, R_C^*, A^*, S_v^*, E_{vC}^*, I_{vC}^*) = (\frac{\Pi_H}{\mu_H}, 0, ..., 0)$.

(ii) $R_{NC} > 1$, the CHIKV only sub-model (6) has a disease-free (mosquito present) equilibrium (NDFE) given by:

$\varepsilon_{0C} = (S_H^*, E_C^*, I_C^*, R_C^*, A^*, S_v^*, E_{vC}^*, I_{vC}^*) = (\frac{\Pi_H}{\mu_H}, 0, 0, 0, \frac{\Pi_v}{(1+\mu_A)}, \frac{\xi(\Pi_v)}{\mu_v(1+\mu_A)}, 0, 0)$.

3.2. **Asymptotic stability of disease-free equilibrium.**

3.2.1. *TDFE.* The following Theorem 3.1 is established and the proof is given in Appendix A1.

Theorem 3.1. *The TDFE of the model (6), denoted by Υ_{0C}, is GAS in Ω whenever $R_{NC} \leq 1$.*

It should be noted that the mosquito-free equilibrium,Υ_{0C}, is ecologically unrealistic, since mosquitoes commonly exist in the CHIKV-endemic regions of interest.

3.2.2. *NDFE.* The linear stability of ε_{0C} will be investigated using the next generation matrix method on the system (6) [44]. It follows that, the basic reproduction number of the CHIKV only sub-model is given by

$$\mathcal{R}_{0C} = \sqrt{\frac{\xi\Pi_v\Pi_H\beta_v\beta_C\mu_H(\eta_v\mu_v+\sigma_v)(\eta_C g_2+\sigma_C)\sigma_H^2\sigma_m^2\mu^2}{g_1g_2g_3\mu_v^2(1+\mu_A)(\mu_H\sigma_m\Pi_v+\mu_v\sigma_H\Pi_H)^2}}, \tag{8}$$

where $g_1 = \sigma_C + \mu_H$, $g_2 = \tau_C + \delta_C + \mu_H$, $g_3 = \sigma_{vC} + \mu_v$.

Lemma 3.2. *The DFE (ε_{0C}), of the CHIKV only sub-model (6) is locally asymptotically stable (LAS) if $\mathcal{R}_{0C} < 1$, and unstable if $\mathcal{R}_{0C} > 1$.*

The threshold quantity $\mathcal{R}_{0C}$ is the basic reproduction number for CHIKV only sub-model. It represents the average number of secondary cases that one infectious individuals (or mosquitoes) would generate over the duration of the infectious period if introduced into a completely susceptible population.

3.2.3. *Endemic equilibria and backward bifurcation.* The endemic equilibrium (EE) of the model (6) in terms of the forces of infections is given by

$$S_H^* = \frac{\Pi_H}{\lambda_C^* + \mu_H}, \quad E_C^* = \frac{\lambda_C^*\Pi_H}{g_1(\lambda_C^* + \mu_H)}, \quad I_C^* = \frac{\sigma_C\lambda_C^*\Pi_H}{g_1g_2(\lambda_C^* + \mu_H)},$$
$$R_C^* = \frac{\tau_C\sigma_C\lambda_C^*\Pi_H}{g_1g_2\mu_H(\lambda_C^* + \mu_H)}, \quad A^* = \frac{\Pi_v}{1+\mu}, \quad S_v^* = \frac{\xi\Pi_v}{(1+\mu)(\lambda_{vC}^* + \mu)},$$
$$E_{vC}^* = \frac{\xi\lambda_{vC}^*\Pi_v}{g_3(1+\mu)(\lambda_{vC}^* + \mu)}, \quad I_{vC}^* = \frac{\Pi_v(\sigma_{vC}\lambda_{vC}\xi + g_3(1-\xi)(\lambda_{vC}+\mu))}{\mu(1+\mu)g_3(\lambda_{vC}^* + \mu)}.$$

We used the center manifold theory to examined the conditions on the parameter values in the model (6) that cause forward or backward bifurcation to occur [3, 4]. The following Theorem is established and the proof is given in Appendix A2.

Theorem 3.3. *The CHIKV only model (6) undergoes backward bifurcation (BB) at $\mathcal{R}_{0C} = 1$ whenever the bifurcation coefficient, a^*, given by Eqn (A2.4) in Appendix A2, is positive, i.e., $a^* > 0$.*

The public health significance of the phenomenon of backward bifurcation is that the classical requirement of $\mathcal{R}_{0C} < 1$, although necessary, is no longer sufficient for effective disease control (or elimination). In such a backward bifurcation (BB) scenario, effective disease control or elimination would depend on the initial sizes of the sub-populations (state variables) of the model (6). In other words, the presence of backward bifurcation in CHIKV only transmission dynamics makes its effective (population-wide) control more difficult.

3.3. **The DENV only sub-model.** The DEN only sub-model can be obtained by setting $E_C = E_{CD} = I_C = I_{CE} = I_{DE} = I_{CD} = E_{CT} = E_{DT} = I_{CT} = I_{DT} = R_C = T = E_{vC} = E_M = I_{vC} = I_{vCE} = I_{vDE} = I_{vM} = 0$ in the model Eqns (1)-(2) is given by

$$\begin{aligned}
\frac{dS_H}{dt} &= \Pi_H - \lambda_D S_H - \mu_H S_H,\\
\frac{dE_D}{dt} &= \lambda_D S_H - (\sigma_D + \mu_H) E_D,\\
\frac{dI_D}{dt} &= \sigma_D E_D - (\tau_D + \delta_D + \mu_H) I_D,\\
\frac{dR_D}{dt} &= \tau_D I_D - \mu_H R_D,\\
\frac{dA}{dt} &= \Pi_v - \xi A - \mu_A A,\\
\frac{dS_v}{dt} &= \xi A - \lambda_{vD} S_v - \mu_v S_v,\\
\frac{dE_{vD}}{dt} &= \lambda_{vD} S_v - (\sigma_{vD} + \mu_v) E_{vD},\\
\frac{dI_{vD}}{dt} &= \sigma_{vD} E_{vD} - \mu_v I_{vD},
\end{aligned} \tag{9}$$

where

$$\begin{aligned}
\lambda_D &= \frac{\beta_D \sigma_m \sigma_H}{\sigma_m N_v + \sigma_H N_H}(\eta_{vD} E_{vD} + I_{vD})\\
\lambda_{vD} &= \frac{\beta_v \sigma_m \sigma_H}{\sigma_m N_v + \sigma_H N_H}(\eta_D E_D + I_D),
\end{aligned} \tag{10}$$

with, $N_H = S_H + E_D + I_D + R_D$ and $N_v = A + S_v + E_{vD} + I_{vD}$.
Consider the region

$$\Omega_D = \left\{(S_H, E_D, I_D, R_D, A, S_v, E_{vD}, I_{vD}) \in \mathbb{R}^8_+ : N_H \leq \tfrac{\Pi_H}{\mu_H}, A \leq \tfrac{\Pi_v}{\mu_A + \xi}, N_I \leq \tfrac{\xi}{\mu_v}\right\},$$

it can be shown, as in section 2, that the region Ω_D is positively-invariant. It is convenient to define the threshold quantity $R_{ND} = \frac{\xi \Pi_v}{\mu_v(\xi + \mu_A)}$ that accounts for eggs production by female adult mosquito.

The DENV only sub-model (9) has two disease-free equilibria, namely the trivial disease-free equilibrium (denoted by TDFE) and a non-trivial disease-free equilibrium (denoted by NDFE) as described below.

(i) If $R_{ND} \leq 1$, the DENV only sub-model (9) has a TDFE (where no mosquito exist) given by: $\Upsilon_{0D} = (S_H^*, E_D^*, I_D^*, R_D^*, A^*, S_v^*, E_{vD}^*, I_{vD}^*) = (\frac{\Pi_H}{\mu_H}, 0, ..., 0)$.

(ii) If $R_{ND} > 1$, the DENV only sub-model (9) has a disease-free (mosquito present) equilibrium (NDFE) given by:
$\varepsilon_{0D} = (S_H^*, E_D^*, I_D^*, R_D^*, A^*, S_v^*, E_{vD}^*, I_{vD}^*) = (\frac{\Pi_H}{\mu_H}, 0, 0, 0, \frac{\Pi_v}{(\mu+\xi)}, \frac{\xi(\Pi_v)}{\mu(\mu+\xi)}, 0, 0)$.

3.4. **Asymptotic stability of disease-free equilibrium.**

3.4.1. *TDFE.* The following theorem is established, and the proof is given in Appendix A3.

