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Research Article

A Dengue Vaccination Model for Immigrants in a Two-Age-Class Population

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We develop a model of dengue transmission with some vaccination programs for immigrants. We classify the host population into child and adult classes, in regards to age structure, and into susceptible, infected and recovered compartments, in regards to disease status. Since migration plays important role in disease transmission, we include immigration and emigration factors into the model which are distributed in each compartment. Meanwhile, the vector population is divided into susceptible, exposed, and infectious compartments. In the case when there is no incoming infected immigrant, we obtain the basic reproduction ratio as a threshold parameter for existence and stability of disease-free and endemic equilibria. Meanwhile, in the case when there are some incoming infected immigrants, we obtain only endemic equilibrium. This indicates that screening for the immigrants is important to ensure the effectiveness of the disease control.

1. Introduction

Dengue fever is an endemic disease in many tropical countries, especially in the urban areas. This disease is caused by the dengue virus, which is transmitted to a human by the bite of infected female *Aedes aegypti* mosquitoes.

There are some epidemiological and demographical factors that contribute to the transmission of the disease. Age factor is among the important demographical factors affecting the transmission of the disease. From a theoretical point of view, age structure affects the dynamics of the disease transmission [1], and hence it should be taken into account in modeling the transmission of the disease to increase the realism of the model and to obtain a more prudent decision derived from the model. From a practical point of view, many

vaccination programs are directed to a certain class of age, unexceptionally in the case of dengue in which the Pediatric Dengue Vaccine Initiative targets children in their vaccination program (http://www.pdvi.org/). A study in [2] shows that a pediatric vaccination would be economically viable and highly cost effective, once a perfect dengue vaccine is made. A similar study shows that an optimal vaccination strategy could be given to only certain classes of age [3].

In literatures, most of the age-structured population models appear in the form of integropartial differential equations [4–6]. Some authors included age structure in epidemic models in the form of discrete compartmental differential equations, such as in [7–9]. The authors in [7] have generalized the model in [10] by separating the human population into age cohorts, and then for each cohort they construct a set of SIR equations. Disease-free and endemic equilibria are found, but there is no stability analysis for these equilibria. In [8], the authors have simplified their model to a two-age-class model. They allowed different transmission rates for the adult and the child classes and found disease-free and endemic equilibria. They also provided the condition for the local stability of the disease-free equilibrium in the general case. The stability condition for the endemic equilibrium has only been found for the special case, in which no infection occurs for the adult class.

The authors in [9] showed that a two-age-class model is a special case of a more general continuous age model for a certain choice of survival function. In their paper they discussed a two-age-class dengue transmission model by dividing the human population into child and adult classes and considered vaccination in the child class only. Many scientists believe that most dengue infections are asymptomatic. For every ten cases we see in the hospital, there should be at least 50–90 cases in the community who have only fever and no complications [11]. In this regards, the authors in [9] also showed that, in some circumstances, if there is an inadvertent vaccination to asymptomatic infectious children, which worsens their condition as the time span of being infectious increases, then paradoxically, vaccination can be counterproductive; that is, vaccination makes the basic reproduction number even bigger. This suggests that, in practice, screening to identify truly susceptibles is needed before implementing a vaccination program.

Beside age factor, another factor that plays important role in disease transmission is immigration. It is easy to understand that immigration of infectious individuals could ignite the spreading of a disease in a virgin populations. Diseases like HIV, SARS, and avian influenza are believed among the examples of diseases that might be caused by the immigrants of infectious individuals [12, 13]. Many mathematical models have been devised as the means to understand and to control those kinds of diseases [6, 14, 15]. The authors in [14] showed that if there is a constant influx of infective immigrants into a population, there will be no disease-free equilibrium.

Although the immigrants are not carrying a disease at all, still they have an impact on the transmission of a disease. The buildup of immigrants (also the locals) can be viewed as the buildup of susceptibles that are ready to be infected by any disease once available or enhance the spreading of the existing disease. In this respect, it is reasonable to enforce a policy to vaccinate incoming immigrant, following a screening, to ensure that they will not contribute to the buildup of susceptible.

There is no commercially dengue vaccine available yet. However, there are some potential dengue vaccines available. A survey in four South-Eastern Asian countries in 2002 revealed that there is a high and urgent perceived need for a dengue vaccine (http://www.pdvi.org/). To simulate vaccination program in gaining some insight on how vaccination would affect the transmission of the disease, even before the vaccine itself is available in

the market, is among the interests of vaccine scientists and policy makers. In this paper we develop a two-age-class model for dengue transmission by considering immigration vaccination strategy, as an anticipative study before the vaccine exists.

The introduction of immigration into the system is plausible since dengue is regarded as an urban disease [16], where the rate of immigration cannot be neglected. Different from [9] in which it is assumed that vaccination targets individuals in the child class, here we look at a scenario where vaccination is given to a portion of newborns (both immigrant and local babies) and a portion of newly arrived mature immigrants, to protect them from being infected by the local dengue disease. In practical point of view, the vaccination strategy proposed in this paper is easier to be implemented than the one in [9].

2. Model Formulation

Let us assume that the host population is classified into the child class and the adult class. Each of the classes is divided into the susceptible, infected, and recovered subclasses. We also assume that the recovered hosts have life-long immunity and there is no wanning effect of the vaccine, which means that the vaccine has a life-long permanent protection. So, the recovered hosts and the vaccinated hosts can be grouped into the recovered class.

We use variables $\widetilde{S}_C$, $\widetilde{I}_C$, and $\widetilde{R}_C$ to denote the size of the susceptible, infected, and recovered of child population, respectively. Similarly, we use the subscript A for the adult population.

We denote the susceptible, exposed, and infected vector populations by S_V, E_V, and I_V, respectively. We consider the latent class E_V, since the incubation period of the disease in mosquitoes is relatively large compared to the life span of the mosquitoes.

