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-exists 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.

1. Introduction. Chikungunya virus (CHIKV), a vector-borne disease transmitted by an infected female mosquito, is an \textit{arboviral} disease caused by a member of the genus \textit{alphavirus} which belongs to the \textit{Togaviridae} family \cite{8,26,28,30,39,49}. Whereas, Dengue virus (DENV) belongs to the genus \textit{Flavivirus}, under the family of \textit{Flaviviridae}, and there are four serotypes of DENV which causes a wide

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spectrum of illness from asymptomatic to symptomatic or severe fatal Dengue Hemorrhagic Fever or Dengue Shock Syndrome (DHF/DSS) (the DHF/DSS has 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 vaccine 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
DYNAMICAL ANALYSIS OF CHIKV AND DENV CO-INFECTION MODEL

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 $N_{CT}(t)$), individuals exposed to DENV but recovered from CHIKV with permanent immunity ($E_{CT}(t)$), individuals exposed to 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 $N_M(t)$, 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:

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

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 ($E_vD(t)$), adult mosquitoes exposed to both CHIKV and DENV viruses ($E_vM(t)$), 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

$$N_I(t) = S_v(t) + E_vC(t) + E_vD(t) + E_vM(t) + I_vC(t) + I_vD(t) + I_{vCE}(t) + I_{vDE}(t) + I_{vM}(t).$$

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

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

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

and

\[
\begin{align*}
\frac{dA}{dt} &= \Pi_v - (1 + \mu_A)A, \\
\frac{dS_v}{dt} &= \xi A - (\lambda_v + \lambda_v D)S_v - \mu_v S_v, \\
\frac{dE_{vC}}{dt} &= \lambda_v C S_v - \omega_1 \lambda_v D E_{vC} - (\sigma_v + \mu_v)E_{vC}, \\
\frac{dE_{vD}}{dt} &= \lambda_v D S_v - \omega_2 \lambda_v C E_{vD} - (\sigma_v + \mu_v)E_{vD}, \\
\frac{dE_M}{dt} &= \omega_1 \lambda_v D E_{vC} + \omega_2 \lambda_v C E_{vD} - (\theta_1 + \theta_2 + \mu_v)E_M, \\
\frac{dI_{vC}}{dt} &= (1 - \xi)A + \sigma_v C E_{vC} - (\lambda_v + \mu_v)I_{vC},
\end{align*}
\]
\[
\begin{align*}
\frac{dI_{vD}}{dt} &= \sigma v D E v D - (\lambda v C + \mu v) I_{vD}, \\
\frac{dI_{vCE}}{dt} &= \theta_1 E_M + \lambda v D I_{vC} - (\rho_1 + \mu v) I_{vCE}, \\
\frac{dI_{vDE}}{dt} &= \theta_2 E_M + \lambda v C I_{vD} - (\rho_2 + \mu v) I_{vDE}, \\
\frac{dI_{vM}}{dt} &= \rho_1 I_{vCE} + \rho_2 I_{vDE} - \mu v I_{vM}.
\end{align*}
\]

where,
\[
\begin{align*}
\lambda_C &= \frac{\beta_C b_1(N_H, N_v)}{N_v} \left[ (\eta_v E_v C + \eta_M E_M) + I_{vC} + I_{vCE} + I_{vM} \right], \\
\lambda_D &= \frac{\beta_D b_1(N_H, N_v)}{N_v} \left[ (\eta_v D E_v D + \eta_M E_M) + I_{vD} + I_{vDE} + I_{vM} \right], \\
\lambda_{vC} &= \frac{\beta_v b_2(N_H, N_v)}{N_H} \left[ (\eta_C(E_C + E_{CT}) + \eta_{CD} E_{CD}) + I_C + I_{CT} + \eta(I_{CD} + I_{CE}) \right], \\
\lambda_{vD} &= \frac{\beta_v b_2(N_H, N_v)}{N_H} \left[ (\eta_D(E_D + E_{DT}) + \eta_{CD} E_{CD}) + I_D + I_{DT} + \eta(I_{CD} + I_{DE}) \right].
\end{align*}
\]

In Eqns (3), \( \beta_C \) and \( \beta_D \) 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,
\[
b_1(N_H, N_v) = \frac{\sigma v m \sigma H N_v}{\sigma v m N_v + \sigma H N_H}, \quad \text{and} \quad b_2(N_H, N_v) = \frac{\sigma v m \sigma H N_H}{\sigma v m N_v + \sigma H N_H}.
\]

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 \( t \).
\[
\begin{align*}
\frac{dN_H}{dt} &= \Pi_H - \mu_H N_H - \delta C I_C - \delta D I_D - \delta_{CD} I_{CD} - \delta_{CE} I_{CE} \\
&\quad - \delta_{DE} I_{DE} - \delta_{CT} I_{CT} - \delta_{DT} I_{DT} \leq \Pi_H - \mu_H N_H,
\end{align*}
\]

and
\[
\frac{dN_v}{dt} = \Pi_v - \mu_A A - \mu_v N_I \leq \Pi_v - \mu_v N_v.
\]

