DETERMINISTIC AND STOCHASTIC DENGUE EPIDEMIC MODEL: EXPLORING THE PROBABILITY OF EXTINCTION

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Abstract. Dengue, a vector-borne disease, threatens the life of humans in tropical and subtropical regions. Hence, the dengue transmission dynamics need to be studied. An important aspect to be investigated is the probability of extinction. In this paper, deterministic and stochastic dengue epidemic models with two-age classes have been developed and analyzed, and the probability of extinction has been determined. For the stochastic approach, we use the Continuous-Time Markov Chain model. The results show that vaccination of adult individuals leads to a lower number of adult infected individuals. Furthermore, the results showed that a higher number of initial infections cause a low probability of dengue extinction. Furthermore, factors contributing to an increase in the infection-related parameters have to be minimized to increase the potential reduction of dengue cases.

Keywords: dengue, modelling, probability of extinction.

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1. INTRODUCTION

Dengue, a vector-borne disease, threatens life of individuals living in tropical and sub-tropical regions. Around 400 million infections occur annually where ninety million have shown clinical symptoms [1]. In the last five decades, dengue incidence has increased [2].

Although a number of strategies have been implemented, they been found less effective and the risk of being infected is possible. Therefore, alternative strategies such as using a vaccine or Wolbachia bacterium have been proposed [3], [4]. A development of dengue vaccine is underway. Research showed that higher efficacy of dengue can be obtained when it has been implemented in individuals age 9–45 years [5] and its efficacy ranges between 60 to 80 percent [6]–[9]. However, the candidates of dengue vaccine are not effective against all dengue serotypes, which potentially cause higher incidence of secondary infections.

Understanding a disease transmission dynamics using a mathematical model is common [10]–[12]. Deterministic and stochastic models have been commonly formulated to study the disease transmission dynamics in the presence of absence of controls [13]–[16]. Several works estimated the reproduction number of dengue and found that the reproduction number of dengue ranges between approximately one to three [16], [17]. When the reproduction number closes to one, it is possible that the disease can die out due to stochastic effects although in deterministic model, it suggests that an outbreak may happen. Therefore, it is important to formulate the stochastic model to study the probability of disease extinction.

The majority of dengue model has been developed either deterministic or stochastic. It is better for both models to be studied at the same time to obtain comprehensive understanding of dengue transmission [18], Champagne et al. [18] formulated deterministic and stochastic models to assess the disease transmission dynamics. It showed that the deterministic models give a good approximation of the mean trajectory with a low computation cost, while the stochastic approach is better to account for simulation and parameter uncertainty. Furthermore, the existing models are generally single or two serotypes with a single age class [11], [15]. Age-dependent mathematical models have been rarely formulated [19]–[23]. Age dependent dengue mathematical model with a single serotype can serve as a basic model for the development of dengue mathematical model for studying various questions of interest. Therefore, this paper aims to analyze the effects of age-dependent structure in dengue transmission dynamics by formulating an age structured model for single serotype with waning immunity. Furthermore, the influential parameters and the probability of disease extinction have been determined. To achieve this, we formulate deterministic and stochastic mathematical models. The contribution of this paper is the probability of extinction of dengue.

2. RESEARCH METHODS

2.1. Formulation of Mathematical Model

A deterministic dengue mathematical model has been formulated by dividing the population into disjoint compartments. The human population comprises susceptible child and adult compartments (\(S_1\) and \(S_2\) respectively), infected child and adult compartments (\(I_1\) and \(I_2\) respectively) and recovered child and adult compartments (\(R_1\) and \(R_2\) respectively). For the mosquito population, it is divided into susceptible (\(S_v\)) and infected (\(I_v\)) groups.