We use the diagram in Figure 1 for the dengue transmission in the population. The parameters P_C and P_A are the incoming immigration recruitment rates for child and adult classes, respectively, some positive fractions f_*, g_*, and h_* of the incoming immigrants are susceptible, infected, and recovered or vaccinated, respectively ($f_* + g_* + h_* = 1$). In practice, it is necessary to undertake screening to identify the susceptibility status of the incoming immigrants. There is also a constant birth recruitment rate B that increases the child population.

The parameters p and q are the fractions of susceptible incoming children (including natural birth) and susceptible incoming adults that are vaccinated; s is the vaccine efficacy; μ_C, μ_A, and μ_V are the child, adult, and vector natural death rates; respectively, ε_C and ε_A are the per capita emigration rates for children and adults, respectively; λ_C, λ_A, and λ_V are the successful infection rates for children, adults, and vectors; respectively, δ is the transition rate from child class to adult class; γ is the recovery rate, P_V and $1/\tau$ are the recruitment rate for vector and the latent period of vectors, respectively.

Using the transmission diagram in Figure 1, we formulate the following 9-dimensional model:

$$\frac{d\widetilde{S}_C}{dt} = (1-ps)\ (f_C P_C + B) - \lambda_C\ \frac{\widetilde{S}_C}{\widetilde{N}_H}\ \widetilde{I}_V - (\delta + \varepsilon_C + \mu_C)\widetilde{S}_C, \tag{2.1}$$

$$\frac{d\widetilde{I}_C}{dt} = g_C P_C + \lambda_C\ \frac{\widetilde{S}_C}{\widetilde{N}_H}\widetilde{I}_V - (\delta + \gamma + \varepsilon_C + \mu_C)\widetilde{I}_C, \tag{2.2}$$

$$\frac{d\widetilde{R}_C}{dt} = h_C P_C + ps\ (f_C P_C + B) + \gamma \widetilde{I}_C - (\delta + \varepsilon_C + \mu_C)\widetilde{R}_C, \tag{2.3}$$

$$\frac{d\widetilde{S}_A}{dt} = (1 - q\ s) f_A P_A + \delta \widetilde{S}_C - \lambda_A \frac{\widetilde{S}_A}{\widetilde{N}_H} \widetilde{I}_V - (\varepsilon_A + \mu_A)\widetilde{S}_A, \tag{2.4}$$

$$\frac{d\widetilde{I}_A}{dt} = g_A P_A + \lambda_A \frac{\widetilde{S}_A}{\widetilde{N}_H} \widetilde{I}_V + \delta \widetilde{I}_C - (\gamma + \varepsilon_A + \mu_A)\widetilde{I}_A, \tag{2.5}$$

$$\frac{d\widetilde{R}_A}{dt} = (h_A + q\ s\ f_A) P_A + \delta \widetilde{R}_C + \gamma \widetilde{I}_A - (\varepsilon_A + \mu_A)\widetilde{R}_A, \tag{2.6}$$

$$\frac{d\widetilde{S}_V}{dt} = P_V - \lambda_V \widetilde{S}_V \frac{\widetilde{I}_C + \widetilde{I}_A}{\widetilde{N}_H} - \mu_V \widetilde{S}_V, \tag{2.7}$$

$$\frac{d\widetilde{E}_V}{dt} = \lambda_V \widetilde{S}_V\ \frac{\widetilde{I}_C + \widetilde{I}_A}{\widetilde{N}_H} - (\tau + \mu_V)\widetilde{E}_V, \tag{2.8}$$

$$\frac{d\widetilde{I}_V}{dt} = \tau \widetilde{E}_V - \mu_V \widetilde{I}_V, \tag{2.9}$$

where $\widetilde{N}_H$ is the total population of host. Furthermore, we use $\widetilde{N}_C = \widetilde{S}_C + \widetilde{I}_C + \widetilde{R}_C$, $\widetilde{N}_A = \widetilde{S}_A + \widetilde{I}_A + \widetilde{R}_A$, and $\widetilde{N}_V = \widetilde{S}_V + \widetilde{E}_V + \widetilde{I}_V$ as the total populations of child, adult, and vector, respectively. These populations are governed by the following equations:

$$\frac{d\widetilde{N}_C}{dt} = P_C + B - (\delta + \varepsilon_C + \mu_C)\widetilde{N}_C, \tag{2.10}$$

$$\frac{d\widetilde{N}_A}{dt} = P_A + \delta \widetilde{N}_C - (\varepsilon_A + \mu_A)\widetilde{N}_A, \tag{2.11}$$

$$\frac{d\widetilde{N}_V}{dt} = P_V - \mu_V \widetilde{N}_V. \tag{2.12}$$

When $t \to \infty$, we have that $\widetilde{N}_C \to (P_C + B)/(\delta + \varepsilon_C + \mu_C)$, $\widetilde{N}_A \to (\delta(P_C + B) + (\delta + \varepsilon_C + \mu_C)P_A)/(\delta + \varepsilon_C + \mu_C)(\varepsilon_A + \mu_A)$, and $\widetilde{N}_V \to P_V/\mu_V$.

First, we consider that the host and vector populations have reached the limiting states; these are $\widetilde{N}_C = (P_C + B)/(\delta + \varepsilon_C + \mu_C)$, $\widetilde{N}_A = (\delta\ (P_C + B) + (\delta + \varepsilon_C + \mu_C)P_A)/(\delta + \varepsilon_C + \mu_C)(\varepsilon_A + \mu_A)$, $\widetilde{N}_V = P_V/\mu_V$, and $\widetilde{N}_H = \widetilde{N}_C + \widetilde{N}_A$. Then, we scale model (2.1)–(2.9) with following transformations $S_C = \widetilde{S}_C/\widetilde{N}_C$, $I_C = \widetilde{I}_C/\widetilde{N}_C$, $R_C = \widetilde{R}_C/\widetilde{N}_C$, $S_A = \widetilde{S}_A/\widetilde{N}_A$, $I_A = \widetilde{I}_A/\widetilde{N}_A$, $R_A = \widetilde{R}_A/\widetilde{N}_A$, $S_V = \widetilde{S}_V/\widetilde{N}_V$, $E_V = \widetilde{E}_V/\widetilde{N}_V$, and $I_V = \widetilde{I}_V/\widetilde{N}_V$. Thus, we obtain the following reduced model:

$$\frac{dS_C}{dt} = (1 - ps)\ (f_C Q_C + T) - \beta_C S_C I_V - (\delta + \varepsilon_C + \mu_C)S_C, \tag{2.13}$$

$$\frac{dI_C}{dt} = g_C Q_C + \beta_C S_C I_V - (\delta + \gamma + \varepsilon_C + \mu_C)I_C, \tag{2.14}$$

