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DYNAMICS OF A DENGUE FEVER MODEL WITH UNREPORTED CASES AND ASYMPTOMATIC INFECTED CLASSES IN SINGAPORE, 2020*

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Abstract This study is devoted to consider a novel model of dengue fever with unreported cases and asymptomatic infected classes, where infected humans is admitted to general and intensive hospitals for treatment. First, the basic reproduction number is calculated by using the next generation matrix method. The disease-free equilibrium is locally asymptotically stable when the basic reproduction number is less than one, but forward bifurcation occurs at the disease-free equilibrium when the basic reproduction number equals one. Then, the endemic equilibrium is consistent persistence when the basic reproduction number is more than one. Next, the existence of the optimal control pair is analyzed, and the mathematical expression of the optimal control is given by using Pontriagin's maximum principle. Finally, based on the dengue fever data in Singapore during the 15-52 weeks of 2020, the best fitting parameters of the model are determined by using Markov Chain Monte Carlo algorithm. The basic reproduction number is 1.6015 (95%CI: (1.5425-1.6675)). Numerical simulation and sensitivity analysis of several parameters are carried out. It is suggested that patients with dengue fever should report and receive treatment in time, which is of great significance for prevention and control of dengue fever.

Keywords Dengue fever, stability, bifurcation, optimal control, unreported case.

MSC(2010) 34D05, 34D20, 34D23, 49J15.

1. Introduction

Dengue fever, one of the most serious mosquito-borne diseases, is spread by the dengue virus, a flavivirus, mainly through the bite of infected Aedes(i.e., Aedes aegypti and Aedes albopictus) [8, 12, 19, 34]. The dengue virus is endemic in at least 80 countries around the world, with approximately 10 to 20 thousands death reported each year and the number of cases has increased 30-fold in the past 50 years, threatening 2.5 billion people [31, 38]. Among many mosquito-borne diseases, it

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ranks only behind malaria in morbidity and mortality, with more than 100 million people diagnosed with both each year [11, 18, 22, 28]. Once bitten by a dengue-carrying mosquito, a person can become infected with the virus, which can cause symptoms ranging from mild muscle pain and headache to severe hemorrhagic fever and even shock. Similarly, mosquitoes can catch the virus by biting a person who carries the virus [38].

Dengue virus are serotyped five, but cross-immunity among them is low. That is, once someone is infected with one serotype recovers, he (or she) will easily infect with another serotype [43,50]. An individual infected with one serotype has an immunity period of about six months before being re-infected with a different serotype [44]. The epidemiological relationship between human and mosquito-borne transmission is as follows. The female Aedes mosquitoes infect with the dengue virus after eating and biting the blood of an infected individual [7, 29]. After a period of about 3 to 14 days, mosquitoes become infectious [5, 27]. Infected mosquitoes have no immunity and can transmit the virus during their life cycle [31,42,43]. Similarly, one susceptible person becomes infectious after being bitten by an infectious mosquito [5, 27, 42]. Mosquitoes go from being infected to being contagious. This period is called the external incubation period (EIP). In turn, the population goes from being infected to being infectious. Such period is known as the internal incubation period (IIP) [5]. The virus can range from classic dengue to secondary dengue, which can lead to dengue haemorrhagic fever or dengue shock syndrome [8, 30]. Symptoms appear between 3 and 7 days, which roughly equivalent to the infection period the period of infection.

In recent decades, many models of dengue fever have been studied to analyze and predict the development trend and transmission dynamics of dengue fever [2, 14, 25, 26, 32, 36, 41, 42, 45, 49]. Xue et al. [42] developed a mathematical model considering optimal control, and showed the optimal conditions for controlling the spread of the disease by numerical simulations. Zheng et al. [49] used a delayed differential equation model to analyze the control effect of the release of infected Wolbachia males on dengue fever, and obtained two critical thresholds for the release of infected Wolbachia males. Their results suggest that even when vaccine efficacy is relatively low, age-specific vaccination may be cost-effective as long as the cost of vaccination is low enough. Cai et al. [2] showed a dengue fever model with bilinear incidence rate and standard incidence rate, and presented a representation of the basic reproduction number. Zhang and Lui [45] considered released Wolbachia infected Aedes *aegypti* mosquitoes to control mosquito populations and prevent the spread of dengue fever. Shim [32] developed a dengue model with age structure and vaccination to study dengue dynamics and vaccine cost-effectiveness in the Philippines. Magal and Webb [23] analyzed the relationship between reported and unreported cases, and showed that the proportion of unreported cases was very high, which is of great significance for taking measures to control the epidemic. Musa et al. [27] established a deterministic model finding a consistent fitting results and equivalent goodness-of-fit when asymptomatic infection was considered. Jing et al. [16] established a non-autonomous influenza model with meteorological factors and unreported cases, and showed that unreported cases accounted for a very high proportion of infected people, and improving the reporting rate of diseases by public health departments could help control the spread of diseases. Jing et al. [17] also studied the influence model with ozone concentration in air, pulse vaccination on influenza and unreported cases, and obtained that increasing the

vaccine coverage rate and appropriately increasing ozone concentration could effectively prevent the spread of influenza, and controlling the number of unreported cases also plays a very important role. In order to demonstrate that the effect of unreported cases, Huo et al. [15] also considered unreported cases in the outbreak in periodic influenza model. However, according to the actual situation, the asymptomatic infected should also become the asymptomatic infected (infectious) through the incubation period, rather than directly becoming the asymptomatic infected from the susceptible.

The purpose of this paper is to extend the known models in order to describe the individuals who are asymptomatic, and the unreported cases who are in the stage of infection. Our model includes both mosquito and human populations. Taking into account the characteristics of dengue fever disease, some of the patients with the first infection will recover by themselves and enter the compartment of infections(I), some of the patients with mild symptoms will be treated and recovered in the general hospital(H_m), and a few of the patients with serious re-infection will enter the severe hospital(H_s) for treatment and recover. The hospital warehouses can be converted to each other.

The rest work of this paper is as follows. In Section 2, a dengue fever model with unreported cases and asymptomatic infection classes is established. In Section 3, the basic number of regenerations is calculated, the stability of the disease-free equilibrium and forward bifurcation are given. The existence of the endemicity equilibrium and consistent persistence of disease are given, and sensitivity analysis are showed in Section 4. In Section 5, Pontryagin's Maximum optimal control strategy is investigated. One case and the corresponding numerical results are obtained in Section 6. A brief discussion is included in the last part.

2. Model Formulation

First, we show a compartmental model based on the susceptible-exposed-infected-recovered (SEIR) structure incorporating the demographic process (i.e.,births and deaths). Assume that the total human population size $N_h(t)$ is divided into eight classes: $S_h(t)$, $E_h(t)$, $I_h(t)$, $A_h(t)$, $P(t)$, $H_m(t)$, $H_s(t)$, $R(t)$. Here, $S_h(t)$ represents the susceptible people, $E_h(t)$ represents the exposed people(infected but not infectious), $I_h(t)$ represents the symptomatically infected people(contain of those having mild symptoms and severe symptoms), $H_m(t)$ represents the mild hospital people, $H_s(t)$ represents the severe hospital people, $A_h(t)$ represents the asymptomatically infected(exposed individuals that becomes infectious without showing any clinical symptoms), $P(t)$ represents the unreported infected people, $R(t)$ represents the recovered people. Further, assume that the total mosquito population $N_m(t)$ is divided into three classes: $S_m(t)$ representing the susceptible mosquito, $E_m(t)$ representing the exposed mosquito(infected but not infectious mosquitoes), $I_m(t)$ represents symptomatic mosquito.

Thus, the model for the dengue virus transmission in the human and mosquito populations is given by the following deterministic ordinary differential equations

(ODE) systems (2.1).

$$\begin{cases} \dfrac{\mathrm{d}S_h}{\mathrm{d}t} = \pi_h - \lambda_h S_h - \mu_h S_h, \\ \dfrac{\mathrm{d}E_h}{\mathrm{d}t} = \lambda_h S_h - \sigma_h E_h - \mu_h E_h, \\ \dfrac{\mathrm{d}A_h}{\mathrm{d}t} = (1-\rho)\sigma_h E_h - (\gamma_a + \delta + \mu_h) A_h, \\ \dfrac{\mathrm{d}I_h}{\mathrm{d}t} = \rho\sigma_h E_h - (\gamma_i + \tau + \xi + \omega + \mu_h) I_h, \\ \dfrac{\mathrm{d}P}{\mathrm{d}t} = \delta A_h + \omega I_h - (\gamma_p + \mu_h) P, \\ \dfrac{\mathrm{d}H_m}{\mathrm{d}t} = \tau I_h + \psi H_s - (\gamma_m + \varphi + \mu_h) H_m, \\ \dfrac{\mathrm{d}H_s}{\mathrm{d}t} = \xi I_h + \varphi H_m - (\gamma_s + \psi + \mu_h) H_s, \\ \dfrac{\mathrm{d}R_h}{\mathrm{d}t} = \gamma_a A_h + \gamma_p P + \gamma_i I_h + \gamma_m H_m + \gamma_s H_s - \mu_h R_h, \\ \dfrac{\mathrm{d}S_m}{\mathrm{d}t} = \pi_m - (\lambda_m + \mu_m) S_m, \\ \dfrac{\mathrm{d}E_m}{\mathrm{d}t} = \lambda_m S_m - (\sigma_m + \mu_m) E_m, \\ \dfrac{\mathrm{d}I_m}{\mathrm{d}t} = \sigma_m E_m - \mu_m I_m. \end{cases} \tag{2.1}$$

Here, the rates of human infection and mosquito infection are given by $\lambda_h = \frac{kl\beta_h I_m}{N_h}$, and $\lambda_m = \frac{kl\beta_m(I_h + A_h + P + H_m + H_s)}{N_h}$, respectively.

The transfer diagram of the model (2.1) is shown in Figure 1.

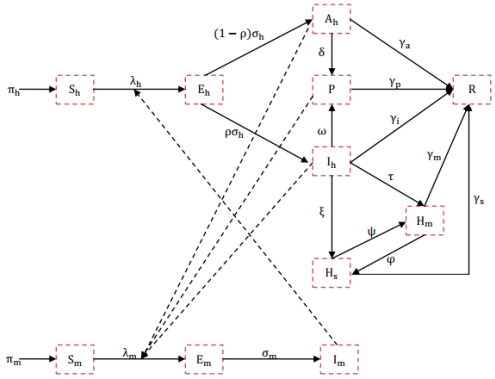


Figure 1. Transfer diagram for the model (2.1).

In model (2.1), these parameters are described in Table 1. Excepting the $1-\rho$ is the fraction of the asymptomatic humans.

Table 1. Descriptions of the parameters in the model (2.1).