Theorem 3.4. *The TDFE of the model (9), denoted by Υ_{0D}, is GAS in Ω whenever $R_{ND} \leq 1$.*

3.4.2. *NDFE.* Let $R_{ND} > 1$ (so that the NDFE, ε_{0D}, exists). The linear stability of ε_{0D} will be investigated using the next generation matrix method on the system (9) [44]. It follows that, the basic reproduction number of the DENV only sub-model is given by using the same approach as in the case of CHIKV only sub-model

$$\mathcal{R}_{0D} = \sqrt{\frac{\xi\Pi_v\Pi_H\beta_v\beta_D\mu_H(\eta_v\mu_v+\sigma_v)(\eta_D h_2+\sigma_D)\sigma_H^2\sigma_m^2}{h_1h_2h_3m(\mu_H\sigma_m\Pi_v+\mu\sigma_H\Pi_H)^2}}, \tag{11}$$

where $h_1 = \sigma_D + \mu_H$, $h_2 = \tau_D + \delta_D + \mu_H$, $h_3 = \sigma_{vD} + \mu$, $m = \xi + \mu$.

Using theorem 2 of [44], the following result is established.

Lemma 3.5. *The DFE (ε_{0D}), of the DENV only sub-model (9) is locally asymptotically stable (LAS) if $\mathcal{R}_{0D} < 1$, and unstable if $\mathcal{R}_{0D} > 1$.*

3.4.3. *Endemic equilibria and backward bifurcation.* The endemic equilibrium (EE) of the model (9) in terms of the forces of infections is given by

$$\begin{aligned}
S_H^* &= \frac{\Pi_H}{\lambda_D^*+\mu_H}, \quad E_D^* = \frac{\lambda_D^*\Pi_H}{h_1(\lambda_D^*+\mu_H)}, \quad I_D^* = \frac{\sigma_D\lambda_D^*\Pi_H}{h_1h_2(\lambda_D^*+\mu_H)},\\
R_D^* &= \frac{\tau_D\sigma_D\lambda_D^*\Pi_H}{h_1h_2\mu_H(\lambda_D^*+\mu_H)}, \quad A^* = \frac{\Pi_v}{m}, \quad S_v^* = \frac{\xi\Pi_v}{m(\lambda_v^*+\mu)},\\
E_{vD}^* &= \frac{\xi\lambda_{vD}^*\Pi_v}{h_3m(\lambda_v^*+\mu)}, \quad I_{vD}^* = \frac{\xi\sigma_{vD}\lambda_v^*\Pi_v}{h_3\mu m(\lambda_v^*+\mu)}.
\end{aligned} \tag{12}$$

We substitute Eqn (12) into Eqn (10), and after simplification, we have

$$\begin{aligned}
\lambda_D^* = [\xi\beta_D\sigma_m\sigma_H\Pi_v h_1h_2\mu_H\lambda_v^*(\eta_{vD}\mu+\sigma_{vD})(\lambda_D^*+\mu_H)]\cdot[(\lambda_v^*+\mu)mh_3]^{-1}\cdot\\
[[\sigma_H\Pi_H\mu(h_1h_2\mu_H+\lambda_D^*(h_2\mu_H+\sigma_D\mu_H+\sigma_D\tau_D))+\sigma_m\Pi_v h_1h_2\mu_H(\lambda_C^*+\mu_H)]]^{-1},
\end{aligned} \tag{13}$$

and

$$\lambda_v^* = \frac{\beta_v\sigma_m\sigma_H\Pi_H\mu\mu_H(\eta_D h_2+\sigma_D)\lambda_D^*}{[\sigma_H\Pi_H\mu(h_1h_2\mu_H+\lambda_D^*(h_2\mu_H+\sigma_D\mu_H+\sigma_D\tau_D))+\sigma_m\Pi_v\mu_H h_1h_2(\lambda_D^*+\mu_H)]}. \tag{14}$$

By substituting Eqn (14) into Eqn (13), it can be shown that the non-zero equilibria of the model satisfy the following quadratic equation (in terms of λ_D^*)

$$a_1(\lambda_D^*)^2 + a_2\lambda_D^* + a_3 = 0, \tag{15}$$

where
$a_1 = m^2(\sigma_H^2\Pi_H^2\mu^2(h_2\mu_H+\sigma_D\mu_H+\sigma_D\tau_D)^2+2\sigma_m\sigma_H\Pi_v\Pi_H\mu\mu_H h_1h_2(h_2\mu_H+\sigma_D\mu_H+\sigma_D\tau_D)+\ \sigma_m^2\Pi_v^2h_1^2h_2^2\mu_H^2+\beta_v\sigma_m\sigma_H^2\Pi_H^2\mu\mu_H(\eta_D h_2+\sigma_D)(h_2\mu_H+\sigma_D\mu_H+\sigma_D\tau_D)$
$+\beta_v\sigma_m^2\sigma_H\Pi_H\Pi_v\mu_H^2h_1h_2(\eta_D h_2+\sigma_D))$,
$a_2 = m^2(2\sigma_H^2\Pi_H^2\mu^2\mu_H h_1h_2h_3(h_2\mu_H+\sigma_D\mu_H+\sigma_D\tau_D)+2\sigma_m\sigma_H\Pi_v\Pi_H\mu\mu_H^2h_1^2h_2^2h_3+$

$2\sigma_m\sigma_H\Pi_v\Pi_H\mu\mu_H^2h_1h_2h_3(h_2\mu_H+\sigma_D\mu_H+\sigma_D\tau_D)+2\sigma_m^2\Pi_v^2\mu_H^3h_1^2h_2^2h_3+$
$\beta_v\sigma_m\sigma_H^2\Pi_H^2\mu\mu_H^2h_3(\eta_Dh_2+\sigma_D)+\beta_v\sigma_m^2\sigma_H^2\Pi_H\Pi_v\mu_H^3h_1h_2h_3(\eta_Dh_2+\sigma_D)-$
$(\mu_Hh_1^2h_2^2h_3)(\mu_H\sigma_m\Pi_v+\mu\sigma_H\Pi_H)^2\mathcal{R}_{0D}^2),$
$a_3=[m^2h_1^2h_2^2h_3\mu_H^2(\mu_H\sigma_m\Pi_v+\mu\sigma_H\Pi_H)^2(1-\mathcal{R}_{0D}^2)].$

Hence, we obtained a positive endemic equilibria of the model (9) by solving equation (12) for λ_D^* and substituting the positive values of λ_D^* into the expression in equation (15). Clearly, the coefficient a_1, of Eqn (15), is always positive, and a_3 is positive (negative) if $\mathcal{R}_{0D}$ is less than (greater than) unity, respectively. Since $a_1>0$, the existence of the positive solutions of quadratic equation (15) will depend on the signs of a_2 and a_3. If $\mathcal{R}_{0D}>1$, then $a_3<0$ and Eqn (15) has only positive solution. Thus, there is unique EE whenever $\mathcal{R}_{0D}>1$. If $\mathcal{R}_{0D}=1$ then $a_3=0$ and Eqn (15) has a unique nonzero solution of $\lambda_D^*=-\frac{a_2}{a_1}$, which is positive if and only if $a_2<0$ and negative solution if $a_2>0$. Therefore, no EE exits if $\mathcal{R}_{0D}=1$ and $a_2>0$. The case $\mathcal{R}_{0D}<1$ makes $a_3>0$. If $a_2<0$, equation (15) has two positive solutions, given by

$$\lambda_D^*=-\frac{a_2+\sqrt{a_2^2-4a_1a_3}}{2a_1},\ \text{and}\quad \lambda_D^*=-\frac{a_2-\sqrt{a_2^2-4a_1a_3}}{2a_1}. \tag{16}$$

Theorem 3.6. *The DENV model (9) has*
i) *a unique EE, if* $a_3<0\iff\mathcal{R}_{0D}>1$*;*
ii) *a unique EE, if* $a_2<0$ *when* $a_3=0$ *or* $a_2^2-4a_1a_3=0$*;*
iii) *two EEs if,* $a_3>0$*,* $a_2<0$ *and* $a_2^2-4a_1a_3>0$*; or*
iv) *no EE, otherwise.*

It is obvious from Theorem 3.6 (Case i) that the model has a unique EE when $\mathcal{R}_{0D}>1$. Further, Case (iii) indicates the possibility of backward bifurcation (where the locally-asymptotically stable DFE co-exists with a locally-asymptotically stable EE when $\mathcal{R}_{0D}<1$) in the model (9) when $\mathcal{R}_{0D}<1$. To check for this, we set the discriminant $(a_2^2-4a_1a_3)$ to zero and simplify for the critical value of $\mathcal{R}_{0D}$, denoted by $\mathcal{R}_c$, given by

$$\mathcal{R}_c=\sqrt{1-\frac{a_2^2}{4a_1[h_1^2h_2^2h_3\mu_H^2m^2(\mu_H\sigma_m\Pi_v+\mu\sigma_H\Pi_H)^2]}}. \tag{17}$$

Thus, the BB would occur for values of $\mathcal{R}_{0D}$ such that $\mathcal{R}_c<\mathcal{R}_{0D}<1$. This is illustrated by simulating the model with the following set of parameter values. Note that these parameters are chosen for illustrative purpose only, and may not necessarily be true epidemiologically: $\Pi_H=20$, $\Pi_v=40$, $\mu_H=0.000004$, $\mu=0.00099$, $\delta_D=2.6$, $\delta_v=0.2$, $\sigma_m=0.3$, $\sigma_H=0.011$, $\sigma_v=0.9$, $\sigma_D=0.303$, $\tau_D=0.47$, $\beta_D=0.78$ and $\beta_v=0.398$, $\eta_D=0.266$, $\eta_v=0.2$ (see Table 3, for the units of the parameters). Hence, $\mathcal{R}_c=0.8990<1$ and $\mathcal{R}_{0D}=0.9552<1$ so that $\mathcal{R}_c<\mathcal{R}_{0D}<1$. Fig. 3 shows the backward bifurcation diagram of the model (9).