Furthermore, consider the region
\[
\Omega = \left\{ (S_H, E_C, E_D, E_{CD}, I_C, I_D, I_{CE}, I_{DE}, I_{CD}, E_{CT}, E_{DT}, I_{CT}, I_{DT}, R_C, R_D, T), \right.
\]
\[
A, S_v, E_v C, E_v D, E_v M, I_v C, I_v D, I_v CE, I_v DE, I_v M) \in \mathbb{R}^+ : N_H \leq \frac{\mu H}{\mu A} : N_v \leq \frac{\mu v}{\mu}\right\}.
\]

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 \( \Omega \) will remain in \( \Omega \) for all time \( t \geq 0 \). Thus, the region \( \Omega \) is positive-invariant, and it is sufficient to consider solutions restricted in \( \Omega \). In this region, the usual existence, uniqueness and continuation results hold for the model Eqns (1)-(2) [21, 43].
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 $E_D = E_{CD} = I_D = I_{CE} = I_{DE} = I_{CD} = E_{CT} = E_{DT} = I_{CT} = I_{DT} = R_D = T = E_v = E_M = I_{vD} = I_{vCE} = I_{vDE} = I_{vM} = 0$ in Eqns (1)-(2)) is given by

$$
\begin{align*}
\frac{dS_H}{dt} &= \Pi_H - \lambda_C S_H - \mu_H S_H, \\
\frac{dE_C}{dt} &= \lambda_C S_H - (\sigma_C + \mu_H)E_C, \\
\frac{dI_C}{dt} &= \sigma_C E_C - (\tau_C + \delta_C + \mu_H)I_C, \\
\frac{dR_C}{dt} &= \tau_C I_C - \mu_H R_C, \\
\frac{dA}{dt} &= \Pi_v - (1 + \mu_A)A,
\end{align*}
$$

(6)
\begin{equation}
\begin{align*}
\frac{dS_v}{dt} &= \xi A - \lambda_v C S_v - \mu_v S_v, \\
\frac{dE_v C}{dt} &= \lambda_v C S_v - (\sigma_v C + \mu_v) E_v C, \\
\frac{dI_v C}{dt} &= (1 - \xi) A + \sigma_v C E_v C - \mu_v I_v C,
\end{align*}
\end{equation}

where,
\begin{align*}
\lambda_C &= \frac{\beta_C \sigma_m \sigma_H}{\sigma_m N_v + \sigma_H N_H} (\eta_v C E_v C + I_v C), \\
\lambda_v C &= \frac{\beta_v \sigma_m \sigma_H}{\sigma_m N_v + \sigma_H N_H} (\eta_v C E_v C + I_v C),
\end{align*}

with \( N_H = S_H + E_C + I_C + R_C \) and \( N_v = A + S_v + E_v C + I_v C \).

Consider the region
\[ \Omega_C = \left\{ (S_H, E_C, I_C, R_C, A, S_v, E_v C, I_v C) \in \mathbb{R}_+^8 : N_H \leq \frac{\Pi_H}{\mu H}, N_v \leq \frac{\Pi_v}{\mu v} \right\}, \]

it can be shown, as in section 2, that the region \( \Omega_C \) is positively-invariant.

Let \( R_{NC} = \frac{\Pi_u}{\mu v (1 + \mu_A)} \) 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: 
\[
Υ_{OC} = (S^*_H, E^*_C, I^*_C, R^*_C, A^*, S^*_v, E^*_vC, I^*_vC) = \left( \frac{Π_v}{μH}, 0, \ldots, 0 \right).
\]

(ii) \( R_{NC} > 1 \), the CHIKV only sub-model (6) has a disease-free (mosquito present) equilibrium (NDFE) given by:
\[
ς_{OC} = (S^*_H, E^*_C, I^*_C, R^*_C, A^*, S^*_v, E^*_vC, I^*_vC) = \left( \frac{Π_v}{μH}, 0, 0, 0, \frac{Π_v}{μ(1+μA)}, \frac{ξ(Π_v)}{μ(1+μA)}, 0, 0 \right).
\]

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 \( ς_{OC} \) 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

\[
R_{OC} = \sqrt{\frac{Π_vΠ_Hβ_vβ_CμH(η_vμ_v + σ_v)(η_cg_2 + σ_C)σ_H^2σ_m^2}{g_1g_2g_3(1 + μ_A)(μHσ_mΠ_v + μ_vσ_HΠ_H)^2}},
\]

where \( g_1 = σ_C + μ_H \), \( g_2 = τ_C + δ_C + μ_H \), \( g_3 = σ_vC + μ_v \).