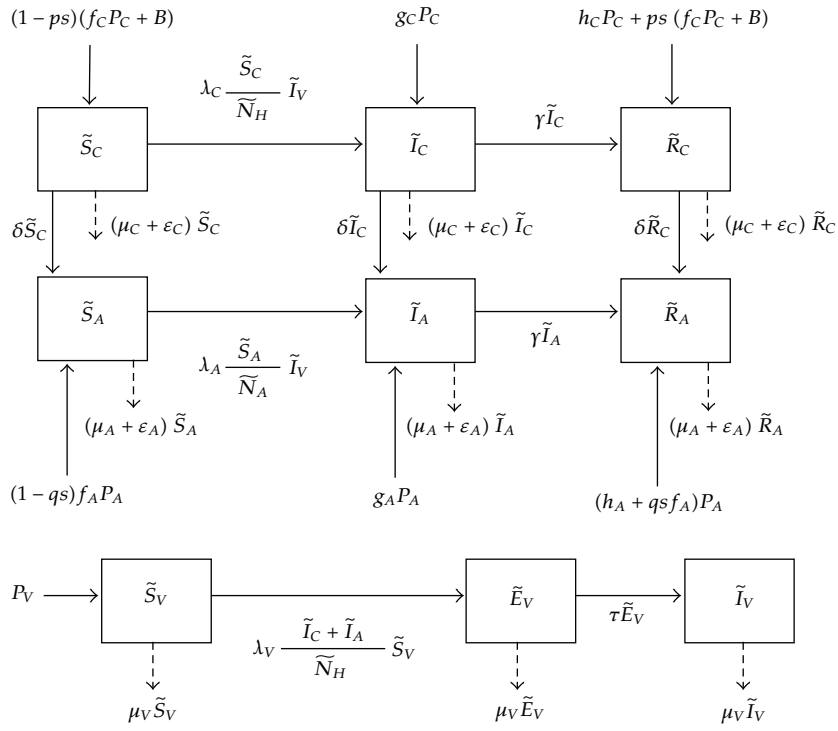


Figure 1: A two-age-class dengue transmission diagram.

$$\frac{dS_A}{dt} = (1-qs)\ f_AQ_A + \delta\sigma S_C - \beta_A S_A I_V - (\varepsilon_A+\mu_A)S_A, \tag{2.15}$$

$$\frac{dI_A}{dt} = g_{_A}Q_A + \beta_A S_A I_V + \delta\sigma I_C - (\gamma+\varepsilon_A+\mu_A)I_A, \tag{2.16}$$

$$\frac{dE_V}{dt} = S_V(\theta_C I_C + \theta_A I_A) - (\tau+\mu_V)E_V, \tag{2.17}$$

$$\frac{dI_V}{dt} = \tau E_V - \mu_V I_V, \tag{2.18}$$

where $T = B/\widetilde{N}_C$, $Q_C = P_C/\widetilde{N}_C$, $\beta_C = \lambda_C\widetilde{N}_V/\widetilde{N}_H$, $\theta_C = \lambda_V\widetilde{N}_C/\widetilde{N}_H$, $\sigma = \widetilde{N}_C/\widetilde{N}_A$, $Q_A = P_A/\widetilde{N}_A$, $\beta_A = \lambda_A\widetilde{N}_V/\widetilde{N}_H$, $\theta_A = \lambda_V\widetilde{N}_A/\widetilde{N}_H$, and $S_V = 1 - E_V - I_V$. The values of R_C and R_A in the limiting state can be evaluated using $R_C = 1 - S_C - I_C$ and $R_A = 1 - S_A - I_A$.

After the scaling, the region of biological interest of model (2.13)–(2.18) is

$$\Omega = \left\{(S_C, I_C, S_A, I_A, E_V, I_V) \in [0,1]^6 : S_C + I_C \le 1,\ S_A + I_A \le 1,\ E_V + I_V \le 1\right\}. \tag{2.19}$$

This region is positive invariant under the flow generated by the vector field of model (2.13)–(2.18), because the vector field on the boundary of Ω does not point out the exterior of Ω.

For the rest of the paper, we will analyze model (2.13)–(2.18) since this reduced model is the limiting system of model (2.1)–(2.9) and has the same asymptotic behavior as the original model [17, 18].

3. Model Analysis

Solving the equilibrium conditions of model (2.13)–(2.18), we obtain the following equations:

$$S_C = \frac{(1-ps)(f_C Q_C + T)}{\beta_C I_V + \delta + \varepsilon_C + \mu_C}, \tag{3.1}$$

$$I_C = \frac{g_C Q_C + \beta_C S_C I_V}{\delta + \gamma + \varepsilon_C + \mu_C}, \tag{3.2}$$

$$S_A = \frac{(1-qs)\ f_A Q_A + \delta\sigma S_C}{\beta_A I_V + \varepsilon_A + \mu_A}, \tag{3.3}$$

$$I_A = \frac{g_A Q_A + \beta_A S_A I_V + \delta\sigma I_C}{\gamma + \varepsilon_A + \mu_A}, \tag{3.4}$$

$$E_V = \frac{\mu_V}{\tau}\ I_V, \tag{3.5}$$

and the variable I_V satisfies $M(I_V) + N(I_V) = 0$, where

$$M(I_V) = \left(c_2\ I_V^2 + c_1\ I_V + c_0\right)\ I_V, \tag{3.6}$$

$$\begin{aligned} N(I_V) = {} & (I_V\beta_A + \varepsilon_A + \mu_A)(I_V\beta_C + \delta + \varepsilon_C + \mu_C)(I_V(\mu_V + \tau) - \tau) \\ & \times [g_A Q_A \theta_A(\gamma + \delta + \varepsilon_C + \mu_C) + g_C Q_C(\theta_C(\gamma + \varepsilon_A + \mu_A) + \theta_A\delta\sigma)], \end{aligned} \tag{3.7}$$

