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Mathematical modeling of mosquito borne diseases with vertical transmissions as applied to Dengue

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Abstract

Aedes aegypti mosquitoes transmit important mosquito borne diseases that include dengue, yellow fever, zika, chikungunya, rift valley, and west nile among others. The dynamics of these diseases are influenced by various factors such as population dynamics of humans and mosquitoes, mosquito behaviour, and transmission modes. This study focuses on multiple transmissions, where both vertical and horizontal modes are considered with application to dengue virus. We therefore present a model that incorporates vertical transmission within the mosquito population. Threshold quantities for the model are computed, with the mosquito extinction equilibrium being globally asymptotically stable when the basic offspring number (N_0) is less than one, also, the disease free equilibrium is shown to be locally asymptotically stable when the basic reproduction number ($\mathcal{R}_0$) is less than one. The model is shown to undergo backward bifurcation, and conditions under which the disease free equilibrium would be globally asymptotically stable is presented. Type reproduction numbers are also computed. Some results of numerical simulations, and sensitivity (both local and global) analysis of the model parameters are shown and computed respectively.

Keywords: Mosquito borne diseases; vertical transmission, basic offspring number; stability; basic reproduction number; Metzler matrix; backward bifurcation.
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1 Introduction

Mosquito borne diseases (MBDs) are viral and parasitic infections that are primarily transmitted through bites by different species of infected mosquitoes, most notably are the aedes (with two specific species of aedes-aegypti and aedes-albopictus), culex (the house mosquitoes), and anopheles. While the aedes transmit diseases such as the dengue, zika, chikungunya, yellow fever and rift valley, the culex transmit diseases like Japanese encephalitis, west nile virus, and lymphatic filariasis. Even though anopheles are known for the transmission of malaria, they also transmit lymphatic filariasis [1, 2]. MBDs pose significant threats to humanity and global public health system, recently there were multiple outbreaks of MBDs in different parts of the world, particularly malaria, zika, yellow fever, and dengue.

Malaria is a parasitic infection that accounts for over 219 million infections and 400 thousand deaths annually [1, 3, 4]. Although malaria is one of the most important MBDs, almost half the population of our planet is exposed to dengue (the most common mosquito viral infection). It is one of the world's most threatening and widespread MBDs, in fact, it has recently accounted for about 390 million new infections annually, with about 96 million of them being symptomatic, it is estimated to cause between 40-50 million new cases annually [5, 6]. Yellow fever (YF) is another viral infection that is endemic in South America, Asia, and Africa, with almost a billion individuals from about 47 countries being exposed to it. YF is estimated to cause between 84,000-170,000 new cases with up to 29,000 mortality every year [7]. Likewise, zika virus was devastating between 2015-2016, it was attributed to the severe neurological defects that affected developing fetuses of women who were infected with the virus [1, 8]. Although Chikungunya virus was first discovered in tropical regions of Africa, it spread east into Asia and north into Europe, and by 2016 there were more than 1.7 million suspected cases of infection in the Americas [1].

While modeling the transmission dynamics of MBDs with horizontal transmissions (through mosquito bites) have been extensively studied as in [8, 9, 10, 11, 12, 13], vertical transmissions (either in human or mosquito population) have received less attention. However, mosquitoes vertical transmission (where they transmits infectious agent to their offspring) within aedes aegypti and albopictus populations have been documented, and this mode of transmission may have significantly influenced the spread and sustenance of the diseases [14, 15, 16, 17, 18]. In fact, vertical transmissions within the mosquito population for some of the diseases is a probable means for their persistence especially during periods that are not favorable for horizontal transmission to thrive [16, 18, 19]. For dengue in particular, it has been a challenge to understand how the virus remain endemic in humans even if there are long periods of extremely low, or zero, incidence [21], this study aimed at broadening the understanding of the transmission dynamics of dengue with vertical transmission.

Mathematical models for the transmission dynamics of dengue fever can be traced back from 1970 with well-known complex epidemiological dynamics, over the years, those models tried to incorporate factors focusing on different aspects of the disease and vectors, which could provide rich dynamical behavior even in the most basic models. The existing models are developed to evaluate, for example, the effect of co-circulation of multiple strains (or variants), the immunological path for disease severity, and the impact of vaccination [20]. The work of [22] incorporated aquatic stage of mosquito development to investigate the impact of vaccination (alone) and in combination with treatment and adulticides control on the population dynamics of dengue in Johor, Malaysia, while [11] investigated the cause(s) of backward bifurcation in a dengue model with and without vaccination. Also, [23] formulated a deterministic model that was used to gain insight into the

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effect of seasonal variations and vector vertical transmission (VT) on the dynamics of dengue, and [24] incorporated VT both for the populations of mosquitoes and humans with a constant rate of recruitment, it should be noted that, though VT of MBDs in humans have been reported and attracted attention especially in zika, its impact is still negligible, as such, it is ignored in this study. Furthermore, the work of [21] constructed and analyzed a mathematical model of dengue with VT in the population of mosquitoes (with both aquatic and non-aquatic stages), however, the model assumed a constant oviposition with a fraction of the laid eggs eventually getting infection. In this work, similar to the work of [21], we would incorporate both horizontal and vertical (within mosquito population) transmissions for dengue by extending the work of [21] with VT captured at the point of oviposition (usually called transovarial transmission). Thus, fraction of eggs laid by the reproductive but infected mosquitoes would acquire infection vertically. This is in addition to assuming that oviposition is proportional to the total number of reproductive mosquitoes, some of which are non-infected while others are infected.

2 Mathematical model

In this section, a thorough description of assumptions and methods involved in the model formulation would be given.

2.1 Model assumptions

At any time t, the total population of humans is denoted by $N(t)$, this is divided into non-intersecting compartments of susceptible $S(t)$, infected $I(t)$, and recovered $R(t)$ humans. Due to the incorporation of vertical transmission, mosquito population would be considered at both aquatic and non-aquatic stages, thus, at a time t, the development stage that includes eggs, larvae and pupae (aquatic stage) is denoted by $A(t)$. Some eggs are infected while others normal, this stage is further sub-divided into infected $A_i(t)$ and non-infected $A_n(t)$ aquatic mosquitoes, such that, a vertically infected egg would pass through the developmental stages as an infected aquatic mosquitoes (the same applies to a non-vertically-infected). Also, the non-aquatic stage has a total population denoted by $N_v(t)$, which is sub-divided into susceptible $S_v(t)$ and infected $I_v(t)$ non-aquatic mosquitoes.

Homogeneous mixing of the human and mosquito populations is assumed, thus, each bite of a mosquito has an equal probability of either acquiring (as susceptible mosquitoes bite infected humans) or transmitting (when infected mosquitoes bite susceptible humans) the disease.

2.2 Incidence function

There are different incidence functions that have been used in epidemiology, the density dependent (mass action incidence), and the frequency dependent (standard incidence) functions have been used more. Although no rule of choice exists in this regards, the standard incidence function has often been preferred in modeling of mosquito borne and sexually transmitted diseases [15, 25].

Let the biting rate of an infected mosquito be b_{vh}, while b_{hv} be the rate at which susceptible mosquitoes bite humans. Also, let ρ_{vh} denote the transmission probability from infectious mosquitoes to susceptible humans, and that of infectious humans to susceptible mosquitoes be ρ_{hv}. Donate $\beta_{vh} = \rho_{vh} b_{vh}$ and $\beta_{hv} = \rho_{hv} b_{hv}$ respectively be transmission rate from infectious mosquito to susceptible humans, and transmission rate from infectious human to susceptible mosquitoes. It

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is a known fact that, for the total number of mosquito bites to be conserved in a community, the total number of bites by mosquitoes at any given time must be equal to the total number of bites received by humans [11, 15, 26, 27, 28]. Therefore,

$$\beta_{vh}(N_h, N_v)N_h = \beta_{hv}(N_h, N_v)N_v \implies N_v = \frac{\beta_{vh}(N_h, N_v)}{\beta_{hv}(N_h, N_v)}N_h, \tag{2.1}$$

and as such, the human and mosquito incidences (force of infections) are

$$\lambda_h = \frac{\beta_{vh}I_v}{N_v}, \text{ and, } \lambda_v = \frac{\beta_{hv}I_h}{N_h}, \tag{2.2}$$

thus, substituting N_v from (2.1) in (2.2) gives,

$$\lambda_h = \frac{\beta_{hv}I_v}{N_h}, \text{ and, } \lambda_v = \frac{\beta_{hv}I_h}{N_h}. \tag{2.3}$$

Note that $\frac{I_v}{N_v}$ and $\frac{I_h}{N_h}$ in (2.2) represent probabilities that contact were made with infectious mosquitoes and humans respectively.