**Lemma 3.2.** The DFE (\( ς_{OC} \)), of the CHIKV only sub-model (6) is locally asymptotically stable (LAS) if \( R_{OC} < 1 \), and unstable if \( R_{OC} > 1 \).
DYNAMICAL ANALYSIS OF CHIKV AND DENV CO-INFECTION MODEL

The threshold quantity $R_{0C}$ 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

$$S^*_H = \frac{\Pi_H}{\lambda^*_C + \mu_H}, \quad E^*_C = \frac{\lambda^*_C \Pi_H}{g_1(\lambda^*_C + \mu_H)}, \quad I^*_C = \frac{\sigma_C \lambda^*_C \Pi_H}{g_1 g_2 (\lambda^*_C + \mu_H)},$$
$$R^*_C = \frac{\tau \sigma_C \lambda^*_C \Pi_H}{g_1 g_2 \mu_H (\lambda^*_C + \mu_H)}, \quad A^* = \frac{\Pi_v}{1 + \mu}, \quad S^*_v = \frac{\xi \lambda^*_v \Pi_v}{g_3 (1 + \mu) (\lambda^*_v + \mu)},$$
$$E^*_v = \frac{\xi \lambda^*_v \Pi_v}{g_3 (1 + \mu) (\lambda^*_v + \mu)}, \quad I^*_v = \frac{\Pi_v (\sigma_v \lambda_v \xi + g_3 (1 - \xi) (\lambda_v + \mu))}{\mu (1 + \mu) g_3 (\lambda^*_v + \mu)}.$$

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 $R_{0C} = 1$ 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 $R_{0C} < 1$, 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 $E_C = E_{CD} = I_C = I_{CE} = I_{DE} = I_{CD} = E_{CT} = E_{DT} = I_{CT} = I_{DT} = R_C = T = E_v = E_M = I_v = I_{vE} = I_{vDE} = I_{vM} = 0$ in the model Eqns (1)-(2) is given by

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

\[9\]
where
\[ \lambda_D = \frac{\beta_D \sigma_m \sigma_H}{\sigma_m N_v + \sigma_H N_H} (\eta_H E_v D + I_v D) \]
\[ \lambda_v D = \frac{\beta_m \sigma_H}{\sigma_m N_v + \sigma_H N_H} (\eta_D E_D + I_D), \]

with, \( N_H = S_H + E_D + I_D + R_D \) and \( N_v = A + S_v + E_v D + I_v D \).

Consider the region
\[ \Omega_D = \left\{ (S_H, E_D, I_D, R_D, A, S_v, E_v D, I_v D) \in \mathbb{R}_+^8 : N_H \leq \frac{\mu_H}{\mu_m}, A \leq \frac{\mu_D}{\mu_A \xi}, N_I \leq \frac{\xi}{\mu} \right\}, \]

it can be shown, as in section 2, that the region \( \Omega_D \) is positively-invariant. It is convenient to define the threshold quantity \( R_{ND} = \frac{\xi \Pi_v}{\mu_m (\xi + \eta)} \) 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: \( \Upsilon_{TD} = (S^*_H, E^*_D, I^*_D, R^*_D, A^*, S^*_v, E^*_v D, I^*_v D) = \left( \frac{\mu_H}{\mu_m}, 0, 0, 0, \frac{\mu_m}{\mu_v (\xi + \eta)}, 0, 0 \right) \).

(ii) If \( R_{ND} > 1 \), the DENV only sub-model (9) has a disease-free (mosquito present) equilibrium (NDFE) given by:
\[ \varepsilon_{ND} = (S^*_H, E^*_D, I^*_D, R^*_D, A^*, S^*_v, E^*_v D, I^*_v D) = \left( \frac{\mu_H}{\mu_m}, 0, 0, 0, \frac{\mu_m}{\mu_v (\xi + \eta)}, 0, 0 \right). \]

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 \( \Upsilon_{TD} \), is GAS in \( \Omega \) whenever \( R_{ND} \leq 1 \).

3.4.2. NDFE. Let \( R_{ND} > 1 \) (so that the NDFE, \( \varepsilon_{ND} \), exists). The linear stability of \( \varepsilon_{ND} \) 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
\[ R_{ND} = \frac{\xi \Pi_v \Pi_H \beta_v \sigma_D \mu_H (\eta_H \mu_v + \sigma_v) (\eta_D h_2 + \sigma_D) \sigma_H^2 \sigma_m^2}{h_1 h_2 h_3 m (\mu_H \sigma_m \Pi_v + \mu \sigma_H \Pi_H)^2}, \]

where \( h_1 = \sigma_D + \mu_H, h_2 = \tau_D + \delta_D + \mu_H, h_3 = \sigma_v + \mu = \xi + \mu \).

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

**Lemma 3.5.** The DFE (\( \varepsilon_{ND} \)), of the DENV only sub-model (9) is locally asymptotically stable (LAS) if \( R_{ND} < 1 \), and unstable if \( R_{ND} > 1 \).

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
\[ S^*_H = \frac{\Pi_H}{\lambda^*_D + \mu_H}, \quad E^*_D = \frac{\lambda^*_D \Pi_H}{h_1 (\lambda^*_D + \mu_H)}, \quad I^*_D = \frac{\sigma_D \lambda^*_D \Pi_H}{h_1 h_2 (\lambda^*_D + \mu_H)}, \]
\[ R^*_D = \frac{\tau_D \sigma_D \lambda^*_D \Pi_H}{h_1 h_2 \mu_H (\lambda^*_D + \mu_H)}, \quad A^* = \frac{\Pi_v}{m}, \quad S^*_v = \frac{\xi \Pi_v}{m (\lambda^*_v + \mu)}, \]
\[ E^*_v D = \frac{\xi \sigma_v \lambda^*_v \Pi_v}{h_3 m (\lambda^*_v + \mu)}, \quad I^*_v D = \frac{\xi \sigma_v \lambda^*_v \Pi_v}{h_3 m (\lambda^*_v + \mu)}. \]