$$\begin{aligned} c_2 = {} & \beta_A\beta_C(\mu_V + \tau)\ \Big[(1-qs)\ f_A Q_A\theta_A(\gamma + \delta + \varepsilon_C + \mu_C) \\ & + \mu_V(\gamma + \varepsilon_A + \mu_A)\ (\gamma + \delta + \varepsilon_C + \mu_C) \\ & + S_C^d\ (\delta + \varepsilon_C + \mu_C)\ (\theta_C(\gamma + \varepsilon_A + \mu_A) + \theta_A\ \delta\ \sigma)\Big], \end{aligned} \tag{3.8}$$

$$\begin{aligned} c_1 = {} & \beta_C(\varepsilon_A + \mu_A)(\gamma + \delta + \varepsilon_C + \mu_C) \\ & \times \left[\mu_V(\gamma + \varepsilon_A + \mu_A)(\mu_V + \tau) - \beta_A\theta_A\tau S_A^d\right] \\ & + \beta_A(\delta + \varepsilon_C + \mu_C)\left[\mu_V(\gamma + \varepsilon_A + \mu_A)(\gamma + \delta + \varepsilon_C + \mu_C)(\mu_V + \tau)\right. \\ & \left. - \beta_C\tau S_C^d(\theta_C(\gamma + \varepsilon_A + \mu_A) + \delta\sigma\theta_A)\right] \\ & + (\varepsilon_A + \mu_A)(\delta + \varepsilon_C + \mu_C)(\mu_V + \tau)\ \Big[\beta_A\theta_A S_A^d(\gamma + \delta + \varepsilon_C + \mu_C) \\ & + \beta_C S_C^d(\theta_C(\gamma + \varepsilon_A + \mu_A) + \delta\sigma\theta_A)\Big] \\ & + \beta_A\beta_C\theta_A\delta\sigma\tau S_C^d\ (\gamma + \delta + \varepsilon_C + \mu_C), \end{aligned} \tag{3.9}$$

$$c_0 = (\delta + \gamma + \varepsilon_C + \mu_C)(\delta + \varepsilon_C + \mu_C)(\varepsilon_A + \mu_A)\ (\gamma + \varepsilon_A + \mu_A)\mu_V\ (\mu_V + \tau)\ (1 - R_0), \tag{3.10}$$

$$S_C^d = \frac{(1 - p\ s)\ (f_C\ Q_C + T)}{\delta + \varepsilon_C + \mu_C}, \tag{3.11}$$

$$S_A^d = \frac{(1 - q\ s)\ f_A\ Q_A + \delta\ \sigma\ S_C^d}{\varepsilon_A + \mu_A}, \tag{3.12}$$

$$R_0 = \frac{\tau\left[\beta_C S_C^d(\theta_C(\gamma + \varepsilon_A + \mu_A) + \theta_A\delta\sigma) + \beta_A\theta_A S_A^d(\delta + \gamma + \varepsilon_C + \mu_C)\right]}{\mu_V\ (\gamma + \varepsilon_A + \mu_A)\ (\delta + \gamma + \varepsilon_C + \mu_C)\ (\mu_V + \tau)}. \tag{3.13}$$

The zeros of the polynomial $M + N$ determine the equilibrium of model (2.13)–(2.18). We analyze the zeros of the polynomial $M + N$ into two cases. The first case is if there is no incoming infected immigrant, so the incoming immigrants are susceptible or have permanent immunity to the dengue infection. In this case, only polynomial M determines the equilibrium. The second case is if there are some incoming infected immigrants. In the second case, both polynomials M and N determine the equilibrium.

3.1. No Incoming Infected Immigrants

In this subsection, we consider the case where there is no incoming infected immigrant or mathematically $g_C = g_A = 0$. Furthermore, the condition $g_C = g_A = 0$ implies that polynomial N becomes a zero polynomial.

In this case, model (2.13)–(2.18) has a disease-free equilibrium; that is, $E^d = (S_C^d, 0, S_A^d, 0, 0, 0)$, where S_C^d and S_A^d are exactly as in (3.11)-(3.12). This equilibrium is obtained by substituting $I_V = 0$ into (3.5).

If the vaccination programme is not implemented ($p = q = 0$) and all immigrants are susceptibles ($f_C = f_A = 1$), then we obtain $S_C^d = S_A^d = 1$ and $R_C^d = R_A^d = 0$. In the limiting case where all susceptible immigrants and all births are vaccinated ($p = q = 1$) and the vaccine efficacy is perfect ($s = 1$), we have $S_C^d = S_A^d = 0$ and $R_C^d = R_A^d = 1$.

Basic reproduction ratio is the expected number of secondary cases per primary case in a "virgin" population [19]. It is an important threshold because it determines whether an initial infection in a virgin population will end up in an endemic. This threshold parameter is given by the spectral radius of the next-generation matrix. The spectral radius of our next-generation matrix is the square root of R_0, where R_0 is exactly as in (3.13). This square root of R_0 can be interpreted as the basic reproduction ratio under vaccination programme.

Next, we explore the existence of the endemic equilibrium of model (2.13)–(2.18) when $g_C = g_A = 0$. Here, we consider the equation $c_2 I_V^2 + c_1 I_V + c_0 = 0$, where the coefficients c_0, c_1, and c_2 are as in (3.8)–(3.10).

It can be seen that c_2 is positive. The coefficient c_0 is positive for $R_0 < 1$, and it is negative for $R_0 > 1$. Moreover, for $R_0 = 1$, we have that $c_0 = 0$ and $c_1, c_2 > 0$. So, model (2.13)–(2.18) cannot exhibit backward bifurcation at $R_0 = 1$.