2.3 Model formulation

Population of susceptible humans is generated through immigration and birth at a rate given by r_h. It decreases as a result of infection through contact with infectious mosquitoes at a rate β_{vh}. They naturally die at a rate μ_h. So that,

$$\frac{dS_h}{dt} = r_h - \frac{\beta_{vh}I_v}{N_v}S_h - \mu_h S_h.$$

Infected humans (I_h) increase through infection of susceptible humans. They recover at a rate ω_h and due to disease induced death at a rate δ_h, and naturally at the rate μ_h. Thus,

$$\frac{dI_h}{dt} = \frac{\beta_{vh}I_v}{N_v}S_h - \delta_h I_h + \omega_h I_h + \mu_h I_h.$$

Recovered human population increase through the recovery of infected humans, and reduces due to natural death. So that

$$\frac{dR_h}{dt} = \omega_h I_h - \mu_h R_h.$$

Non-infected aquatic mosquitoes population (A_n) is generated via oviposition by susceptible (S_v) or infectious (I_v) mosquitoes at ρ_v and $\zeta_v\rho_v$ respectively, where ρ_v is the oviposition rate while ζ_v is a proportion of non-infected eggs laid by infected mosquitoes. It decreases through maturation at a rate b_v, die naturally at μ_A and due to density death (growing logistically with $\mathcal{K}$ as their carrying capacity). Similar to the assumptions in [13, 29], $\mathcal{K} \propto N_h$; $\mathcal{K} = mN_h$, thus,

$$\frac{dA_n}{dt} = \rho_v(1 - \frac{A}{\mathcal{K}})(S_v + \zeta_v I_v) - b_v A_n - \mu_A A_n$$

Infected aquatic mosquitoes (A_i) are generated via laying of eggs by infectious mosquitoes, this leads to transmission of the disease vertically at a rate $1 - \zeta_v$. They mature at the rate b_v and die naturally at μ_A. This gives

$$\frac{dA_i}{dt} = \rho_v(1 - \frac{A}{\mathcal{K}})(1 - \zeta_v)I_v - b_v A_i - \mu_A A_i.$$

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where, $A = A_n + A_i$.
Through maturation of non-infected aquatic mosquitoes, population of susceptible adult mosquitoes (S_v) is generated, they decrease through infection at the rate λ_v, and natural death (at μ_v). So that

$$\frac{dS_v}{dt} = b_v A_n - \frac{\beta_{hv} I_h}{N_h} S_v - \mu_v S_v.$$

Infectious adult mosquitoes population (I_v) is generated by maturation of infected aquatic mosquitoes and infection of susceptible mosquitoes. They decrease due to natural death, thus

$$\frac{dI_v}{dt} = \frac{\beta_{hv} I_h}{N_h} S_v + b_v A_i - \mu_v I_v.$$

2.4 Model equations

The following non-linear system of equations represent the model for the transmission of mosquito borne diseases with vertical transmission in mosquito population.

$$\begin{aligned}
\frac{dS_h}{dt} &= r_h - \frac{\beta_{vh} I_v}{N_v} S_h - \mu_h S_h, \\
\frac{dI_h}{dt} &= \frac{\beta_{vh} I_v}{N_v} S_h - (\delta_h + \omega_h + \mu_h) I_h, \\
\frac{dR_h}{dt} &= \omega_h I_h - \mu_h R_h, \\
\frac{dA_n}{dt} &= \rho_v (1 - \frac{A}{\mathcal{K}})(S_v + \zeta_v I_v) - b_v A_n - \mu_A A_n, \\
\frac{dA_i}{dt} &= \rho_v (1 - \frac{A}{\mathcal{K}})(1 - \zeta_v) I_v - b_v A_i - \mu_A A_i, \\
\frac{dS_v}{dt} &= b_v A_n - \frac{\beta_{hv} I_h}{N_h} S_v - \mu_v S_v, \\
\frac{dI_v}{dt} &= \frac{\beta_{hv} I_h}{N_h} S_v + b_v A_i - \mu_v I_v.
\end{aligned} \tag{2.4}$$

The model variables are described in Table 1, while the model parameters are in Table 2 and assumed to be positive with non-negative initial conditions.

3 Model analysis

Basic analysis of the model (2.4) with initial condition and non-negative parameters would be conducted in this section.

Lemma 3.1. *The following set denoted by Ω defines a biologically feasible region which is positively invariant with respect to the model given by (2.4),*

$$\begin{aligned}
\Omega = \Big\{ S_h, I_h, R_h, A_n, A_i, S_v, I_v \in \mathbb{R}^7_+ : \frac{r_h}{\delta_h + \mu_h} \leq N_h \leq \frac{r_h}{\mu_h}, \quad A_n \leq \mathcal{K}, \quad A_i \leq \mathcal{K}, \\
S_v + I_v \leq \frac{\mathcal{K} b_v}{\mu_v} \Big\}.
\end{aligned} \tag{3.1}$$

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Table 1: Variables of the model given by (2.4) and their descriptions.

Variables	Description
S_h	Total number of susceptible humans
I_h	Total number of infectious humans
R_h	Total number of recovered humans
N_h	Cumulative number of humans
A_n	Total number of non-infected aquatic mosquitoes
A_i	Total number of infected aquatic mosquitoes
S_v	Total number of susceptible mosquitoes
I_v	Total number of infectious mosquitoes
A	Cumulative number of aquatic mosquitoes
N_v	Cumulative number of adult female mosquitoes

Proof. Clearly, the system given by (2.4) is C^1 in $\mathbb{R}^7_+$, therefore, the local existence and uniqueness of solution follow. Likewise, since at any time, $A_n(t) + A_i(t) \leq \mathcal{K}$ then $A_n(t) \leq \mathcal{K}$ and $A_i(t) \leq \mathcal{K}$. Let $N_v = S_v + I_v$, then by Gronwall's lemma we have

$$N_h(0)e^{-(\delta_h+\mu_h)} + \frac{r_h}{(\delta_h+\mu_h)}(1-e^{-l_h}) \leq N_h(t) \leq N_h(0)e^{-\mu_h} + \frac{r_h}{\mu_h}(1-e^{-\mu_h})$$

and (3.2)

$$N_V(t) \leq N_V(0)e^{-\mu_v} + \frac{\mathcal{K}r_h}{\mu_v}(1-e^{-\mu_v}),$$

hence, from (3.2), the total populations are bounded and thus, solutions exist for all $t \geq 0$. In addition, $N_h(t) \geq \frac{r_h}{\delta_h+\mu_h}$ if $N_h(0) \geq \frac{r_h}{\delta_h+\mu_h}$, $N_h(t) \leq \frac{r_h}{\mu_h}$ if $N_h(0) \leq \frac{r_h}{\mu_h}$, and $N_v(t) \leq \frac{\mathcal{K}b_v}{\mu_v}$ if $N_v(0) \leq \frac{\mathcal{K}b_v}{\mu_v}$. Consequently, the unique solution to the model given by (2.4) with initial conditions in Ω remains in Ω for all $t \geq 0$.

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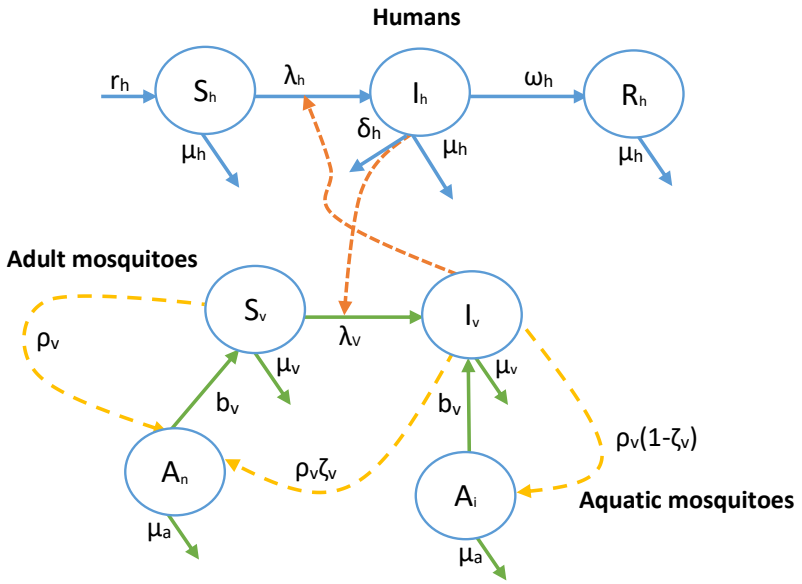

Figure 1: Model diagram for the model given by (2.4).

3.1 Mosquito only population

Here, we analyse the sub-model obtained by removing human population (mosquito only component) from the model given by (2.4). Therefore, in the absence of humans, the mosquito component reduced to

$$\begin{aligned}
\frac{dA_n}{dt} &= \rho_v(1-\frac{A}{\mathcal{K}})(S_v+\zeta_v I_v) - b_v A_n - \mu_A A_n,\\
\frac{dA_i}{dt} &= \rho_v(1-\frac{A}{\mathcal{K}})(1-\zeta_v) I_v - b_v A_i - \mu_A A_i,\\
\frac{dS_v}{dt} &= b_v A_n - \lambda_v S_v - \mu_v S_v,\\
\frac{dI_v}{dt} &= \lambda_v S_v + b_v A_i - \mu_v I_v.
\end{aligned} \tag{3.3}$$

In the presence of abundant resources, and space (i.e. $A < \mathcal{K}$), on average, throughout the lifespan of a female mosquito, it would produce a certain number of offspring often called the basic offspring

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number and denoted by N_0. Thus, N_0 is given by,

$$N_0 = \frac{b_v \rho_v}{(b_v + \mu_A)\mu_v}. \tag{3.4}$$

Interpretation of the basic offspring number: Aquatic female mosquitoes will mature to adulthood with a probability given by $\frac{b_v}{b_v+\mu_A}$, where $\frac{1}{b_v+\mu_A}$ represents an average period at the aquatic stage, and b_v is the maturation rate of aquatic mosquitoes to adulthood. The adult mosquito has an average life expectancy of $\frac{1}{\mu_v}$, with ρ_v being their rate of oviposition, so that, on average, a female mosquito would lay $\frac{\rho_v}{\mu_v}$ number of eggs. Therefore on average, equation (3.4) represents the number of offspring that a susceptible female mosquito would produce during her life time, named the basic offspring number.