(11)
We substitute Eqn (12) into Eqn (10), and after simplification, we have
\[
\lambda_D = \left[\beta_D \sigma_m \sigma_H \Pi_v h_1 h_2 \mu_H \lambda^*_v (\eta_D \mu + \sigma_v D) (\lambda_D' + \mu_H)\right] \cdot \left[(\lambda_D^* + \mu) \eta h_3\right]^{-1}.
\]

\[
[\sigma_H \Pi_H h_1 h_2 \mu_H + \lambda_D' (h_2 \mu_H + \sigma_D \mu_H + \sigma_D \tau_D)] + \sigma_m \Pi_v h_1 h_2 \mu_H (\lambda_D^* + \mu_H)]^{-1},
\]
and
\[
\lambda^*_v = \frac{\beta_v \sigma_m \sigma_H \Pi_H \mu_H (\eta_D h_2 + \sigma_D)}{[\sigma_H \Pi_H h_1 h_2 \mu_H + \lambda_D' (h_2 \mu_H + \sigma_D \mu_H + \sigma_D \tau_D)] + \sigma_m \Pi_v h_1 h_2 (\lambda_D^* + \mu_H)]}.
\]

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 \(\lambda_D^*\))
\[
a_1 (\lambda_D^*)^2 + a_2 \lambda_D^* + a_3 = 0,
\]
where
\[
a_1 = m^2 (\sigma_D^2 \Pi_H^2 \mu_H^2 (\sigma_D^2 + \sigma_D \tau_D)^2 + 2 \sigma_m \sigma_H \Pi_v \Pi_H \mu_H \mu_H h_1 h_2 (h_2 \mu_H + \sigma_D \mu_H + \sigma_D \tau_D) + \sigma_m^2 \Pi_H^2 \mu_H^2 + \beta_v \sigma_m \sigma_H \Pi_H \mu_H (\eta_D h_2 + \sigma_D))(h_2 \mu_H + \sigma_D \mu_H + \sigma_D \tau_D) + \beta_v \sigma_m^2 \sigma_H \Pi_H \mu_\Pi h_1 h_2 (\eta_D h_2 + \sigma_D) + 2 \sigma_m^2 \Pi_H^2 \mu_H^2 h_1 h_2 h_3 + 2 \sigma_m \sigma_H \Pi_H \mu_\Pi^2 h_1 h_2 h_3 + (\mu_H \mu_H h_2 h_3) + (\mu_H \mu_H h_2 h_3) (\lambda_D^* + \mu_D H) + \sigma_D^2 \Pi_D^2 h_2 h_3 (\eta_D h_2 + \sigma_D) - (\mu_H \mu_H h_2 h_3) (\mu_H \sigma_m \Pi_v + \mu_H \Pi_H)^2 R_D^2).
\]
\[
a_3 = [m^2 h_2^2 h_3^2 (\mu_H \sigma_m \Pi_v + \mu_H \Pi_H) (1 - R_D^2)]
\]

Hence, we obtained a positive endemic equilibria of the model (9) by solving equation (12) for \(\lambda_D^*\) and substituting the positive values of \(\lambda_D^*\) 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
\[
\lambda_D^* = -\frac{a_2 + \sqrt{a_2^2 - 4a_1 a_3}}{2a_1}, \quad \text{and} \quad \lambda_D^* = -\frac{a_2 - \sqrt{a_2^2 - 4a_1 a_3}}{2a_1}.
\]

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 \(a_2 < 0 \iff a_3 = 0 \text{ or } a_2^2 - 4a_1 a_3 = 0\);
iii) two EEs if \(a_3 > 0, a_2 < 0 \text{ and } a_2^2 - 4a_1 a_3 > 0\) 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 \((a_2^2 - 4a_1 a_3)\) to zero and simplify for the critical value of \(\mathcal{R}_{0D}\), denoted
by $R_c$, given by

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

---

**Figure 3.** Backward bifurcation diagram of the model (9).

Thus, the BB would occur for values of $R_{0D}$ such that $R_c < R_{0D} < 1$. 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, $R_c = 0.8990 < 1$ and $R_{0D} = 0.9552 < 1$ so that $R_c < R_{0D} < 1$. 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 $R_c < R_{0D} < 1$.

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 $R_{0D} = 1$.

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.

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 Eqs (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 Eqs (1)-(2) has a TDFE (where no mosquito exist) given by

\[
\mathcal{Y}_{0CD} = \left( S_H^*, E_C^*, E_D^*, E_{CT}^*, E_{DT}^*, I_C^*, I_D^*, I_{CD}^*, I_{CT}^*, I_{DT}^*, R_C^*, R_D^*, T, A, S_v^*, E_v^*, E_M^*, I_v^*, I_{vC}^*, I_{vD}^*, I_{vCE}^*, I_{vDE}^*, I_{vM}^* \right) = \left( \frac{\Pi_H}{\mu_H}, 0, \ldots, 0 \right) \subset \mathbb{R}_+^{26}.
\]

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

\[
\mathcal{Y}_{0CD} = \left( S_H^*, E_C^*, E_D^*, E_{CT}^*, E_{DT}^*, I_C^*, I_D^*, I_{CD}^*, I_{CT}^*, I_{DT}^*, R_C^*, R_D^*, T, A, S_v^*, E_v^*, E_M^*, I_v^*, I_{vC}^*, I_{vD}^*, I_{vCE}^*, I_{vDE}^*, I_{vM}^* \right) = \left( \frac{\Pi_H}{\mu_H}, 0, \ldots, 0, \frac{\xi \Pi_v}{\mu_v (1 + \mu_A)}, 0, \ldots, 0 \right) \subset \mathbb{R}_+^{26}.
\]

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

**Theorem 4.1.** The TDFE of the full model Eqs (1)-(2), denoted by \( \mathcal{Y}_{0CD} \), is GAS in \( \Omega \) whenever \( R_{NCD} \leq 1 \).