For $R_0 \leq 1$, we have following inequalities:

$$\begin{aligned} \beta_C\tau S_C^d(\theta_C(\gamma + \varepsilon_A + \mu_A) + \delta\sigma\theta_A) &< \mu_V(\gamma + \varepsilon_A + \mu_A) \\ (\delta + \gamma + \varepsilon_C + \mu_C)(\mu_V + \tau)\beta_A\theta_A\tau S_A^d &\\ < \mu_V(\gamma + \varepsilon_A + \mu_A)(\mu_V + \tau). & \end{aligned} \tag{3.14}$$

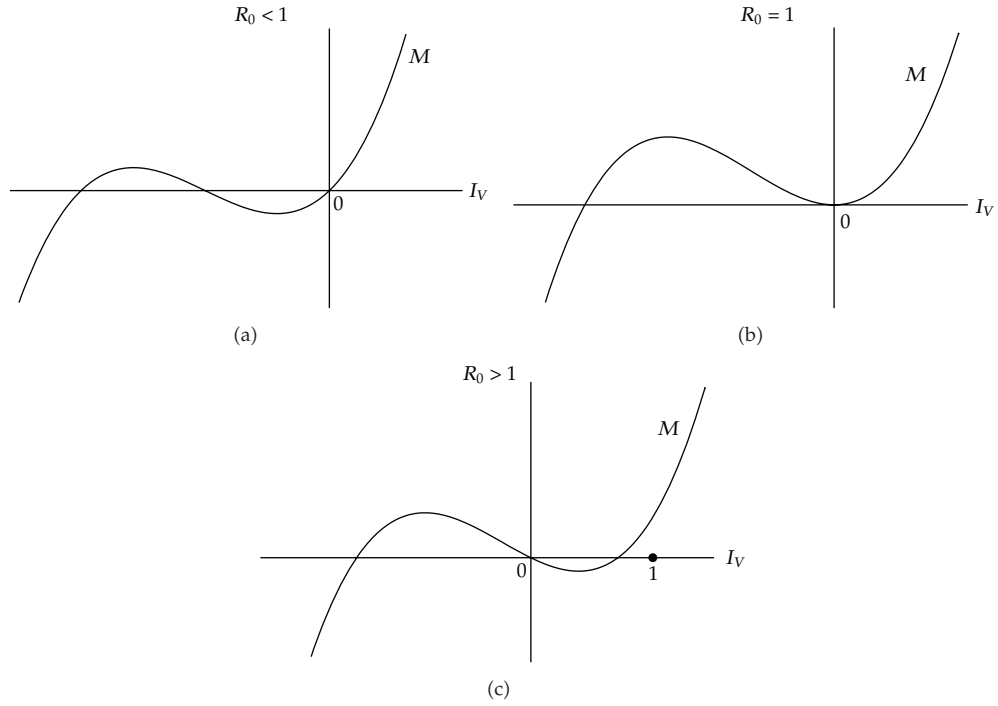


Figure 2: Three typical graphs of the polynomial M in (3.6) with respect to three conditions of R_0.

These inequalities imply $c_1 > 0$ for $R_0 \leq 1$. Thus, there is no positive root I_V of the equation $c_2\, I_V^2 + c_1\, I_V + c_0 = 0$ for $R_0 \leq 1$. And there is a unique positive root I_V^e of the equation $c_2\, I_V^2 + c_1\, I_V + c_0 = 0$ which is always less than one for $R_0 > 1$. Figure 2 gives three qualitative graphs of M with respect to the three conditions of R_0.

It can be verified that the equilibrium $E^e = (S_C^e, I_C^e, S_A^e, I_A^e, E_V^e, I_V^e)$ whose coordinates satisfy equations (3.5) is in int(Ω) if and only if $R_0 > 1$. We summarized these results in the following proposition.

Proposition 3.1. *Let $g_C = g_A = 0$. Model* (2.13)–(2.18) *always has a unique disease-free equilibrium E^d in Ω. For $R_0 > 1$, model* (2.13)–(2.18) *also has a unique positive endemic equilibrium E^e in* int(Ω) *whose components satisfy* (3.5)*, and I_V satisfies $c_2 I_V^2 + c_1 I_V + c_0 = 0$, where the coefficients c_0, c_1, c_2 are as in* (3.8)–(3.10).

The next proposition gives the stability of equilibrium E^d.

Proposition 3.2. *Let $g_C = g_A = 0$. The disease-free equilibrium E^d is locally asymptotically stable if $R_0 < 1$ and it is unstable if $R_0 > 1$.*

Proof. The linearization of model (2.13)–(2.18) at point E^d gives the Jacobian matrix:

$$A = \begin{pmatrix} A_1 & A_3 \\ 0 & A_2 \end{pmatrix}, \tag{3.15}$$

where

$$A_1 = \begin{pmatrix} -\delta - \varepsilon_C - \mu_C & 0 \\ \delta\ \sigma & -\varepsilon_A - \mu_A \end{pmatrix},$$

$$A_2 = \begin{pmatrix} -\delta - \gamma - \varepsilon_C - \mu_C & 0 & 0 & \beta_C S_C^d \\ \delta\ \sigma & -\gamma - \varepsilon_A - \mu_A & 0 & \beta_A S_A^d \\ \theta_C & \theta_A & -\mu_V - \tau & 0 \\ 0 & 0 & \tau & -\mu_V \end{pmatrix}. \tag{3.16}$$

Moreover, the eigenvalues of matrices A_1 and A_2 determine the local stability of E^d.

