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A Host-Vector Model for Dengue Transmission: Integrating Microscopic and Macroscopic Dynamics

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Abstract. Dengue fever poses a significant global health threat, with millions of infections annually. This work introduces a preliminary mathematical model for studying dengue reinfections, aiming at the Antibody-Dependent Enhancement phenomenon. We employ a vector-host modeling approach, incorporating viral and antibody micro-dynamics to define new infections. In addition, we explore a specific case that leads to a delayed model, analyzing its endemic equilibrium through theoretical and numerical studies. The results confirm expected epidemiological behavior, supporting the model's applicability in dengue research.

Keywords. Dengue Fever, Mathematical Models, Delay Differential Equations, Numerical Simulations

1 Introduction

Dengue fever is a viral infection transmitted from person to person through the Aedes aegypti mosquito. Approximately half of the global population is now at risk of dengue, with an estimated 100 to 400 million infections occurring each year [6], which makes this disease a significant public health concern in many regions of the world. In addition, there is no specific treatment for dengue or severe dengue, highlighting the importance of studying mathematical models and control strategies for this disease [1, 2].

Humans can experience symptomatic dengue virus (DENV) infections more than once due to four antigenically distinct serotypes: DENV1, DENV2, DENV3, and DENV4 [5]. This diversity complicates efforts to combat the disease. Furthermore, a secondary infection with a different serotype significantly increases the risk of developing a severe disease. This phenomenon is known as Antibody-Dependent Enhancement (ADE), where preexisting antibodies can facilitate the new infection [3–5]. In this work, we propose a preliminary model to study dengue fever reinfections and subsequently allow the study of the ADE phenomenon.

Different modeling approaches exist in dengue epidemiological studies, including host-to-host, vector-host, and within-host models [1]. We present a vector-host model to represent the interaction between the vector, virus, and host populations. The micro-dynamics are taken into account to define new infections, which will be essential for the study of the ADE phenomenon. In this work, microscopic dynamics, or within-host dynamics [3], consider the variation of viral load and the antibody level (Ab). However, for an initial investigation, we consider the viral load and antibodies of only a single dengue serotype.

Therefore, we present a general model and a particular case that leads to an associated delayed model. In this particular case, we present the theoretical and numerical study of the endemic

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equilibrium. The numerical results also allow us to conclude that the model presents expected behavior to the dynamics analyzed.

2 The Model

Let us suppose a constant population N_h and N_v of hosts and vectors, respectively. The host population is divided into susceptible $S(t)$, exposed $E(t)$ (infected host population with the virus at intrinsic incubation stage), infected $I(t, z, y)$ (with some viral load z and antibody level y), and recovered $R(t, y)$. The vector population is divided only into the exposed vectors $E_v(t)$ (infected vector with the virus in the extrinsic incubation stage) and the infected vectors $I_v(t)$. If necessary, the susceptible vectors can be computed by $S_v = N_v - E_v(t) - I_v(t)$.

The dynamics is described by

$$\partial_t I(t,z,y) + \partial_z \left(a_1 I(t,z,y) z - a_2 I(t,z,y) y\right) + \partial_y \left(-a_3 I(t,z,y) y + a_4 I(t,z,y) z\right) = 0, \tag{1a}$$

$$\partial_t R(t,y) + \partial_y \left(-a_5 R(t,y)\right) = -I(t,z_0,y)\big(a_1 z_0 - a_2 y\big), \tag{1b}$$

$$\dot{S}(t) = -b I_v(t) \frac{S(t)}{N_h} + a_5 R(t, y_0), \tag{1c}$$

$$\dot{E}(t) = b I_v(t) \frac{S(t)}{N_h} - \frac{1}{\tau_h} E(t), \tag{1d}$$

$$\dot{E}_v(t) = \left(\int_0^\infty \int_{z_0}^\infty \frac{I(t,z,y)}{N_h} \gamma(z) dz dy \right) \left(N_v - E_v(t) - I_v(t)\right) - \frac{1}{\tau_v} E_v(t) - \mu_v E_v(t), \tag{1e}$$

$$\dot{I}_v(t) = \frac{1}{\tau_v} E_v(t) - \mu_v I_v(t), \tag{1f}$$

where $t, z, y \in [0, +\infty)$, $I : [0, +\infty) \times [z_0, +\infty) \times [0, +\infty) \to [0, N_h]$ and $R : [0, +\infty) \times [y_0, +\infty) \to [0, N_h]$. The descriptions of the parameters are given in Table 1, where $[\cdot]$ denotes the desired unit of measurement for viral load $[z]$ and Ab level $[y]$.