Let \(\alpha\) be the progression rate from child to adult and \(\alpha = \frac{1}{T}\) where \(T\) is the age at which individuals in child class move to the adult class. The parameters \(e\) is vaccine efficacy adult individuals, \(m\) is the number of mosquitoes per human. Parameter \(\gamma\) is the recovery rate. \(B_h\) and \(B_v\) are the recruitment rates of human and mosquitoes, respectively. A dengue mathematical model in the form of system of differential equation is given by the following equation.

\[
\frac{ds_1}{dt} = B_h - \alpha s_1 - \frac{mbv}{N_v} s_1 - \mu_h s_1 + q_1 r_1,
\]

\[
\frac{ds_2}{dt} = \alpha s_1 + \frac{mbv}{N_v} s_2 - \varepsilon v s_2 - \mu_h s_2 + q_2 r_2,
\]

\[
\frac{dl_1}{dt} = \frac{mbv}{N_v} s_1 - (\gamma + \mu_h) l_1.
\]
\[
\begin{align*}
\frac{dI_2}{dt} &= \frac{mbp_{hv}}{N_v}S_2 - (\gamma + \mu_h)I_2, \\
\frac{dR_1}{dt} &= \gamma I_1 - (\mu_h + q_1)R_1, \\
\frac{dR_2}{dt} &= \gamma I_2 - (\mu_h + q_2)R_2, \\
\frac{dS_v}{dt} &= B_v - \frac{bp_{vh}(I_1 + I_2)}{N_h}S_v - \mu_v S_v, \\
\frac{dI_v}{dt} &= \frac{bp_{vh}(I_1 + I_2)}{N_h}S_v - \mu_v I_v.
\end{align*}
\]

with non-negative initial conditions \( S_1 \geq 0, S_2 \geq 0, I_1 \geq 0, I_2 \geq 0, R_1 \geq 0, R_2 \geq 0, S_v \geq 0, I_v \geq 0 \). We can verify that the solutions of the Model (1) with non-negative initial conditions remain non-negative. Note that the sum of human compartments gives the total population of human, that is,

\[
\frac{dN_h}{dt} = B_h - \mu_h N_h.
\]

It follows that \( \limsup_{t \to \infty} N_h(t) = \frac{B_h}{\mu_h} \). We can also see for the mosquito population we obtain

\[
\frac{dN_v}{dt} = B_v - \mu_v N_v.
\]

It follows that \( \limsup_{t \to \infty} N_v(t) = \frac{B_v}{\mu_v} \). Hence the feasible region

\[
\Omega = \left\{ S_1, S_2, I_1, I_2, R_1, R_2, S_v, I_v \in \mathbb{R}^8 \mid S_1 + S_2 + I_1 + I_2 + R_1 + R_2 \leq \frac{B_h}{\mu_h}, S_v + I_v \leq \frac{B_v}{\mu_v} \right\}
\]

is positively invariant with respect to Model (1). The disease-free equilibrium is obtained as follows:

\[
S_1^* = \frac{B_h}{\alpha + \mu_h}, \quad S_2^* = \frac{(\mu_h + q_2)\alpha B_h}{\mu_h (\alpha + \mu_h + q_2)(\alpha + \mu + q_2)}, \quad I_1^* = I_2^* = R_1^* = I_v^* = 0,
\]

\[
R_2^* = \frac{\alpha \epsilon v B_h}{\mu_h (\alpha + \mu_h + q_2)(\alpha + \mu + q_2)}, \quad S_v^* = \frac{B_v}{\mu_v}.
\]

### 2.2. Reproduction Number

The reproduction number, denoted by \( R_0 \), and is generated using the concept of the next generation matrix [24]. First, the transmission and transition matrices have been constructed. The transmission matrix, \( T \) and the transition matrix, \( \Sigma \), are

\[
T = \begin{pmatrix}
0 & 0 & \frac{mbp_{hw}}{N_v}S_1 \\
0 & 0 & \frac{mbp_{hw}}{N_v}S_2 \\
\frac{bp_{vh}}{N_h}S_v & \frac{bp_{vh}}{N_h}S_v & 0
\end{pmatrix}, \quad \Sigma = \begin{pmatrix}
-(\gamma + \mu_h) & 0 & 0 \\
0 & -(\gamma + \mu_h) & 0 \\
0 & 0 & -\mu_v
\end{pmatrix}
\]
We then take the inverse of the transition matrix $\Sigma^{-1}$ and obtain

$$\Sigma^{-1} = \begin{pmatrix}
-\frac{1}{(\gamma + \mu_h)} & 0 & 0 \\
0 & -\frac{1}{(\gamma + \mu_h)} & 0 \\
0 & 0 & -\frac{1}{\mu_v}
\end{pmatrix}.$$ 