On equating the right hand side of the sub-model given by (3.3) we obtained an extinction DFE given by $\mathcal{G}_0$, and non-extinction DFE also denoted by $\mathcal{G}_1$ as follows

$$\mathcal{G}_0 = (A_n^*, A_i^*, S_v^*, I_V^*) = (0, 0, 0, 0), \tag{3.5}$$

and

$$\mathcal{G}_1 = (A_n^*, A_i^*, S_v^*, I_V^*) = (\mathcal{K}(1 - \frac{1}{N_0}), 0, \frac{b_v \mathcal{K}}{\mu_v}(1 - \frac{1}{N_0}), 0). \tag{3.6}$$

For stability of $\mathcal{G}_0$ and $\mathcal{G}_1$, the following Theorem from [30] and some references therein is essential.

Consider an autonomous dynamical system given by $\dot{x} = k(x)$, where $\Omega^* \subseteq \mathbb{R}_+^n$ and $k : \Omega^* \longrightarrow \mathbb{R}_+^n$ is continuous [15, 30]. Then

Theorem 3.2. *Let $e, f \in \Omega^*$ be such that $e < f$, $[e, f] \subseteq \Omega^*$ and $k(f) \leq 0 \leq k(e)$. Then the system defines a (positive) dynamical system on $[e, f]$. Moreover, if $[e, f]$ contains a unique equilibrium j, then j is globally asymptotically stable on $[e, f]$ [15, 30].*

The sub-model given by (3.3) can be written in form of $\dot{x} = k(x)$, where $k : \Omega^* \longrightarrow \mathbb{R}_+^4$ and $\Omega^* \subseteq \mathbb{R}_+^4$. Consequently, we have

Theorem 3.3. *The extinction DFE given by $\mathcal{G}_0$ is GAS provided $N_0 \leq 1$ and unstable when $N_0 > 1$. The non-extinction DFE given by $\mathcal{G}_1$ exists and it is LAS whenever $N_0 > 1$.*

Proof. Consider $[e, f] = [0, f] \subseteq \mathbb{R}_+^4$, where $f = (p, \frac{(b_v+\mu_A)p}{\rho_v})$ and $p > 0$. Thus, $k(0) = 0$, and

$$k(f) = \begin{pmatrix} -(b_v + \mu_A)\frac{p^2}{\mathcal{K}} \\ \\ b_v p(1 - \frac{1}{N_0}) \end{pmatrix} \text{ therefore } k(f) < 0 \text{ provided } N_0 \leq 1. \tag{3.7}$$

Thus, $k(f) \leq 0 \leq k(0)$ provided that $N_0 < 1$, and by Theorem (3.2) above, the mosquito only model given by (3.3) is a positive dynamical system that is defined on $[0, f]$ and therefore, $\mathcal{G}_0$ is globally asymptotically stable on $[0, f]$. Moreover, because p is arbitrary, there is no restriction on the choice of f, it can extend to any number bigger than $x \in \mathbb{R}_+^2$. Thus, the result stands on $\mathbb{R}_+^2$. Through linearization, the second part follows.

Biologically, based on Theorem (3.3), provided N_0 is less one, the mosquito population would go extinct and both vertical and horizontal transmissions of MBDs can be avoided.

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3.2 Disease free equilibrium when $N_0 < 1$

Denote by $\mathcal{G}_2$, the DFE of the model given by (2.4) which is obtained whenever $N_0 < 1$, such that,

$$\mathcal{G}_2 = (S_h^*, E_h^*, I_h^*, R_h^*, A_n^*, A_i^*, S_v^*, E_V^*, I_V^*) = (\frac{r_h}{\mu_h}, 0, 0, 0, 0, 0, 0, 0). \tag{3.8}$$

Using the next generation matrix approach as described by [31], local asymptotic stability of $\mathcal{G}_2$ is established. The approach described by [10, 27, 31, 32] is used in computing the next generation matrix (K).

The matrix for new infection is denoted by F, while that of transmission terms is denoted by V. They are respectively given by,

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_{hv} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\rho_v(1-\zeta_v)}{N_0} \\ 0 & 0 & 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} S_1 & 0 & 0 & 0 \\ -\omega_h & \mu_h & 0 & 0 \\ 0 & 0 & S_2 & 0 \\ 0 & 0 & -b_v & \mu_v \end{pmatrix}, \tag{3.9}$$

where, $S_1 = \omega_h + \mu_h + \delta_h$, $S_2 = \mu_A + b_v$.

The next generation matrix with large domain (K_L) is

$$\begin{aligned} K_L = FV^{-1} &= \begin{pmatrix} 0 & 0 & 0 & \beta_{hv} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\rho_v(1-\zeta_v)}{N_0} \\ 0 & 0 & 0 & 0 \end{pmatrix} \times \begin{pmatrix} \frac{1}{S_1} & 0 & 0 & 0 \\ \frac{\omega_h}{S_1\mu_h} & \frac{1}{\mu_h} & 0 & 0 \\ 0 & 0 & \frac{1}{S_2} & 0 \\ 0 & 0 & \frac{b_v}{S_2\mu_v} & \frac{1}{\mu_v} \end{pmatrix} \\ &= \begin{pmatrix} 0 & 0 & \frac{\beta_{hv}b_v}{S_2\mu_v} & \frac{\beta_{hv}}{\mu_v} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\rho_v(1-\zeta_v)b_v}{N_0S_2\mu_v} & \frac{\rho_v(1-\zeta_v)}{N_0\mu_v} \\ 0 & 0 & 0 & 0 \end{pmatrix}. \end{aligned} \tag{3.10}$$

Therefore, using the method of [32] where Q is an auxiliary matrix, the next generation matrix (K) is

$$K = Q^T K_L Q = Q^T F V^{-1} Q = \begin{pmatrix} 0 & \frac{\beta_{hv}b_v}{S_2\mu_v} \\ 0 & \frac{\rho_v(1-\zeta_v)b_v}{N_0S_2\mu_v} \end{pmatrix}, \quad \text{where } Q = \begin{pmatrix} 1 & 0 \\ 0 & 0 \\ 0 & 1 \\ 0 & 0 \end{pmatrix}. \tag{3.11}$$

Unlike K_L (the next generation matrix with large domain), the next generation matrix (K) removes unnecessarily information and hence, the threshold obtained can easily be given a detailed biological interpretation [32]. For mosquito extinction DFE, the dominant eigenvalue of K denoted by $\mathcal{R}_1$ is given by

$$\mathcal{R}_1 = \frac{\rho_v(1-\zeta_v)b_v}{N_0(b_v+\mu_A)\mu_v} = 1 - \zeta_v, \text{ with, } 0 < 1 - \zeta_v < 1.$$

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Table 2: Parameters for the model given by (2.4) and their descriptions.

Parameter	Description
ρ_v	Rate of adult mosquitoes oviposition
μ_A	Rate of natural death of aquatic mosquitoes
μ_v	Adult mosquito population natural death
ζ_v	Proportion of non-vertically transmitted laid eggs
$\mathcal{K}$	Carrying capacity of mosquito population
m	Ratio of mosquitoes to humans
b_v	Rate at which mosquitoes mature
r_h	Rate of human recruitment
ω_h	Rate of recovery in human population
μ_h	Rate of natural death in human population
δ_h	Death rate due to disease in human population
b_{vh}	Bite rate of infectious mosquitoes
b_{hv}	Bite rate of susceptible mosquitoes
ρ_{vh}	Transmission probability from I_v to S_h
ρ_{hv}	Transmission probability from I_h to S_v
β_{vh}	Rate of transmissions from I_v to S_h
β_{hv}	Rate of transmission from I_h to S_v

3.3 Disease free equilibrium when $N_0 > 1$

If the DFE of the model given by (2.4) when $N_0 > 1$ is denoted by $\mathcal{G}_3$ be , then

$$\mathcal{G}_3 = (S_h^*, I_h^*, R_h^*, A_n^*, A_i^*, S_v^*, I_v^*) = (\frac{r_h}{\mu_h}, 0, 0, \mathcal{K}(1 - \frac{1}{N_0}), 0, \frac{b_v}{\mu_v}\mathcal{K}(1 - \frac{1}{N_0}), 0) \tag{3.12}$$

using similar method as above, the next generation matrix (K) is given by

$$K = \begin{pmatrix} 0 & \frac{\beta_{hv}S_h^*}{N_h^*\mu_v} \\ \frac{\beta_{hv}S_v^*}{N_h^*S_1} & \frac{\rho_v(1-\zeta_v)b_v}{N_0S_2\mu_v} \end{pmatrix} = \begin{pmatrix} 0 & \mathcal{R}_{vh} \\ \mathcal{R}_{hv} & \mathcal{R}_{vv} \end{pmatrix}. \tag{3.13}$$

Thus, the dominant eigenvalue of K, which is the basic reproduction number denoted by $\mathcal{R}_0$ is given by

$$\mathcal{R}_0 = \frac{1}{2}(\mathcal{R}_{vv} + \sqrt{\mathcal{R}_{vv}^2 + 4\mathcal{R}_{hv}\mathcal{R}_{vh}}), \tag{3.14}$$

where, $\mathcal{R}_{hv} = \frac{\beta_{hv}S_v^*}{N_h^*S_1}$, $\mathcal{R}_{vh} = \frac{\beta_{hv}S_h^*}{N_h^*\mu_v} = \frac{\beta_{hv}}{\mu_v}$, and $\mathcal{R}_{vv} = 1 - \zeta_v$.