Table 1: Description of the parameters.

Parameter	Description	Unit
a_1	Virus growth rate in the host	day^{-1}
a_2	Viral load decay rate	$[z]/([y]\text{day})^{-1}$
a_3	Ab titer decay rate on Infected host	day^{-1}
a_4	Ab titer production rate in the presence of virus	$[y]([z]\text{day})^{-1}$
a_5	Ab titer decay rate on Recovered host	$[y]\text{day}^{-1}$
b	Vector to host transmission rate	day^{-1}
z_0	Minimum detectable viral load	$[z]$
y_0	Minimum Ab level associated to z_0	$[y]$
$\gamma(z)$	Host to vector transmission rate	$([z][y]\text{day})^{-1}$
τ_h	Intrinsic incubation period	day
τ_v	Extrinsic incubation period	day
μ_v	Vector death rate	day^{-1}

In our equations, we assume that the vectors die, but their population is rapidly replenished, considering that the populations remain constant.

The boundary conditions for the PDE of this model are zero, except for

$$I(t, z = z_0, y \in [0, y_0]) = \frac{1}{(a_1 z_0 - a_2 y^\star)\tau_h} E(t) g(y), \tag{2}$$

where $g(y)$ is a distribution function with center of mass $y^\star \in [0, y_0]$. The boundary condition (2) represents the exposed part of the population, which has a significant viral load and antibodies to enter the dynamics of infected population I, according to the distribution $g(y)$.

The transport terms in (1) determine what is the exact microscopic temporal dynamics of the viral load vs. the antibody level. They were chosen to be the simplest possible, while still describing the desired dynamics [5], which in our case takes the explicit form of the characteristic equations (3). However, more detailed behaviors could be modeled, by adapting the transport terms (and thus, the characteristic's trajectories) accordingly. Still, it is challenging to conduct a more detailed study on the temporal variation of antibody levels with respect to viral load, since such detailed descriptions are not yet available in the literature.

Given the complexity of the model (1) for theoretical analysis, let us assume that our entire population has the same characteristics, that is, that our distribution function $g(y)$ is a Dirac delta $\delta(y - y^\star)$. Therefore, we can build an associated delay model for a particular study.

2.1 Associated Delay Model

From the model (1), we know that after the incubation period, some individuals enter the compartment I with a viral load z_0 and some Ab level, as described by the boundary condition (2). Let us say that all individuals leaving compartment E and entering compartment I are concentrated at a single point $(t, z_0, y^\star)$. Thus, the I's compartment dynamics is described by a single characteristic curve of equation (1a), starting from $(t, z_0, y^\star)$. According to the dynamics of system (1), this characteristic curve remains in the set $(z, y) \in [z_0, +\infty) \times [0, +\infty)$ for a period τ_1. After this time, the characteristic reaches a point $(t + \tau_1, z_0, y^+)$. By the characteristic equations of (1a), we know that τ_1 and y^+ are computed by the smaller value of $\xi > 0$ such that

$$\begin{cases} z_0 = e^{\frac{a_1 - a_3}{2}\xi}\Big(z_0 \cos\beta\xi + \Big(z_0\dfrac{a_1 + a_3}{2\beta} - \dfrac{y^\star a_2}{\beta}\Big)\sin\beta\xi\Big), \\ y^+ = e^{\frac{a_1 - a_3}{2}\xi}\Big(y^\star \cos\beta\xi + \Big(\dfrac{a_4 z_0}{\beta} - y^\star\dfrac{a_1 + a_3}{2\beta}\Big)\sin\beta\xi\Big), \end{cases} \tag{3}$$

for given z_0 and $y^\star$, where $\beta = \frac{1}{2}\sqrt{4a_2a_4 - (a_1 + a_3)^2}$. Numerically, we can compute the variables τ_1 and y^+ by Newton's method.