Lemma 3.7. *The model (9) undergoes BB when case (iii) of Theorem 3.6 holds and* $\mathcal{R}_c<\mathcal{R}_{0D}<1$.

Although, the BB has been first shown to exist in DENV by Garba *et al.* [16], and the causes have been explained in detail by Gumel [19]. Thus, the analysis in this section show that the BB property of the DENV only sub-model (9) can be removed whenever $\mathcal{R}_{0D}=1$.

4. Analysis of CHIKV-DENV co-infection model.

Having analyzed the dynamics of the two sub-models, the full model Eqns (1)-(2) is now considered.

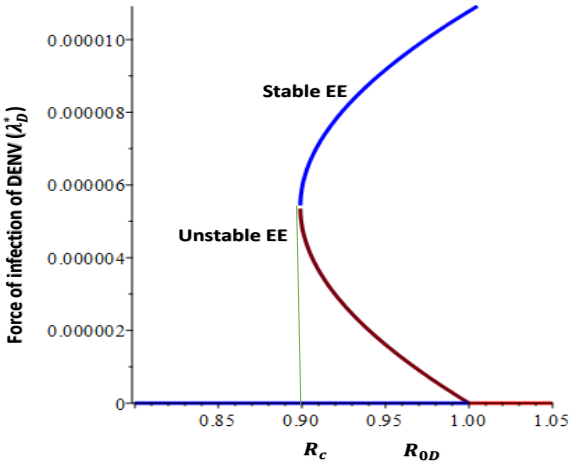

FIGURE 3. Backward bifurcation diagram of the model (9).

4.1. **Asymptotic stability of DFE.**

4.1.1. *TDFE.* Let the threshold quantity be given by $R_{NCD} = \frac{\xi\Pi_v}{\mu_v(1+\mu_A)}$. The full model Eqns (1)-(2) has two disease-free equilibria, namely the trivial disease-free equilibrium (denoted by TDFE) and a non-trivial disease-free equilibrium (denoted by NDFE) as described below:

(i) $R_{NCD} \leq 1$, the full model Eqns (1)-(2) has a TDFE (where no mosquito exist) given by

$$\Upsilon_{0CD} = (S_H^*, E_C^*, E_D^*, E_{CD}^*, E_{CT}^*, E_{DT}^*, I_C^*, I_D^*, I_{CE}^*, I_{DE}^*, I_{CD}^*, I_{CT}^*, I_{DT}^*, R_C^*, R_D^*, T,$$
$$A, S_v^*, E_{vC}^*, E_{vD}^*, E_M^*, I_{vC}^*, I_{vD}^*, I_{vCE}^*, I_{vDE}^*, I_{vM}^*) = \left(\frac{\Pi_H}{\mu_H}, 0, ..., 0\right) \subset \mathbb{R}_+^{26}.$$

(ii) $R_{NCD} > 1$, the full model Eqns (1)-(2) has a disease-free (mosquito present) equilibrium (NDFE) given by

$$\begin{aligned}\varepsilon_{0CD} =& (S_H^*, E_C^*, E_D^*, E_{CD}^*, E_{CT}^*, E_{DT}^*, I_C^*, I_D^*, I_{CE}^*, I_{DE}^*, I_{CD}^*, I_{CT}^*, I_{DT}^*, R_C^*, R_D^*, T,\\ & A, S_v^*, E_{vC}^*, E_{vD}^*, E_M^*, I_{vC}^*, I_{vD}^*, I_{vCE}^*, I_{vDE}^*, I_{vM}^*)\\ =& \left(\frac{\Pi_H}{\mu_H}, 0, ..., 0, \frac{\Pi_v}{1+\mu_A}, \frac{\xi\Pi_v}{\mu_v(1+\mu_A)}, 0, ..., 0\right) \subset \mathbb{R}_+^{26}.\end{aligned}$$

The following theorem is established, and the proof is given in Appendix A4.

Theorem 4.1. *The TDFE of the full model Eqns (1)-(2), denoted by Υ_{0CD}, is GAS in Ω whenever $R_{NCD} \leq 1$.*

4.1.2. *NDFE.* The CHIKV-DENV co-infection model Eqns (1)-(2) has a NDFE, given by,

$$\begin{aligned}\varepsilon_{0CD} =&(S_H^*, E_C^*, E_D^*, E_{CD}^*, E_{CT}^*, E_{DT}^*, I_C^*, I_D^*, I_{CD}^*, I_{CE}^*, I_{DE}^*, I_{CT}^*, I_{DT}^*, R_C^*, R_D^*, T,\\ & A, S_v^*, E_{vC}^*, E_{vD}^*, E_M^*, I_{vC}^*, I_{vD}^*, I_{vCE}^*, I_{vDE}^*, I_{vM}^*)\\ =&\left(\frac{\Pi_H}{\mu_H}, 0, ..., 0, \frac{\Pi_v}{1+\mu_A}, \frac{\xi\Pi_v}{\mu_v(1+\mu_A)}, 0..., 0\right) \subset \mathbb{R}_+^{26}.\end{aligned}$$

Following [20, 35], the basic reproduction number of the CHIKV-DENV model Eqns (1)-(2), denoted by,

$$\mathcal{R}_{0CD} = \max\left(\mathcal{R}_{0C}, \mathcal{R}_{0D}\right),$$

where $\mathcal{R}_{0C}$ and $\mathcal{R}_{0D}$ are defined in section 3. Using Theorem 2 of [44], the following result is established.

Lemma 4.2. *The NDFE, (ε_{0CD}), of the CHIKV-DENV model in Eqns (1)-(2) is locally asymptotically stable (LAS) if $\mathcal{R}_{0CD} < 1$, and unstable if $\mathcal{R}_{0CD} > 1$.*

The threshold quantity $\mathcal{R}_{0CD}$ is the basic reproduction number of the CHIKV-DENV co-infection model Eqns (1)-(2).

TABLE 2. Interpretation of the parameters of the model Eqns (1)-(2).

Parameter	Interpretation/Description
Π_H, Π_V	Recruitment rate of humans and mosquitoes, respectively
μ_H	Natural death rate of humans
μ_A	Death rate of immature mosquitoes
μ_v	Death rate of adult mosquitoes
λ_C	Rates of CHIKV force of infection in humans
λ_D	Rates of DENV force of infection in humans
λ_{vC}	Rates of CHIKV force of infection in mosquitoes
λ_{vD}	Rates of DENV force of infection in mosquitoes
β_C	Transmission probability for CHIKV to humans
β_D	Transmission probability for DENV to humans
β_v	Transmission probability from an infectious human to a susceptible adult mosquitoes
b_1	Number of bites per human per unit time
b_2	Number of bites per mosquitoes per unit time
ξ	Fraction of immature mosquitoes becoming susceptible adult
α_1	Modification parameter for the heterogeneity of DENV infection between susceptible humans and humans exposed to CHIKV
α_2	Modification parameter for the heterogeneity of CHIKV infection between susceptible humans and humans exposed to DENV
ω_1	Modification parameter for the heterogeneity of DENV infection between susceptible adult mosquitoes and those exposed to CHIKV
ω_2	Modification parameter for the heterogeneity of CHIKV infection between susceptible adult mosquitoes and those exposed to DENV
σ_m	Number of times a mosquito bites humans *per* unit time
σ_H	Maximum number of mosquito bites a human can receive *per* unit time
σ_C	Progression rate of humans from exposed state of CHIKV to the infectious state of CHIKV
σ_D	Progression rate of humans from exposed state of DENV to the infectious state of DENV
σ_{vC}	Progression rate of adult mosquitoes from exposed state of CHIKV to the infectious state of CHIKV
σ_{vD}	Progression rate of adult mosquitoes from exposed state of DEN to the infectious state of DENV
$\gamma_i (i = 1, 4)$	Progression rates of humans to active CHIKV classes
$\gamma_j (j = 2, 3)$	Progression rates of humans to active DENV classes
θ_1, ρ_2	Progression rates of adult mosquitoes to active CHIKV classes
θ_2, ρ_1	Progression rates of adult mosquitoes to active DENV classes
τ_C	Recovery rate of humans from infectious state of CHIKV to the recovered state of CHIKV
τ_D	Recovery rate of humans from infectious state of DENV to the recovered state of DENV
η	Modification parameters for the increase in infectiousness of dually-infected humans in comparison to mono-infected humans
$\eta_C, \eta_D, \eta_{CD}, \eta_{vC}, \eta_{vD}, \eta_M$	Modification parameters for the increase in infectiousness for the exposed classes in humans and mosquitoes, respectively
$\delta_C, \delta_D, \delta_{CE}, \delta_{DE}, \delta_{CD}, \delta_{CT}, \delta_{DT}$	Disease-induced death rates for humans

