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# Mathematical Modeling and Analysis of the Dynamics of Chikungunya in Bangladesh

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**Abstract** Chikungunya is one of the major public health problems in Bangladesh and the effect of the disease is more virulent over the country. In this paper, a compartmental mathematical model has been proposed that describes the transmission of chikungunya disease in Bangladesh. Nonlinear incidence rate is considered for disease transmission and the system of nonlinear differential equations is developed to represent the model. Furthermore, the treatment term has been introduced in the model. The basic reproduction number, which is a biological threshold parameter for this disease, has been computed for the model by the method of next generation matrix. Disease free and endemic equilibrium points are calculated and the stability of the model has been analyzed using the basic reproduction number. The global stability has been established using the Lyapunov function theory. In this study, we have calculated the numerical value of the basic reproduction number and performed numerical simulations based on the real data collected from several health institutes of Bangladesh. Both analytical and numerical results provide a pattern about the dynamics of the disease in Bangladesh. Based on the effects, some important policies and insights are provided that might help in reducing the number of infected cases of chikungunya.

**Keywords** Chikungunya disease; nonlinear incidence rate; basic reproduction number; Lyapunov function; stability analysis.

**Mathematics Subject Classification** 92D30, 93A30, 93D20

## 1 Introduction

Mosquito-borne diseases are due to the cause of pathogen transmission among hosts by mosquitoes (the vector). Chikungunya fever is a growing epidemic-prone and vector-borne infectious disease. Chikungunya virus (CHIKV) is a kind of alphavirus that is disseminated to humans by the bite of mosquitos named Aedes albopictus and Aedes aegypti. The indications of chikungunya are abrupt high fever, extreme joints soreness, and a galling skin impetuous [1]. Some patients endured from a long-term swelling skin [2]. The crucial upsurge of chikungunya was in 2005 on the island of Reunion in which a total of 244,000 populations (out of 775,000

populations) had testified the signs of the diseases [3-5]. CHIKV is prevalent in some of the tropical regions.

The chikungunya outburst was firstly identified in southern Tanzania in the year 1952 [11]. In Asia, the first case was identified in Thailand in 1960, then India in 1964, Sri Lanka in 1969, Vietnam in 1975, Myanmar in 1975, and Indonesia in 1982 [6]. It emanated in the greater attention during an endemic occurrence in Reunion Island in 2006. The disease has come to light as one of the key public health problems in most of the Asian countries [10-12, 14]. The pervasiveness of infections is growing expeditiously and the huge numbers of cases were reported in Malaysia, Singapore, Thailand, India, Maldives, and Bangladesh [6-7]. In these regions, it is nourished in the human population by a human-to-mosquito-to-human spreading rotation [8]. Chikungunya has been recognized over the sixty countries of Asia, Africa, Europe, and America [9].

Mathematical models are broadly utilized to study the dynamics of infectious diseases. It helps to describe the transmission and pattern of the disease. By these models, one can develop strategies for prevention, control, and treatments for the disease. Several mathematical models are suggested to describe the spread of chikungunya. The outbreak for a specific country like Thailand, Mexico, the USA, and India has been studied by several authors [9-10, 13-15]. A study of the outbreak and characterization of chikungunya has been performed in Colombia through a mathematical model [17]. In this study, Gonzalez-Parra [17] presented a model considering constant population size and provides an analysis and spread of characterization for the chikungunya.

The outbreak of the chikungunya virus has become a major concern among the people of Bangladesh. The first outburst of chikungunya in Bangladesh was in the Rajshahi district and around 32 peoples were affected by this disease [11]. After that, each year a considerable number of chikungunya patients have been reported into several medical centers of Bangladesh [11-12]. Although the outbreak of chikungunya is a major civic health apprehensions in Bangladesh, there is limited literature available for the study of the disease in Bangladesh. Furthermore, most of the researches are accomplished in an empirical way and as medical case studies in Bangladesh [11-12]. As a consequence, it is necessary to develop the mathematical model and investigate the dynamical behavior of the disease in Bangladesh to identify the transmission pattern that is when diseases die out and when it becomes endemic as well as the treatment effect. Stability analysis is also an essential part to explore a mathematical model that means without mathematical stability analysis it may really difficult to ascertain whether a model is appropriate or not to describe the dynamics of the disease. Therefore, a mathematical model is developed here with stability analysis which describes the chikungunya diseases from the viewpoint of Bangladesh. As a part of the development, in this model, we have considered the combined effect of the saturated incidence rate and the treatment function. It will help us for studying and understanding the dynamical behavior of the disease. The model has been analyzed both theoretically and numerically. For the purpose of numerical simulations and results, we have visited several major hospitals and medical centers of the country for collecting data. We have collected the real data which are used to calculate the values of different parameters.