Then, the number of individuals that enter the new compartment R at position $(t + \tau_1, z_0, y^+)$ can be written as:

$$I(t + \tau_1, z_0, y^+) = \frac{1}{(a_1 z_0 - a_2 y^\star)\tau_h} E(t) g(y). \tag{4}$$

As before, we can consider that the information travels from position $(t + \tau_1, z_0, y^+)$ along compartment R, until it reaches compartment $S(t)$. Let τ_2 be the period in which the individuals remain in compartment R. Then, τ_2 is defined from the characteristic equation of (1b), by:

$$\tau_2 = \frac{y^+ - y_0}{a_5}. \tag{5}$$

The number of individuals who leave the compartment R at time $t + \tau_1 + \tau_2$ and enter the compartment S is

$$R(t + \tau_1 + \tau_2, y_0) = \frac{1}{a_5 \tau_h} E(t), \tag{6}$$

for a given time t.

Finally, we consider the host to vector transmission rate γ as constant and write the associated delay model (respecting mass conservation):

$$\dot{S}(t) = -bI_v(t)\frac{S(t)}{N_h} + \frac{1}{a_5\tau_h}E(t-\tau_1-\tau_2), \tag{7a}$$

$$\dot{E}(t) = bI_v(t)\frac{S(t)}{N_h} - \frac{1}{a_5\tau_h}E(t), \tag{7b}$$

$$\dot{E_v}(t) = \frac{\gamma}{N_h}\Big(N_h - S(t) - E(t) - \frac{1}{a_5\tau_h}\int_{y_0}^{y^+} E\Big(t-\tau_1-\frac{y^+-y}{a_5}\Big)dy\Big)\left(N_v - E_v(t) - I_v(t)\right) - \frac{1}{\tau_v}E_v(t) - \mu_v E_v(t), \tag{7c}$$

$$\dot{I}_v(t) = \frac{1}{\tau_v}E_v(t) - \mu_v I_v(t). \tag{7d}$$

3 Endemic Equilibrium

Let a point of the system (7) be denoted by (S, E, E_v, I_v), we will study the endemic equilibrium (s^*, e^*, e_v^*, i_v^*) of this associated delay model.

In order to analyze system (7), let $s = S/N_h$, $e = E/N_h$, $e_v = E_v/N_v$, $i_v = I_v/N_v$, and $m = N_v/N_h$. So, the system (7) can be reduced to the following equations:

$$\dot{s}(t) = -bm\, i_v(t)s(t) + \frac{1}{a_5\tau_h}e(t-\tau_1-\tau_2), \tag{8a}$$

$$\dot{e}(t) = bm\, i_v(t)s(t) - \frac{1}{a_5\tau_h}e(t) \tag{8b}$$

$$\dot{e_v}(t) = \gamma\Big(1 - s(t) - e(t) - \frac{1}{a_5\tau_h}\int_{y_0}^{y^+} e\Big(t-\tau_1-\frac{y^+-y}{a_5}\Big)dy\Big)\left(1 - e_v(t) - i_v(t)\right) - \frac{1}{\tau_v}e_v(t) - \mu_v e_v(t), \tag{8c}$$

$$\dot{i}_v(t) = \frac{1}{\tau_v}e_v(t) - \mu_v i_v(t). \tag{8d}$$

Solving (8) in its steady state, we have the following solution:

$$s^* = \frac{i_v^*(\gamma+\mu_v)(\mu_v\tau_v+1) - \gamma}{\gamma[{i_v^*}^2 a_5 bm(\mu_v\tau_v+1)(\tau_2+\tau_h) + i_v^*(-a_5 bm(\tau_2+\tau_h) + \mu_v\tau_v + 1) - 1]}, \tag{9a}$$

$$e^* = \frac{a_5\tau_h bm i_v^*[i_v^*(\gamma+\mu_v)(\mu_v\tau_v+1) - \gamma]}{\gamma[{i_v^*}^2 a_5 bm(\mu_v\tau_v+1)(\tau_2+\tau_h) + i_v^*(-a_5 bm(\tau_2+\tau_h) + \mu_v\tau_v + 1) - 1]}, \tag{9b}$$

$$e_v^* = \mu_v\tau_v i_v^*. \tag{9c}$$

for an arbitrary i_v^*.