5. Numerical results and sensitivity Analysis.

5.1. **Model fittings.** We fitted each of the sub-model and the full model to the data using the Pearson's Chi-square and the least square methods (using the R statistical software) [21, 35]. Firstly, each of the sub-models (6) and (9) were fitted to the cumulative number of human cases from 2010-2017 (see Table A1 for the number of CHIKV and DENV cases) using the data obtained from National Vector Borne Disease Control Programme (NVBDCP) India [31]. The demographic time series are obtained from World Bank [41]. The demographic parameters (e.g., Π_H and μ_H) are given by the average number of population in India which is given by

TABLE 3. Values and ranges of the parameters of the model Eqns (1)-(2).

Parameter	Baseline; (Range)	Unit	Source(s)
μ_H	3.9×10^{-5}; $(3.6, 4.0) \times 10^{-5}$	day^{-1}	[1, 34]
μ_v	0.05714; $(0.01, 0.1)$	day^{-1}	[37]
μ_A	0.174; $(0.0143, 0.33)$	day^{-1}	[23, 50]
β_C	0.375; $(0.001, 0.54)$	day^{-1}	[8, 42]
β_D	0.75; $(0.1, 0.95)$	day^{-1}	[26]
β_v	0.375; $(0.1, 0.5)$	day^{-1}	[8, 9, 27, 36]
σ_m	0.5; $(0.33, 1)$	day^{-1}	[7, 26]
σ_H	1; $(0.1, 10)$	day^{-1}	[16]
σ_C	0.35; $(0, 1)$	day^{-1}	Estimated [1, 16]
σ_D	0.5; $(0, 1)$	day^{-1}	Estimated [16]
σ_{vC}	0.25; $(0.1, 1)$	day^{-1}	Estimated [16]
$\sigma_{vD}, \sigma_{CT}, \sigma_{DT}$	0.2; $(0, 1)$	day^{-1}	Estimated [16]
τ_C	0.2(0.1429, 0.3333)	day^{-1}	Estimated [15]
τ_D	0.25; $(0.01, 0.3)$	day^{-1}	[15]
Π_H	2.5; $(1, 5)$	day^{-1}	[16]
Π_v	5000; $(2500, 6000)$	day^{-1}	[16]
γ_1	0.23; $(0, 1)$	Dimensionless	Assumed
γ_2	0.25; $(0, 1)$	Dimensionless	Assumed
γ_3	0.3; $(0, 1)$	Dimensionless	Assumed
γ_4	0.23; $(0, 1)$	Dimensionless	Assumed
δ_C, δ_D	1×10^{-3}; $(0.0005, 0.0015)$	day^{-1}	[16]
δ_{CE}, δ_{DE}	1.5×10^{-3}; $(0.00051, 0.0015)$	day^{-1}	Assumed
δ_{CD}, δ_{CT}, δ_{DT}	1.2×10^{-3}; $(0.0005, 0.002)$	day^{-1}	Assumed
η_C,η_M	0.1; $(0, 1)$	Dimensionless	Estimated [16]
η_{CD},η_{vC},η_{vD}	0.1; $(0, 1)$	Dimensionless	Estimated [16]
η,η_D	0.12; $(0, 1)$	Dimensionless	Assumed
ξ	0.01; $(0.001, 0.021)$	Dimensionless	Estimated [8]
α_1	0.03; $(0.15, 0.99)$	Dimensionless	Estimated
α_2	0.018; $(0.10, 1)$	Dimensionless	Estimated
ω_1	0.015; $(0, 1)$	Dimensionless	Assumed
ω_2	0.013; $(0, 0.9)$	Dimensionless	Assumed
θ_1	0.01; $(0.005, 0.016)$	Dimensionless	Assumed
θ_2, ρ_1	0.01; $(0.005, 0.018)$	Dimensionless	Assumed
ρ_2	0.01; $(0.0051, 0.01)$	Dimensionless	Assumed

1,143,638,692, and the average life expectancy in India is 59.4 years [40]. Therefore, the term $\mu_H^{-1} = 59.4$ years, implies that $\frac{\Pi_H}{\mu_H} = 1,143,638,692$, so that $\Pi_H = 52,748$ per day. Note that all other parameters are fixed as in Table 3. Figs 4 and 5 show the fitting results of the models (6) and (9), respectively.

Secondly, the full model Eqns (1)-(2) is also fitted to the human cases from 2007-2012 (see Table A2 for the number of CHIKV-DENV co-infection cases), using the data obtained from [13]. The demographic time series are obtained from World Bank [41]. The demographic parameters (e.g., Π_H and μ_H) are given by the average number of population in India which is given by 1,222,063,475, and the average life expectancy in India is 66.4 years [40]. Therefore, term $\mu_H^{-1} = 66.4$ years), implies

that $\frac{\Pi_H}{\mu_H} = 1,222,063,475$, so that $\Pi_H = 50,423$ per day. Note that all other parameters are fixed as in Table 3. Fig. 6 show the fitting results of the full model Eqns (1)-(2).

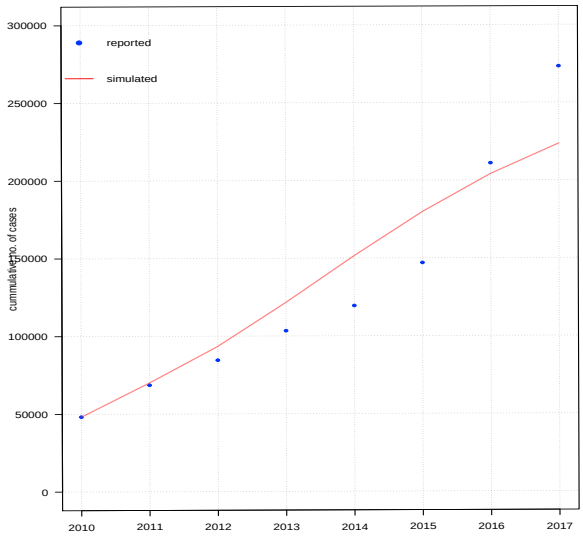

FIGURE 4. Fitting result of the CHIKV only sub-model (6). We used the parameter values from Table 3 and the following initial conditions: $S_H(0) = 1.3 \times 10^9$, $E_C(0) = 1.2 \times 10^5$, $I_C(0) = 48176$, $R_C(0) = 4000$, $A(0) = 8 \times 10^7$, $S_v(0) = 5 \times 10^7$, $E_{vC}(0) = 2 \times 10^5$ and $I_{vC}(0) = 10^4$. The vertical axes indicate the cumulative number of CHIKV cases in India from 2010 to 2017.

5.2. **Sensitivity analysis.** Sensitivity analysis is used in order to establish what factors affect both sub-models and the full model outcomes. A partial rank correlation coefficient (PRCC), a method of conducting sensitivity analysis adapted from [15, 29], is used for ranking the importance of each parameter-output. This gives an insight into designing a meaningful control strategy.

Firstly, 5,000 random samples are taken for each model parameter from uniform distributions using the parameter ranges of values in Table 3. Each sub-model is simulated for each random parameter values to obtain the target biological quantities (in this case, the basic reproduction number and the infection attack rate). Further, the PRCCs were computed between each parameter and target biological quantities. The PRCCs results highlighted that mosquito removal rates, i.e., μ_v and μ_A, the transmission rates, i.e., β_v, β_C, and β_D, and the mosquito maturation rate, i.e., ξ, are the top control parameters for combating CHIKV, DENV and CHIKV-DENV co-infection outbreaks.