The model formulation and analysis for chikungunya transmission has been presented in Section 2. Section 3 describes the details of data collection. Numerical simulations are presented in Section 4. Finally, Section 5 concludes the work.

## 2 Mathematical Model Formulation of Chikungunya Transmission

The formulation of our proposed model for the transmission of chikungunya disease has been discussed here. We have considered that there is an interaction between two populations (Human-Vector), and entire human and vector populations are homogeneously mixing with one another. Table 1 and Table 2 contains variables and parameters which are used to describe the model.

Table 1: List of Variables

| Variable | Description |
|---|---|
| $S_h$ | The number of susceptible human population at a time $t$ |
| $I_h$ | The number of infected human population at a time $t$ |
| $R_h$ | The number of recovered human population at a time $t$ |
| $S_m$ | The number of susceptible mosquito population at a time $t$ |
| $I_m$ | The number of infected mosquito population at a time $t$ |

Table 2: List of Parameters

| Parameter | Description |
|---|---|
| $\beta_m$ | The disease contact rate of susceptible human population due to infected mosquito population |
| $\beta_h$ | The disease contact rate of susceptible mosquito population due to infected human population |
| $\lambda_h$ | Birth rate of human population |
| $\lambda_m$ | Birth rate of mosquito population |
| $r_h$ | The transmission rate from infected human to recovered class |
| $\gamma_h$ | The transmission rate from recovered class to susceptible class |
| $\mu_h$ | Natural death rate for human population |
| $\mu_m$ | Natural death rate for mosquito population |
| $\alpha_1, \alpha_2$ | Holling type II functional response parameters between human and vector population |
| $u$ | The capacity of treatment for infective |

The total number of human populations ($N_h$) are separated into three compartments specifically susceptible ($S_h$), infected ($I_h$), and recovered ($R_h$). That is, $N_h = S_h + I_h + R_h$. The mosquito populations ($N_m$) are divided into two compartments namely susceptible ($S_m$), and

infected ($I_m$). That is, $N_m(t) = S_m(t) + I_m(t)$. All new born humans are infection free and become the susceptible human. Accordingly, all new born mosquitoes are susceptible mosquito. The birth and natural death rate of human populations are $\lambda_h$ and $\mu_h$ respectively, and the birth and natural death rate of mosquito populations are $\lambda_m$ and $\mu_m$ respectively.

As we know the chikungunya disease is transferred by the direct contact of infected vector population, so the susceptible humans become infected by the bite of infected mosquito. As a consequences, when a susceptible mosquito emanates in contact with an infected human then this mosquito becomes infected. The human death due to the cause of chikungunya disease is very rare in Bangladesh, therefore, we didn't consider any disease related death rate in our developed model. We have also assumed the loss of permanent immunity of human population (no lifelong immunity for human), that is, a proportion of recovered human populations have a chance to be infected again due to the bite of vector (mosquito).

We have developed the model considering saturated incidence rate that is by integrating a standard force of infection with the portion of antibody generated by the human in response to the contagion instigated by vector (mosquito). Actually the requirement of including the saturated incidence term in the Chikungunya model is mainly the number of efficacious contact between the susceptible and the infectious populations (both for humans and mosquitoes) might be saturated at high infectious level due to the crowd of the infectious individuals in the population. If the human infection saturates at high infection level then due to preventive measures (behavioural changes), the effective contact between human and mosquito decreases and the infection level saturate for mosquito population [24]. Therefore, we have used here the Holling type II functional response for expressing the nonlinear saturated incidence. As far as we know there is no clinical evidence of the study that the amount of antibody created by vector in response to the occurrence of infection triggered by human, therefore, in the model we have assumed parameter $\alpha_1$ as Holling type II functional response for human population and parameter $\alpha_2$ as Holling type II functional response for vector population. We have also considered $T(I_h)$ as a treatment function in the model. Figure 1 shows the schematic diagram represents our proposed model.