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

\[
\mathcal{Y}_{0CD} = \left( S_H^*, E_C^*, E_D^*, E_{CT}^*, E_{DT}^*, I_C^*, I_D^*, I_{CD}^*, I_{CT}^*, I_{DT}^*, R_C^*, R_D^*, T, A, S_v^*, E_v^*, E_M^*, I_v^*, I_{vC}^*, I_{vD}^*, I_{vCE}^*, I_{vDE}^*, I_{vM}^* \right) = \left( \frac{\Pi_H}{\mu_H}, 0, \ldots, 0, \frac{\xi \Pi_v}{\mu_v (1 + \mu_A)}, 0, \ldots, 0 \right) \subset \mathbb{R}_+^{26}.
\]

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

\[ R_{0CD} = \max (R_{0C}, R_{0D}) \]

where \( R_{0C} \) and \( R_{0D} \) are defined in section 3. Using Theorem 2 of [44], the following result is established.

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

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., \( \Pi_H \) and \( \mu_H \)) are given by the average number of population in India which is given by
The term $\frac{\mu_H}{\mu_H} = 59.4$ years, implies that $\frac{H}{H} = 1,143,638,692$, 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., $\Pi_H$) 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 $\mu_H = 66.4$ years), implies that $\frac{H}{H} = 1,222,063,475$, 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).

### 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., $\mu_V$ and $\mu_A$, the transmission rates, i.e., $\beta_V$, $\beta_C$, and $\beta_D$, and the mosquito maturation rate, i.e., $\xi$, are the top control parameters for combating CHIKV, DENV and CHIKV-DENV co-infection outbreaks.

| Parameter | Interpretation/Description |
|-----------|----------------------------|
| $\mu_H$   | Bacteriological rate of human and mosquitoes, respectively |
| $\beta$   | Natural death rate of human |
| $\mu_A$   | Death rate of immature mosquitoes |
| $\kappa$  | Death rate of adult mosquitoes |
| $\lambda_C$ | Rates of CHIKV force of infection in human |
| $\lambda_D$ | Rates of DENV force of infection in human |
| $\lambda_{CD}$ | Rates of CHIKV force of infection in mosquitoes |
| $\lambda_{DP}$ | Rates of DENV force of infection in mosquitoes |
| $\Pi$     | Transmission parameter for DENV to human |
| $\beta_D$ | Transmission parameter from an infectious human to a susceptible adult mosquitoes |
| $\theta$  | Number of bites per human per unit time |
| $\eta$    | Number of bites per mosquitoes per unit time |
| $\xi$     | Fraction of immature mosquitoes becoming susceptible adult |
| $\gamma_1$ | Modification parameter for the heterogeneity of DENV infection between susceptible humans and human exposed to CHIKV |
| $\gamma_2$ | Modification parameter for the heterogeneity of CHIKV infection between susceptible humans and human exposed to DENV |
| $\gamma_3$ | Modification parameter for the heterogeneity of DENV infection between susceptible adult mosquitoes and those exposed to CHIKV |
| $\gamma_4$ | Modification parameter for the heterogeneity of CHIKV infection between susceptible adult mosquitoes and those exposed to DENV |
| $\gamma_5$ | Number of times a mosquito bites human per unit time |
| $\gamma_6$ | Maximum number of mosquito bites a human can receive per unit time |
| $\gamma_7$ | Progression rate of human from exposed state of CHIKV to the infectious state of CHIKV |
| $\gamma_8$ | Progression rate of human from exposed state of DENV to the infectious state of DENV |
| $\gamma_9$ | Progression rate of adult mosquitoes from exposed state of CHIKV to the infectious state of CHIKV |
| $\gamma_{10}$ | Progression rate of adult mosquitoes from exposed state of DENV to the infectious state of DENV |
| $\gamma_{11}$ | Progression rate of humans to active CHIKV classes |
| $\gamma_{12}$ | Progression rates of humans to active DENV classes |
| $\gamma_{13}$ | Progression rate of adult mosquitoes to active CHIKV classes |
| $\gamma_{14}$ | Progression rates of adult mosquitoes to active DENV classes |
| $\xi$     | Disease-induced death rates for humans |
| $\beta_M$ | Modification parameter for the heterogeneity of CHIKV infection between susceptible adult mosquitoes and those exposed to CHIKV |
| $\beta_D$ | Modification parameter for the heterogeneity of DENV infection between susceptible adult mosquitoes and those exposed to CHIKV |
| $\beta_C$ | Modification parameter for the heterogeneity of DENV infection between susceptible adult mosquitoes and those exposed to CHIKV |
| $\beta_{M}^D$ | Modification parameter for the heterogeneity of CHIKV infection between susceptible adult mosquitoes and those exposed to CHIKV |
| $\beta_{M}^C$ | Modification parameter for the heterogeneity of CHIKV infection between susceptible adult mosquitoes and those exposed to CHIKV |