Lemma 3.4. *The mosquito persistent DFE of the model denoted by $\mathcal{G}_3$ is locally asymptotically stable when $\mathcal{R}_0 < 1$, and unstable otherwise [31].*

3.3.1 Interpretation of $\mathcal{R}_0$

The basic reproduction number $(\mathcal{R}_0)$ represents an average number of new secondary cases that is generated by a single infectious individual (throughout its infectivity period) when it is introduced into a wholly susceptible population of humans and mosquitoes. $\mathcal{R}_0$ is interpreted as follows.

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Susceptible humans would get infection through contact (bite) with an infected mosquitoes, such bite should be capable of transmitting the disease. So that, for an infected mosquito, the number of infections it generates (near DFE) is given by the product of its rate of infection ($\frac{\beta_{hv}}{N_h^*}$) with the average time it spends in the infectious stage ($\frac{1}{\mu_v}$). Thus, (with $S_h^* = N_h^*$)

$$\mathcal{R}_{hv} = \frac{\beta_{hv}}{N_h^* \mu_v} S_h^* = \frac{\beta_{hv}}{\mu_v}. \tag{3.15}$$

Likewise, a susceptible mosquito acquires infection through bite on an infectious human (I_h). So that, the number of infections that an adult susceptible mosquito would acquire from an infectious human (near the DFE) is equivalent to the product of infection rate of infectious humans ($\frac{\beta_{hv}}{N_h^*}$) and the average period of infectivity ($\frac{1}{S_1}$). Therefore

$$\mathcal{R}_{vh} = \frac{\beta_{hv}}{N_h^* S_1} S_v^* \tag{3.16}$$

Some percentage of eggs laid per oviposition by infectious mosquitoes would be infected, thus, that would give the number of mosquito vertical transmissions (which occurs at the point of lay) given by ($\mathcal{R}_{vv} = 1 - \zeta_v$). Therefore, the basic reproduction number is given by (3.14).

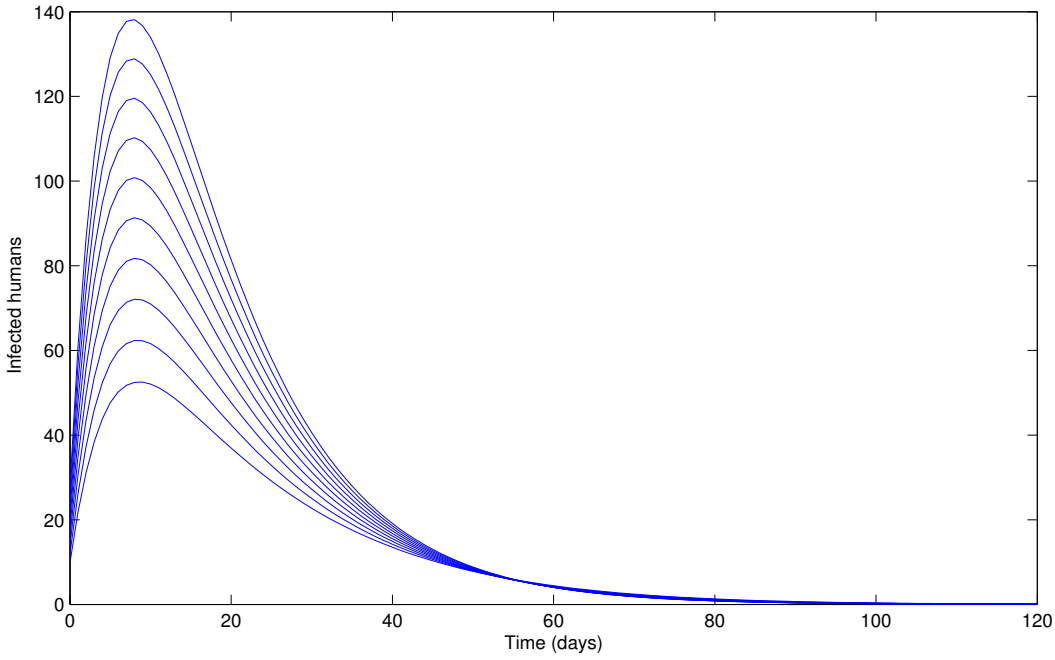

Figure 2: Simulation showing infected humans with different initial conditions converging to the DFE when $\mathcal{R}_0 < 1$.

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3.4 Type reproduction number

In the case of non-vector borne diseases (where a population under study is homogeneous), the basic reproduction number defines the threshold quantity that can easily be used to control or eliminate the disease under consideration. It may not necessarily be the case for a vector borne disease (heterogeneous population), in fact, the basic reproduction number may not be important especially if effort in control is targeted at a particular population, or as cycle of infection goes through other populations [33]. Thus, it is important to calculate another threshold quantity called the type reproduction number.

Here, we compute another threshold quantity that can be used in correctly determining critical control effort needed for a heterogeneous population known as the type-reproduction number (T) [34]. A strategy to estimating effort(s) needed to control an infectious disease by targeting a specific sub-population of hosts, with the fact that infection will pass through other sub-populations before causing secondary infections was described by [15, 33, 34].

If K denote the next generation matrix with large domain, and the host type 1 denote the populations of I_h, while the host type 2 denote the population of I_v. The type j reproduction number is given by

$$T_j = g^T K(I-(I-M)K)^{-1}g, \tag{3.17}$$

where I and M are respectively an identity and projection matrices, g is a unit vector with all elements equal to zero except the jth. Let

$$K = \begin{pmatrix} 0 & 0 & K_{13} & K_{14} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & K_{33} & K_{34} \\ K_{41} & 0 & 0 & 0 \end{pmatrix},$$

where,

$$K_{13} = \frac{\beta_{hv} b_v}{S_2 \mu_v}, \quad K_{14} = \frac{\beta_{hv}}{\mu_v},$$
$$K_{33} = \frac{\rho_v(1-\zeta_v)b_v}{N_0 S_2 \mu_v}, \quad K_{34} = \frac{\rho_v(1-\zeta_v)}{N_0 \mu_v}, \quad K_{41} = \frac{\beta_{hv} S_v^*}{N_h^* S_1}.$$

So that from (3.17) the type-reproduction number for infectious human is given by

$$T_1 = \frac{K_{13}K_{34}K_{41}}{1-K_{33}} + K_{41}K_{14} = \frac{\mathcal{R}_{hv}\mathcal{R}_{vh}}{1-\mathcal{R}_{vv}} = \frac{\mathcal{R}_{hv}\mathcal{R}_{vh}}{\zeta_v} = \frac{S_v^* \beta_{hv}^2}{N_h^* S_1 \mu_v \zeta_v}. \tag{3.18}$$

The threshold above is the expected number of cases in the human population that is caused by one infectious human in a completely susceptible population, the infection might be through chains of infections or directly, it singles out the required control effort when targeting the human population [15, 34].

If $\mathcal{R}_0 > 1$, It can easily be shown that,

$$\mathcal{R}_{hv}\mathcal{R}_{vh} > 1 - \mathcal{R}_{vv} = \zeta_v.$$

Similarly, $\mathcal{R}_0 < 1$ implies that

$$\mathcal{R}_{hv}\mathcal{R}_{vh} < 1 - \mathcal{R}_{vv} = \zeta_v.$$

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Table 3: Parameter values used in numerical calculations with their references, with low baseline values that gives $R_0 < 1$, while $R_0 > 1$ for the high baseline

Parameter	Range	Low baseline	High baseline	References
r_h	$1 - 10^3$ day^{-1}	30 day^{-1}	10 day^{-1}	[11, 26, 36]
μ_h	$0.02 - 0.05$ years	0.015 years	0.02 years	[4, 12, 13]
δ_h	$0 - 10^{-3}$ day^{-1}	0.001 day^{-1}	0.001 day^{-1}	[11]
b_{vh}	$0.1 - 1$ day^{-1}	0.3 day^{-1}	0.5 day^{-1}	[4, 11, 42]
b_{hv}	$0.1 - 1$ day^{-1}	0.414 day^{-1}	0.823 day^{-1}	[11, 42]
ρ_{vh}	$0.1 - 0.75$ day^{-1}	0.25 day^{-1}	0.55 day^{-1}	[41, 42]
ρ_{hv}	$0.5 - 1$ day^{-1}	0.35 day^{-1}	0.45 day^{-1}	[41, 42]
β_{hv}	$0.2 - 1$ day^{-1}	0.145 day^{-1}	0.37 day^{-1}	[11, 41]
ω_h	$0.07 - 0.33$ day^{-1}	0.14 day^{-1}	0.08 day^{-1}	[40]
m	$1 - 10$	2	5	[29, 41]
ζ_v	$0 - 1$ day^{-1}	0.95 day^{-1}	0.90 day^{-1}	[14, 21]
ρ_v	$1 - 14$ day^{-1}	4 day^{-1}	6 day^{-1}	[12, 13]
b_v	$0.05 - 0.5$ day^{-1}	0.05 day^{-1}	0.1 day^{-1}	[13, 29]
μ_A	$0.25 - 0.33$ days	0.3 days	0.2 days	[12, 13]
μ_v	$0.03 - 0.25$ days	0.14 days	0.05 days	[41, 42]

Thus, $T_1 < 1 \Longleftrightarrow \mathcal{R}_0 < 1$. In the same vain, the type-reproduction number for infected mosquitoes is

$$T_2 = \frac{K_{14}K_{41}}{1 + K_{14}K_{41}} = \frac{\mathcal{R}_{hv}\mathcal{R}_{vh}}{1 + \mathcal{R}_{hv}\mathcal{R}_{vh}}. \tag{3.19}$$

It is the expected number of cases within the adult mosquito population that is caused by one infected adult mosquito in a population of susceptible mosquitoes. It is straightforward to see that $T_2 < 1$.