As we want a positive endemic equilibrium, we conclude that the possible solutions of the system (9), are given by:

$$i_v^* \in \left(0,\ \frac{\gamma}{(\gamma+\mu_v)(\mu_v\tau_v+1)}\right).$$

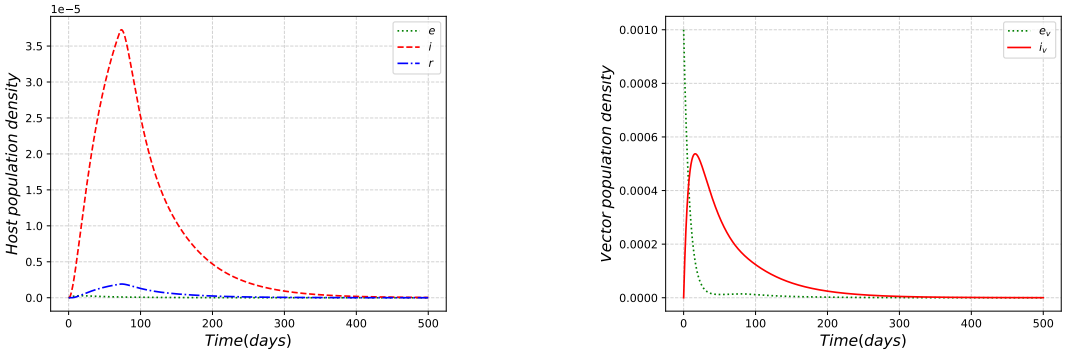
For simplicity, we denote only the solution concerning the admissible set of i_v.

4 Numerical Studies

In our numerical study, we consider the model (8), which has the disease-free equilibrium point $DFE = (1, 0, 0, 0)$. We assume our initial condition as a small perturbation on DFE, that is, $(1, 0, 0.001, 0)$. We fix the following parameters: $a_1 = 0.9$, $a_2 = 0.4$, $a_3 = 0.0001$, $a_4 = 0.9$, $a_5 = 0.05$, $\gamma = 0.1$, $\tau_v = 10$, $\mu_v = 0.025$, $z_0 = 1$, and we take $b \in \{0.005; 0.05\}$ and we vary m. We compute the delay values τ_1 and τ_2 present in (8) only once. The first delay value τ_1 is calculated from (3) by Newton's method, with initial shooting $\xi_0 = 2.5$. The second delay value τ_2 is explicitly given by (5).

The derivatives on (8) are approximated by the explicit Euler method (which is sufficient for our present purposes), and the integral term is approximated by the generalized left rectangle rule. The final time evaluated is set as $T_f = 1000$, the time step is $\Delta t = 0.1$, and the number of discrete points for each variable is $N_T = T_f/\Delta t$. To compute the normalized recovered host population r, we consider the integral of equation (6) over the interval $[t^n+\tau_1, t^n+\tau_1+\tau_2]$, for each $t^n = n\Delta t$, with $n = 0, \ldots, N_T$. This integral is also approximated by the generalized left rectangle rule. Finally, to represent the normalized infected host population i, we assume a constant normalized total host population, that is, $i(t^n)+r(t^n)+s(t^n)+e(t^n) = 1$ for $n = 0, \ldots, N_T$, then we isolate the term $i(t^n)$. In the same way, we compute the susceptible vector population from $s_v(t^n) + i_v(t^n) + e_v(t^n) = 1$.

It was possible to observe a stable and constant numerical solution in relation to the DFE point (for sufficiently large t), as shown in Fig. 1 for $m = 0.3$. The higher the value of m (up to 0.5), the longer it takes t to achieve DFE.



(a) Host dynamics for e, i and r.

(b) Vector dynamics for e_v and i_v.

Figure 1: Dynamics of compartments exposed (e, e_v), infected (i, i_v) and recovered (r), with $m = 0.3$. Source: produced by the author.

From values of $m > 0.5$ we have a growing number of infections. That is, as we increase the proportion of mosquitoes to humans, the model leads to a significant increase in infections, even though the parameters b and γ are small.

The Fig. 2 shows the dynamics for host and vector populations, respectively, assuming $m = 5.0$. We observe that the numerical solution seems to stabilize at one of the endemic equilibria.