Parameter	Description
π_h/π_m	The constant recruitment rate of the population/mosquitoes
μ_h/μ_m	The natural death rate of the population/mosquitoes
k	Mosquitoes biting rate
l	Maximum number of bites a human can receive per unit time
β_h	Transmission probability from infectious mosquitoes to susceptible population
β_m	Transmission probability from infectious humans to susceptible mosquitoes
σ_h/σ_m	Progression rate of exposed humans/mosquitoes to infectious huams/mosquitoes
ρ	Fration of symptomatically infected humans that are exposed
δ	Progression rate from A_h to P
ω	Progression rate from I_h to P
τ, ξ	Progression rate from I_h to mild hospital, severe hospital, respectively
φ, ψ	The transformation between mild hospital and severe hospital
γ_a,γ_p,γ_i,γ_m,γ_s	Recovery rate of infectious huams from A_h, P, I_h, H_m, H_s,respectively

Define

$$\begin{aligned}\Omega =&\{(S_h, E_h, A_h, I_h, P, H_m, H_s, R_H, S_m, E_m, I_m) \in R_+^{11} :\\ &0 \le S_h, E_h, A_h, I_h, P, H_m, H_s, R_H \le N_h \le \frac{\pi_h}{\mu_h}, 0 \le S_h, E_h, A_h \le N_m \le \frac{\pi_m}{\mu_m}\}.\end{aligned}$$

Lemma 2.1. *The solutions of model* (2.1) *are bounded and the set* Ω *is a positive invariant set.*

Proof. Adding the first eight equations and the last three equations in the model (2.1), we have

$$\frac{\mathrm{d}N_h}{\mathrm{d}t} = \frac{\mathrm{d}S_h}{\mathrm{d}t} + \frac{\mathrm{d}E_h}{\mathrm{d}t} + \frac{\mathrm{d}A_h}{\mathrm{d}t} + \frac{\mathrm{d}I_h}{\mathrm{d}t} + \frac{\mathrm{d}P}{\mathrm{d}t} + \frac{\mathrm{d}H_m}{\mathrm{d}t} + \frac{\mathrm{d}H_s}{\mathrm{d}t} + \frac{\mathrm{d}R_h}{\mathrm{d}t} = \pi_h - \mu_h N_h,$$

and

$$\frac{\mathrm{d}N_m}{\mathrm{d}t} = \frac{\mathrm{d}S_m}{\mathrm{d}t} + \frac{\mathrm{d}E_m}{\mathrm{d}t} + \frac{\mathrm{d}I_m}{\mathrm{d}t} = \pi_m - \mu_m N_m.$$

That means

$$0 \le N_h(t) \le \frac{\pi_h}{\mu_h} + N_h(0)e^{-\mu_h t}, 0 \le N_m(t) \le \frac{\pi_m}{\mu_m} + N_m(0)e^{-\mu_m t},$$

where $N_h(0)$ and $N_m(0)$ are the initial values of the total population and the initial values of the total mosquito, respectively. Hence, $0 \le \lim\limits_{t\to\infty} \sup N_h(t) \le \frac{\pi_h}{\mu_h}$ and $0 \le \lim\limits_{t\to\infty} \sup N_m(t) \le \frac{\pi_m}{\mu_m}$. So, we get a positive invariant set of model (2.1) defined by Ω. This completes the proof. □

Thus, we consider dynamics of model (2.1) on the set Ω in rest part of this paper.

3. The Disease-free Equilibrium and its Dynamics

3.1. The Basic Number and Stability of the Disease-free Equilibrium

Obviously, the model (2.1) always has a disease-free equilibrium given by: $E_0 = (S_h^0, E_h^0, A_h^0, I_h^0, P^0, H_m^0, H_s^0, R_h^0, S_m^0, E_m^0, I_m^0) = (\frac{\pi_h}{\mu_h}, 0, 0, 0, 0, 0, 0, 0, \frac{\pi_m}{\mu_m}, 0, 0)$. In this part, the basic reproduction number will be obtained by the method of the next generation matrix in reference [37]. Model (2.1) can be written as

$$\frac{\mathrm{d}x}{\mathrm{d}t} = \mathcal{F}(x) - \mathcal{V}(x),$$

where $x = (E_h, A_h, I_h, P, H_m, H_s, E_m, I_m)^T$, $\mathcal{F}(x) = (\lambda_h S_h, 0, 0, 0, 0, 0, \lambda_m S_m, 0)^T$, $\mathcal{V}(x) = (-u_1 E_h, (1-\rho)\sigma_h E_h - u_2 A_h, \rho\sigma_h E_h - u_3 I_h, \delta A_h + \omega I_h - u_4 P, \psi H_s + \tau I_h - u_5 H_m, \xi I_h + \varphi H_m - u_6 H_s, -v_1 E_m, \sigma_m E_m - \mu_m I_m)^T$, here $u_1 = \sigma_h + \mu_h$, $u_2 = \gamma_a + \delta + \mu_h$, $u_3 = \gamma_i + \tau + \xi + \omega + \mu_h$, $u_4 = \gamma_p + \mu_h$, $u_5 = \gamma_m + \varphi + \mu_h$, $u_6 = \gamma_s + \psi + \mu_h$, $v_1 = \sigma_m + \mu_m$.

According to the method of reference [37], the basic reproduction number R_0 is

$$R_0 = R_h * R_m,$$

where

$$R_h = \sqrt{\frac{\mu_h k l \beta_h \sigma_h}{\pi_h}\left[\frac{(1-\rho)(\delta+u_4)}{u_1 u_2 u_4} + \frac{\rho(\omega+u_4)}{u_1 u_3 u_4} + \frac{\rho(\psi\xi + \tau u_6 + \tau\varphi + u_5\xi)}{(u_5 u_6 - \psi\varphi) u_1 u_3}\right]},$$

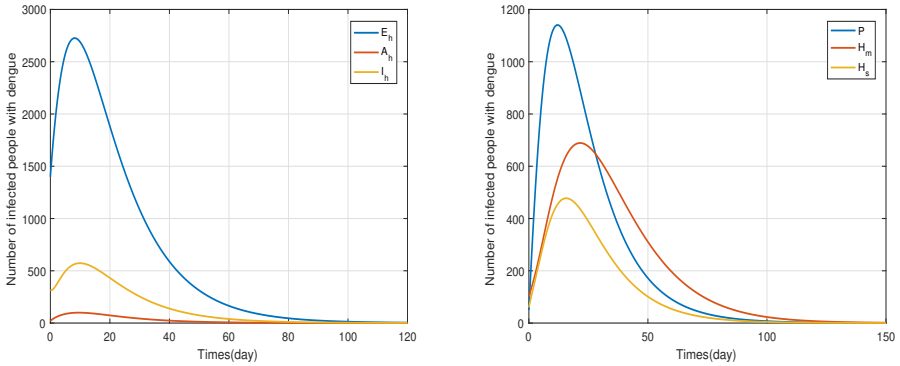
$$R_m = \sqrt{\frac{k l \beta_m \sigma_m S_m^0}{v_1 \mu_m}}.$$

We all know that the ability of one virus to spread in the early stages of an epidemic is measured by the basic reproductive number R_0, which refers to the average number of secondary cases produced by a case during its infection period. Further, using the Theorem 2 of reference [37], we can get the local stability of the disease-free equilibrium.

Theorem 3.1. *The disease-free equilibrium E_0 is locally asymptotically stable when $R_0 < 1$, but unstable when $R_0 > 1$.*

According to Lemma 3.1 and the threshold theory established by Van den Dreessche [37], when $R_0 < 1$, the disease-free equilibrium of model (2.1) is locally asymptotically stable. That is, the disease will be controlled and become extinct as time goes on. When $R_0 > 1$, the disease-free equilibrium of model (2.1) is unstable, that is, the disease will spread and become the endemic. Based on the characteristic equation and Hurwitz criterion, the proof can be given easily. But, the dimension of this model is higher. Thus, we omit the process of proof.

We give some numerical simulations to illustrate and extend our results. The parameter values except $\beta_h = 0.04843$ are given in the Table 2. It is easily obtained that $R_0 = 0.4057 < 1$. Thus the disease free equilibrium E_0 of model (2.1) is locally asymptotically stable according to the Theorem 3.1(see Figure 2).



(a) (b)

Figure 2. Disease free equilibrium E_0 is locally asymptotically stable. (a)E_h, A_h and I_h; (b)P, H_m and H_s.

3.2. Forward Bifurcation

The bifurcation analysis is carried out by using the center manifold theory as shown in reference [3]. Thus, we have the following result.

Theorem 3.2. *The disease-free equilibrium of model* (2.1) *undergoes forward bifurcation at* $R_0 = 1$ *whenever the bifurcation coefficients* a *and* b *are negative and positive, respectively.*

Proof. By the central manifold theory described in reference [3], let $S_h = x_1, E_h = x_2, A_h = x_3, I_h = x_4, P = x_5, H_m = x_6, H_s = x_7, R_h = x_8, S_m = x_9, E_m = x_{10}$ and $I_m = x_{11}$. Then the model (2.1) becomes

$$\begin{cases} \dfrac{\mathrm{d}x_1}{\mathrm{d}t} = \pi_h - \lambda_h x_1 - \mu_h x_1 := f_1, \\ \dfrac{\mathrm{d}x_2}{\mathrm{d}t} = \lambda_h x_1 - \sigma_h x_2 - \mu_h x_2 := f_2, \\ \dfrac{\mathrm{d}x_3}{\mathrm{d}t} = (1-\rho)\sigma_h x_2 - (\gamma_a + \delta + \mu_h)x_3 := f_3, \\ \dfrac{\mathrm{d}x_4}{\mathrm{d}t} = \rho\sigma_h x_2 - (\gamma_i + \tau + \xi + \omega + \mu_h)x_4 := f_4, \\ \dfrac{\mathrm{d}x_5}{\mathrm{d}t} = \delta x_3 + \omega x_4 - (\gamma_p + \mu_h)x_5 := f_5, \\ \dfrac{\mathrm{d}x_6}{\mathrm{d}t} = \tau x_4 + \psi x_7 - (\gamma_m + \varphi + \mu_h)x_6 := f_6, \\ \dfrac{\mathrm{d}x_7}{\mathrm{d}t} = \xi x_4 + \varphi x_6 - (\gamma_s + \psi + \mu_h)x_7 := f_7, \\ \dfrac{\mathrm{d}x_8}{\mathrm{d}t} = \gamma_a x_3 + \gamma_p x_5 + \gamma_i x_4 + \gamma_m x_6 + \gamma_s x_7 - \mu_h x_8 := f_8, \\ \dfrac{\mathrm{d}x_9}{\mathrm{d}t} = \pi_m - (\lambda_m + \mu_m)x_9 := f_9, \end{cases} \tag{3.1}$$

$$\vdots$$

$$\begin{cases} \vdots \\ \dfrac{\mathrm{d}x_{10}}{\mathrm{d}t} = \lambda_m x_9 - (\sigma_m + \mu_m)x_{10} := f_{10}, \\ \dfrac{\mathrm{d}x_{11}}{\mathrm{d}t} = \sigma_m x_{10} - \mu_m x_{11} := f_{11}. \end{cases} \tag{3.1}$$