3.5 Endemic equilibrium and backward bifurcation

Here, the endemic equilibrium (EE) of the model (2.4) would be computed, and direction of bifurcation at $\mathcal{R}_0 = 1$ is also analyzed.

3.5.1 Endemic equilibrium

By letting

$$A_i + A_n = A$$

and

$$\lambda_h^{**} = \frac{\beta_{hv} I_V^{**}}{N_h^{**}}, \quad \lambda_V^{**} = \beta_{hv} \frac{I_h^{**}}{N_h^{**}},$$

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it can be shown that the model given by (2.4) has a unique endemic equilibrium provided that $N_0 > 1$ given by

$$\begin{aligned}
S_h^{**} &= \frac{r_h}{\lambda_h^{**} + \mu_h}, \quad I_h^{**} = \frac{\lambda_h^{**} r_h}{S_1(\lambda_h^{**} + \mu_h)}, \quad R_h^{**} = \frac{\lambda_h^{**} r_h \omega_h}{S_1(\lambda_h^{**} + \mu_h)\mu_h}, \\
A^{**} &= \frac{\mathcal{K} S_2 \mu_v (N_0 - 1)}{b_v}, \quad A_n^{**} = \frac{\rho_v (1 - \frac{A^{**}}{\mathcal{K}})[S_V^{**} + \zeta_v I_V^{**}]}{S_2}, \\
A_i^{**} &= \frac{\rho_v (1 - \frac{A^{**}}{\mathcal{K}})[1 - \zeta_v] I_V^{**}}{S_2}, \quad S_V^{**} = \frac{b_v \rho_v (1 - \frac{A^{**}}{\mathcal{K}})[S_V^{**} + \zeta_v I_V^{**}]}{(\lambda_V^{**} + \mu_v) S_2}, \\
I_V^{**} &= \frac{\lambda_V^{**} S_2 b_v A^{**} + b_v \mu_v \rho_v (1 - \frac{A^{**}}{\mathcal{K}})[1 - \zeta_v] I_V^{**}}{(\lambda_V^{**} + \mu_v) S_2 \mu_v},
\end{aligned} \tag{3.20}$$

where

$$N_h^{**} = S_h^{**} + I_h^{**} + R_h^{**}.$$

It is worth mentioning that there are many mosquito born disease models that were shown to undergo backward bifurcation (a phenomenon where stable DFE coexists with a stable EE even when $\mathcal{R}_0 < 1$), some of which include [8, 9, 11, 15, 35, 37]. Thus, we explore the existence or otherwise of it.

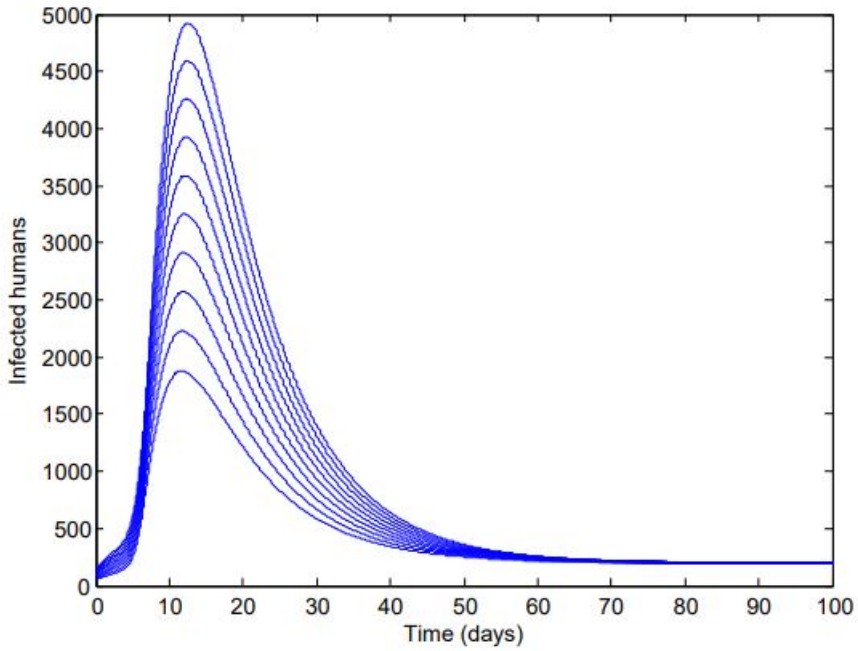

Figure 3: Simulation showing infected humans converging to the EE when $\mathcal{R}_0 > 1$ with multiple initial conditions.

3.5.2 Backward bifurcation analysis

By applying the method described by [31, 38]. It is based on center manifold theory.

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Theorem 3.5. *The model given by (2.4) undergoes BB at $\mathcal{R}_0 = 1$ whenever the bifurcation coefficient $\boldsymbol{a}$ given by (A.4) is positive.*

Proof of the Theorem is in Appendix A.

The epidemiological implication of the backward bifurcation is that, the classical requirement that $\mathcal{R}_0 < 1$ is sufficient for disease elimination does not stands. Although it is still a necessarily condition, but it is not sufficient to eliminate dengue. In such situation, elimination of dengue in a population would depend on the initial sizes of the sub-populations given in the model [11].

Theorem 3.6. *The model (2.4) does not undergoes backward bifurcation at $\mathcal{R}_0 = 1$ if $\delta_h = 0$.*

Proof. Note that, if $\delta_h = 0$ then $S_1 = \omega_h + \mu_h$, and,

$$\frac{5(\mu_h+\omega_h)}{\mu_h} - \frac{3S_1}{\mu_h}\left(\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{(1-\mathcal{R}_{VV})}\right) = \frac{(\mu_h+\omega_h)}{\mu_h}\left(5 - 3\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{(1-\mathcal{R}_{VV})}\right),$$

and since $\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{(1-\mathcal{R}_{VV})} \leq 1$, then,

$$\mathbf{a} \leq \frac{-2w_2^2 v_2}{N_h^* \mathcal{K}}\left(\frac{S_v^* 8\beta_{hv}^2 \mathcal{K}}{\mu_v \zeta_v} + \frac{\beta_{hv}^2 S_v^* \mathcal{K} S_1}{\mu_v \mu_h \zeta_v} + \beta_{hv} S_v^* \mathcal{K}\frac{S_1}{\mu_h}\left(5 - 3\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{(1-\mathcal{R}_{VV})}\right)\right) < 0 \tag{3.21}$$

The result of the Corollary (3.6) above is similar to that obtained numerically by Chitnis et. al [9] in their Malaria model without incorporating aquatic stages of mosquitoes.

3.6 Global stability of the DFE ($\mathcal{G}_3$)

Using the method of [39], conditions for which the disease free equilibrium ($\mathcal{G}_3$) will be globally asymptotically stable with respect to the invariant region Ω defined by (3.1) are obtained.

Theorem 3.7. *The disease free equilibrium ($\mathcal{G}_3$) of the model (2.4) with respect to Ω is GAS if $\mathcal{R}_{HV}\mathcal{R}_{VH}\frac{N_h^* N_0(\delta_h+\mu_h)}{r_h(N_0-1)} + \mathcal{R}_{VV} \leq 1$.*

Notice that $\mathcal{R}_{HV}\mathcal{R}_{VH}\frac{N_h^* N_0(\delta_h+\mu_h)}{r_h(N_0-1)} + \mathcal{R}_{VV} \leq 1$ is equivalent to $\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{(1-\mathcal{R}_{VV})} \leq \frac{r_h(N_0-1)}{N_h^* N_0(\delta_h+\mu_h)}$, since $\frac{r_h}{\delta_h+\mu_h} \leq N_h^*$ and $N_0 - 1 < N_0$, then $\mathcal{R}_{HV}\mathcal{R}_{VH}\frac{N_h^* N_0(\delta_h+\mu_h)}{r_h(N_0-1)} + \mathcal{R}_{VV} \leq 1$ suffices that $\mathcal{R}_0 \leq 1$.

Next, we look into a case for another positively invariant set $\Omega^* \subset \Omega$ given as follows.