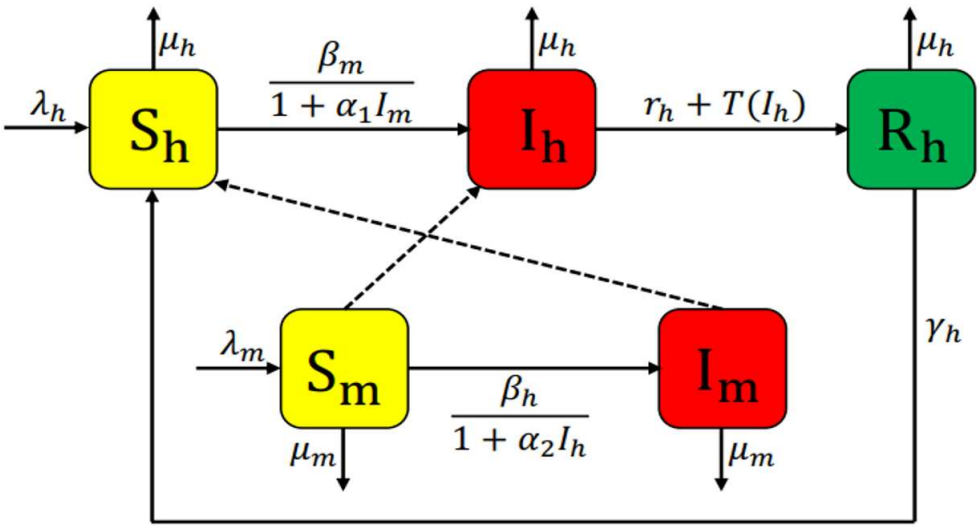


Figure 1: Schematic Diagram of Chikungunya Model with Nonlinear Incidence Rate

The following systems of nonlinear differential equations are developed to describe the model.

$$\begin{aligned}
\frac{dS_h}{dt} &= \lambda_h - \beta_m \frac{S_h I_m}{1+\alpha_1 I_m} - \mu_h S_h + \gamma_h R_h \\
\frac{dI_h}{dt} &= \beta_m \frac{S_h I_m}{1+\alpha_1 I_m} - r_h I_h - \mu_h I_h - T(I_h) \\
\frac{dR_h}{dt} &= r_h I_h - \mu_h R_h + T(I_h) - \gamma_h R_h \\
\frac{dS_m}{dt} &= \lambda_m - \beta_h \frac{S_m I_h}{1+\alpha_2 I_h} - \mu_m S_m \\
\frac{dI_m}{dt} &= \beta_h \frac{S_m I_h}{1+\alpha_2 I_h} - \mu_m I_m
\end{aligned} \tag{1}$$

Here the treatment function $T(I_h)$ is defined as

$$T(I_h) = \begin{cases} u, \text{ if } I_h > 0 \\ 0, \text{ if } I_h = 0, \end{cases}$$

where $u > 0$ is a positive constant represents the capacity of treatment for infective. Summing the first three equations of (1), we obtain the total human population fulfill the equation, $dN_h/dt = \lambda_h - \mu_h N_h$, and its solution is

$$N_h(t) = N_h^0 e^{-\mu_h t} + \frac{\lambda_h}{\mu_h}\left(1 - e^{-\mu_h t}\right).$$

Again, adding the last two equations of (1), we obtain $dN_m/dt = \lambda_m - \mu_m N_m$ whose solution is

$$N_m(t) = {N_m}^0 e^{-\mu_m t} + \frac{\lambda_m}{\mu_m}\left(1 - e^{-\mu_m t}\right).$$

Both solution indicates the total number of population varies with time. In absence of disease, the total number of human population ($N_h$) converges to the equilibrium $\lambda_h/\mu_h$, and the total number of mosquito population ($N_m$) converges to the equilibrium $\lambda_m/\mu_m$. Thus, the positively invariant region for the model is the set,

$$\Gamma = \left\{(S_h, I_h, R_h, S_m, I_m) \in \mathbb{R}_+^5 : 0 \le S_h + I_h + R_h \le \frac{\lambda_h}{\mu_h}, 0 \le S_m + I_m \le \frac{\lambda_m}{\mu_m}\right\}.$$

### 2.1 Equilibrium Points and Basic Reproduction Number of Chikungunya Model

In this section, the equilibrium points and the basic reproduction number of our proposed model (1) have been discussed. Based on the work of Cai and Li [18], we have assumed the initial value of the human population is

$${N_h}^0(t) = {S_h}^0(t) + {I_h}^0(t) + {R_h}^0(t) = \frac{\lambda_h}{\mu_h}$$

in order to get a population of a constant size. Using, $R_h = \frac{\lambda_h}{\mu_h} - S_h - I_h$ in (1) we can eliminate the third equation. In addition, using the property of treatment function, we obtain the reduced model is as follows.

$$\begin{aligned}
\frac{dS_h}{dt} &= \lambda_h - \beta_m \frac{S_h I_m}{1+\alpha_1 I_m} - \mu_h S_h + \gamma_h \left(\frac{\lambda_h}{\mu_h} - S_h - I_h\right) \\
\frac{dI_h}{dt} &= \beta_m \frac{S_h I_m}{1+\alpha_1 I_m} - r_h I_h - \mu_h I_h - u \\
\frac{dS_m}{dt} &= \lambda_m - \beta_h \frac{S_m I_h}{1+\alpha_2 I_h} - \mu_m S_m \\
\frac{dI_m}{dt} &= \beta_h \frac{S_m I_h}{1+\alpha_2 I_h} - \mu_m I_m
\end{aligned} \tag{2}$$

To find equilibrium points of the proposed model we set

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dS_m}{dt} = \frac{dI_m}{dt} = 0$$

in (2).