The next generation matrix is obtained by $T \Sigma^{-1}$ that is

$$T \Sigma^{-1} = \begin{pmatrix}
0 & 0 & \frac{mbp_{ih}}{N_v} S_i \\
0 & 0 & \frac{mbp_{ih}}{N_v} S_2 \\
\frac{bp_{ih} S_i}{N_h(\gamma + \mu_h)} & \frac{bp_{ih} S_v}{N_h(\gamma + \mu_h)} & 0
\end{pmatrix}.$$ 

The reproduction number is the spectral radius of the next generation matrix. We obtain the reproduction number $R_0$ is

$$R_0 = \sqrt{mb^2 p_{ih} p_{ih}} \text{ or } R_0^2 = \frac{mb^2 p_{ih} p_{ih}}{\mu_v(\gamma + \mu_h)}.$$ 

(2)

2.3. The Stability of Disease-Free Equilibrium Point

**Theorem 1.** If $R_0 < 1$ then the disease-free equilibrium is locally asymptotically stable.

**Proof.** The Jacobian of Model (1) is given by the following matrix

$$J(E_0) =
\begin{pmatrix}
-(\alpha + \mu_h) & 0 & 0 & 0 & q_1 & 0 & 0 & -\frac{mbp_{ih} B_i}{N_i(\alpha + \mu_h)} \\
\alpha & -(\mu_h + \lambda) & 0 & 0 & 0 & q_2 & 0 & -\frac{mbp_{ih} \alpha B_i (\mu_h + q_2)}{m(\alpha + \mu_h)(\mu_h + q_2 + \lambda)} \\
0 & 0 & -(\gamma + \mu_h) & 0 & 0 & 0 & 0 & \frac{mbp_{ih} B_i}{N_i(\alpha + \mu_h)} \\
0 & 0 & 0 & -\gamma & 0 & -(\mu_h + q_1) & 0 & 0 \\
0 & \lambda & 0 & \gamma & 0 & -(\mu_h + q_1) & 0 & 0 \\
0 & \lambda & 0 & \gamma & 0 & -(\mu_h + q_1) & 0 & 0 \\
0 & -\frac{bp_{ih} B_i}{\mu N_h} & -\frac{bp_{ih} B_i}{\mu N_h} & 0 & 0 & -\mu & 0 & 0 \\
0 & -\frac{bp_{ih} B_i}{\mu N_h} & -\frac{bp_{ih} B_i}{\mu N_h} & 0 & 0 & -\mu & 0 & 0 \\
0 & 0 & -\frac{bp_{ih} B_i}{\mu N_h} & -\frac{bp_{ih} B_i}{\mu N_h} & 0 & 0 & -\mu & 0 \\
0 & 0 & -\frac{bp_{ih} B_i}{\mu N_h} & -\frac{bp_{ih} B_i}{\mu N_h} & 0 & 0 & -\mu & 0
\end{pmatrix}$$
From the Jacobian matrix $J(E_0)$ we find five negative eigenvalues $-(\alpha + \mu_h), -(\mu_h + q_1), -\mu_h, -\mu_v, \text{and } -(\mu_h + q_2 + \epsilon\nu)$. The three others eigenvalues are the solution of equation

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0,$$

(3)

where

$$a_1 = 2(\gamma + \mu_h) + \mu_v \geq 0,$$

$$a_2 = (\gamma + \mu_h)(\gamma + \mu_h + 2\mu_v(1-R_0^2)),$$

$$a_3 = (\gamma + \mu_h)^2\mu_v(1-R_0^2).$$

Obviously, the equation (3) has a real negative part if $R_0 < 1$. Hence, the disease-free equilibrium $E_0$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. This completes the proof.