The eigenvalues of matrix A_1 are $-(\delta+\varepsilon_C+\mu_C)$ and $-(\varepsilon_A+\mu_A)$. The matrix $-A_2$ is an M-matrix. The real parts of all eigenvalues of matrix $-A_2$ are positive if and only if $\det(-A_2) > 0$ (see [20]). Furthermore, all eigenvalues of A_2 have negative real parts if and only if $\det(A_2) > 0$. The determinant of matrix A_2 is given by

$$\det(A_2) = -\mu_V(\gamma + \varepsilon_A + \mu_A)(\delta + \gamma + \varepsilon_C + \mu_C)\ (\mu_V + \tau)\ (R_0 - 1). \tag{3.17}$$

Thus, if $R_0 < 1$, then the equilibrium E^d is locally asymptotically stable and it is unstable if $R_0 > 1$. □

Let the endemic equilibrium $E^e = (S_C^e, I_C^e, S_A^e, I_A^e, E_V^e, I_V^e)$ exists. Linearization of model (2.13)–(2.18) at point E^e gives following Jacobian matrix:

$$J = \begin{pmatrix} \Delta_1 & 0 & 0 & 0 & 0 & -\beta_C S_C^e \\ \beta_C\ I_V^e & \Delta_2 & 0 & 0 & 0 & \beta_C S_C^e \\ \delta\ \sigma & 0 & \Delta_3 & 0 & 0 & -\beta_A S_A^e \\ 0 & \delta\ \sigma & \beta_A\ I_V^e & \Delta_4 & 0 & \beta_A S_A^e \\ 0 & S_V^e \theta_C & 0 & \theta_A\ S_V^e & \Delta_5 & -(\theta_A I_A^e + \theta_C I_C^e) \\ 0 & 0 & 0 & 0 & \tau & -\mu_V \end{pmatrix}, \tag{3.18}$$

where $\Delta_1 = -(\beta_C\ I_V^e + \delta + \varepsilon_C + \mu_C)$, $\Delta_2 = -(\gamma + \delta + \varepsilon_C + \mu_C)$, $\Delta_3 = -(\beta_A I_V^e + \varepsilon_A + \mu_A)$, $\Delta_4 = -(\gamma + \varepsilon_A + \mu_A)$, $\Delta_5 = -(\theta_A I_A^e + \theta_C I_C^e + \mu_V + \tau)$, and $S_V^e = 1 - E_V^e - I_V^e$.

It is not easy to prove analytically that all eigenvalues of J have negative real parts for $R_0 > 1$. However, from our numerical simulations (case $R_0 > 1$) all of the eigenvalues have negative real parts. Figure 3 gives the projection of three orbits of three different initial conditions when $R_0 > 1$ on the $I_C - I_A$ plane. The component (I_C, I_A) of the equilibrium E^e is

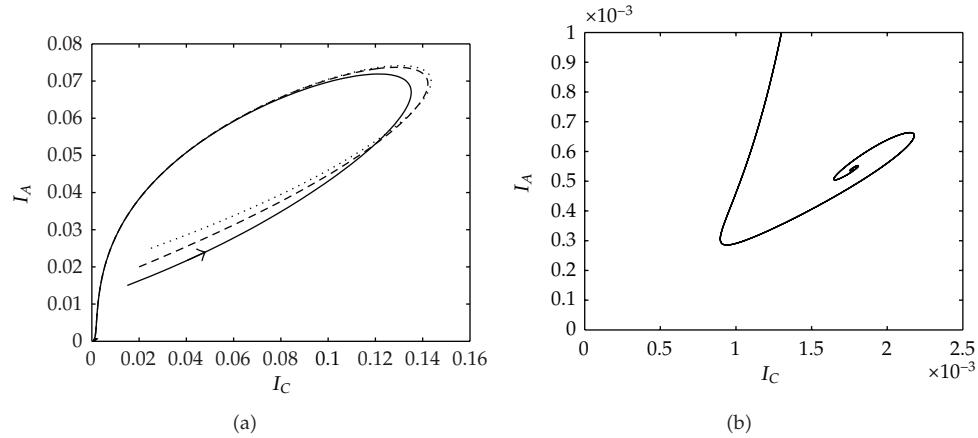


Figure 3: Projection of three orbits of model (2.13)–(2.18) on $I_C - I_A$ plane (a) and the projection near the equilibrium state (b). This projection indicates the local stability of the endemic equilibrium E^e when $R_0 > 1$.

not $(0, 0)$. This simulation indicates that the endemic equilibrium E^e is locally asymptotically stable when $R_0 > 1$.

3.2. Some Incoming Immigrants Are Infected

Here, we consider the case that there are some infected incoming immigrants; that is, g_C or g_A is larger than zero. In this case, we have following proposition.

Proposition 3.3. *Let g_C or g_A be larger than zero. Model* (2.13)–(2.18) *always has a unique positive endemic equilibrium E^f in* int(Ω) *whose components satisfy* (3.5) *and I_V satisfies $M(I_V)+N(I_V) = 0$.*

We will give the outline of proof of Proposition 3.3.

Outline of proof. When g_C or g_A or both are larger than zero, the cubic polynomial N in (3.7) always has two negative zeros and one positive zero which is less than one. Figure 4 gives the graph of the polynomial N.

The cubic polynomial M in (3.6) always has a trivial zero. Depending on R_0, the other two zeros could be negative, zero, or positive. Figure 2 illustrates three typical graphs of the polynomial M with respects to R_0.

Figure 5 gives the graph of polynomial $M + N$. The graph always has two negative zeros and one positive zero which is less than one. This positive zero is the component I_V of endemic equilibrium E^f.

From Proposition 3.3, there is no disease-free equilibrium and there is only endemic equilibrium if there are always some infected incoming child or adult immigrants. So, it is very important to do screening for the child and adult immigrants. The infected immigrants should be quarantined as long as they are ill. Otherwise, we will lose the disease-free condition. Here, we get a similar conclusion as in [14]. In [14], the authors did not separate the child class and the adult class in their model.

Figures 6 and 7 show the values of the equilibrium infected child population I_C and the equilibrium infected adult population I_A as the function of the portion of infected child

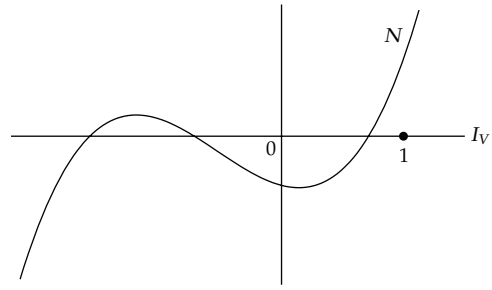


Figure 4: Graph of the polynomial N.