### Table 2. Interpretation of the parameters of the model Eqns (1)-(2).
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 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., $R_{NC}$, $R_{ND}$, or $R_{NCD}$) 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
Figure 4. Fitting result of the CHIKV only sub-model (6). We used the parameter values from Table 3 and the following initial conditions: \(S_H(0) = 1.3 \times 10^9\), \(E_C(0) = 1.2 \times 10^9\), \(I_C(0) = 48176\), \(R_C(0) = 4000\), \(A(0) = 8 \times 10^7\), \(S_v(0) = 5 \times 10^7\), \(E_vC(0) = 2 \times 10^5\) and \(I_vC(0) = 10^4\). The vertical axes indicate the cumulative number of CHIKV cases in India from 2010 to 2017.

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., \(R_c\), 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).

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., \(\mu_v\), \(\mu_A\), \(\beta_C\), \(\beta_D\), \(\beta_v\), and \(\xi\)) 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.

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
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: $S_H(0) = 1179681900$, $E_C(0) = 1200$, $E_D(0) = 1200$, $E_{CD}(0) = 10$, $I_C(0) = 8$, $I_D(0) = 8$, $I_{CE}(0) = 8$, $I_{DE}(0) = 8$, $R_C(0) = 8$, $R_D(0) = 8$, $T(0) = 3$, $A(0) = 8 \times 10^{10}$, $S_v(0) = 5 \times 10^{10}$, $E_{vC}(0) = 2 \times 10^5$, $E_{vD}(0) = 2 \times 10^5$, $E_M(0) = 2 \times 10^5$, $I_{vC}(0) = 10^4$, $I_{vD}(0) = 10^4$, $I_{vCE}(0) = 100$, $I_{vDE}(0) = 10^4$, and $I_{vM}(0) = 10^4$. The vertical axes indicate the cumulative number of DENV cases in India since 2007 to 2012.

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

where

- $K_1 = \frac{\beta_{vH} \sigma_m \sigma_H (\eta_{vC} E_v + I_C)}{\sigma_m N_v + \sigma_H N_H}$,
- $K_2 = \frac{\beta_{vH} \sigma_m \sigma_H \eta_{vC} S_v}{\sigma_m N_v + \sigma_H N_H}$,
- $K_3 = \frac{\beta_{vH} \sigma_m \sigma_H S_v}{\sigma_m N_v + \sigma_H N_H}$,
- $K_4 = \frac{\beta_{vH} \sigma_m \sigma_H (\eta_{vC} E_v + I_C)}{\sigma_m N_v + \sigma_H N_H}$,
- $K_5 = \frac{\beta_{vH} \sigma_m \sigma_H S_v}{\sigma_m N_v + \sigma_H N_H}$, and
- $K_6 = \frac{\beta_{vH} \sigma_m \sigma_H S_v}{\sigma_m N_v + \sigma_H N_H}$,

and $G = (\Pi_H, 0, ..., 0)^T \in \mathbb{R}_+^8$. Let $R_{NC} \leq 1$, so that the model (6) has only the TDFE, $\Upsilon_{0C}$. 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
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.

model (6) with variables \( Z_i (i = 1, \ldots, 8) \). 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 \( W = (1, 1, 1, \frac{1}{\mu_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}) > 0 \) \([34]\). Thus \( V(Z) > 0 \), except at \( Z = TDFE_Z \), so that

\[
\frac{dV(Z)}{dt} = \langle W, B(Z)Z \rangle = -(Z_6 + Z_7 + Z_8) - \frac{1 + \mu_A}{\Pi_v} Z_5 + \frac{1}{\mu_v} Z_5
\]

\[
= -(Z_6 + Z_7 + Z_8) - \frac{1 + \mu_A}{\Pi_v} (1 - R_{NC}) Z_5.
\]

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, \( \Upsilon_{0C} \) 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 \( \psi \) is the bifurcation parameter, \( f \) is continuously differentiable at least twice in both \( x \) and \( \psi \). The disease-free equilibrium is the line \( (x_0, \psi) \) and the local stability of the disease-free equilibrium changes at the point \( (x_0, \psi) \) \([44]\). Now it shall show that there are non trivial equilibrium near the bifurcation point \( (x_0, \psi) \).
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.