Theorem 3.8. *The disease free equilibrium ($\mathcal{G}_3$) of the model (2.4) with respect to Ω^* is globally asymptotically stable if $\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{(1-\mathcal{R}_{VV})} \leq \frac{r_h}{N_h^*(\delta_h+\mu_h)}$, where,*

$$\Omega^* = \{S_h, I_h, R_h, A_n, A_i, S_v, I_v \in \mathbb{R}_+^7 : \frac{r_h}{\delta_h+\mu_h} \leq N_h \leq \frac{r_h}{\mu_h}, \quad A_n \leq \mathcal{K}, \quad A_i \leq \mathcal{K},$$
$$S_v \leq S_v^* = \frac{\mathcal{K}b_v}{\mu_v}(1-\frac{1}{N_0}), \quad \leq S_v + I_v \leq \frac{\mathcal{K}b_v}{\mu_v}\}.$$

Corollary 3.9. *The disease free equilibrium ($\mathcal{G}_3$) of the model given by (2.4) in respect of Ω^* is GAS whenever $\delta_h = 0$, and $\mathcal{R}_0 \leq 1$.*

If $\delta_h = 0$, the condition reduces to $\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{1-\mathcal{R}_{VV}} \leq 1$. It should also be observed that for the subset Ω^*, $\bar{A}_{22} = A_{22}(\bar{x}_1, 0)$. Thus, the condition for GAS of $\mathcal{G}_3$ is $\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{1-\mathcal{R}_{VV}} \leq 1 \implies \mathcal{R}_0 \leq 1$.

Similar proof has been done in [39].

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4 Numerical simulation and sensitivity analysis

Here, some simulations are conducted for the model (2.4), and sensitivity analysis (both local and global) for the basic reproduction number ($\mathcal{R}_0$) are performed with respect to the model parameters. Values for parameter ranges are given in Table 3 for high ($\mathcal{R}_0 > 1$) and low ($\mathcal{R}_0 < 1$) transmissions are used.

4.1 Numerical simulations

Using parameter values as presented in Table 3, the low baseline referred to the case when $\mathcal{R}_0 < 1$ and $N_0 = 3$, while high transmission baseline is when $\mathcal{R}_0 > 1$ and $N_0 = 34$. Some numerical simulations for the model (2.4) are performed for the two baseline parameter values. Figure 3 show the simulation of infected humans (I_h) with different initial conditions approaching the endemic equilibrium when $\mathcal{R}_0 > 1$, while Figure 2 depict simulation of the model showing population of infected humans (I_h) approaching the disease free equilibrium when $\mathcal{R}_0 < 1$.

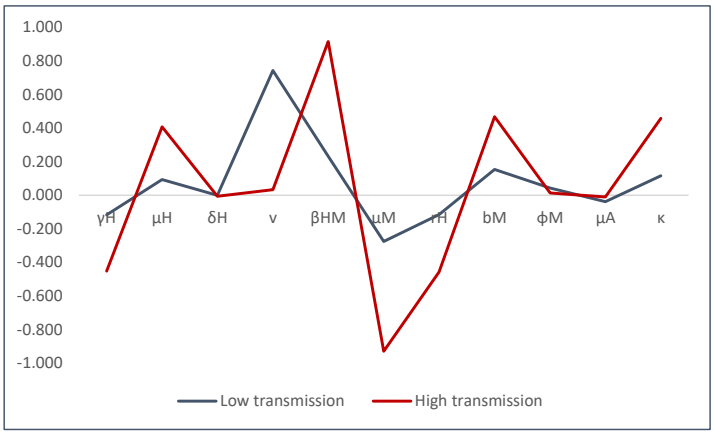

Figure 4: Plot for the local sensitivity indices of $\mathcal{R}_0$ for the model parameters with $\mathcal{R}_0 < 1$(low transmission) and $\mathcal{R}_0 > 1$ (high transmission). Where $v = 1 - \zeta_v$.

4.2 Local sensitivity analysis of $\mathcal{R}_0$

Relative change in a function due to parameter changes (local sensitivity indices) can be measured using elasticity index [44, 45]. Given a multi-variable function f and any arbitrary parameter

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say ω, the elasticity index is given by

$$\Upsilon_{\omega}^{f} = \frac{\partial f}{\partial \omega} \times \frac{\omega}{f}. \tag{4.1}$$

With the explicit value for the basic reproduction number, the above formula (4.1) is used to analyze elasticity (local sensitivity) indices of $\mathcal{R}_0$ with respect to the parameters of model (2.4).

With the use of parameter values presented in Table 3, sensitivity index of the basic reproduction number for both low and high transmission are obtained. For the sake of easy comparison, a plot for the sensitivity index is given in Figure 4. This index is obtained when other parameters are kept constant, thus, there is need for more robust sensitivity index.

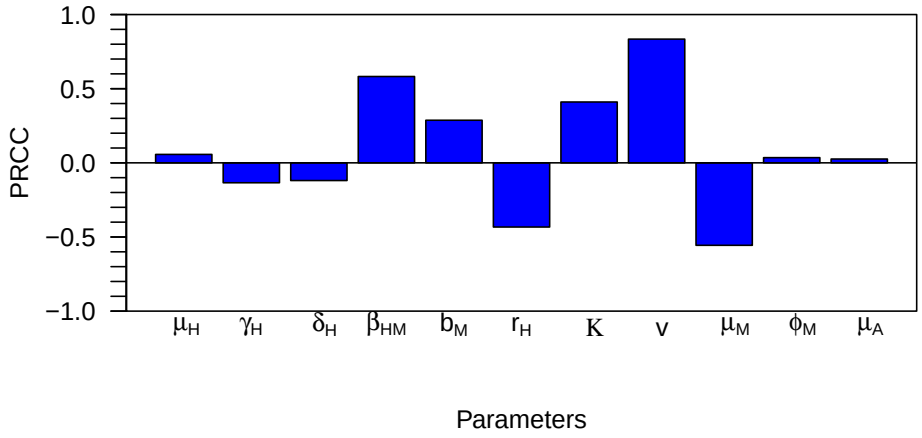

Figure 5: Partial Rank Correlation Coefficients (PRCC) of $\mathcal{R}_0$ for the model parameters.

4.3 Global sensitivity analysis

Although easier to compute, but because local sensitivity indices are more suited for parameters known with certainty, it is necessarily to perform a more reliable analysis which allows for the variation of other parameters as effect of a particular parameter is gauged.

Using parameter ranges given in Table 3, the partial rank correlation coefficient (PRCC) of the model parameters are computed and presented in Figure 5 with the basic reproduction number as the R_0. Likewise Table 4 shows the sensitivity index, bias, error and confidence interval for each parameter.

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Table 4: Partial Rank Correlation Coefficients (PRCC) of the parameters (Par) of $\mathcal{R}_0$ where $v = 1 - \zeta_v$.

Par	Sensitivity	Bias	Std error	Min. C.I	Max. C.I
μ_h	+0.0568229	-0.001056	0.03434093	-0.0075356	+0.12541
ω_h	-0.13438731	-0.000729	0.03080668	-0.1971379	-0.074570
δ_h	-0.11941843	+0.00178	0.03205131	-0.1877505	-0.058661
β_{hv}	+0.5822655	-0.000888	0.01898839	+0.546284	+0.62179
b_v	+0.2872719	-0.000173	0.02971285	+0.227748	+0.34781
r_h	-0.43285879	-0.000464	0.02730825	-0.4861036	-0.382255
κ	+0.4103448	+0.00078	0.02517658	+0.360995	+0.46169
v	+0.8342995	+0.00015	0.01241340	+0.810207	+0.85844
μ_v	-0.55622995	-0.000434	0.02038602	-0.5970142	-0.514089
ρ_v	+0.0351035	-0.000558	0.03430478	-0.0298447	+0.10249
μ_A	+0.0257274	-0.002854	0.03544698	-0.0403328	+0.09836

Conclusion

The dengue model that incorporates both mosquito vertical transmission is constructed and analyzed. Major findings include.

- There exists a threshold quantity for the mosquito population called the basic offspring number (N_0). It plays major role in extinction or otherwise of the mosquito population, with $N_0 \leq 1$ indicating the extinction of mosquitoes which persist otherwise.
- For the full model (with both mosquitoes and humans), dengue virus can be controlled if the associated basic reproduction number is less than or equal to unity and $N_0 \leq 1$.
- The full model (with both mosquitoes and humans) undergoes backward bifurcation (at $\mathcal{R}_0 = 1$) whenever $N_0 > 1$. It was further shown that, the bifurcation is caused by disease induced death rate of humans.
- The type reproduction numbers were also computed and their relationship with the basic reproduction number presented. Conditions for global asymptotic stability of the DFE were computed. Also, β_{hv} and μ_v are respectively the most positively and negatively correlated parameters to the basic reproduction.