Furthermore, to study the global stability of the DFE, we follow Castillo-Chavez [25] as follows. First, the system (1) is written in the form

$$\frac{dX}{dt} = F(X,0), \frac{dx}{dt} = G(X,0), G(X,0) = 0,$$

where $F(X,0)$ is the right-hand side of $\frac{dS_1}{dt}, \frac{dS_2}{dt}, \frac{dR_1}{dt}, \frac{dR_2}{dt}, \frac{dS_v}{dt}$ when $I_1 = I_2 = I_v = 0$ and $G(X,0)$ is the right hand side of $\frac{dI_1}{dt}, \frac{dI_2}{dt}, \frac{dI_v}{dt}$. Then it is assumed that the following condition C1 and C2 are satisfied

1. $G(X,0) = 0$,
2. $G(X,1) = D_{x_1}G(X_1^*,0)X_2 - G(X,1), \hat{G}(X,1) \geq 0, (X_1, X_2) \in \Omega$.

Where:

$$X^*_1 = \left( \frac{B_h}{\alpha + \mu_h}, \frac{(\mu_h + q_1)\alpha B_h}{\mu_h^2(\nu + \mu_h + q_2)(\alpha + \mu_h)}, 0, 0, 0, \frac{\alpha\epsilon\nu B_h}{\mu_h(\epsilon + \mu_h + q_2)(\alpha + \mu_h)}, \frac{B_i}{\mu_i} \right),$$

$D_{x_1}G(X^*_1,0)$ is an $M$-matrix with non-negative off-diagonal, which is obtained from the Jacobian $G(X,1)$ at $(X_1,0)$ and $\Omega$ is the biologically feasible region for the DFE of system (1). Then, we claim the following theorem.

**Theorem 2** The disease-free equilibrium $E_0$ is globally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

**Proof.** From system (1) we have
\[
F(X_1, 0) = \begin{bmatrix}
B_h - (\alpha + \mu_h)S_1 \\
\alpha S_1 - (\tilde{\varepsilon}v + \mu_h)S_2 + q_2R_2 \\
-(\mu_h + q_1)R_1 \\
\varepsilon v S_2 - (\mu_h + q_2)R_2 \\
B_v - \mu_v S_v
\end{bmatrix}
\]

and
\[
D_{x_2} G(X_1^*, 0) = \begin{bmatrix}
-(\gamma + \mu_h) & 0 & \frac{mbp_h S_1^*}{N_v} \\
0 & -(\gamma + \mu_h) & \frac{mbp_h S_2^*}{N_v} \\
\frac{bp_{vh} S_v}{N_h} & \frac{bp_{vh} S_v}{N_h} & -\mu_v
\end{bmatrix}
\]

From condition C2, we get
\[
\dot{G}(X_1, X_2) = D_{x_2} G(X_1^*, 0) - G(X_1, X_2)
\]
\[
= \begin{bmatrix}
mbp_h I_v S_1^* \left(1 - \frac{S_1}{N_v} \frac{N_v}{S_1^*}\right) \\
mbp_h I_v S_2^* \left(1 - \frac{S_2}{N_v} \frac{N_v}{S_2^*}\right) \\
bp_{vh} (I_1 + I_2) S_v^* \left(1 - \frac{S_v}{N_v} \frac{N_v}{S_v^*}\right)
\end{bmatrix}
\]