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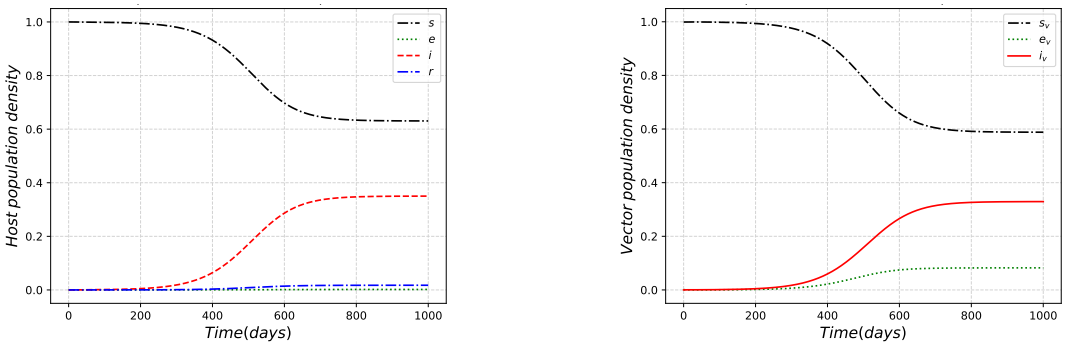
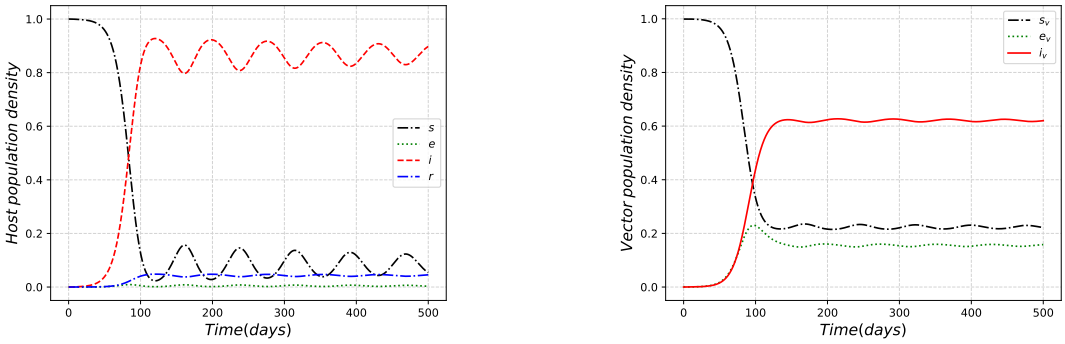


(a) Host dynamics. (b) Vector dynamics.

Figure 2: Dynamics for system (8), with $m = 5.0$. Source: produced by the author.

Keeping the vector-to-host ratio $m = 5.0$, we modify the b infection rate to try to visualize the oscillations associated with new infections. The Fig. 3 allows us to observe the increase in the infected hosts, in which almost the entire population becomes infected. Even though it is not a real case, we can visualize the expected dynamics and we can notice a tendency towards an equilibrium.



(a) Host dynamics. (b) Vector dynamics.

Figure 3: Dynamics for system (8), $m = 5.0$ and $b = 0.05$. Source: produced by the author.

5 Conclusion

In this work, we have considered a model which integrates microscopic dynamics (in the form of the joint evolution of the viral load and the antibody levels, given in (1) and described by (3)) with the more traditional macroscopic compartments appearing in (1) and (7). Macroscopic properties are influenced by the detailed dynamics between viral load and antibody level. In this work, we have only considered a simple form for the microscopic dynamics. It remains to be explored how different micro dynamics would alter or influence the macroscopic outcomes – which would represent a fuller integration of the micro and macro aspects.

We can observe that the numerical results are in line with the theoretical results presented for the study of the endemic equilibrium of the proposed model. Furthermore, at the qualitative level, the model is consistent with the real behavior of dengue infections, which will allow us to

study questions such as the existence of a basic reproduction number R_0, and the stability of the equilibria in the future.

In future work, we intend to further investigate the proposed micro-macro interactions, as well as to extend the model to more than one serotype (and their corresponding antibodies), with the objective to study the ADE phenomenon.

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