The Jacobian matrix $J(E_0)$ at the disease-free equilibrium E_0 is

$$J(E_0) = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -kl\beta_h \\ 0 & -u_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & kl\beta_h \\ 0 & (1-\rho)\sigma_h & -u_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \rho\sigma_h & 0 & -u_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta & \omega & -u_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau & 0 & -u_5 & \psi & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \xi & 0 & \varphi & -u_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_a & \gamma_i & \gamma_p & \gamma_m & \gamma_s & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & -A^0 & -A^0 & -A^0 & -A^0 & -A^0 & 0 & -\mu_m & 0 & 0 \\ 0 & 0 & A^0 & A^0 & A^0 & A^0 & A^0 & 0 & 0 & -v_1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \sigma_m & -\mu_m \end{pmatrix}$$

where, $A^0 = kl\beta_m \frac{\pi_m \mu_h}{\mu_m \pi_h}$. Further, β_h is chosen as bifurcation parameter. The value of parameter β_h is defined as β_h^* when $R_0 = 1$. According to the Jacobian matrix $J(E_0)$, it is clear that zero is a simple eigenvalue of $J(E_0)$. Hence, the theory of reference [3] is used to analyze the system (3.1) when $\beta_h = \beta_h^*$. When $R_0 = 1$, the $J(E_0)$ has a right eigenvector (matching up the zero eigenvalue), defined by $\boldsymbol{m} = (m_1, m_2, \cdots, m_{11})^T$, where

$$\begin{aligned} m_1 &= -\frac{T_1}{\mu_h} m_{11}, m_2 = \frac{T_1}{u_1} m_{11}, m_3 = \frac{(1-\rho)\sigma_h T_1}{u_1 u_2} m_{11}, m_4 = \frac{\rho\sigma_h T_1}{u_1 u_3} m_{11}, \\ m_5 &= \frac{1}{u_4}\Big(\frac{(1-\rho)\sigma_h \delta T_1}{u_1 u_2} + \frac{\omega\rho\sigma_h T_1}{u_1 u_3}\Big) m_{11}, m_6 = \frac{(u_6+\psi)\rho\sigma_h T_1}{u_1 u_3 (u_5 u_6 - \varphi\psi)} m_{11}, \\ m_8 &= \frac{1}{\mu_h}\Big(\frac{\gamma_a(1-\rho)\sigma_h T_1}{u_1 u_2} + \frac{\gamma_i \rho\sigma_h T_1}{u_1 u_3} + \gamma_p\Big(\frac{\delta(1-\rho)\sigma_h T_1}{u_1 u_2 u_4} + \frac{\omega\rho\sigma_h T_1}{u_1 u_3 u_4}\Big) \\ &\quad + \frac{\gamma_m(u_6+\psi)\rho\sigma_h T_1}{u_1 u_3 (u_5 u_6 - \varphi\psi)} + \frac{\gamma_s(\varphi+u_5)\rho\sigma_h T_1}{u_1 u_3 (u_5 u_6 - \varphi\psi)}\Big) m_{11}, \\ m_7 &= \frac{(\varphi+u_5)\rho\sigma_h T_1}{u_1 u_3 (u_5 u_6 - \varphi\psi)} m_{11}, m_9 = -\frac{v_1}{\sigma_m} m_{11}, m_{10} = \frac{\mu_m}{\sigma_m} m_{11}, m_{11} > 0, \end{aligned}$$

here $T_1 = kl\beta_h^*$. Similarly, the constituent part of the left eigenvector of $J(E_0)$ (matching up the zero eigenvalue) is denoted by $\boldsymbol{n} = (n_1, n_2, \cdots, n_{11})$, where

$$n_1 = 0, n_2 = \frac{\mu_m}{T_1} n_{11}, n_3 = \frac{B}{u_2},$$

$$
\begin{aligned}
&n_4 = \frac{1}{u_3}\omega B + \frac{1}{u_3}\Big(\frac{\tau(\varphi+u_6)}{u_5u_6-\phi\psi} + \frac{\xi(\psi+u_5)}{u_5u_6-\phi\psi} + 1\Big)\frac{\sigma_m}{v_1}A^0 n_{11},\\
&n_5 = B, n_6 = \frac{(\varphi+u_6)}{u_5u_6-\varphi\psi}\frac{\sigma_m}{v_1}A^0 n_{11}, n_7 = \frac{(\psi+u_5)}{u_5u_6-\varphi\psi}\frac{\sigma_m}{v_1}A^0 n_{11},\\
&n_8 = n_9 = 0, n_{10} = \frac{\sigma_m}{v_1}n_{11}, n_{11} > 0,
\end{aligned}
$$

here $B = \big(\frac{[-u_3(1-\rho)\sigma_h - u_2\rho\sigma_h(\tau(\psi+u_6)+\xi(\psi+u_5)+1)]\sigma_m A^0}{v_1(u_2\rho\sigma_h\omega+u_3\delta(1-\rho)\sigma_h)(u_5u_6-\varphi\psi)} + \frac{u_1u_2u_3\mu_m}{T_1}\big)n_{11}$. Note that the free variables are selected to be $m_{11} = \frac{1}{Q_1+Q_2}$ and $n_{11} = 1$ respectively, where

$$
\begin{aligned}
Q_1 =& 1 + \frac{\mu_m}{u_1} + \frac{B(1-\rho)\sigma_h T_1}{u_1u_2^2} + \frac{u_3(1-\rho)\sigma_h\mu_m}{u_2} + \frac{\rho\sigma_h T_1}{u_1u_3^2} + \frac{B(1-\rho)\sigma_h\delta T_1}{u_1u_2u_4}\\
&+ \frac{B\omega\rho\sigma_h T_1}{u_1u_3u_4} + \frac{(1-\rho)\sigma_h\delta T_1}{u_3u_4\mu_m} + \frac{\omega\rho\sigma_h}{u_2u_4\mu_m} + \frac{\mu_m}{v_1},\\
Q_2 =& \frac{\rho\sigma T_1\sigma_m}{u_1u_3^2v_1}\Big(\frac{\tau(\varphi+u_6)}{u_5u_6-\psi\varphi} + \frac{\xi(\psi+u_5)}{u_5u_6-\psi\varphi} + 1\Big)A^0\\
&+ \left(\frac{(u_6+\psi)(u_6+\varphi)\rho\sigma_h T_1}{u_1u_3(u_5u_6-\psi\varphi)^2} + \frac{(u_5+\psi)(u_5+\varphi)\rho\sigma_h T_1}{u_1u_3(u_5u_6-\psi\varphi)^2}\right)\frac{\sigma_m}{v_1}A^0.
\end{aligned}
$$

Hence, $\boldsymbol{n}\cdot\boldsymbol{m} = 1$.

Next, we calculate the branching coefficients a and b, respectively, which are given by

$$
a = \sum_{k,i,j=1}^{11} n_k m_i m_j \frac{\partial^2 f_k(0,0)}{\partial x_i \partial x_j}, \quad b = \sum_{k,i=1}^{11} n_k m_i \frac{\partial^2 f_k(0,0)}{\partial x_i \partial \beta_h},
$$

where

$$
\begin{aligned}
a =& -2m_{11}^2\frac{kl\beta_m\pi_h}{\mu_h}\Big(\frac{(1-\rho)\sigma_h}{u_1u_2} + \frac{\rho\sigma_h}{u_1u_3} + \frac{1}{u_4}\Big(\frac{(1-\rho)\sigma_h\delta}{u_1u_2} + \frac{\omega\rho\sigma_h}{u_1u_3}\Big)\\
&+ \frac{(u_6+\psi)\rho\sigma_h}{u_1u_3(u_5u_6-\varphi\psi)} + \frac{(\varphi+u_5)\rho\sigma_h}{u_1u_3(u_5u_6-\varphi\psi)}\Big) < 0.\\
b =& \frac{\mu_h k^2 l^2\beta_m\sigma_h\sigma_m\pi_m}{\pi_h v_1\mu_m}\Big(\frac{1-\rho}{u_1u_2} + \frac{\delta(1-\rho)}{u_1u_2u_4} + \frac{\omega\rho}{u_1u_3u_4}\Big)\\
&+ \frac{\mu_h k^2 l^2\beta_m\sigma_h\sigma_m\pi_m}{\pi_h v_1\mu_m}\Big(\frac{\rho}{u_1u_3} + \frac{\rho(\psi\xi+\tau u_6+\tau\varphi+u_5\xi)}{(u_5u_6-\psi\varphi)u_1u_3}\Big) > 0.
\end{aligned}
$$

We find that the coefficient b is always positive and a is often negative. In line with Theorem 4.1 of [3], the model (2.1) appears a forward bifurcation when $R_0 = 1$. This completes the proof. □

The forward bifurcation diagram of model (2.1) is illustrated in Figure 3. According to Theorem 3.2, the model (2.1) exists forward bifurcation at $R_0 = 1$. There is a local asymptotically stable disease-free equilibrium point when $R_0 < 1$, which indicates that the disease has not broken out. There are unstable disease-free equilibrium point and the locally asymptotically stable endemic equilibrium point coexist when $R_0 > 1$, which indicates that the disease tends to be stable.

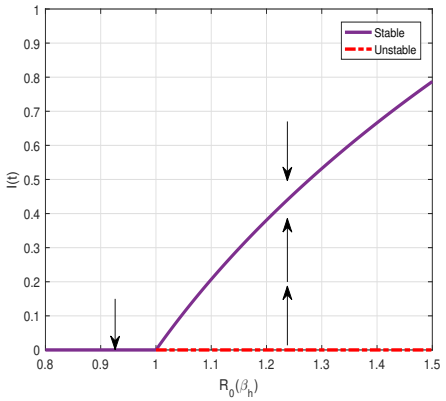


Figure 3. Forward bifurcation diagram taking one parameter β_h in R_0 as bifurcation parameter.

4. The Endemic Equilibrium and its Dynamics

4.1. Existence of the Endemic Equilibrium

If $R_0 > 1$ and $u_5u_6 - \psi\varphi > 0$, then there exists one unique endemic equilibrium of model (2.1), which is $E^* = (S_h^*, E_h^*, A_h^*, I_h^*, P^*, H_m^*, H_s^*, R_h^*, S_m^*, E_m^*, I_m^*)$, where λ_h^* and λ_m^* are given by

$$S_h^* = \frac{\pi_h}{\lambda_h + \mu_h}, E_h^* = \frac{\lambda_h \pi_h}{u_1(\lambda_h + \mu_h)}, A_h^* = \frac{(1-\rho)\sigma_h \lambda_h \pi_h}{u_2 u_1(\lambda_h + \mu_h)}, I_h^* = \frac{\rho\sigma_h \lambda_h \pi_h}{u_3 u_1(\lambda_h + \mu_h)},$$

$$P^* = \frac{\lambda_h \pi_h \sigma_h}{u_4 u_1(\lambda_h + \mu_h)}[\frac{\delta(1-\rho)}{u_2} + \frac{\omega\rho}{u_3}], H_m^* = \frac{I_h^* u_6}{u_5 u_6 - \psi\varphi}(\frac{\psi\xi}{u_6} + \tau),$$

$$H_s^* = \frac{I_h^*}{u_6}[\xi + \frac{\varphi}{u_5 u_6 - \psi\varphi}(\frac{\psi\xi}{u_6} + \tau)], S_m^* = \frac{\pi_m}{\lambda_m + \mu_m},$$

$$E_m^* = \frac{\lambda_m \pi_m}{v_1(\lambda_m + \mu_m)}, I_m^* = \frac{\sigma_m \lambda_m \pi_m}{\mu_m v_1(\lambda_m + \mu_m)},$$

$$R_h^* = \frac{\pi_h \lambda_h}{\mu_h(\lambda_h + \mu_h)}[\frac{\gamma_a(1-\rho)\sigma_h}{u_1 u_2} + \frac{\gamma_p \sigma_h}{u_1 u_4}(\frac{\delta(1-\rho)}{u_2} + \frac{\omega\rho}{u_3}) + \frac{\gamma_i \rho \sigma_h}{u_1 u_3}$$
$$+ \frac{\gamma_m u_6 \rho \sigma_h}{u_1 u_3(u_5 u_6 - \psi\varphi)}(\frac{\psi\xi}{u_6} + \tau) + \frac{\gamma_s \rho\sigma}{u_1 u_3 u_6}(\xi + \frac{\varphi}{u_5 u_6 - \psi\varphi}(\frac{\psi\xi}{u_6} + \tau))].$$

4.2. Uniform Persistence

Here, the uniform persistence of model (2.1) is demonstrated by the theory of uniform persistence proposed by Zhao [48]. There is at least one positive solution of model (2.1) which is uniformly persistent when $R_0 > 1$. Thus, we have the following result.