where \( S_1^* = \frac{B_h}{\alpha + \mu_h} \), \( S_2^* = \frac{(\mu_h + q_2)\alpha B_h}{\mu_h(\tilde{\varepsilon}v + \mu_h + q_2)(\alpha + \mu_h)} \), and \( S_v^* = N_v^* \). In region \( \Omega \), we have \( S_1 \leq S_1^* \), \( S_2 \leq S_2^* \), and \( S_v \leq S_v^* \). Since \( S_v \leq N_v \), then inequality
\[
1 - \frac{S_1}{N_v} \frac{N_v}{S_1^*} > 0, \quad 1 - \frac{S_2}{N_v} \frac{N_v}{S_2^*} > 0, \quad \text{and} \quad 1 - \frac{S_v}{N_v} \frac{N_v}{S_v^*} > 0
\]
hold if the human and mosquito population are at equilibrium level, such that \( G(X_1, X_2) \geq 0 \). Therefore, by the theorem in Castilo-Chavez et al [25] the DFE is globally asymptotically stable. This ends the proof. This means that the disease-free equilibrium is always stable starting from any nearby initial conditions. The endemic equilibrium is complex and do not present here.

2.4. Formulation of Stochastic Dengue Mathematical Model

2.4.1. Continuous Time Markov Chain

We consider a discrete valued random vector:
\[
X(t) = (S_1(t), S_2(t), I_1(t), I_2(t), R_1(t), R_2(t), S_v(t), I_v(t))
\]

where the components of \( S_1(t), S_2(t), I_1(t), I_2(t), R_1(t), R_2(t), S_v(t), I_v(t) \) are the discrete random value for the number of child susceptible human, adult susceptible human, child infected human, adult infected human, child recovered human, adult recovered human, susceptible mosquitoes, and infected mosquitoes. The continuous time Markov chain (CTMC) at the small period (\( \Delta t \)) has been formulated. Table 1 presents the transition and their corresponding rates.
### Table 1. Transition of individuals between compartment and its rates

<table>
<thead>
<tr>
<th>Description</th>
<th>Transition</th>
<th>Rates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth of $S_1$</td>
<td>$S_1 \rightarrow S_1 + 1$</td>
<td>$B_h$</td>
</tr>
<tr>
<td>Birth of $S_2$</td>
<td>$(S_1, S_2) \rightarrow (S_1 - 1, S_2 + 1)$</td>
<td>$\alpha S_i$</td>
</tr>
<tr>
<td>Infection of $S_1$</td>
<td>$(S_1, I_1) \rightarrow (S_1 - 1, I_1 + 1)$</td>
<td>$\frac{mbp_{h}}{N_v} I_v S_1$</td>
</tr>
<tr>
<td>Death of $S_1$</td>
<td>$S_1 \rightarrow S_1 - 1$</td>
<td>$\mu_i S_1$</td>
</tr>
<tr>
<td>Loss of $R_1$</td>
<td>$(R_1, S_1) \rightarrow (R_1 - 1, S_1 + 1)$</td>
<td>$q_i R_i$</td>
</tr>
<tr>
<td>Infection of $S_2$</td>
<td>$(S_2, I_2) \rightarrow (S_2 - 1, I_2 + 1)$</td>
<td>$\frac{mbp_{h}}{N_v} I_v S_2$</td>
</tr>
<tr>
<td>Vaccination of $S_2$</td>
<td>$(S_2, R_1) \rightarrow (S_2 - 1, R_2 + 1)$</td>
<td>$\epsilon_v S_2$</td>
</tr>
<tr>
<td>Death of $S_2$</td>
<td>$S_2 \rightarrow S_2 - 1$</td>
<td>$\mu_i S_2$</td>
</tr>
<tr>
<td>Loss of $R_2$</td>
<td>$(R_2, S_2) \rightarrow (R_2 - 1, S_2 + 1)$</td>
<td>$q_i R_2$</td>
</tr>
<tr>
<td>Recovery of $I_1$</td>
<td>$(I_1, R_1) \rightarrow (I_1 - 1, R_1 + 1)$</td>
<td>$\gamma I_1$</td>
</tr>
<tr>
<td>Death of $I_1$</td>
<td>$I_1 \rightarrow I_1 - 1$</td>
<td>$\mu_i I_1$</td>
</tr>
<tr>
<td>Recovery of $I_2$</td>
<td>$(I_2, R_2) \rightarrow (I_2 - 1, R_2 + 1)$</td>
<td>$\gamma I_2$</td>
</tr>
<tr>
<td>Death of $I_2$</td>
<td>$I_2 \rightarrow I_2 - 1$</td>
<td>$\mu_i I_2$</td>
</tr>
<tr>
<td>Death of $R_1$</td>
<td>$R_1 \rightarrow R_1 - 1$</td>
<td>$\mu_i R_1$</td>
</tr>
<tr>
<td>Death of $R_2$</td>
<td>$R_2 \rightarrow R_2 - 1$</td>
<td>$\mu_i R_2$</td>
</tr>
<tr>
<td>Birth of $S_\nu$</td>
<td>$S_\nu \rightarrow S_\nu + 1$</td>
<td>$B_\nu$</td>
</tr>
<tr>
<td>Infection of $S_\nu$</td>
<td>$(S_\nu, I_\nu) \rightarrow (S_\nu - 1, I_\nu + 1)$</td>
<td>$\frac{bp_{i\nu}(I_1 + I_2)}{N_\nu} S_\nu$</td>
</tr>
<tr>
<td>Death of $I_\nu$</td>
<td>$I_\nu \rightarrow I_\nu - 1$</td>
<td>$\mu_i I_\nu$</td>
</tr>
</tbody>
</table>