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Appendix A: Backward bifurcation

Proof of the existence of backward bifurcation

Let,

$$(S_h, I_h, R_h, A_n, A_i, S_v, I_v) = (z_1, z_2, z_3, z_4, z_5, z_6, z_7),$$

so that the total human population, aquatic and adult mosquito populations are:

$$N_h = z_1 + z_2 + z_3, \quad A = z_4 + z_5, \quad \text{and} \quad N_V = z_6 + z_7.$$

Then, model (2.4) as transformed is represented by,

$$\begin{aligned}
\frac{dz_1}{dt} &= r_h - (\frac{\beta_{hv} z_7}{z_1 + z_2 + z_3})z_1 - \mu_h z_1, \\
\frac{dz_2}{dt} &= (\frac{\beta_{hv} z_7}{z_1 + z_2 + z_3})z_1 - \delta_h z_2 - \omega_h z_2 - \mu_h z_2, \\
\frac{dz_3}{dt} &= \omega_h z_2 - \mu_h z_3, \\
\frac{dz_4}{dt} &= \rho_v (1 - \frac{z_4 + z_5}{\mathcal{K}})(z_6 + \zeta_v z_7) - b_v z_4 - \mu_A z_4, \\
\frac{dz_5}{dt} &= \rho_v (1 - \frac{z_4 + z_5}{\mathcal{K}})(1 - \zeta_v) z_7 - b_v z_5 - \mu_A z_5, \\
\frac{dz_6}{dt} &= b_v z_4 - \frac{\beta_{hv} z_2}{z_1 + z_2 + z_3} z_6 - \mu_v z_6, \\
\frac{dz_7}{dt} &= \frac{\beta_{hv} z_2}{z_1 + z_2 + z_3} z_6 + b_v z_5 - \mu_v z_7.
\end{aligned} \tag{A.1}$$

and,

$$\lambda_h = \frac{\beta_{hv} z_7}{z_1 + z_2 + z_3} z_1, \quad \lambda_V = \frac{\beta_{hv} z_2}{z_1 + z_2 + z_3} z_6.$$

The Jacobian matrix at the DFE and $\beta_{hv} = \beta^*_{hv}$ is:

$$J^* = \begin{pmatrix}
-\mu_h & 0 & 0 & 0 & 0 & 0 & -\beta^*_{HV} \\
0 & -S_1 & 0 & 0 & 0 & 0 & \beta^*_{HV} \\
0 & \omega_h & -\mu_h & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & -\frac{\rho_v b_v}{\mu_v} & -S_2(N_0 - 1) & \frac{S_2 \mu_v}{b_v} & \frac{S_2 \mu_v \zeta_v}{b_v} \\
0 & 0 & 0 & 0 & -S_2 & 0 & \frac{S_2 \mu_v (1 - \zeta_v)}{b_v} \\
0 & -\frac{\beta^*_{HV} S^*_v}{S^*_h} & 0 & b_v & 0 & -\mu_v & 0 \\
0 & \frac{\beta^*_{HV} S^*_v}{S^*_h} & 0 & 0 & b_v & 0 & -\mu_v
\end{pmatrix}.$$

At $\mathcal{R}_0 = 1$ which implies $\mathcal{R}_{HV}\mathcal{R}_{VH} = 1 - \mathcal{R}_{VV}$, the Jacobian J^* has left (v_i) and right (w_i)

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eigenvectors associated with the zero eigenvalue respectively given by

$$\begin{aligned} v_1 &= 0, \quad v_2 = \frac{1}{S_h^* S_2 \mu_V^2 \zeta_v^2 \mu_h^2 + S_v^* \beta_{hv}^2 \mu_h^2 ((1-\zeta_v) + S_2 \zeta_v)}, \quad v_3 = 0 \\ v_4 &= 0, \quad v_5 = \frac{\beta_{hv} b_v}{S_2 \mu_v \zeta_v} v_2, \quad v_6 = 0, \quad v_7 = \frac{\beta_{hv}}{\mu_v \zeta_v} v_2, \end{aligned} \tag{A.2}$$

and

$$\begin{aligned} w_1 &= \frac{-\mathcal{R}_{HV}\mathcal{R}_{VH} S_1}{\zeta_v \mu_h} w_2, \quad w_2 = S_h^* S_2 \mu_h^2 \mu_V^2 \zeta_v^2, \quad w_3 = \frac{\omega_h w_2}{\mu_h}, \quad w_4 = \frac{-\beta_{hv} S_v^* (1-\zeta_v)}{S_h^* b_v \zeta_v} w_2, \\ w_5 &= \frac{\beta_{hv} S_v^* (1-\zeta_v)}{S_h^* b_v \zeta_v} w_2, \quad w_6 = -\frac{\beta_{hv} S_v^*}{S_h^* \mu_v \zeta_v} w_2, \quad w_7 = \frac{\beta_{hv} S_v^*}{S_h^* \mu_v \zeta_v} w_2. \end{aligned} \tag{A.3}$$

Using the vectors in (A.2) and (A.3) we have

$$\begin{aligned} \mathbf{a} = \sum_{k,i,j=1}^{n} V_k W_i W_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) = \frac{-2 w_2^2 v_2}{N_h^* \mathcal{K}} \Big[8\beta_{hv}^2 \frac{S_v^* \mathcal{K}}{\mu_v \zeta_v} + \frac{\beta_{hv}^2 S_v^* \mathcal{K}(\omega_h + \mu_h)}{\mu_v \mu_h \zeta_v} + \\ \beta_{hv} S_v^* \mathcal{K} \big(\frac{4\mu_h + 5\omega_h}{\mu_h} - 3 \frac{S_1}{\mu_h} \{ \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{(1-\mathcal{R}_{VV})} \} \big) \Big] \end{aligned} \tag{A.4}$$

while,

$$\mathbf{b} = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \rho_v}(0,0) = \frac{S_v^* w_2 v_2}{S_h^*} \big(\frac{\beta_{hv}}{\mu_v \zeta_v} + 8 \big) > 0. \tag{A.5}$$

Observe that a can be positive or negative.

Appendix B: Global asymptotic stability of $\mathcal{G}_3$

Here, the global stability of the DFE $\mathcal{G}_3$ is done using a method described by [39]. The model (2.4) is first transformed into a pseudo-triangular form.
If $\mathbf{p}_1 = (S_h, A_n, S_v)^T$ is the populations of the susceptible components of (2.4), and $\bar{\mathbf{p}_1} = (S_h^*, A_n^*, S_v^*)^T$ represents the DFE. Based on the properties of the disease free equilibrium, equations of the model can be simplified as,

$$\begin{aligned} \frac{dS_h}{dt} &= r_h - \beta_{hv} \frac{I_v}{N_h} S_h - \mu_h S_h, \\ &= -\mu_h (S_h - S_h^*) - \beta_{hv} \frac{I_v}{N_h} S_h, \end{aligned} \tag{B.1}$$

similarly,

$$\begin{aligned} \frac{dA_n}{dt} &= \rho_v (1 - \frac{A}{\mathcal{K}})(S_v + \zeta_v I_v) - S_2 A_n, \\ &= -(A_n - A_n^*)(S_2 + \rho_v \frac{S_v}{\mathcal{K}}) + \rho_v (1 - \frac{A}{\mathcal{K}})(S_v - S_v^* + \zeta_v I_v) - \rho_v \frac{S_v}{\mathcal{K}} A_i, \end{aligned} \tag{B.2}$$

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and,

$$\begin{aligned}\frac{dS_v}{dt} &= b_v A_n - \beta_{hv}\frac{I_h}{N_h}S_v - \mu_v S_v,\\ &= -\mu_v(S_v - S_v^*) + b_v(A_n - A_n^*) - \beta_{hv}\frac{I_h}{N_h}S_v.\end{aligned} \tag{B.3}$$

From the above, the following coefficient matrices are obtained

$$A_{11}(\mathbf{p}) = \begin{pmatrix} -\mu_h & 0 & 0 \\ 0 & -(S_2 + \rho_v\frac{S_v}{\mathcal{K}}) & \rho_v(1-\frac{A_n^*}{\mathcal{K}}) \\ 0 & b_v & -\mu_v \end{pmatrix},$$

$$A_{12}(\mathbf{p}) = \begin{pmatrix} 0 & 0 & 0 & -\beta_{hv}\frac{S_h}{N_h} \\ 0 & 0 & -\rho_v\frac{S_v}{\mathcal{K}} & \rho_v\zeta_v(1-\frac{A}{\mathcal{K}}) \\ -\beta_{hv}\frac{S_v}{N_h} & 0 & 0 & 0 \end{pmatrix}, \tag{B.4}$$

Also, if $\mathbf{p}_2 = (I_h, R_h, A_i, I_v)^T$ is the population of infectious component of (2.4), with the following simplification

$$\begin{aligned}\frac{dA_i}{dt} &= \rho_v(1-\frac{(A_i+A_n)}{\mathcal{K}})(1-\zeta_v)I_v - b_v A_i - \mu_A A_i,\\ &= \rho_v(1-\zeta_v)I_v - \rho_v(1-\zeta_v)I_v\frac{A_i}{\mathcal{K}} - \rho_v(1-\zeta_v)I_v\frac{A_n}{\mathcal{K}} - S_2 A_i,\\ &= -A_i(S_2 + \rho_v(1-\zeta_v)\frac{I_v}{\mathcal{K}}) + \rho_v(1-\zeta_v)I_v(1-\frac{A_n}{\mathcal{K}}).\end{aligned} \tag{B.5}$$

the following matrix is also obtained,

$$A_{22}(\mathbf{p}) = \begin{pmatrix} -S_1 & 0 & 0 & \beta_{hv}\frac{S_h}{N_h} \\ \omega_h & -\mu_h & 0 & 0 \\ 0 & 0 & -m_{33} & m_{34} \\ \beta_{hv}\frac{S_v}{N_h} & 0 & b_v & -\mu_v \end{pmatrix}, \tag{B.6}$$

where, $m_{33} = S_2 + \rho_v(1-\zeta_v)\frac{I_v}{\mathcal{K}}$ and $m_{34} = \rho_v(1-\zeta_v)(1-\frac{A_n^*}{\mathcal{K}})$. So that, the system is re-written as

$$\begin{cases} \dot{\mathbf{p}_1} &= A_{11}(\mathbf{p})(\mathbf{p}_1 - \bar{\mathbf{p}_1}) + A_{12}(\mathbf{p})\mathbf{p}_2 \\ \\ \dot{\mathbf{p}_2} &= A_{22}(\mathbf{p})\mathbf{p}_2. \end{cases} \tag{B.7}$$