Theorem 4.1. *If $R_0 > 1$, then there exists a positive constant $\epsilon > 0$ such that the solution of model (2.1) with each initial value $\Gamma(0)$ satisfied*

$$\lim_{t\to\infty} inf(E_h(t), A_h(t), I_h(t), P(t), H_m(t), H_s(t), E_m(t), I_m(t)) \geq (\epsilon, \epsilon, \epsilon, \epsilon, \epsilon, \epsilon, \epsilon, \epsilon).$$

Proof. Define

$$X = \{(S_h, E_h, A_h, I_h, P, H_m, H_s, R_h, S_m, E_m, I_m) \in \Omega\},$$
$$X_0 = \{(S_h, E_h, A_h, I_h, P, H_m, H_s, R_h, S_m, E_m, I_m) \in X : X_0 \in \Re_+^{11}\}.$$

Thus, $\partial X_0 = X - X_0$. It is easily that both X and X_0 are positively invariant for model (2.1), and X_0 is relatively closed in X. In addition, by Theorem 3.1, the model (2.1) is point dissipative. Hence, the model (2.1) exists a globally attractor.

Next, set

$$\begin{aligned} M_\partial =&\{(S_h(0), E_h(0), A_h(0), I_h(0), P(0), H_m(0), H_s(0), R_h(0), S_m(0),\\ &E_m(0), I_m(0)) \in \partial X_0 : (S_h(t), E_h(t), A_h(t), I_h(t), P(t), H_m(t),\\ &H_s(t), R_h(t), S_m(t), E_m(t), I_m(t)) \in \partial X_0, \forall t \geq 0\}. \end{aligned}$$

We will prove that $M_\partial = \{(S_h, 0, 0, 0, 0, 0, 0, 0, S_m, 0, 0) \in \partial X_0 : S_h \geq 0, S_m \geq 0\} \triangleq M'_\partial$. It is clearly that $M'_\partial \subseteq M_\partial$. We only need to show the vality of $M_\partial \subseteq M'_\partial$. Let $\Gamma(t)$ be a solution of model (2.1) with initial condition $\Gamma(0)$. Hence, for any

$$\Gamma(t)=(S_h(t), E_h(t), A_h(t), I_h(t), P(t), H_m(t), H_s(t), R_h(t), S_m(t), E_m(t), I_m(t)) \in M_\partial$$

and $\Gamma(t) \neq M'_\partial$, for $\forall t > 0$, the following inequality are given

$$\begin{aligned} E_h(t) &= e^{-(\sigma_h+\mu_h)}\big[E_h(0) + \int_0^t (kl\beta_h(\nu)I_m(\nu)/S_h(\nu) + E_h(\nu) + A_h(\nu)\\ &\quad + I_h(\nu) + P(\nu) + H_m(\nu) + H_s(\nu))S_h(\nu)d\nu\big] > 0,\\ A_h(t) &= e^{-(\gamma_a+\delta+\mu_h)}\big[A_h(0) + \int_0^t (1-\rho)\sigma_h E_h(\nu)d\nu\big] > 0,\\ I_h(t) &= e^{-(\gamma_i+\tau+\xi+\omega+\mu_h)}\big[I_h(0) + \int_0^t \rho\sigma_h E_h(\nu)d\nu\big] > 0,\\ P(t) &= e^{-(\gamma_p+\mu_h)}\big[P(0) + \int_0^t (\delta A_h(\nu) + \omega I_h(\nu))d\nu\big] > 0,\\ H_m(t) &= e^{-(\gamma_m+\phi+\mu_h)}\big[H_m(0) + \int_0^t (\psi H_s(\nu) + \tau I_h(\nu))d\nu\big] > 0,\\ H_s(t) &= e^{-(\gamma_s+\psi+\mu_h)}\big[H_s(0) + \int_0^t (\varphi H_m(\nu) + \xi I_h(\nu))d\nu\big] > 0,\\ E_m(t) &= e^{-(\sigma_m+\mu_m)}\big[E_m(0) + \int_0^t (kl\beta_m(\nu)(I_h(\nu) + A_h(\nu) + P(\nu) + H_m(\nu)\\ &\quad + H_s(\nu))/S_h(\nu) + E_h(\nu) + A_h(\nu) + I_h(\nu) + P(\nu) + H_m(\nu)\\ &\quad + H_s(\nu))S_m(\nu)d\nu\big] > 0,\\ I_m(t) &= e^{-\mu_m}\big[I_m(0) + \int_0^t \sigma_m E_m(\nu)d\nu\big] > 0. \end{aligned}$$

There exists at least one of $E_h(t), A_h(t), I_h(t), P(t), H_m(t), H_s(t), E_m(t)$, and $I_m(t)$ which is not one. This means that $\Gamma(t) \notin \partial X_0$ for $t > 0$, which contradicts the hypothesis that $\Gamma(t) \in M_\partial$. Therefore, we can get that $M_\partial \subseteq M'_\partial$ which indicates $M_\partial = M'_\partial$. We can obtain M_∂ only has the $E_0(S_h^0, 0, 0, 0, 0, 0, 0, 0, S_m^0, 0, 0)$ and E_0

is isolate and compact invariant. And then we prove that $W^s(E_0) \cap X_0 = \emptyset$, where $W^s(E_0)$ indicates the stable manifold of E_0. Then, there exists a positive constant ϵ such that under the initial condition $\Gamma(0) \in X_0$, the follow inequality is true for any solution $\Gamma_t(\Gamma(0))$ of system

$$D(\Gamma_t(\Gamma(0)), E_0)^\infty \geq \epsilon,$$

where D is a distance function in X_0. Inverse, for $\forall \bar{\epsilon} > 0$, we assume that $D(\Gamma_t(\Gamma(0)), E_0)^\infty < \bar{\epsilon}$. For $\forall \bar{\epsilon} > 0$, there exists $T > 0$ such that $\frac{\pi_h}{\mu_h} - \bar{\epsilon} \leq S_h(t) \leq \frac{\pi_h}{\mu_h} + \bar{\epsilon}$, $0 \leq E_h(t) \leq \bar{\epsilon}$, $0 \leq A_h(t) \leq \bar{\epsilon}$, $0 \leq I_h(t) \leq \bar{\epsilon}$, $0 \leq P(t) \leq \bar{\epsilon}$, $0 \leq H_m(t) \leq \bar{\epsilon}$, $0 \leq H_s(t) \leq \bar{\epsilon}$, $0 \leq R_h(t) \leq \bar{\epsilon}$, $\frac{\pi_m}{\mu_m} - \bar{\epsilon} \leq S_m(t) \leq \frac{\pi_m}{\mu_m} + \bar{\epsilon}$, $0 \leq E_m(t) \leq \bar{\epsilon}$, $0 \leq I_m(t) \leq \bar{\epsilon}$.

Further, we consider the following comparison system

$$\begin{cases} \dfrac{\mathrm{d}\tilde{E}_h}{\mathrm{d}t} = \tilde{\lambda}_h(\dfrac{\pi_h}{\mu_h} - \bar{\epsilon}) - \sigma_h\tilde{E}_h - \mu_h\tilde{E}_h, \\ \dfrac{\mathrm{d}\tilde{A}_h}{\mathrm{d}t} = (1-\rho)\sigma_h\tilde{E}_h - (\gamma_a + \delta + \mu_h)\tilde{A}_h, \\ \dfrac{\mathrm{d}\tilde{I}_h}{\mathrm{d}t} = \rho\sigma_h\tilde{E}_h - (\gamma_i + \tau + \xi + \omega + \mu_h)\tilde{I}_h, \\ \dfrac{\mathrm{d}\tilde{P}}{\mathrm{d}t} = \delta\tilde{A}_h + \omega\tilde{I}_h - (\gamma_p + \mu_h)\tilde{P}, \\ \dfrac{\mathrm{d}\tilde{H}_m}{\mathrm{d}t} = \tau\tilde{I}_h + \psi\tilde{H}_s - (\gamma_m + \varphi + \mu_h)\tilde{H}_m, \\ \dfrac{\mathrm{d}\tilde{H}_s}{\mathrm{d}t} = \xi\tilde{I}_h + \varphi\tilde{H}_m - (\gamma_s + \psi + \mu_h)\tilde{H}_s, \\ \dfrac{\mathrm{d}\tilde{E}_m}{\mathrm{d}t} = \tilde{\lambda}_m(\dfrac{\pi_m}{\mu_m} - \bar{\epsilon}) - (\sigma_m + \mu_m)\tilde{E}_m, \\ \dfrac{\mathrm{d}\tilde{I}_m}{\mathrm{d}t} = \sigma_m\tilde{E}_m - \mu_m\tilde{I}_m, \end{cases} \tag{4.1}$$

where $\tilde{\lambda}_h = kl\beta_h\frac{\tilde{I}_m}{\tilde{N}_h}$, and $\tilde{\lambda}_m = kl\beta_m\frac{\tilde{A}_h+\tilde{I}_h+\tilde{P}+\tilde{H}_m+\tilde{H}_s}{\tilde{N}_h}$. System (4.1) can be represented as

$$x' = \bar{F}(\bar{\epsilon})x,$$

where

$$\bar{F}(\bar{\epsilon}) = \begin{pmatrix} -u_1 & 0 & 0 & 0 & 0 & 0 & 0 & -kl\beta_h S_h^0\bar{\epsilon} \\ (1-\rho)\sigma_h & -u_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ \rho\sigma_h & 0 & -u_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & \delta & \omega & -u_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau & 0 & -u_5 & \psi & 0 & 0 \\ 0 & 0 & \xi & 0 & \varphi & -u_6 & 0 & 0 \\ 0 & \Theta\bar{\epsilon} & \Theta\bar{\epsilon} & \Theta\bar{\epsilon} & \Theta\bar{\epsilon} & \Theta\bar{\epsilon} & -v_1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma_m & \mu_m \end{pmatrix},$$

here $\Theta = -kl\beta_m S_h^0$. Let $U(\bar{F}(\bar{\epsilon}))$ be the stability modulus of matrix $\bar{F}(\bar{\epsilon})$, which is defined by

$$U(\bar{F}(\bar{\epsilon})) = \max\{Re\ \aleph : \aleph \text{ is an eigenvalue of } U(\bar{F}(\bar{\epsilon}))\}.$$

Obviously, $\bar{F}(\bar{\epsilon})$ has non-negative off-diagonal elements and it is irreducible. Hence, according to Theorem A. 5 in Zhao [47], $U(\bar{F}(\bar{\epsilon}))$ is a simple eigenvalue of $\bar{F}(\bar{\epsilon})$ and has a positive eigenvector. By using the Theorem 2 in reference [37] and the Lemma 2.1 in reference [40], we know that $U(\bar{F}(\bar{\epsilon})) < 0$ if and only if $R_0 < 1$, inverse $U(\bar{F}(\bar{\epsilon})) > 0$ if and only if $R_0 > 1$.