6. **Discussion.** A CHIKV and DENV co-infection model is constructed and used to assess the impact of the co-endemicity of these diseases on the transmission dynamics of each disease. In each of the CHIKV and DENV sub-models and the full

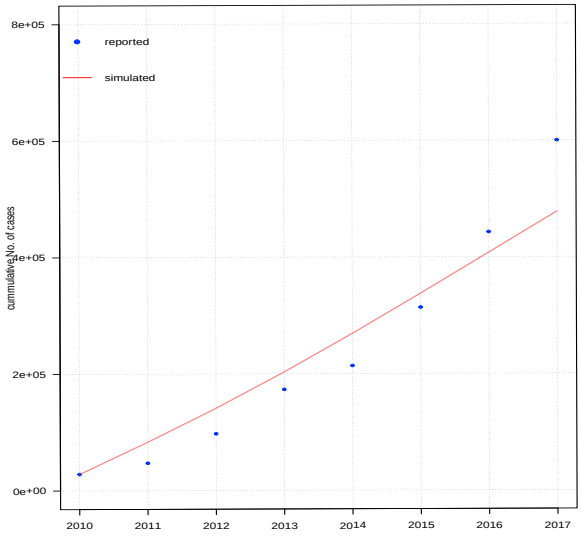

FIGURE 5. Fitting result of the DENV only sub-model (9). We used the parameter values from Table 3 and the following initial conditions: $S_H(0) = 1.3 \times 10^9$, $E_D(0) = 1.1 \times 10^5$, $I_D(0) = 28292$, $R_D(0) = 3000$, $A(0) = 8 \times 10^6$, $S_v(0) = 5 \times 10^6$, $E_{vD}(0) = 2 \times 10^5$ and $I_{vD}(0) = 10^5$. The vertical axes indicate the cumulative number of DENV cases in India from 2010 to 2017.

model, it has been shown, using Lyapunov function theory and LaSalle's Invariance Principle, that the trivial disease-free equilibrium (TDFE) is globally asymptotically stable (GAS) whenever a certain associated threshold quantity (i.e., $\mathcal{R}_{NC}$, $\mathcal{R}_{ND}$, or $\mathcal{R}_{NCD}$) is less than unity. The mosquito-present disease-free equilibrium of the full model (or its associated sub-models) is shown to be locally-asymptotically stable (LAS) whenever the associated reproduction number is less than unity, and is unstable if it exceeds unity. For the scenario where the basic reproduction number is greater than one, each of the sub-models is shown to have a unique endemic equilibrium. The CHIKV and DENV sub-models and the full model each exhibit the phenomenon of backward bifurcation, where the stable non-trivial disease-free equilibrium (NDFE) co-exists with the stable endemic equilibrium when the reproduction number is below one. The epidemiological consequences of the phenomenon of the backward bifurcation is that making the basic reproduction number to be less than one is no longer a key requirement for the disease control, therefore, it is important to consider some other features such as making the basic reproduction number even less than a critical value (i.e., $\mathcal{R}_c$, in the case of DENV) so that the disease eradication could be achieved.

We fitted each of the sub-models and the full model with realistic data for CHIKV, DENV and CHIKV-DENV coinfection cases (obtained from, the National Vector Borne Disease Control Programme (NVBDCP) in India [31] and from [13], see Figs. 4, 5, and 6).

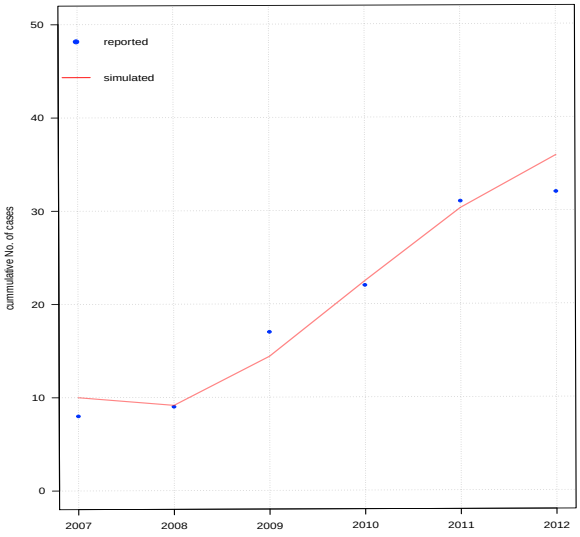

FIGURE 6. Fitting result of the full model of Eqns (1)-(2). We used the parameter values from Table 3 and the following initial conditions: $S_H(0) = 1179681900$, $E_C(0) = 1200$, $E_D(0) = 1200$, $E_{CD}(0) = 10$, $I_C(0) = 8$, $I_D(0) = 8$, $I_{CE}(0) = 8$, $I_{DE}(0) = 8$, $I_{CD}(0) = 6$, $E_{CT}(0) = 12$, $E_{DT}(0) = 12$, $I_{CT}(0) = 8$, $I_{DT}(0) = 8$, $R_C(0) = 8$, $R_D(0) = 8$, $T(0) = 3$, $A(0) = 8\times10^{10}$, $S_v(0) = 5\times10^{10}$, $E_{vC}(0) = 2\times10^5$, $E_{vD}(0) = 2\times10^5$, $E_M(0) = 2\times10^5$, $I_{vC}(0) = 10^4$, $I_{vD}(0) = 10^4$, $I_{vCE}(0) = 100$, $I_{vDE}(0) = 10^4$, and $I_{vM}(0) = 10^4$. The vertical axes indicate the cumulative number of DENV cases in India since 2007 to 2012.

The sensitivity analysis results using the partial ranked correlation coefficient (PRCC) with 95% confidence interval show the top-ranked parameters of the model (i.e., μ_v, μ_A, β_C, β_D, β_v, and ξ) that are sensitive to our main results, and should be prioritized in combating CHIKV, DENV and CHIKV-DENV co-infection outbreaks. The PRCCs results highlighted that, for any control strategy for combating CHIKV, DENV and CHIKV-DENV co-infection outbreaks to be effective, policymakers should focus on reducing mosquitoes in the environment (for example, spraying insecticide, proper sanitation and clearing mosquito breeding sites, etc.) and also reduce human-mosquito contact rate by taking some protection measures such as using insecticide-treated bed nets and curtains, insect repellent, etc.

Finally, this study can be extended by (i) including the possibility of simultaneous transmission of both diseases; and (ii) incorporating seasonality in order to get insight of its effect on the transmissions dynamics of each disease and the co-infection.

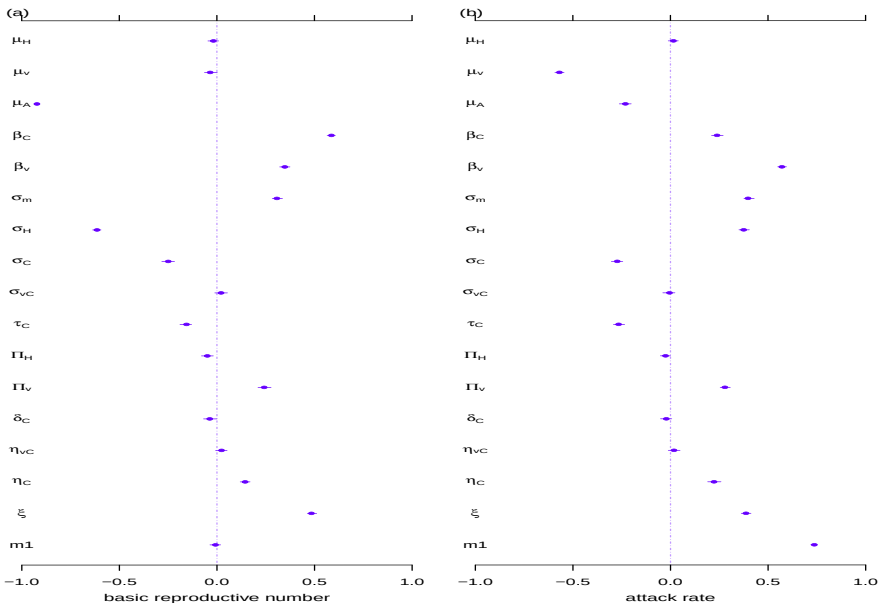

FIGURE 7. The PRCCs (of the CHIKV only sub-model (6)) of basic reproduction number (panel (a)) and infection attack rate (panel (b)) with respect to the model parameters. m_1 denotes the mosquito to human ratio. The blue dots are the estimated correlations and the bars represent the 95% CIs. The ranges of parameters are given in Table 3.

Appendices

A1. The Proof of Theorem 3.1.