#### 2.4.2. Branching Process Approximation

The nonlinear CTMC dynamics near the disease free equilibrium has been approximated using the multi type branching process [26]–[29]. The $I_1$, $I_2$, and $I_\nu$ are the source of infections. The $u_1$, $u_2$, and $u_3$ has been described as dummy variables for the infection states. The probability of extinction is determined using offspring probability generating function.

The offspring probability generating function for $I_1$ where $I_1(0) = 1$, $I_2(0) = 0$, $I_\nu(0) = 0$ are

$$f_1(u_1, u_2, u_3) = \frac{\beta_i u_1 u_2 + \gamma + \mu_i}{\beta_i + \gamma + \mu_i}$$

where $\beta_i = bp_{i\nu}/\hat{S}_\nu$. The term $\beta_i / (\beta_i + \gamma + \mu_i)$ is the probability that infected human (child) creates infected mosquitoes. The term $(\gamma + \mu_i) / (\beta_i + \gamma + \mu_i)$ is the probability which an infected human die or is out of compartment.

The offspring probability generating function for $I_2$ where $I_2(0) = 1$, $I_1(0) = 0$, $I_\nu(0) = 0$ are

$$f_2(u_1, u_2, u_3) = \frac{\beta_i u_2 u_3 + \gamma + \mu_i}{\beta_i + \gamma + \mu_i}$$
where \( \beta_1 = bp_{ih} \hat{S}_h / \hat{N}_h \). The term \( \beta_1 / (\beta_1 + \gamma + \mu_h) \) is the probability that infected human (adult) creates infected mosquito. The term \( (\gamma + \mu_h) / (\beta_1 + \gamma + \mu_h) \) is the probability that an infected human dies or is out of compartment.

The offspring probability generating function for \( I_v \) where \( I_v(0) = 1, I_1(0) = 0, I_2(0) = 0 \) are

\[
f(u_1, u_2, u_3) = \frac{\beta_1 u_1 + \beta_2 u_2 + \beta_3 u_3 + \mu_v}{\beta_2 + \beta_3 + \mu_v}
\]

where \( \beta_2 = mbp_{hv} \hat{S}_i / \hat{N}_v \) and \( \beta_3 = mbp_{hv} \hat{S}_i / \hat{N}_v \). The terminology \( \beta_1 / (\beta_2 + \beta_3 + \mu_v) \) denotes the probability that an infected mosquito causes infected child. The terminology \( \beta_3 / (\beta_2 + \beta_3 + \mu_v) \) denotes the probability that an infected mosquito causes infected adult. The \( \mu_v / (\beta_2 + \beta_3 + \mu_v) \) is the probability that an infected mosquito dies. We now construct the expectation matrix as follows