Since $U(\bar{F}(\bar{\epsilon}))$ is continuous about $\bar{\epsilon}$. Let $\bar{\epsilon}$ be small enough such that $U(\bar{F}(\bar{\epsilon})) > 0$. So, assume that $x(t) = (E_h(t), A_h(t), I_h(t), P(t), H_m(t), H_s(t), E_m(t), I_m(t))^T$ is a positive solution of the comparison system (4.1), which is strictly increasing with $x_i(t) \to +\infty$ as $t \to +\infty$, $i = E_h(t), A_h(t), \cdots, I_m(t)$. By comparison principle, we have

$$\lim_{t\to\infty} E_h(t) = +\infty, \lim_{t\to\infty} A_h(t) = +\infty, \lim_{t\to\infty} I_h(t) = +\infty, \lim_{t\to\infty} P(t) = +\infty,$$
$$\lim_{t\to\infty} H_n(t) = +\infty, \lim_{t\to\infty} H_s(t) = +\infty, \lim_{t\to\infty} E_m(t) = +\infty, \lim_{t\to\infty} I_m(t) = +\infty.$$

This contracts with our assumption. Then, there exists $W^s(E_0) \cap X_0 = \emptyset$. Hence, the model (2.1) is uniformly persistent when $R_0 > 1$. This completes the proof. □

4.3. Sensitivity Analysis

We observe that the five parameters, namely, β_h, β_m, μ_m, γ_i and l are the pivotal parameters which regulate the basic reproduction number R_0. The sensitivity of the basic reproduction number R_0 to these parameters is given as follows:

$$\begin{aligned}
\frac{\mathrm{d}R_0}{\mathrm{d}\beta_m} =& \frac{R_h}{2R_m}\frac{kl\pi_m}{v_1\mu_m^2} > 0,\\
\frac{\mathrm{d}R_0}{\mathrm{d}\mu_m} =& -\frac{R_h}{2R_m}\Big[\frac{kl\beta_m\pi_m}{(\sigma_m+\mu_m)^2\mu_m^2} + \frac{kl\beta_m\pi_m}{(\sigma_m+\mu_m)\mu_m^3}\Big] < 0,\\
\frac{\mathrm{d}R_0}{\mathrm{d}\beta_h} =& \frac{R_m}{2R_h}\Big[\frac{\mu_h kl\sigma_h}{\pi_h}\Big(\frac{1-\rho}{u_1u_2} + \frac{\delta(1-\rho)}{u_1u_2u_4} + \frac{\omega\rho}{u_1u_3u_4} + \frac{\rho}{u_1u_3}\Big)\\
&+ \frac{\mu_h kl\sigma_h}{\pi_h(u_5u_6-\psi\varphi)}\frac{\rho(\psi\xi+\tau u_6+\tau\varphi+u_5\xi)}{u_1u_3}\Big] > 0,\\
\frac{\mathrm{d}R_0}{\mathrm{d}l} =& \frac{R_m}{2R_h}\Big[\frac{\mu_h k\beta_h\sigma_h}{\pi_h}\Big(\frac{1-\rho}{u_1u_2} + \frac{\delta(1-\rho)}{u_1u_2u_4} + \frac{\omega\rho}{u_1u_3u_4} + \frac{\rho}{u_1u_3}\Big)\\
&+ \frac{\mu_h k\beta_h\sigma_h}{\pi_h(u_5u_6-\psi\varphi)}\frac{\rho(\psi\xi+\tau u_6+\tau\varphi+u_5\xi)}{u_1u_3}\Big] > 0,\\
\frac{\mathrm{d}R_0}{\mathrm{d}\gamma_i} =& -\frac{R_m}{2R_h}\Big[\frac{\mu_h kl\beta_h\sigma_h}{\pi_h}\Big(\frac{\omega\rho}{u_1u_3^2u_4} + \frac{\rho}{u_1u_3^2}\Big)\\
&+ \frac{\mu_h kl\beta_h\sigma_h}{\pi_h(u_5u_6-\psi\varphi)}\frac{\rho(\psi\xi+\tau u_6+\tau\varphi+u_5\xi)}{u_1u_3^2}\Big] < 0.
\end{aligned}$$

When the partial derivative is positive, the basic reproduction number R_0 increases with the increase of parameters. When the partial derivative is negative, the basic reproduction number R_0 decreases with the increase of the parameter.

In order to understand the effect of the proportional changes of these parameters on R_0, we calculate the elasticity [35]. Elasticity is nothing more than a proportional response to a proportional disturbance.

$$
\begin{aligned}
E_{\beta_m} &= \frac{\beta_m}{R_0}\frac{\partial R_0}{\partial \beta_m} = \frac{1}{2R_0}\frac{R_h}{R_m}\frac{kl\beta_m\pi_m}{v_1\mu_m^2},\\
E_{\mu_m} &= \frac{\mu_m}{R_0}\frac{\partial R_0}{\partial \mu_m} = -\frac{1}{2R_0}\frac{R_h}{R_m}\Big(\frac{kl\beta_m\pi_m}{(\sigma_m+\mu_m)^2\mu_m} + \frac{kl\beta_m\pi_m}{(\sigma_m+\mu_m)\mu_m^2}\Big),\\
E_{\beta_h} &= \frac{\beta_h}{R_0}\frac{\partial R_0}{\partial \beta_h} = \frac{1}{2R_0}\frac{R_m}{R_h}\Big[\frac{\mu_h kl\sigma_h\beta_h}{\pi_h}\Big(\frac{1-\rho}{u_1u_2} + \frac{\delta(1-\rho)}{u_1u_2u_4} + \frac{\omega\rho}{u_1u_3u_4} + \frac{\rho}{u_1u_3}\Big)\\
&\quad + \frac{\mu_h kl\sigma_h\beta_h}{\pi_h(u_5u_6-\psi\varphi)}\frac{\rho(\psi\xi+\tau u_6+\tau\varphi+u_5\xi)}{u_1u_3}\Big],\\
E_l &= \frac{l}{R_0}\frac{\partial R_0}{\partial l} = \frac{1}{2R_0}\frac{R_m}{R_h}\Big[\frac{\mu_h kl\beta_h\sigma_h}{\pi_h}\Big(\frac{1-\rho}{u_1u_2} + \frac{\delta(1-\rho)}{u_1u_2u_4} + \frac{\omega\rho}{u_1u_3u_4} + \frac{\rho}{u_1u_3}\Big)\\
&\quad + \frac{\mu_h kl\beta_h\sigma_h}{\pi_h(u_5u_6-\psi\varphi)}\frac{\rho(\psi\xi+\tau u_6+\tau\varphi+u_5\xi)}{u_1u_3}\Big],\\
E_{\gamma_i} &= \frac{\gamma_i}{R_0}\frac{\partial R_0}{\partial \gamma_i} = -\frac{1}{2R_0}\frac{R_m}{R_h}\Big[\frac{\mu_h kl\beta_h\gamma_i\sigma_h}{\pi_h}\Big(\frac{\omega\rho}{u_1u_3^2u_4} + \frac{\rho}{u_1u_3^2}\Big)\\
&\quad + \frac{\mu_h kl\beta_h\sigma_h\gamma_i}{\pi_h(u_5u_6-\psi\varphi)}\frac{\rho(\psi\xi+\tau u_6+\tau\varphi+u_5\xi)}{u_1u_3^2}\Big].
\end{aligned}
$$

From these expressions, it can be seen that β_h, β_m and l have a promoting effect on R_0, while the rest have a inhibiting effect on R_0. That is, R_0 is positively correlated with β_h, β_m and l, but negatively correlated with μ_m and γ_i. In Figures 4 and 5, the influence of parameters on R_0 is more intuitively shown.

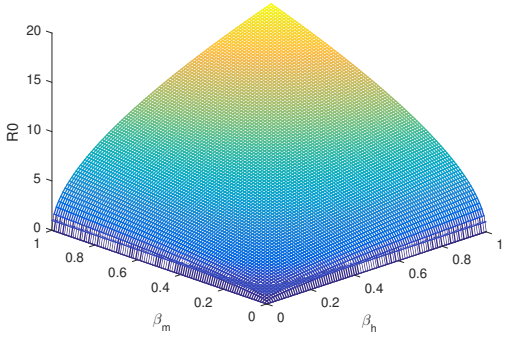


Figure 4. The influence of β_h and β_m on R_0

From Figure 4, it shows that the effects of parameters β_h and β_m on R_0 are positive. This suggests that controlling transmission between mosquitoes and human can help curb the spread of the disease. From Figure 5(a), β_h has more influence on R_0 than γ_i. Similarly, Figure 5(b) shows that increasing the mortality rate of

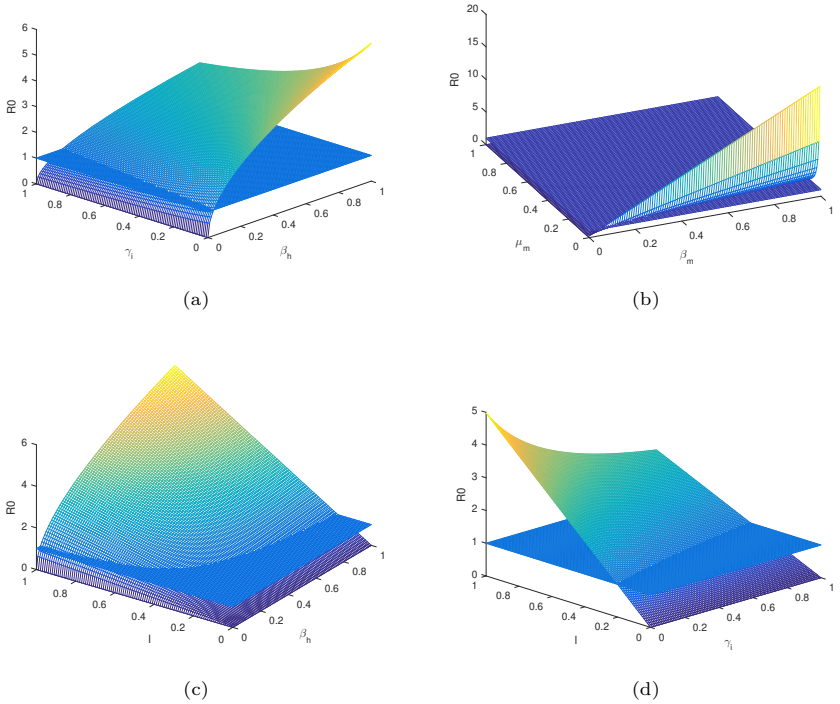


Figure 5. The influence of each parameter on R_0. (a) β_h and γ_i; (b) β_m and μ_m; (c) β_h and l; (d) γ_i and l.

mosquitoes can greatly control the spread of dengue fever. Figure 5(c) shows that the effects of l and β_h on R_0 are basically equal. The effects of l and γ_i on R_0 are shown in Figure 5(d). From Figures 4 and 5, we can conclude that control policies should aim to reduce transmission rates and increase the mortality of mosquitoes and the recovery rate from treatment.