Proof. Following [34], the model (6) can be re-written in matrix form or vector form as $\frac{dY}{dt} = A(Y)Y + G$, where $Y = (S_H, E_C, I_C, R_C, A, S_v, E_v, I_v)^T$, and $A(Y)_{8\times 8}$ is a M-matrix (Metzler Matrix) given by

$$A(Y) = \begin{bmatrix} -K_1-\mu_H & 0 & 0 & 0 & 0 & 0 & -K_5 & -K_6 \\ K_1 & -g_1 & 0 & 0 & 0 & 0 & K_5 & K_6 \\ 0 & \sigma_C & -g_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_C & -\mu_H & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -g_4 & 0 & 0 & 0 \\ 0 & -K_2 & -K_3 & 0 & \xi & -K_4-\mu_v & 0 & 0 \\ 0 & K_2 & K_3 & 0 & 0 & K_4 & -g_3 & 0 \\ 0 & 0 & 0 & 0 & g_5 & 0 & \sigma_{vC} & -\mu_v \end{bmatrix}$$

where
$K_1 = \frac{\beta_C\sigma_m\sigma_H(\eta_{vC}E_v+I_v)}{\sigma_m N_v+\sigma_H N_H}$, $K_2 = \frac{\beta_v\sigma_m\sigma_H\eta_C S_v}{\sigma_m N_v+\sigma_H N_H}$, $K_3 = \frac{\beta_v\sigma_m\sigma_H S_v}{\sigma_m N_v+\sigma_H N_H}$,
$K_4 = \frac{\beta_v\sigma_m\sigma_H(\eta_C E_C+I_C)}{\sigma_m N_v+\sigma_H N_H}$, $K_5 = \frac{\beta_C\sigma_m\sigma_H\eta_{vC}S_H}{\sigma_m N_v+\sigma_H N_H}$, and $K_6 = \frac{\beta_C\sigma_m\sigma_H S_H}{\sigma_m N_v+\sigma_H N_H}$,
and $G = (\Pi_H, 0, ..., 0)^T \subset \mathbb{R}^8_+$. Let $R_{NC} \leq 1$, so that the model (6) has only

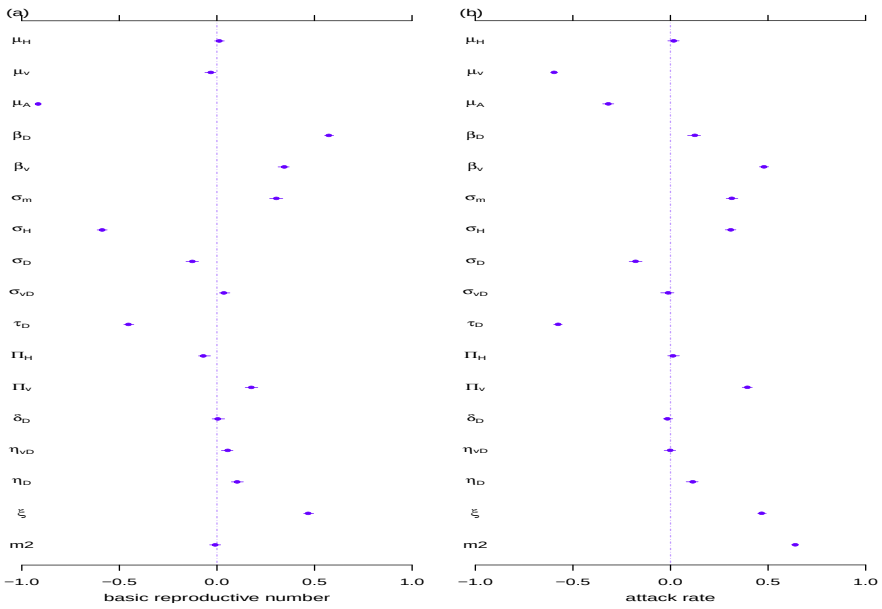

FIGURE 8. The PRCCs (of the DENV only sub-model (9)) of basic reproduction number (panel (a)) and infection attack rate (panel (b)) with respect to the model parameters. m_2 denotes the mosquito to human ratio. The blue dots are the estimated correlations and the bars represent the 95% CIs. The ranges of parameters are given in Table 3.

the TDFE, Υ_{0C}. Furthermore, let $Z = Y - TDFE$. Thus, the equation $\frac{dY}{dt} = A(Y)Y + G$ can be re-written as $\frac{dZ}{dt} = B(Z)Z$, where $B(Z)$ is the coefficients of the model (6) with variables $Z_i (i = 1, ..., 8)$. It is clear that $TDFE_Z = (0, ..., 0) \subset \mathbb{R}^8_+$ is the only equilibrium of the system $\frac{dZ}{dt} = B(Z)Z$. Consider a Lyapunov function $V(Z) = \langle W, Z\rangle$ with positive coefficient vector $W = (1, 1, 1, 1, \frac{1}{\Pi_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}) > 0$ [34]. Thus $V(Z) > 0$, except at $Z = TDFE_Z$, so that

$$\begin{aligned}\frac{dV(Z)}{dt} &= \langle W, B(Z)Z\rangle = -(Z_6 + Z_7 + Z_8) - \frac{1+\mu_A}{\Pi_v}Z_5 + \frac{1}{\mu_v}Z_5 \\ &= -(Z_6 + Z_7 + Z_8) - \frac{1+\mu_A}{\Pi_v}(1 - R_{NC})Z_5.\end{aligned}$$

Since $R_{NC} \leq 1$ in $C([0], \mathbb{R}^8_+)$, it follows that $V'(Z) \leq 0$. Following the LaSalle's Invariance Principle (Theorem 6.4 of [24]), we have that, the maximal invariant set contained in $V/V'(Z) \leq 0$ is the $TDFE_Z$. Thus, the transformed equilibrium, $TDFE_Z$, is GAS in $C([0], \mathbb{R}^8_+)$ if $R_{NC} \leq 1$. Hence, Υ_{0C} is also GAS in $C([0], \mathbb{R}^8_+)$ whenever $R_{NC} \leq 1$. □

A2. **The Proof of Theorem 3.3.** The proof is based on using centre manifold theory [3, 4]. Consider the system $\frac{dx}{dt} = f(x, \psi)$, where ψ is the bifurcation parameter, f is continuously differentiable at least twice in both x and ψ. The disease-free equilibrium is the line (x_0, ψ) and the local stability of the disease-free equilibrium

changes at the point (x_0, ψ) [44]. Now it shall show that there are non trivial equilibrium near the bifurcation point (x_0, ψ).

Now, consider the case when $\mathcal{R}_{0C} = 1$. Suppose, further, that $\beta_C = \beta_C^*$ is chosen as a bifurcation parameter. Solving for $\mathcal{R}_{0C} = 1$ gives

$$\beta_C = \beta_C^* = \frac{g_1 g_2 g_3 g_4 \mu_v^2 (\mu_H \sigma_m \Pi_v + \mu_v \sigma_H \Pi_H)^2}{\xi \Pi_v \Pi_H \beta_v \mu_H (\eta_v \mu_v + \sigma_v)(\eta_C g_2 + \sigma_C) \sigma_H^2 \sigma_m^2 \mu^2}.$$

By Lemma 3.2, the disease-free equilibrium E_0 is locally stable when $\beta_C < \beta_C^*$ and unstable when $\beta_C > \beta_C^*$. Here $\beta_C = \beta_C^*$ is a bifurcation value.

For computational convenience, we let $S_H = x_1$, $E_C = x_2$, $I_C = x_3$, $R_C = x_4$, $A = x_5$, $S_v = x_6$, $E_{vC} = x_7$, $I_{vC} = x_8$, so that $N_H = x_1 + x_2 + x_3 + x_4$ and $N_v = x_5 + x_6 + x_7 + x_8$. Further, by adopting the same vector notations with $x = (x_1, x_2, ..., x_8)^T$, the model (6) can be written in the form $\frac{dX}{dt} = F(X)$ where $F = (f_1, f_2, ..., f_8)^T$ as follows:

$$\begin{aligned}
f_1 &= \frac{dx_1}{dt} = \Pi_H - \lambda_C x_1 - \mu_H x_1, \\
f_2 &= \frac{dx_2}{dt} = \lambda_C x_1 - g_1 x_2, \\
f_3 &= \frac{dx_3}{dt} = \sigma_C x_2 - g_2 x_3, \\
f_4 &= \frac{dx_4}{dt} = \tau_C x_3 - \mu_H x_4, \\
f_5 &= \frac{dx_5}{dt} = \Pi_v - g_4 x_5, \\
f_6 &= \frac{dx_6}{dt} = \xi x_5 - \lambda_{vC} x_6 - \mu_v x_6, \\
f_7 &= \frac{dx_7}{dt} = \lambda_{vC} x_6 - g_3 x_7, \\
f_8 &= \frac{dx_8}{dt} = g_5 x_5 + \sigma_{vC} x_7 - \mu_v x_8,
\end{aligned} \tag{A2.1}$$

with the associated forces of infection given by

$$\lambda_C = \frac{\beta_C \sigma_m \sigma_H (\eta_{vC} x_7 + x_8)}{\sigma_m \sum_{i=5}^{8} x_i + \sigma_H \sum_{i=1}^{4} x_i}, \lambda_{vC} = \frac{\beta_v \sigma_m \sigma_H (\eta_C x_2 + x_3)}{\sigma_m \sum_{i=5}^{8} x_i + \sigma_H \sum_{i=1}^{4} x_i}. \tag{A2.2}$$