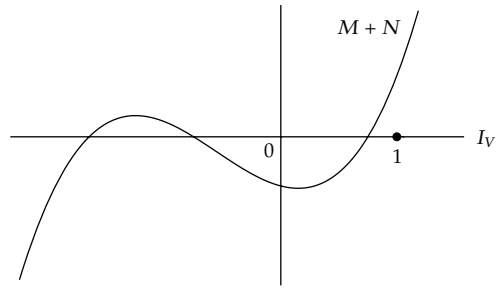


Figure 5: Graph of the polynomial $M + N$.

g_C and adult immigrants g_A. In Figure 6, we use parameters which produce $R_0 < 1$ around $g_C = g_A = 0$. Note that the lowest point $(g_C = g_A = 0)$ corresponds to the components I_C^d and I_A^d of the disease-free equilibrium E^d. When $g_C, g_A \neq 0$, the points in the surface correspond to the components I_C^f and I_A^f of the endemic equilibrium E^f. However, in Figure 7, we use parameters which produce $R_0 > 1$ around $g_C = g_A = 0$. Here, the lowest point $(g_C = g_A = 0)$ corresponds to the components I_C^e and I_A^e of the endemic equilibrium E^e. When $g_C, g_A \neq 0$, the points in the surface correspond to the components I_C^f and I_A^f of endemic equilibrium E^f. Despite the difference in the resulting properties of the basic reproduction number, and since both I_A and I_C constitute the endemic equilibrium E_f, the figures in fact indicate the existence of this endemic equilibrium when g_C and g_A are not zero.

The stability of the endemic equilibrium E^f is not easy to be obtained analytically. Numerical simulations indicate the local stability of the equilibrium E^f. Figure 8 gives three orbits of three different sets of parameter values. This simulation indicates that the equilibrium E^f is locally asymptotically stable.

4. Numerical Simulation

In the following numerical simulations, we use data in Table 1.

In Figure 9, we simulate four different scenarios, relative to no vaccination scenario and low screening level, that is, $g_a = g_c = 20\%$. The situation is described as follow, first if we raise the level of screening twice, that is, reduction of g_a and g_c from 20% to 10%, the infection will decrease from 100% to 85.7% for I_a and 94% for I_c. If we gain the screening process up to four times, we have the infection decreasing from 100% to 82.1% for I_a and 91.5% for I_c.

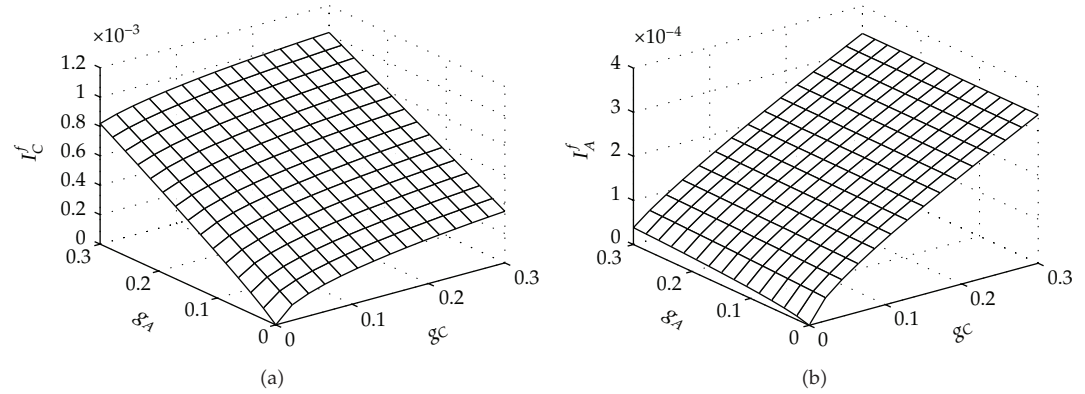


Figure 6: Component I_C(a) and component I_A(b) of endemic equilibria E^f. Here, $R_0 < 1$ when $g_C = g_A = 0$.

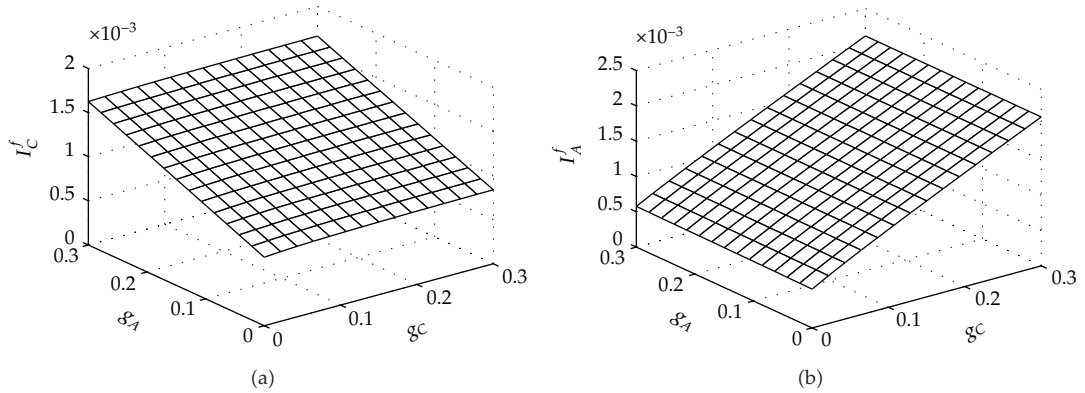


Figure 7: Component I_C(a) and component I_A(b) of endemic equilibria E^f. Here, $R_0 > 1$ when $g_C = g_A = 0$.