5. The Optimal Control

5.1. The Existence of Optimal Control

In the previous part, we mainly focus on the dynamical behavior of the model (2.1). In this section, we will use three control variables. The control of using bed nets to avoid exposure to mosquitoes is denoted as $g_1(t)$. Thus, the force of infection is reduced by a factor of $1 - g_1(t)$. The control variable $g_2(t)$ represents promoting awareness of humans to protect themselves from dengue infection, such as increasing the coverage of recovers, and $g_3(t)$ represents reducing mosquito population. Here, we assume that the control set is

$$U = \{(g_1, g_2, g_3) \mid g_i(t) \in L^\infty[0, t_f], 0 \le g_i(t) \le c_i, 0 < c_i \le 1, i = 1, 2, 3\}.$$

The optimal control model is given as

$$\begin{cases} \dfrac{\mathrm{d}S_h}{\mathrm{d}t} = \pi_h - (1-g_1(t))\lambda_h S_h - \mu_h S_h - g_2(t)S_h, \\ \dfrac{\mathrm{d}E_h}{\mathrm{d}t} = (1-g_1(t))\lambda_h S_h - \sigma_h E_h - \mu_h S_h, \\ \dfrac{\mathrm{d}A_h}{\mathrm{d}t} = (1-\rho)\sigma_h E_h - (\gamma_a + \delta + \mu_h)A_h, \\ \dfrac{\mathrm{d}I_h}{\mathrm{d}t} = \rho\sigma_h E_h - (\gamma_i + \tau + \xi + \omega + \mu_h)I_h, \\ \dfrac{\mathrm{d}P}{\mathrm{d}t} = \delta A_h + \omega I_h - (\gamma_p + \mu_h)P, \\ \dfrac{\mathrm{d}H_m}{\mathrm{d}t} = \tau I_h + \psi H_s - (\gamma_m + \varphi + \mu_h)H_m, \\ \dfrac{\mathrm{d}H_s}{\mathrm{d}t} = \xi I_h + \varphi H_m - (\gamma_s + \psi + \mu_h)H_s, \\ \dfrac{\mathrm{d}R_h}{\mathrm{d}t} = \gamma_a A_h + \gamma_p P + \gamma_i I_h + \gamma_m H_m + \gamma_s H_s + g_2(t)S_h - \mu_h R_h, \\ \dfrac{\mathrm{d}S_m}{\mathrm{d}t} = \pi_m - (\lambda_m + \mu_m)S_m - g_3(t)S_m, \\ \dfrac{\mathrm{d}E_m}{\mathrm{d}t} = \lambda_m S_m - (\sigma_m + \mu_m)E_m - g_3(t)E_m, \\ \dfrac{\mathrm{d}I_m}{\mathrm{d}t} = \sigma_m E_m - \mu_m I_m - g_3(t)I_m. \end{cases} \tag{5.1}$$

For nonnegative initial conditions and bounded Lebesgue measurable controls, system (5.1) has nonnegative bounded solutions. We consider an optimal control problem to minimize the objective function

$$\begin{aligned} J(g_1, g_2, g_3) = \int_0^{t_f} & [E_h(t) + A_h(t) + I_h + P + H_m + H_s + E_m + I_m \\ & + \frac{1}{2}c_1 g_1^2(t) + \frac{1}{2}c_2 g_2^2(t) + \frac{1}{2}c_3 g_3^2(t)]dt. \end{aligned} \tag{5.2}$$

Next, the existence of the optimal control pair in the system (5.1) is obtained by using the results of Fleming and Rishel [9].

Theorem 5.1. *Under the initial conditions, the system* (5.1) *exists an optimal control pair* $\{(g_1^*, g_2^*, g_3^*) \in U, t \in [0, t_f]\}$ *such that*

$$J(g_1^*, g_2^*, g_3^*) = \min_{g_i(t)\in U, i=1,2,3} J(g_1, g_2, g_3).$$

Proof. To prove the existence of optimal control, the following conditions must be satisfied:
(1) The control and related state variables are non-negative values.
(2) The control set U is convex and closed.
(3) The right-hand of the state system (5.1) is bounded and it is a linear function of the control and the state variable.
(4) The integrand of the objective function on U is convex.
(5) There exist constants $b_1, b_2 > 0$ and $\alpha > 1$ such that the integrant of the

objective functional

$$\begin{aligned} L(t, g_1, g_2, g_3) =& E_h(t) + A_h(t) + I_h + P + H_m + H_s + E_m + I_m \\ &+ \frac{1}{2}(c_1 g_1^2(t) + c_2 g_2^2(t) + c_3 g_3^2(t)) \end{aligned}$$

satisfies

$$L(t, g_1, g_2, g_3) \geq b_1[u_1^2(t) + u_2^2(t) + u_3^2(t)]^{\frac{\alpha}{2}} - b_2.$$

It is obvious the conditions (1), (2) and (4) are satisfied. As for condition (3), we have already proved that eleven state variable are bounded, therefore

$$\begin{aligned} &\frac{\mathrm{d}S_h}{\mathrm{d}t} \leq \pi_h, \quad \frac{\mathrm{d}E_h}{\mathrm{d}t} \leq (1 - g_1(t))\lambda_h S_h, \quad \frac{\mathrm{d}A_h}{\mathrm{d}t} \leq (1-\rho)\sigma_h E_h, \\ &\frac{\mathrm{d}I_h}{\mathrm{d}t} \leq \rho\sigma_h E_h, \quad \frac{\mathrm{d}P}{\mathrm{d}t} \leq \delta A_h + \omega I_h, \quad \frac{\mathrm{d}H_m}{\mathrm{d}t} \leq \tau I_h + \psi H_s, \\ &\frac{\mathrm{d}S_m}{\mathrm{d}t} \leq \pi_m, \quad \frac{\mathrm{d}E_m}{\mathrm{d}t} \leq \lambda_m S_m, \quad \frac{\mathrm{d}I_m}{\mathrm{d}t} \leq \sigma_m E_m, \\ &\frac{\mathrm{d}H_s}{\mathrm{d}t} \leq \xi I_h + \varphi H_m, \quad \frac{\mathrm{d}R_h}{\mathrm{d}t} \leq \gamma_a A_h + \gamma_p P + \gamma_i I_h + \gamma_m H_m + \gamma_s H_s + g_2(t) S_h. \end{aligned}$$

For the condition (5), there exist $b_1 = min\{\frac{c_1}{2}, \frac{c_2}{2}\}, b_2 \in R^+$ and $\alpha = 2$ such that

$$L(t, g_1, g_2, g_3) \geq b_1[g_1^2(t) + g_2^2(t) + g_3^2(t)]^{\frac{\alpha}{2}} - b_2,$$

which completes the existence of an optimal control. □

5.2. Characterization of Optimal Control

According to the Pontryagin's Maximum Principle [4], the necessary conditions for the existence of optimal control of the system (5.1) are obtained. Now, we will formulate the Hamiltonian from the governing dynamics and the objective functional to get the optimality conditions. So, we have

$$\begin{aligned} &H(S_h, E_h, A_h, I_h, P, H_m, H_s, R_h, S_m, E_m, I_m, g_1, g_2, g_3, \lambda_i) \\ =& L(E_h, A_h, I_h, P, H_m, H_s, E_m, I_m, g_1, g_2, g_3) \\ &+ \lambda_1[\pi_h - (1 - g_1(t))\lambda_h S_h - \mu_h S_h - g_2(t) S_h] \\ &+ \lambda_2[(1 - g_1(t))\lambda_h S_h - \sigma_h E_h - \mu_h E_h] + \lambda_3[(1-\rho)\sigma_h E_h - (\gamma_a + \delta + \mu_h) A_h] \\ &+ \lambda_4[\rho\sigma_h E_h - (\gamma_i + \tau + \xi + \omega + \mu_h) I_h] + \lambda_5[\delta A_h + \omega I_h - (\gamma_p + \mu_h) P] \\ &+ \lambda_6[\tau I_h + \psi H_s - (\gamma_m + \varphi + \mu_h) H_m] + \lambda_7[\xi I_h + \varphi H_m - (\gamma_s + \psi + \mu_h) H_s] \\ &+ \lambda_8[\gamma_a A_h + \gamma_p P + \gamma_i I_h + \gamma_m H_m + \gamma_s H_s + g_2(t) S_h - \mu_h R_h] \\ &+ \lambda_9[\pi_m - (\lambda_m + \mu_m) S_m - g_3(t) S_m] + \lambda_{10}[\lambda_m S_m - (\sigma_m + \mu_m) E_m - g_3(t) E_m] \\ &+ \lambda_{11}[\sigma_m E_m - \mu_m I_m - g_3(t) I_m], \end{aligned}$$

where $\lambda_i, i = 1, 2, \cdots, 11$ are adjoint variables.

Theorem 5.2. *There is an optimal control pairs* $(g_1^*,\ g_2^*,\ g_3^*)$. *Let* $S_h, E_h, A_h, I_h, P, H_m, H_s, R_h, S_m, E_m,$ *and* I_m *be the state solutions of system* (5.1). *Then there exists adjoint variables* $\lambda_i, i = 1, 2, \cdots, 11$ *satisfying*

$$-\frac{d\lambda_i}{dt} = \frac{\partial H}{\partial i} \tag{5.3}$$

and with the terminal conditions

$$\lambda_i(t_f) = 0,\ i = 1, 2, \cdots, 11. \tag{5.4}$$

The optimality conditions is given by

$$\frac{\partial H}{\partial g_j} = 0,\ j = 1,\ 2,\ 3. \tag{5.5}$$

Further, the control $(g_1^*,\ g_2^*,\ g_3^*)$ *can be obtained from the following equations:*

$$g_1^* = \min\{1,\ \max\{0,\ g_1^*(t)\}\}, \quad g_2^* = \min\{1,\ \max\{0,\ g_2^*(t)\}\},$$
$$g_3^* = \min\{1,\ \max\{0,\ g_3^*(t)\}\}.$$