The Jacobian matrix of the system (A2.1) , evaluated at the DFE (ε_{0C}) with $\beta_C = \beta_C^*$ (denoted by $J_{\varepsilon_{0C}}$), is given by

$$J(\varepsilon_{0C}) = \begin{bmatrix}
-\mu_H & 0 & 0 & 0 & 0 & 0 & -s_1 & -s_2 \\
0 & -g_1 & 0 & 0 & 0 & 0 & s_1 & s_2 \\
0 & \sigma_C & -g_2 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \tau_C & -\mu_H & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -g_4 & 0 & 0 & 0 \\
0 & -s_3 & -s_4 & 0 & \xi & -\mu_v & 0 & 0 \\
0 & s_3 & s_4 & 0 & 0 & 0 & -g_3 & 0 \\
0 & 0 & 0 & 0 & g_5 & 0 & \sigma_{vC} & -\mu_v
\end{bmatrix}, \tag{A2.3}$$

where,
$s_1 = \frac{\beta_C \sigma_m \sigma_H \eta_{vC} \Pi_H \mu_v (1+\mu_A)}{(\Pi_H (1+\mu_A)\sigma_H + \sigma_m \Pi_v \mu_H)\mu_v + \sigma_m \Pi_v \mu_H \xi}$, $s_2 = \frac{\beta_C \sigma_m \sigma_H \Pi_H \mu_v (1+\mu_A)}{(\Pi_H (1+\mu_A)\sigma_H + \sigma_m \Pi_v \mu_H)\mu_v + \sigma_m \Pi_v \mu_H \xi}$,
$s_3 = \frac{\beta_v \sigma_m \sigma_H \eta_C \xi \Pi_v \mu_H}{\sigma_m \Pi_v (\mu_v+\xi)\mu_H + \sigma_H \Pi_H \mu_v (1+\mu_A)}$, $s_4 = \frac{\beta_v \sigma_m \sigma_H \xi \Pi_v \mu_H}{\sigma_m \Pi_v (\mu_v+\xi)\mu_H + \sigma_H \Pi_H \mu_v (1+\mu_A)}$.
The Jacobian, $J(\varepsilon_{0C})$, of the linearized system has a simple zero eigenvalue (with all other eigenvalues having negative real part). Hence, the Center Manifold Theory [3, 4] can be used to analyze the dynamics of the system (A2.1) around $\beta_C = \beta_C^*$. In particular, a theorem 4.1 in [4]. Using the notation in [4], the following computations are carried out.

Eigenvectors of $J(\varepsilon_{0C})_{\beta_C=\beta_C^*}$: For the case when $\mathcal{R}_{0C} = 1$ it can be shown that the $J(\varepsilon_{0C})$ has a right eigenvector (corresponding to the zero eigenvalue), given by $w = [w_1, w_2, ..., w_8]^T$, where

$$w_1 = -[\frac{s_1}{g_3}(\frac{s_3 g_2}{\sigma_C} + s_4) + \frac{s_2 \sigma_{vC}}{\mu_v g_3}(\frac{s_3 g_2}{\sigma_C} + s_4)]\frac{w_3}{\mu_H}, w_2 = \frac{g_2}{\sigma_C} w_3, w_3 > 0, w_4 = \frac{\tau_C}{\mu_H} w_3,$$
$$w_5 = 0, w_6 = -\frac{s_3 g_2 + \sigma_C s_4}{\mu_v \sigma_C} w_3, w_7 = \frac{s_3 g_2 + \sigma_C s_4}{g_3 \sigma_C} w_3, w_8 = \frac{(\sigma_{vC})(s_3 g_2 + \sigma_C s_4)}{\mu_v \sigma_C g_3} w_3.$$

Similarly, the components of the left eigenvector of $J(\varepsilon_{0C})$ (corresponding to the zero eigenvalue), denoted by $v = [v_1, v_2, ..., v_8]$, are given by

$$v_1 = 0, \quad v_2 = \frac{\sigma_C s_4 + s_3 g_2}{s_4 g_1} v_3, \quad v_3 > 0, \quad v_4 = 0, \quad v_5 = \frac{(g_5 s_2)(\sigma_C s_4 + s_3 g_4)}{g_1 g_4 \mu_v s_4} v_3,$$
$$v_6 = 0, \quad v_7 = \frac{g_2}{s_4} v_3, \quad v_8 = \frac{s_2 s_4 \sigma_C + s_2 s_3 g_4}{g_1 s_4 \mu_v} v_3.$$

Note that the free right eigenvectors, w_3 and left eigenvector, v_3 , are chosen to be $v_3 = 1$, and $w_3 = \frac{1}{A_1 + A_2}$, where, $A_1 = \frac{g_2 s_4 \sigma_C + s_3 g_2^2 + g_1 s_4 \sigma_C}{g_1 s_4 \sigma_C}$,
$A_2 = \frac{g_2(s_3 g_2 + s_4 \sigma_C)}{g_3 s_4 \sigma_C} + \frac{(s_2 \sigma_{vC})(s_4 \sigma_C + s_3 g_4)(s_3 g_2 + s_4 \sigma_C)}{g_1 g_3 \mu_v^2 s_4 \sigma_C}$, so that $v \cdot w = 1$ (in line with [4]).

It can be shown, by computing the non-zero partial derivatives of the right-hand side functions, $f_i (i = 1, ..., 8)$, that the associated backward bifurcation coefficients, a^* and b^*, are given, respectively, by (see Theoram 4.1 in [4]).

$$a^* = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k(0,0)}{\partial x_i \partial x_j} = \frac{q_1 + q_2}{q_3}, \tag{A2.4}$$

where,
$q_1 = 2(-g_4 p_1((p_2 \sigma_H + \sigma_m p_3) v_2 \beta_C - v_7 w_6 \beta_v \sigma_H p_4)\Pi_H +$
$\mu_H (v_2 w_1 p_5 \beta_C + v_7 w_6 \beta_v p_6)\Pi_v \sigma_m)\mu_v$,
$q_2 = \mu_H \Pi_v (v_2 w_1 \sigma_m p_7 \beta_C - (p_8 \sigma_H + \sigma_m p_9) v_7 p_{10} \beta_v)\xi)(1 + \mu_A)\mu_v \mu_H \sigma_H \sigma_m$,
$q_3 = ((\Pi_H \mu_v g_4 \sigma_H + \sigma_m \Pi_v \mu_H (\mu_v + \xi))^2)$, $p_1 = (w_8 + w_7 \eta_{vC})$, $p_2 = (w_3 + w_4 + w_2)$,
$p_3 = (w_7 + w_8 + w_6)$, $p_4 = (w_3 + w_2 \eta_C)$, $p_5 = (w_8 + w_7 \eta_{vC})$, $p_6 = (w_3 + w_2 \eta_C)$,
$p_7 = (w_8 + w_7 \eta_{vC})$, $p_8 = (w_2 + w_1 + w_3 + w_4)$, $p_9 = (w_7 + w_8)$ and $p_{10} = (w_3 + w_2 \eta_C)$,
and

$$b^* = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k(0,0)}{\partial x_i \partial \beta_v^*} = \frac{(w_2 \eta_C + w_3)\Pi_v \xi \mu_H \sigma_H \sigma_m v_7}{\Pi_H \mu_v (1 + \mu_A)\sigma_H + \sigma_m \Pi_v \mu_H (\mu_v + \xi)}. \tag{A2.5}$$

Since the coefficient b^* is always positive, it follows that the model (6), or it is transformed equivalent (A2.1), will undergo backward bifurcation if the coefficient a^* is positive.