Theorem 4.1. *Consider (2.4). Let $\Omega \subset \mathbb{R}_+^{n_1+n_2}$ be a positively-invariant set. If*

1. *The system (2.4) is defined on the positively invariant set $\Omega \subset \mathbb{R}_+^{n_1+n_2}$.*
2. *The sub-system $\dot{p} = A_{11}(p)(z_1 - \bar{z_1})$ is globally asymptotically stable at the equilibrium $\bar{z_1}$.*
3. *For any $p \in \Omega$, the matrix $A_{22}(p)$ is Metzler and irreducible.*

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4. *There exists an upper bound matrix* $\bar{A_{22}}$ *for the set* $\mathcal{W} = \{A_{22}(p)/p \in \Omega\}$, *with the property that either* $\bar{A_{22}} \notin \mathcal{W}$ *or if* $\bar{A_{22}} \in \mathcal{W}$*(i.e.,* $\bar{A_{22}} = max_{\Omega}\mathcal{W}$*), then for* $\bar{p} \in \Omega$ *such that* $\bar{A_{22}} = A_{22}(\bar{p})$, *then* $\bar{p} \in \mathbb{R}^7 \times \{0\}$ *(the DFE sub-manifold contains the points where the maximum is attained).*

5. *The stability modulus of* $\bar{A_{22}}$ *satisfies* $\alpha(\bar{A_{22}}) \leq 0$.

Then, the associated DFE is GAS in Ω *[12, 15, 39].*

We have already shown that Ω was is positively invariant with respect to (2.4) (Lemma 3.1). For the fixed point $\mathcal{G}_3$, where $A^* = \mathcal{K}(\frac{N_0-1}{N_0})$, the associated eigenvalues of the matrix $(A_{11}(p)\ )$ given by (B.4) are as follows

$$
\begin{aligned}
&-\mu_h,\\
&-\frac{1}{2}(S_2+\mu_v+\frac{\rho_v S_v}{\mathcal{K}})+\frac{1}{2}\sqrt{(S_2+\mu_v+\frac{\rho_v S_v}{\mathcal{K}})^2-4\frac{\rho_v S_v}{\mathcal{K}}\mu_v},\\
&-\frac{1}{2}(S_2+\mu_v+\frac{\rho_v S_v}{\mathcal{K}})-\frac{1}{2}\sqrt{(S_2+\mu_v+\frac{\rho_v S_v}{\mathcal{K}})^2-4\frac{\rho_v S_v}{\mathcal{K}}\mu_v}.
\end{aligned}
$$

Via some computations, it can be shown that $(S_2+\mu_v+\frac{\rho_v S_v}{\mathcal{K}})^2-4\frac{S_v}{\mathcal{K}}\rho_v\mu_v > 0$, and $(S_2+\mu_v+\frac{\rho_v S_v}{\mathcal{K}})^2-4\frac{S_v}{\mathcal{K}}\rho_v\mu_v < (S_2+\mu_v+\frac{\rho_v S_v}{\mathcal{K}})^2$, thus, the eigenvalues of the Metzler matrix $A_{11}(\mathbf{p})$ are all real and negative. Therefore the subsystem $\dot{\mathbf{p}_1} = A_{11}(\mathbf{p})(\mathbf{p}_1-\bar{\mathbf{p}_1})$ is globally asymptotically stable. Any square matrix $\mathbf{U}$ in the following form is reducible

$$
\mathbf{U} = \begin{pmatrix} \mathbf{U}_1 & \mathbf{U}_2 \\ 0 & \mathbf{U}_3 \end{pmatrix} \tag{B.8}
$$

where $\mathbf{U}_1$ and $\mathbf{U}_3$ are square matrices of order at least 1 or if $\mathbf{A}$ can be transformed into the form (B.8) by simultaneous permutations of rows and columns. It is irreducible otherwise. Alternatively, a square matrix is irreducible if and only if its associated digraph is strongly connected [43].

Lemma 4.2. *Let* $\mathcal{W}$ *be a Metzler matrix which is block decomposed as follows*

$$
\mathcal{W} = \begin{pmatrix} A_1 & A_2 \\ A_3 & A_4 \end{pmatrix} \tag{B.9}
$$

A_1 *and* A_4 *are square matrices. Then* $\mathcal{W}$ *is Metzler stable if and only if* A_1 *and* $A_4 - A_3A_1^{-1}A_2$ *are Metzler stable.*

Let $M_h^* = \frac{r_h}{\delta_h+\mu_h}$, then $N_h \geq M_h^*$ and $S_h \leq N_h$, so that $\frac{1}{M_h^*} \geq \frac{1}{N_h}$ and $\frac{S_h}{N_h} \leq 1$ in Ω with equality at the DFE. Furthermore $A_n \leq \mathcal{K}$ and $S_v \leq S_v^*\frac{N_0}{N_0-1}$ in Ω. Also, from the definition of Metzler reducible matrix follows that $A_{22}(\mathbf{p})$ is irreducible, hence conditions 1-3 of Theorem (4.1) are satisfied. The following matrix $\bar{A_{22}}(\mathbf{p})$ given by

$$
\bar{A_{22}}(\mathbf{p}) = \begin{pmatrix} -S_1 & 0 & 0 & \beta_{hv} \\ \omega_h & -\mu_h & 0 & 0 \\ 0 & 0 & -S_2 & \rho_v(1-\zeta_v)\frac{1}{N_0} \\ \frac{\beta_{hv}S_v^*N_0}{(N_0-1)M_h^*} & 0 & b_v & -\mu_v \end{pmatrix} \tag{B.10}
$$

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is Metzler and an upper bound of $A_{22}(\mathbf{p}) \in \Omega$ provided $\mathcal{R}_{HH} < 1$. Thus, condition 4 of Theorem (4.1) is satisfied. In the case of the matrix $\bar{A_{22}}$ we have

$$\mathbb{A} = \begin{pmatrix} -S_1 & 0 \\ \omega_h & -\mu_h \end{pmatrix}, \quad \mathbb{B} = \begin{pmatrix} 0 & \beta_{hv} \\ 0 & 0 \end{pmatrix},$$
$$\mathbb{C} = \begin{pmatrix} 0 & 0 \\ \frac{\beta_{hv} S_v^* N_0}{(N_0-1)M_h^*} & 0 \end{pmatrix}, \quad \mathbb{D} = \begin{pmatrix} -S_2 & \rho_v(1-\zeta_v)\frac{1}{N_0} \\ b_v & -\mu_v \end{pmatrix}. \tag{B.11}$$

Under the condition that $\mathcal{R}_{HH} < 1$, it is easy to verify that $\mathbb{A}$ is Metzler stable. Also

$$\mathbb{D} - \mathbb{C}\mathbb{A}^{-1}\mathbb{B} = \begin{pmatrix} -S_2 & \rho_v(1-\zeta_v)\frac{1}{N_0} \\ b_v & -\mu_v(1 - \frac{\beta_{hv}^2 S_v^* N_0}{S_1 M_h^* \mu_v (N_0-1)}) \end{pmatrix}. \tag{B.12}$$

Let $Z = \frac{N_h^* N_0}{M_h^*(N_0-1)}$. Then $\mathbb{D} - \mathbb{C}\mathbb{A}^{-1}\mathbb{B}$ is Metzler if

$$\frac{\beta_{hv}^2 S_v^* N_h^* N_0}{S_1 N_h^* M_h^* (N_0-1)\mu_v} = \mathcal{R}_{HV}\mathcal{R}_{VH} Z < 1. \tag{B.13}$$

and Metzler stable if

$$S_2\mu_v(1 - \mathcal{R}_{HV}\mathcal{R}_{VH} Z - \mathcal{R}_{VV}) \geq 0 \implies \mathcal{R}_{HV}\mathcal{R}_{VH} Z + \mathcal{R}_{VV} \leq 1. \tag{B.14}$$

Notice that for $N_0 > 1$, $Z = \frac{N_h^* N_0}{M_h^*(N_0-1)} > 1$, therefore, the two conditions given by (B.13) and (B.14) are equivalent to

$$\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{1 - \mathcal{R}_{VV}} \leq \frac{1}{Z} = \frac{M_h^*(N_0-1)}{N_h^* N_0} < 1 \text{ and, } \mathcal{R}_{HV}\mathcal{R}_{VH} < \frac{M_h^*(N_0-1)}{N_h^* N_0}.$$

Thus, the necessarily and sufficient conditions for the GAS of $\mathcal{G}_3$ with respect to Ω is that $\mathcal{R}_{HV}\mathcal{R}_{VH} Z + \mathcal{R}_{VV} \leq 1$.

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