Proof. By differentiating the Hamiltonian, we obtain the adjoint system can be written as:

$$-\frac{\mathrm{d}\lambda_1}{\mathrm{d}t} = \frac{\partial H}{\partial S_h},\ \lambda_1(t_f) = 0, \qquad -\frac{\mathrm{d}\lambda_2}{\mathrm{d}t} = \frac{\partial H}{\partial E_h},\ \lambda_2(t_f) = 0,$$
$$-\frac{\mathrm{d}\lambda_3}{\mathrm{d}t} = \frac{\partial H}{\partial A_h},\ \lambda_3(t_f) = 0, \qquad -\frac{\mathrm{d}\lambda_4}{\mathrm{d}t} = \frac{\partial H}{\partial I_h},\ \lambda_4(t_f) = 0,$$
$$-\frac{\mathrm{d}\lambda_5}{\mathrm{d}t} = \frac{\partial H}{\partial P},\ \lambda_5(t_f) = 0, \qquad -\frac{\mathrm{d}\lambda_6}{\mathrm{d}t} = \frac{\partial H}{\partial H_m},\ \lambda_6(t_f) = 0,$$
$$-\frac{\mathrm{d}\lambda_7}{\mathrm{d}t} = \frac{\partial H}{\partial H_s},\ \lambda_7(t_f) = 0, \qquad -\frac{\mathrm{d}\lambda_8}{\mathrm{d}t} = \frac{\partial H}{\partial R_h},\ \lambda_8(t_f) = 0,$$
$$-\frac{\mathrm{d}\lambda_9}{\mathrm{d}t} = \frac{\partial H}{\partial S_m},\ \lambda_9(t_f) = 0, \qquad -\frac{\mathrm{d}\lambda_{10}}{\mathrm{d}t} = \frac{\partial H}{\partial E_m},\ \lambda_{10}(t_f) = 0,$$
$$-\frac{\mathrm{d}\lambda_{11}}{\mathrm{d}t} = \frac{\partial H}{\partial I_m},\ \lambda_{11}(t_f) = 0.$$

The state adjoint system is given by

$$\frac{\mathrm{d}\lambda_1}{\mathrm{d}t} = [(1 - g_1(t))\lambda_h + \mu_h + g_2(t)]\lambda_1(t) - (1 - g_1(t))\lambda_h\lambda_2(t) - g_2(t)\lambda_8,$$
$$\frac{\mathrm{d}\lambda_2}{\mathrm{d}t} = -1 + (\sigma_h + \mu_h)\lambda_2(t) - (1 - \rho)\sigma_h\lambda_3(t) - \rho\sigma_h\lambda_4(t),$$
$$\frac{\mathrm{d}\lambda_3}{\mathrm{d}t} = -1 + (\gamma_a + \delta + \mu_h)\lambda_3(t) - \delta\lambda_5(t) - \gamma_a\lambda_8(t),$$
$$\frac{\mathrm{d}\lambda_4}{\mathrm{d}t} = -1 + (\gamma_i + \tau + \xi + \omega + \mu_h)\lambda_4(t) - \omega\lambda_5(t) - \gamma_a\lambda_8(t),$$
$$\frac{\mathrm{d}\lambda_5}{\mathrm{d}t} = -1 + (\gamma_p + \mu_h)\lambda_5(t) - \gamma_p\lambda_8(t),$$
$$\frac{\mathrm{d}\lambda_6}{\mathrm{d}t} = -1 + (\gamma_m + \varphi + \mu_h)\lambda_6(t) - \varphi\lambda_7(t) - \gamma_m\lambda_8(t),$$
$$\frac{\mathrm{d}\lambda_7}{\mathrm{d}t} = -1 + (\gamma_s + \psi + \mu_h)\lambda_7(t) - \gamma_s\lambda_8(t),$$
$$\frac{\mathrm{d}\lambda_8}{\mathrm{d}t} = \mu_h\lambda_8(t),$$
$$\frac{\mathrm{d}\lambda_9}{\mathrm{d}t} = (\lambda_m + \mu_h + g_3(t))\lambda_9(t) - \lambda_m\lambda_{10}(t),$$

$$\frac{\mathrm{d}\lambda_{10}}{\mathrm{d}t} = -1 + (\sigma_m + \mu_m + g_3(t))\lambda_{10}(t) - \sigma_m\lambda_{11}(t),$$
$$\frac{\mathrm{d}\lambda_{11}}{\mathrm{d}t} = -1 + (\mu_m + g_3(t))\lambda_{11}(t).$$

Further, by differentiating the Hamiltonian with respect to the controls, we have the following optimality conditions:

$$\frac{\partial H}{\partial g_1} = \xi_1 g_1^*(t) + (\lambda_1(t) - \lambda_2(t))\lambda_h S_h = 0,$$
$$\frac{\partial H}{\partial g_2} = \xi_2 g_2^*(t) - (\lambda_1(t) - \lambda_8(t))S_h = 0,$$
$$\frac{\partial H}{\partial g_3} = \xi_3 g_3^*(t) - \lambda_9(t)S_m - \lambda_{10}(t)E_m - \lambda_{11}(t)I_m = 0.$$

Thus, we have that

$$g_1^* = \frac{[-(\lambda_1(t) - \lambda_2(t))\lambda_h S_h]}{\xi_1}, g_2^* = \frac{[(\lambda_1(t) - \lambda_8(t))S_h]}{\xi_2},$$
$$g_3^* = \frac{[\lambda_9(t)S_m + \lambda_{10}(t)E_m + \lambda_{11}(t)I_m]}{\xi_3}.$$

This completes the proof. □

6. One Case

6.1. Numerical Results

Numerical simulations are carried out by using MATLAB in this section. According to the theory in reference [13], we estimate the parameters of our model on the basis of the dengue data in Singapore in 2020 by carrying out the Markov Chain Monte Carlo (MCMC) procedure. The actual infection is shown in Figure 6.

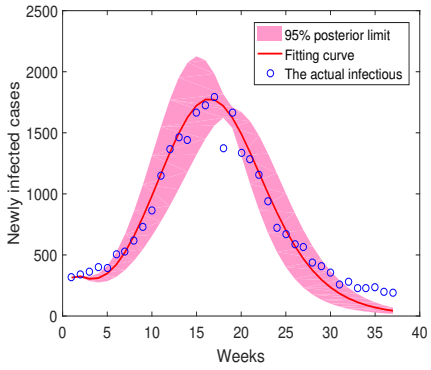


Figure 6. The actual number of people infected in Singapore from 15 to 52 weeks in 2020 years.

In order to calculate the basic reproduction number R_0 of dengue in Singapore and predict changes in the next few years, it is essential to estimate the unknown parameters of the model (2.1). The population size of the Singapore at the end of

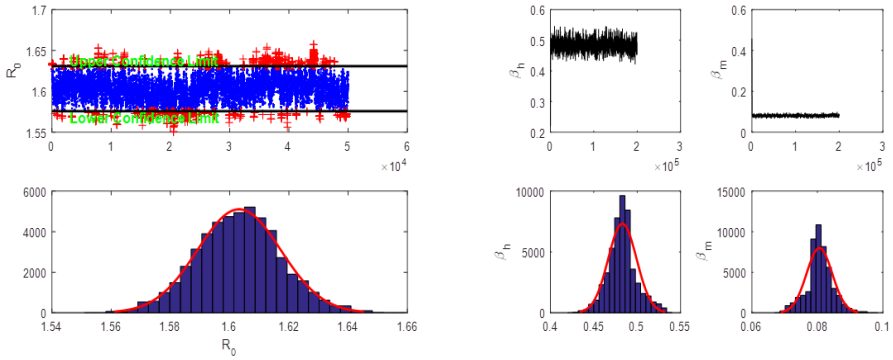
2020 is 568.0058 million. According to the relevant data of the Ministry of Health Singapore (2020), we can get the birth rate in at the end of 2020 is 8.8682 per thousand. Thus, we obtain that the yearly birth population of Singapore is about 49362, and the number of births per day is 133. The average life expectancy of the population of Singapore is 75. So, we conclude that the yearly natural mortality rate of the population in Singapore is approximately $\mu = 1/75$ [39]. The incubation period of dengue is $3-14$ days. We assume that the average progress time of latent individuals $1/\gamma_a$ is 7 days and $1/\gamma_i$ is 6 days, then the daily progress rate γ_a is $1/7$ and γ_i is $1/6$. We choose a set of values of other parameters in Table 2.

Table 2. The parameters description of the Dengue model.

Parameters	Mean value	Std	95% CI	Reference
π_h	133	-	-	[39]
π_m	5000	-	-	[27]
μ_h	1/75	-	-	[39]
μ_m	1/14.49	-	-	[10]
k	0.66	-	-	[51]
l	0.4997	-	-	[46]
σ_h	1/6	-	-	[21, 27]
σ_m	1/7	-	-	[33]
ρ	0.82	-	-	[27]
φ	0.0341	-	-	assumed
γ_a	1/7	-	-	[21]
γ_i	1/7	-	-	[1, 6]
γ_s	0.18	-	-	[27]
β_h	0.4843	0.0177	[0.4497, 0.5190]	MCMC
β_m	0.0803	0.0042	[0.0722, 0.0884]	MCMC
δ	0.66	2.8944×10^{-05}	[0.6599, 0.6601]	MCMC
ω	0.18	0.08244	[0, 0.2466]	MCMC
τ	0.1259	3.6396×10^{-06}	[0.1242, 0.1257]	MCMC
ξ	0.1799	2.5406×10^{-04}	[0.1797, 0.1807]	MCMC
ψ	0.8692	1.3915×10^{-05}	[0.6691, 0.8692]	MCMC
γ_p	0.3096	1.2497×10^{-04}	[0.3093, 0.3098]	MCMC
γ_m	0.06	7.9459×10^{-06}	[0, 0.198]	MCMC

According to the Table 2, infection rate of infected mosquito individuals to susceptible persons is $\beta_h = 0.4843$. Infection rate of infected people individuals to susceptible mosquito is $\beta_m = 0.0803$. Thus, β_h is more bigger than β_m. This suggests that people with dengue in infectious period may be more infectious than those with dengue in the mosquito. We give the number of samples and the frequency distribution of R_0 by using MCMC procedure (seen Figure 7).

From Figure 7(a), we can clearly know that the basic reproductive number R_0 satisfies the normal distribution. The value of the basic reproduction number R_0 is estimated as 1.6015 (95%CI: (1.5425-1.6675)), which is shown in Figure 7(a). This



(a) (b)

Figure 7. Sample values of R_0 and infectivity parameters and sample distributions. (a)The Markov chain of the last 300000 samples of R_0. The blue dots indicate the value of R_0 within the 95% credible intervals, the red pluses indicate the value of R_0 outside the 95% credible intervals, and the black lines indicate the upper and lower credible limits. The frequency distribution of R_0. The red curve is the probability density function curve of R_0. (b)β_h and β_m's Markov Chain Monte Carlo (MCMC) parameters distribution.

means that dengue should be taken seriously in Singapore. From Figure 7(b), it is the Markov chain sampling of β_h and β_m. It can be clearly seen that the two samples obey normal distribution and the estimation effect is suitable.