A3. **Proof of Theorem 3.4.**

Proof. Following [34], the model (9) can be re-written as $\frac{dY^*}{dt} = A^*(Y^*)Y^* + G^*$, where $Y^* = (S_H, E_D, I_D, R_D, A, S_v, E_{vD}, I_{vD})^{T^*}$, $G^* = (\Pi_H, 0, ..., 0)^{T^*} \subset \mathbb{R}^8_+$, and $A^*(Y^*)_{8\times 8}$ is an M-matrix (Metzler Matrix) given by

$$A^*(Y^*) = \begin{bmatrix} -L_1-\mu_H & 0 & 0 & 0 & 0 & 0 & -L_5 & -L_6 \\ L_1 & -h_1 & 0 & 0 & 0 & 0 & L_5 & L_6 \\ 0 & \sigma_D & -h_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_D & -\mu_H & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -m & 0 & 0 & 0 \\ 0 & -L_2 & -L_3 & 0 & \xi & -L_4-\mu_v & 0 & 0 \\ 0 & L_2 & L_3 & 0 & 0 & L_4 & -h_3 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma_{vD} & -\mu_v \end{bmatrix}$$

with $L_1 = \frac{\beta_D\sigma_m\sigma_H(\eta_{vD}E_v+I_v)}{\sigma_m N_v+\sigma_H N_H}$, $L_2 = \frac{\beta_v\sigma_m\sigma_H\eta_D S_v}{\sigma_m N_v+\sigma_H N_H}$, $L_3 = \frac{\beta_v\sigma_m\sigma_H S_v}{\sigma_m N_v+\sigma_H N_H}$,
$L_4 = \frac{\beta_v\sigma_m\sigma_H(\eta_D E_D+I_D)}{\sigma_m N_v+\sigma_H N_H}$, $L_5 = \frac{\beta_D\sigma_m\sigma_H\eta_{vD}S_H}{\sigma_m N_v+\sigma_H N_H}$, $L_6 = \frac{\beta_D\sigma_m\sigma_H S_H}{\sigma_m N_v+\sigma_H N_H}$,
Let $R_{ND} \leq 1$ (so the model (9) has only the TDFE, Υ_{0D}). Furthermore, let $Z^* = Y^* - TDFE$. Thus, equation $\frac{dY^*}{dt} = A^*(Y^*)Y^* + G^*$ can now be re-written as $\frac{dZ^*}{dt} = B^*(Z^*)Z^*$, where $B^*(Z^*)$ is the coefficients of the model (9) with variables $Z_i^*(i = 1, ..., 8)$. It is clear that $TDFE_Z^* = (0, ..., 0) \subset \mathbb{R}^8_+$ is the only equilibrium of the system $\frac{dZ^*}{dt} = B^*(Z^*)Z^*$. Consider a Lyapunov function $V^*(Z^*) = \langle W^*, Z^*\rangle$ with positive coefficient vector $W^* = (1, 1, 1, 1, \frac{1}{\Pi_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}) > 0$ [34]. Thus $V^*(Z^*) > 0$, except at $Z^* = TDFE_{Z^*}$, so that

$$\begin{aligned}\frac{dV^*(Z^*)}{dt} &= \langle W^*, B^*(Z^*)Z^*\rangle = -(Z_6^* + Z_7^* + Z_8^*) - \frac{\xi+\mu_A}{\Pi_v}Z_5^* + \frac{\xi}{\mu_v}Z_5^* \\ &= -(Z_6^* + Z_7^* + Z_8^*) - \frac{\xi+\mu_A}{\Pi_v}(1 - R_{ND})Z_5^*.\end{aligned}$$

Since $R_{ND} \leq 1$ in $C([0], \mathbb{R}^8_+)$, it follows that $V^{*\prime}(Z^*) \leq 0$. Following the LaSalle's Invariance Principle (Theorem 6.4 of [24]), we have that, the maximal invariant set contained in $V^*/V^{*\prime}(Z^*) \leq 0$ is the $TDFE_{Z^*}$. Thus, the transformed equilibrium, $TDFE_{Z^*}$, is GAS in $C([0], \mathbb{R}^8_+)$ if $R_{ND} \leq 1$. Hence, Υ_{0D} is also GAS in $C([0], \mathbb{R}^8_+)$ whenever $R_{ND} \leq 1$. □

A4. **Proof of Theorem 4.1.**

Proof. Following [34], the model Eqns (1)-(2) can be re-written as $\frac{dY^{**}}{dt} = A^{**}(Y^{**})Y^{**} + G^{**}$, where

$$\begin{aligned}Y^{**} =&(S_H, E_C, E_D, E_{CD}, E_{CT}, E_{DT}, I_C, I_D, I_{CD}, I_{CE}, I_{DE}, I_{CT}, I_{DT}, R_C, R_D, T,\\ &A, S_v, E_{vC}, E_{vD}, E_M, I_{vC}, I_{vD}, I_{vCE}, I_{vDE}, I_{vM})^{T^{**}},\end{aligned}$$

$A^{**}(Y^{**})_{26\times 26}$ is a M-matrix (Metzler Matrix) which is not given here to save space and $G^{**} = (\Pi_H, 0, ..., 0)^{T^{**}} \subset \mathbb{R}^{26}_+$. Let $R_{NCD} \leq 1$ (so that the model Eqns (1)-(2) has only the TDFE, Υ_{0CD}). Further, let $Z^{**} = Y^{**} - TDFE$. Thus, the equation $\frac{dY^{**}}{dt} = A^{**}(Y^{**})Y^{**} + G^{**}$ can be re-written as $\frac{dZ^{**}}{dt} = B^{**}(Z^{**})Z^{**}$, where $B(Z)$ is a matrix of coefficients of the model Eqns (1)-(2) with variables $Z_i^{**}(i = 1, ..., 26)$. It is clear that $TDFE_Z^{**} = (0, ..., 0) \subset \mathbb{R}^{26}_+$ is the only equilibrium of the system

$\frac{dZ^{**}}{dt} = B^{**}(Z^{**})Z^{**}$. Consider a Lyapunov function $V^{**}(Z^{**}) = \langle W^{**}, Z^{**}\rangle$ with positive coefficient vector $W^{**} = (1, ..., 1, \frac{1}{\Pi_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}) > 0$, where $W^{**} \subset \mathbb{R}^{26}_+$. Thus $V(Z) > 0$, except at $Z^{**} = TDFE_{Z^{**}}$, so that

$$\frac{dV^{**}(Z^{**})}{dt} = \langle W^{**}, B^{**}(Z^{**})Z^{**}\rangle = -\frac{\mu_A}{\Pi_v}Z^{**}_{17} - \frac{1}{1+\mu_A} -$$

$$(Z^{**}_{18} + Z^{**}_{19} + Z^{**}_{21} + Z^{**}_{22} + Z^{**}_{23} + Z^{**}_{24} + Z^{**}_{25} + Z^{**}_{26}) + \left(\frac{\mu_v(1+\mu_A)}{\xi\Pi_v} + 1\right) R_N$$

$$= -\frac{\mu_A}{\Pi_v}Z^{**}_{17} - \frac{1}{1+\mu_A} - \sum_{i=18}^{26} Z^{**}_i + (\frac{\mu_v(1+\mu_A)}{\xi\Pi_v} + 1)R_{NCD}.$$

Since $R_{NCD} \leq 1$ in $C([0], \mathbb{R}^{26}_+)$, it follows that $V'(Z^{**}) \leq 0$. Following the LaSalle's Invariance Principle (Theorem 6.4 of [24]), we have that, the maximal invariant set contained in $V^{**}/V^{**\prime}(Z^{**}) \leq 0$ is the $TDFE_{Z^{**}}$. Thus, the transformed equilibrium, $TDFE_{Z^{**}}$, is GAS in $C([0], \mathbb{R}^{26}_+)$ if $R_{NCD} \leq 1$. Hence, Υ_{0CD} is also GAS in $C([0], \mathbb{R}^{26}_+)$ whenever $R_{NCD} \leq 1$.

It is worth mentioning that, the mosquito-free equilibrium in the CHIKV only sub-model, DENV only sub-model, and the full model, given by Υ_{0C}, Υ_{0D}, and Υ_{0CD}, respectively, is ecologically unrealistic. This is because, mosquitoes are present exist in the (CHIKV or DENV endemic) regions of interest). □

A5. **CHIKV, DENV and CHIKV-DENV reported cases time series in India.** The numbers of CHIKV, DENV and CHIKV-DENV cases in India used for model fitting are summarized in Tables A1 and A2, respectively.

TABLE A1. **Human reported CHIKV and DENV cases in India** [31].

Year	CHIKV	DENV
2010	48176	28292
2011	20402	18860
2012	15977	50222
2013	18840	75808
2014	16049	40571
2015	27553	99913
2016	64057	129166
2017	62268	157220

TABLE A2. **Human reported CHIKV-DENV co-infection cases in India** [13].

Year	No. of cases
2007	8
2008	1
2009	8
2010	5
2011	9
2012	1

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