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

\[
\beta_C = \beta_C^* = \frac{g_1g_2g_3g_4\mu_v^2(\mu_H\sigma_m\Pi_v + \mu_v\sigma_H\Pi_H)^2}{\xi\Pi_v\Pi_H\beta_C^*\mu_H(\eta_c\mu_v + \sigma_v)(\eta_Cg_2 + \sigma_C)\sigma_H^2\sigma_m^2\mu_v^2}.
\]

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:
\[ f_1 = \frac{dx_1}{dt} = \Pi_H - \lambda_C x_1 - \mu_H x_1, \]
\[ f_2 = \frac{dx_2}{dt} = \lambda_C x_1 - g_1 x_2, \]
\[ f_3 = \frac{dx_3}{dt} = \sigma_C x_2 - g_2 x_3, \]
\[ f_4 = \frac{dx_4}{dt} = \tau_C x_3 - \mu_H x_4, \]
\[ f_5 = \frac{dx_5}{dt} = \lambda_v - g_4 x_5, \]
\[ f_6 = \frac{dx_6}{dt} = \xi x_5 - \lambda_v x_6, \]
\[ f_7 = \frac{dx_7}{dt} = \lambda_v x_6 - g_3 x_7, \]
\[ f_8 = \frac{dx_8}{dt} = g_5 x_5 + \sigma_v x_7 - \mu_v x_8, \]  
\[ (A2.1) \]

with the associated forces of infection given by

\[ \lambda_C = \frac{\beta_C \sigma_m \sigma_H (\eta_C x_7 + x_8)}{\sigma_m \sum_{i=5}^{8} x_i + \sigma_H \sum_{i=1}^{4} x_i}, \]
\[ \lambda_v = \frac{\beta_v \sigma_m \sigma_H (\eta_C x_2 + x_3)}{\sigma_m \sum_{i=5}^{8} x_i + \sigma_H \sum_{i=1}^{4} x_i}. \]  
\[ (A2.2) \]

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

\[ J(\xi_{0C}) = \begin{bmatrix}
-\mu_H & 0 & 0 & 0 & 0 & -s_1 & -s_2 \\
0 & -g_1 & 0 & 0 & 0 & s_1 & s_2 \\
0 & \sigma_C & -g_2 & 0 & 0 & 0 & 0 \\
0 & 0 & \tau_C & -\mu_H & 0 & 0 & 0 \\
0 & 0 & 0 & -g_4 & 0 & 0 & 0 \\
0 & -s_3 & -s_4 & 0 & \xi & -\mu_v & 0 \\
0 & s_3 & s_4 & 0 & 0 & 0 & -g_3 \\
0 & 0 & 0 & 0 & g_5 & 0 & \sigma_v - \mu_v
\end{bmatrix}, \]  
\[ (A2.3) \]

where,

\[ s_1 = \frac{\beta_C \sigma_m \sigma_H \eta_C \Pi_H \mu_v (1 + \mu_A)}{(\Pi_H (1 + \mu_A) \sigma_H + \sigma_m \Pi_H \mu_v + \sigma_m \Pi_H \mu_H \xi)}, \]
\[ s_2 = \frac{\beta_C \sigma_m \sigma_H \Pi_H \mu_v (1 + \mu_A)}{(\Pi_H (1 + \mu_A) \sigma_H + \sigma_m \Pi_H \mu_v + \sigma_m \Pi_H \mu_H \xi)}, \]
\[ s_3 = \frac{\sigma_m \Pi_H (\mu_v + \xi) \mu_H + \sigma_m \Pi_H \mu_v (1 + \mu_A)}{\mu_v + \sigma_m \Pi_H \mu_H \xi}, \]
\[ s_4 = \frac{\sigma_m \Pi_H (\mu_v + \xi) \mu_H + \sigma_m \Pi_H \mu_v (1 + \mu_A)}{\mu_v + \sigma_m \Pi_H \mu_H \xi}. \]

The Jacobian, \(J(\xi_{0C})\), 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 \(J(\xi_{0C})\) \(\beta_C = \beta_C^*\): For the case when \(R_{0C} = 1\) it can be shown that the \(J(\xi_{0C})\) has a right eigenvector (corresponding to the zero eigenvalue), given by
Similarly, the components of the left eigenvector of $J(\varepsilon_0 C)$ (corresponding to the zero eigenvalue), denoted by $v = [v_1, v_2, \ldots, v_8]$, are given by

\begin{align*}
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 v_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} v_3.
\end{align*}

Note that the free right eigenvectors, $w_3$ and left eigenvector, $v_4$, are chosen to be $v_3 = 1$ and $w_3 = \frac{1}{A_1 + A_2}$, where, $A_1 = \frac{g_2 s_4 \sigma_C + s_3 g_2 + g_1 s_4 \sigma_C}{g_3 s_4 \sigma_C}$, $A_2 = \frac{g_2(s_2 g_2 + s_2 \sigma_C)}{g_3 s_4 \sigma_C} + \frac{(s_2 \sigma_C)(s_4 \sigma_C + s_3 g_4)(s_2 g_2 + s_2 \sigma_C)}{g_1 g_2 \mu \sigma_C}$, 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, $f_i (i = 1, \ldots, 8)$, that the associated backward bifurcation coefficients, $a^*$ and $b^*$, are given, respectively, by (see Theorem 4.1 in [4]).