6.2. Uncertainty Analysis

We study uncertainty and sensitivity analyses by using a Latin Hypercube Sampling (LHS) method and evaluating the Partial Rank Correlation Coefficients (PRCCs) [13,24] in this part. From Figure 8, the sensitivity analysis with the selected baseline

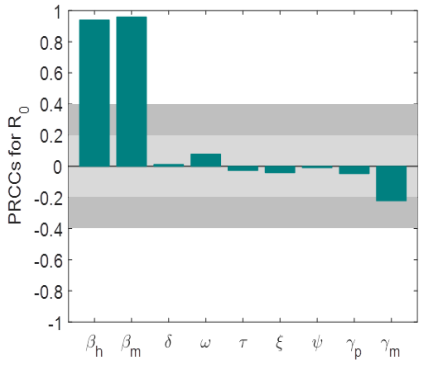


Figure 8. The Partial Rank Correlation Coefficients of R_0 in model (2.1).

values shows that the most sensitive parameter is the rate of transmission between humans and mosquitoes, where the transmission probability from infectious humans to susceptible mosquitoes (β_m) is slightly higher than that of mosquitoes to humans (β_h). A positive sensitivity index indicates that R_0 increases as the parameter

increases. On the contrary, a negative sensitivity index means that R_0 decreases as the parameter increases. In summary, R_0 decreases with the increases of the recovery rates for mild hospital individuals (γ_m) and severe hospital individuals (γ_s), the progression rate from I_h to mild hospital (τ) and severe hospital (ξ). Moreover, R_0 increases with the increase of the probability of pathogens being transmitted from infectious mosquitoes to susceptible humans (β_m), the probability of pathogens being transmitted from infectious humans to susceptible mosquitoes (β_h), the progression rate from A_h to unreported cases P (δ) and the progression rate from I_h to unreported cases P (ω).

According to the Figure 8, we already know the effect of the parameters β_h, β_m, γ_p, γ_m on the basic reproduction of reproduction R_0. However, Figure 9 demonstrates that the changing trend of single parameter and R_0 is more clearly and intuitively.

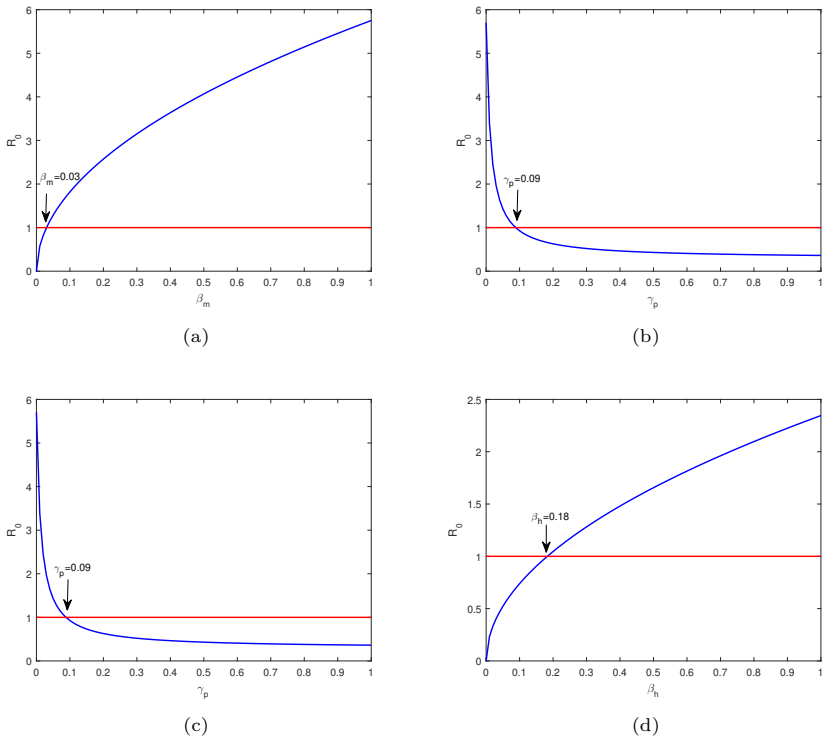


Figure 9. The influence of partial parameter variation on R_0. (a) The variation trend between β_m and R_0. (b) The variation trend between γ_p and R_0. (c) The variation trend between γ_m and R_0. (d) The variation trend between β_h and R_0.

In each subgraph, we change only one parameter and the other parameters shown in Table 2. It can be seen from Figure 9(a) that R_0 is less than 1 only when $\beta_m < 0.03$, which means that the disease can be controlled only when the transmission power of humans to mosquitoes is less than 0.03. Figure 9(b) reveals that the recovery rate of unreported cases is greater than 0.09. This can effectively reduce the spread of the disease. In Figures 9(c) and 9(d), R_0 decreases with an increase in γ_m and a decrease in β_h. When γ_m increases to 0.49 or β_h decreases

to 0.18, R_0 is less than 1, which indicates that increased treatment and reduced transmission from mosquitoes to humans can effectively control the spread of the epidemic.

6.3. Optimal Control

According to the theory in reference [20], we will give the optimal solution of the model (5.1) by using the numerical method. The initial values of model (2.1) are $S_h(0) = 5000000$, $E_h(0) = 10, A_h(0) = 10, I_h(0) = 100, P(0) = 10$, $H_m(0) = 10, H_s(0) = 10$, $R(0) = 10, S_m(0) = 10000000$, $E_m(0) = 10000$ and $I_m(0) = 10000$. We also select the parameter values are in Table 2. The period of the control is 50 weeks. In order to reveal the effect of the control strategies considered in our paper, we will give the graphes of evolution in different compartment populations under different controls. The number of people in different compartments when the weights on objective function are $c_1 = 100, c_2 = 500, c_3 = 1000$ and the different values of u_1, u_2 and u_3 (seen Figure 10).

It can be seen from Figure 10 that the system with control is obviously better than the system without control. When $u_1 = 0, u_2 = 0, u_3 = 0$, the number of people in different compartments is the highest. However, when $u_1 = 0, u_2 = 0.5, u_3 = 0$, the number of people decreased significantly. But, dual control and optimal control have almost the same effect and they are better than single control u_2, while the single control u_2 is better than no control.

7. Discussions and Conclusions

We showed a dengue model with unreported cases and asymptomatic infected classes. The basic reproduction number R_0 is obtained by using the next generation matrix. Stability of the disease-free equilibrium and existence of the endemic equilibrium are derived. Using the Pontryagin's maximum principle, we get the existence of the optimal control pair and the mathematical expression of the optimal control. The best-fit parameter values in our model are identified by the MCMC algorithm based on the data of dengue fever in Singapore from 15 to 52 weeks. We also estimate that the basic reproduction number R_0 is 1.6015 (95%CI: (1.5425-1.6675)). Some numerical simulations and sensitivity analyses are carried out to illustrate our main results, which show that reducing the infection rate and increasing the reporting rate are beneficial to the control of dengue fever. This is consistent with the results of in Musa et al. [27] and Xue et al. [42], which is also better understood and a useful guide provided for better control of dengue fever in the future.

Generally speaking, the epidemic situation in Singapore is grim. On the one hand, the previous epidemics in Singapore were mainly dengue virus serotype 1 and dengue virus serotype 2, so most residents were resistant to dengue virus serotype 1 and dengue virus serotype 2, but not to the current epidemic dengue virus serotype 3. As a result, the infected population of dengue virus serotype 3 was further enlarged by the expansion of Aedes mosquitoes. On the other hand, everyone was isolated and the mosquito control work could not be carried out normally, resulting in a large number of Aedes mosquitoes. At the same time, a large amount of medical resources were used to deal with the other epidemic. Thus, it was difficult to get timely treatment. Dengue fever began to spread.

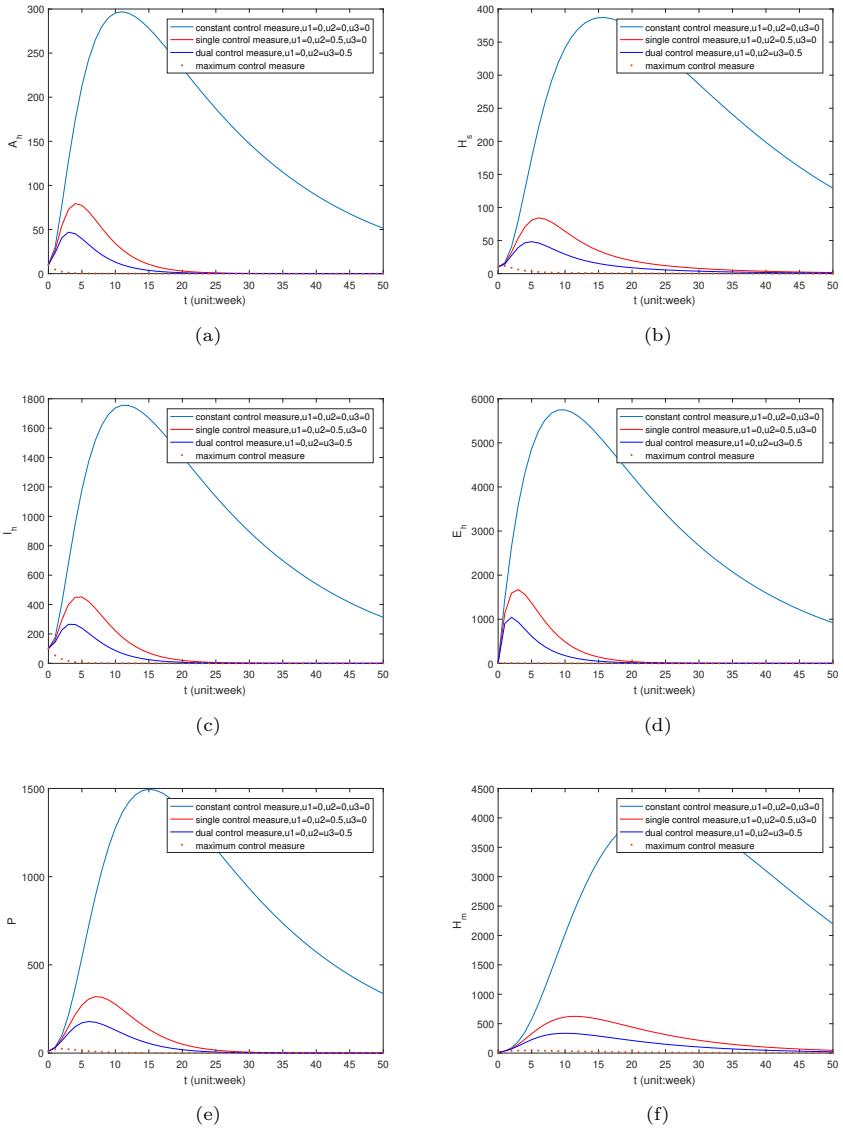


Figure 10. Number of people in different compartment with $c_1 = 100; c_2 = 500; c_3 = 1000$ and different optimal control strategies. (a) A_h; (b) H_s; (c) I_h; (d) E_h; (e) P; (f) H_m.

Our model is not a case study. To simplify the model, we consider some parameters as constants, but these parameters are rarely constants. In fact, there must be a time interval between the spread of infection, and the spread from an infected person to a susceptible population cannot be instantaneous. Similarly, infected people may take some time to become infectious and symptomatic, and there may be a time lag in recovery from the disease. In addition, the disease is subject to temperature changes and is cyclical. Therefore, in the future work, these time-delay and periodic behaviors can be incorporated into the system and its dynamic behavior can be analyzed. So the model would be more realistic and more biologically

meaningful. We will leave that for future research.

Conflict of interest. The authors declare there is no conflict of interest.

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