\[
M = \begin{bmatrix}
\frac{df_1}{du_1} & \frac{df_2}{du_1} & \frac{df_3}{du_1} \\
\frac{df_1}{du_2} & \frac{df_2}{du_2} & \frac{df_3}{du_2} \\
\frac{df_1}{du_3} & \frac{df_2}{du_3} & \frac{df_3}{du_3}
\end{bmatrix}
= \begin{bmatrix}
\frac{\beta_1}{\beta_1 + \gamma + \mu_h} & 0 & \frac{\beta_2}{\beta_2 + \beta_3 + \mu_v} \\
0 & \frac{\beta_1}{\beta_1 + \gamma + \mu_h} & \frac{\beta_3}{\beta_2 + \beta_3 + \mu_v} \\
\frac{\beta_1}{\beta_1 + \gamma + \mu_h} & \frac{\beta_3}{\beta_2 + \beta_3 + \mu_v} & \frac{\beta_2 + \beta_3 + \mu_v}{\beta_2 + \beta_3 + \mu_v}
\end{bmatrix}
\]

where \( u = (u_1, u_2, u_3) \). The spectral radius of the expectation matrix is

\[
\rho(M) = \frac{(\gamma + \mu_h + \beta_1)(\beta_2 + \beta_3) + \beta_1(\beta_2 + \beta_3 + \mu_v)}{(\gamma + \mu_h + \beta_1)(\beta_2 + \beta_3 + \mu_v)}
\]

(4)

The largest eigenvalue of the expectation matrix \( \rho(M) \) is a threshold for persistence or extinction of the disease in the stochastic model. If \( \rho(M) < 1 \), the disease is eliminated from the population and if \( \rho(M) > 1 \), the probability of major outbreak is non-zero. It has been shown that \( \rho(M) \) is similar to the reproduction number \( R_0 \) in the deterministic model. That is \( R_0 < 1 \leftrightarrow \rho(M) < 1 \)[28].

If the process is subcritical, that is, \((u_1, u_2, u_3) = (1, 1, 1)\) is the only critical point. If the process is supercritical, that is \( \rho(M) > 1 \), there is another fixed point \((u_1^*, u_2^*, u_3^*) \in (0, 1) \)[26]–[28], [30]. The fixed point can be used to estimate the probability of disease extinction, that is

\[
P_0 = \begin{cases} 
1 & \text{if } \rho(M) \leq 1 \\
u_1^0 & \mu_v \text{ if } \rho(M) > 1
\end{cases}
\]

(5)

where \( u_1^0, u_2^0, u_3^0 \) are the initial conditions for child infected children, infected adult and infected vector respectively. The analytical expression for the fixed point are the following

\[
u_1 = \frac{(\beta_2 + \beta_3 + \mu_v)(\gamma + \mu_h)}{(\beta_1 + \gamma + \mu_h)(\beta_2 + \beta_3)}
\]

\[
u_2 = \frac{(\beta_2 + \beta_3 + \mu_v)(\gamma + \mu_h)}{(\beta_1 + \gamma + \mu_h)(\beta_2 + \beta_3)}
\]

\[
u_3 = \frac{\mu_v (\beta_1 + \gamma + \mu_h)}{\beta_1 (\beta_2 + \beta_3 + \mu_v)}
\]
Therefore, the probability of extinction is calculated using Equation (5) and the fixed point is given in Equation $u_1, u_2, u_3$. Therefore, the probability of major outbreak is

$$1 - P_0 = 1 - u_1^\theta u_2^\theta u_3^\theta$$

3. RESULTS AND DISCUSSION

The dynamics of ODE model and CTMC are illustrated in this section. First, we explore the deterministic model and the reproduction number and then we explore the probability of extinction. For the numerical simulations of the ODE model, we use the nonstandard finite difference scheme approach.