\begin{equation}
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}
\end{equation}

where,

\begin{align*}
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) \Pi_H \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 \sigma_H \sigma_m, \\
q_3 &= ((\Pi_H \mu_v p_4 \sigma_H + \sigma_m \Pi_v \mu_H (\mu_v + \xi))^2), \\
p_1 &= (w_8 + w_7 \eta_v), \\
p_2 &= (w_3 + w_4 + w_2), \\
p_3 &= (w_7 + w_6 + w_5), \\
p_4 &= (w_3 + w_2 \eta_C), \\
p_5 &= (w_6 + w_7 \eta_v), \\
p_6 &= (w_3 + w_2 \eta_C), \\
p_7 &= (w_8 + w_7 \eta_v), \\
p_8 &= (w_2 + w_1 + w_3 + w_4), \\
p_9 &= (w_7 + w_8) \text{ and } p_{10} = (w_3 + w_2 \eta_C),
\end{align*}

and

\begin{equation}
b^* = \sum_{k, i, j = 1}^n v_k w_i \frac{\partial^2 f_k(0, 0)}{\partial x_i \partial x_j} = \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}
\end{equation}

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 $Y^* = (S_H, E_D, I_D, R_D, A, S_v, E_v, I_v)^T$, $G^* = (\Pi_H, 0, \ldots, 0)^T \in \mathbb{R}^n_+$, and $A^*(Y^*)_{8 \times 8}$ is an M-matrix (Metzler Matrix) given by
TDFE is a matrix of coefficients of the model Eqns (1)-(2) with variables $A_4$. Invariance Principle (Theorem 6.4 of [24]), we have that, the maximal invariant set $\mathcal{M}$ of $Y = Z = 1$ in $A, S, T, C, E, I, M$ can be re-written as $Z^* = B^*(Z^*)Z^*$, where $B^*(Z^*)$ is the coefficients of the model (9) with variables $Z^*_i (i = 1, ..., 8).$ It is clear that $TDFAE^*_Z = (0, 0)^T \in \mathbb{R}_+^8$ 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 $W^* = (1, 1, 1, \frac{1}{\mu c}, \frac{1}{\mu e}, \frac{1}{\mu}, \frac{1}{\mu e}, \frac{1}{\mu e}, \frac{1}{\mu e}, \frac{1}{\mu e}, > 0$ [34]. Thus $V^*(Z^*) > 0$, except at $Z^* = TDFAE^*_Z$. So that

$$
\frac{dV^*(Z^*)}{dt} = \langle W^*, B^*(Z^*)Z^* \rangle = -(Z^*_6 + Z^*_7 + Z^*_8) - \frac{\xi + \mu A}{\mu v} Z^*_6 + \frac{\xi}{\mu v} Z^*_5
$$

Since $R_{ND} \leq 1$ 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 $TDFAE^*_Z$. Thus, the transformed equilibrium, $TDFAE^*_Z$, is GAS in $C([0], \mathbb{R}_+^8)$ if $R_{ND} \leq 1$. Hence, $\Upsilon_0D$ 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

$$
Y^* = \begin{bmatrix} S_H, E_C, E_D, E_CT, E_RT, I_C, I_D, I_CD, I_CE, I_DE, I_CT, I_DT, R_C, R_D, T, A, S_v, E_v, E_vD, E_M, I_vC, I_vD, I_vCE, I_vDE, I_vM \end{bmatrix}^T,
$$

$A^*(Y^*)$ is a M-matrix (Metzler Matrix) which is not given here to save space and $G^* = (\Pi_H, 0, 0, 0)^T \in \mathbb{R}_+^8$. Let $R_{NCM} \leq 1$ (so that the model Eqns (1)-(2) has only the $TDFAE$, $\Upsilon_0CD$). Further, let $Z^* = Y^* - TDFAE$. Thus, the equation

$$
\frac{dZ^*}{dt} = A^*(Y^*)Y^* + G^*
$$

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 $TDFAE^*_Z = (0, 0, 0)^T \in \mathbb{R}_+^8$ 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 $W^* = (1, 1, 1, \frac{1}{\mu c}, \frac{1}{\mu e}, \frac{1}{\mu}, \frac{1}{\mu e}, \frac{1}{\mu e}, \frac{1}{\mu e}, \frac{1}{\mu e}, \frac{1}{\mu e}, \frac{1}{\mu e}, > 0$,
where $W^{**} \subset \mathbb{R}^{26}$. Thus $V(Z) > 0$, except at $Z^{**} = TDFE_{Z^{**}}$, so that

$$
\frac{dV^{**}(Z^{**})}{dt} = (W^{**}, B^{**}(Z^{**})Z^{**}) = -\frac{\mu_A}{\Pi_v} Z^{**}_{17} - \frac{1}{1 + \mu_A} \left( Z_{18}^{**} + Z_{19}^{**} + Z_{21}^{**} + Z_{22}^{**} + Z_{23}^{**} + Z_{24}^{**} + Z_{25}^{**} + Z_{26}^{**} + \left( \frac{\mu_v(1 + \mu_A)}{\xi \Pi_v} + 1 \right) R_N \right)
$$

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

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 $V^{**}/V^{**'}(Z^{**}) \leq 0$ is the $TDFE_{Z^{**}}$. Thus, the transformed equilibrium, $TDFE_{Z^{**}}$, is GAS in $C([0, \mathbb{R}^{26}])$ if $R_{NCD} \leq 1$. Hence, $\Upsilon_{0CD}$ 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 $\Upsilon_{0C}$, $\Upsilon_{0D}$, and $\Upsilon_{0CD}$, 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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