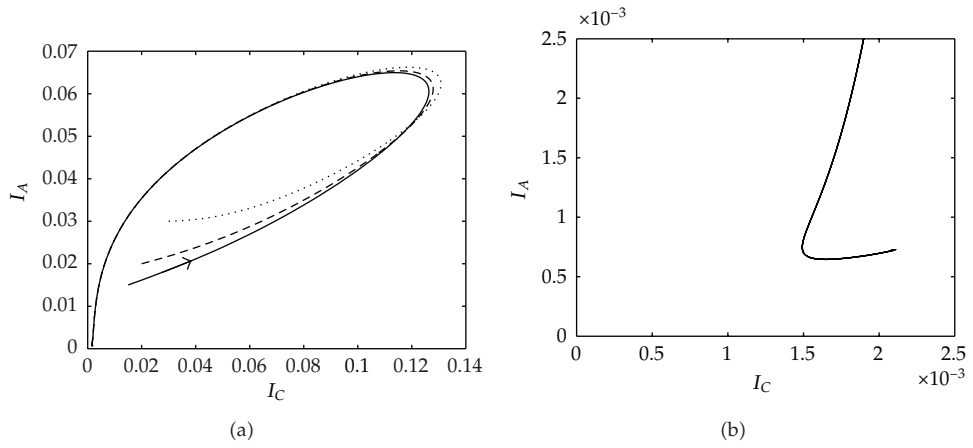


Figure 8: Projection of three orbits of model (2.13)–(2.18) on $I_C - I_A$ plane (a) and the projection near the equilibrium state (b). This projection indicates the local stability of the endemic equilibrium E^f.

Table 1: Data for numerical simulations (y represents years).

Par.	Value	Par.	Value	Par.	Value
P_C	200/y	P_A	2000/y	P_V	5000/y
λ_C	1168/y	λ_A	584/y	λ_V	2190/y
$1/\mu_C$	14.8 y	$1/\mu_A$	60 y	$1/\mu_V$	1/12 y
f_C	0.75	f_A	0.75	$1/\tau$	1/24 y
g_C	0.05	g_A	0.05	B	200/y
ε_C	0.2/y	ε_A	0.1/y	γ	36.5/y
s	0.95	$1/\delta$	15 y	—	—
p	0.9	q	0.9	—	—

Table 2: Percentages of endemicity. We use the first scenario as a reference scenario for the other four scenarios.

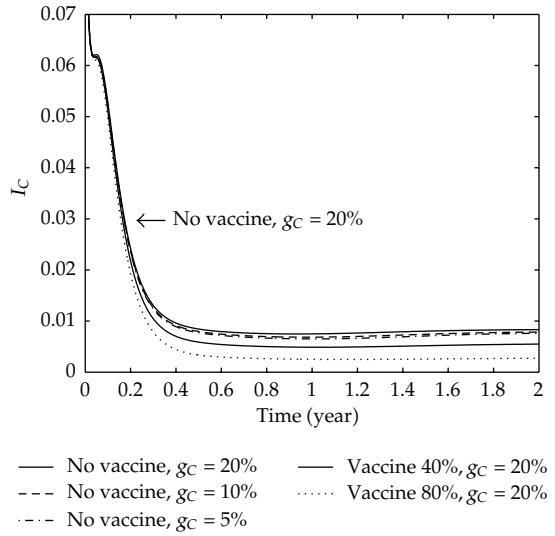
Scenarios	I_A	I_C	Percentage of I_A	Percentage of I_C
No vaccination, $g_a = g_c = 20\%$	0.0028	0.0083	100.0%	100.0%
No vaccination, $g_a = g_c = 10\%$	0.0024	0.0078	85.7%	94%
No vaccination, $g_a = g_c = 5\%$	0.0023	0.0076	82.1%	91.5%
Vac. $p = q = 40\%$, $g_a = g_c = 20\%$	0.002	0.0055	71.4%	66.3%
Vac. $p = q = 80\%$, $g_a = g_c = 20\%$	0.0012	0.0027	42.9%	32.5%

Hence, increasing the level of screening will decrease the endemicity. But if we vaccine 40% of children and adult ($p = q = 40\%$), the decreasing level of infection is 71.4% for I_a and 66.3% for I_c, and if we raise the coverage of vaccination to 80% ($p = q = 80\%$), we can reduce the infection up to 42.9% for I_a and 32.5% for I_c. So, increasing the coverage of vaccination will also decrease the endemicity. The summary of the scenarios can be seen in Table 2.

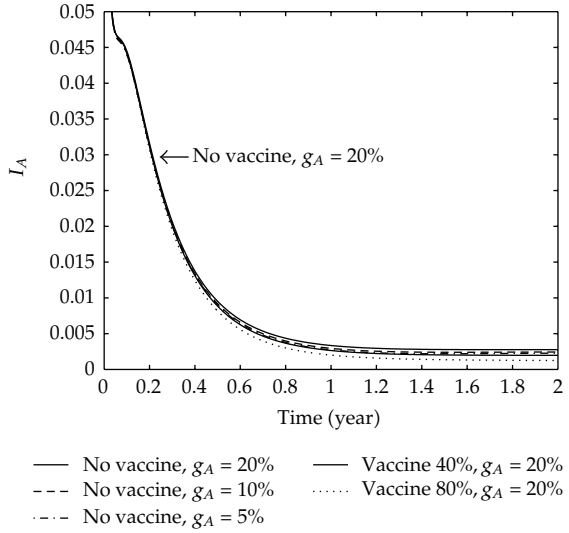
5. Conclusion

In this paper we derive a mathematical model of dengue transmission with vaccination program. The model incorporates two-age classes and migration. We also consider a susceptibility distribution in the incoming migrants.

From the analysis of the model, we obtain a conclusion that the susceptibility distribution is an important factor for the existence of disease-free equilibrium. If there is no incoming infected immigrant, then we have a unique disease-free equilibrium and a unique endemic equilibrium which depend on the basic reproduction ratio. Moreover, the stability of the equilibria also depends on the basic reproduction ratio. However, if some of the incoming immigrants are infected, then we only have a unique endemic equilibrium. Hence, screening for the incoming immigrants must be done. The incoming infected immigrants should be



(a)



(b)

Figure 9: Dynamics of some scenarios. Here, we take (0.3, 0.1, 0.5, 0.1, 0.1, 0.1) as the initial condition.

quarantined until they are recovered. Otherwise, we will lose the disease-free state from the population.

From the sensitivity analysis of the level of screening and the coverage of vaccination, increasing one of these parameters will give the reduction of endemic level. Increasing both parameters will give larger reduction of endemic level. The resulting simulation could give prior information for policy maker in setting the scale of vaccination and understanding the effect of vaccination in the reduction of endemic level.

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