3.1. Sensitivity analysis of reproduction number

In this section, we aim to determine the influential parameters on the reproduction number. The Latin Hypercube Sampling in conjunction with Partial Rank Correlation Coefficient Multivariate analysis have been used to determine the influential parameters on the reproduction number [31], [32]. We run 2000 samples obtained from triangular probability distribution.

Figure 1. showed the PRCC indices of the parameters of the reproduction numbers. It reveals that the infection-related parameters $m, b, p_{bh}, p_{vh}$ and the recovery rate $\gamma$ and the mosquito death rate $\mu_v$ are the influential parameters on the reproduction number. The last two parameters have positive relationship and the first one has negative relationship. It also shows that the vaccination rate has negative negative relationship which implies that an increase in the vaccination rate would reduce the reproduction number.

![PRCC values of the reproduction number](image)

3.2. Numerical Solution

The numerical simulations are presented in this section. In simulation, the parameter values are $\mu_h = 1/(65 \times 52)$, $\alpha = 1/(9 \times 52)$, $m = 2$, $b = 1$, $p_{bh} = 0.75$, $v = 0.01$, $q_1 = 1/24$, $\epsilon = 0.5$, $\gamma = 1$, $\mu_v = 1/2$, $p_{vh} = 0.75$, $q_2 = 1/52$, $N_h = 500$ and the initial conditions are $S_1(0) = 500$, $S_2(0) = 0$, $I_1(0) = I_2(0) = 2$, $R_1(0) = R_2(0) = 0$, $S_v(0) = 1000$, $I_v(0) = 2$. 
Figure 2 shows that condition when the reproduction number is greater than one. It can be seen that the number of children infected by dengue is higher than that of adult. This is because the adult individuals are vaccinated and the children is not.

3.3. Exploring the probability of extinction

We now perform the probability of extinction. The estimation of reproduction number for dengue showed the variation of $R_0$ between 1.1 to 4.22 [17], [33]–[35]. Here we the probability of extinction assuming the reproduction number is above unity which implies that the spectral radius of expectation matrix is also greater than unity [28]. The parameter values used are $\mu_h = 1/(65 \times 52), b = 1, m = 2, \gamma = 7/5.5, \mu_v = 1/2$ [17], [33]. The results are given in the Table 2.

<table>
<thead>
<tr>
<th>$i^0_1$</th>
<th>$i^0_2$</th>
<th>$i^0_v$</th>
<th>$P_0$</th>
<th>Approx.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0.3926</td>
<td>0.3614</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0.5256</td>
<td>0.4968</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>2</td>
<td>0.2933</td>
<td>0.2812</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>2</td>
<td>0.1541</td>
<td>0.1527</td>
</tr>
</tbody>
</table>

Table 2 showed that the probability of extinction using Formula (5) and the simulations provides the similar results. For the simulation, we simulate 1000 samples and the probability of extinction is calculated by the number of samples that hits zero infected individuals that is divided by the total samples. The results showed that an increase in the number of initial infections results in the low probability of extinction or higher probability of major outbreak. For example, when the initial condition of $I_1^0 = I_2^0 = I_v^0 = 1$, the probability of extinction using the formula given in Equation (5) is 0.3926, and 0.3614 using the simulations. It is clear that when the number of initial conditions increases, the probability of extinction decreases. This means that the initial conditions affect the probability of disease extinction.
4. CONCLUSION

In this paper, deterministic and stochastic epidemic model have been developed with age-structure framework. The probability of disease extinction is determined and sensitivity analysis has been performed. The results showed that the probability of dengue extinction is lower when the initial infections are higher. Furthermore, infection-related parameters determine the reproduction numbers, which govern the transmission dynamics of dengue. This means that factors contributing to an increase in the values of infections parameters needs to be controlled, which can reduce the potential for dengue transmission. The results implies that initial conditions determine the probability of disease extinction or probability of outbreak. When the initial infected individuals are higher, the probability of disease extinction is low. Furthermore, vaccination adult individuals would lead the lower infected adult individuals.

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