### 2.1.1 Disease Free Equilibrium Point

Disease free equilibrium (DFE) exists if viruses are absent among the populations. Using $I_h = I_m = 0$ and $T(I_h) = 0$ when $I_h = 0$ in (2), we obtain DFE point of the model

$$E_c^0 = \left(S_h^0, I_h^0, S_m^0, I_m^0\right) = \left(\frac{\lambda_h}{\mu_h}, 0, \frac{\lambda_m}{\mu_m}, 0\right).$$

### 2.1.2 Endemic Equilibrium Point

Endemic equilibrium point (EEP) arises when viruses present among the population. In the case of EEP the infection class is not zero. Let us denote the endemic equilibrium point by $E_c^* = (S_h^*, I_h^*, S_m^*, I_m^*)$, where $S_h^*$, $I_h^*$, $S_m^*$, and $I_m^*$ are the positive solutions of system (2). We get the following endemic equilibrium point.

$$\begin{aligned}
S_h^* &= \frac{\left(1+a_1 I_m^*\right)\left(\lambda_h\left(\mu_h+\gamma_h\right) - \gamma_h \mu_h I_h^*\right)}{\mu_h\left(\left(\mu_h+\gamma_h\right) + \left(\alpha_1\left(\mu_h+\gamma_h\right)+\beta_m\right) I_m^*\right)}, \\
I_h^* &= \frac{\left(\mu_h+\gamma_h\right)\left(\lambda_h\beta_m I_m^* - u\mu_h\left(1+\alpha_1 I_m^*\right)\right) + \beta_m\mu_h\left(u-\gamma_h I_h^*\right) I_m^*}{\mu_h\left(r_h+\mu_h\right)\left(\left(\mu_h+\gamma_h\right) + \left(\alpha_1\left(\mu_h+\alpha_h\right)+\beta_m\right) I_m^*\right)}, \\
S_m^* &= \frac{\lambda_m\left(1+\alpha_2 I_h^*\right)}{\mu_m + \left(\beta_h + a_2\mu_m\right) I_h^*}, \\
I_m^* &= \frac{\lambda_m \beta_h I_h^*}{\mu_m\left(\mu_m + \left(\beta_h + a_2\mu_m\right) I_h^*\right)}.
\end{aligned}$$

### 2.1.3 Basic Reproduction Number

To obtain the basic reproduction number of our proposed model, the next generation matrix method is used. This method is introduced by Diekmann *et al.*, 1990 [16]. In this method, the

basic reproduction number (R) is defined as the spectral radius (dominant eigenvalue) of the next generation operator. The basic reproduction number of chikungunya model (2) is

$$R = \sqrt{\frac{\beta_m \beta_h S_h^0 S_m^0}{\mu_m \left(r_h + \mu_h\right)}}.$$

See appendix for details how to obtain the above result. This dimensionless number is a very useful quantity to clinch the epidemic status of an infectious disease. Based on the value of basic reproduction number, it can be concluded that at which instances the disease dies out and the disease exist in the population. In addition, the analysis of the local and the global stability of a model and the simulations can be deduced using the basic reproduction number.

### 2.2 Stability Analysis of Chikungunya Model

The stability analysis of the model is discussed in this section. From the system of equations of (2) the Jacobian matrix of the model is

$$J = \begin{bmatrix} -\dfrac{\beta_m I_m}{1+\alpha_1 I_m} - \mu_h - \gamma_h & -\gamma_h & 0 & -\dfrac{\beta_m S_h}{\left(1+\alpha_1 I_m\right)^2} \\ \dfrac{\beta_m I_m}{1+\alpha_1 I_m} & -r_h - \mu_h & 0 & \dfrac{\beta_m S_h}{\left(1+\alpha_1 I_m\right)^2} \\ 0 & -\dfrac{\beta_h S_m}{\left(1+\alpha_2 I_h\right)^2} & -\dfrac{\beta_h I_h}{1+\alpha_2 I_h} - \mu_m & 0 \\ 0 & \dfrac{\beta_h S_m}{\left(1+\alpha_2 I_h\right)^2} & \dfrac{\beta_h I_h}{1+\alpha_2 I_h} & -\mu_m \end{bmatrix}. \tag{3}$$

#### 2.2.1 Local Stability of Disease Free Equilibrium

Using $I_h = I_m = 0$, the Jacobian matrix (3) at disease free equilibrium point $E^0$ is,

$$J^0 = \begin{bmatrix} -\mu_h - \gamma_h & -\gamma_h & 0 & -\beta_m {S_h}^0 \\ 0 & -r_h - \mu_h & 0 & \beta_m {S_h}^0 \\ 0 & -\beta_h {S_m}^0 & -\mu_m & 0 \\ 0 & \beta_h {S_m}^0 & 0 & -\mu_m \end{bmatrix}. \tag{4}$$

The characteristic polynomial in the case of disease free equilibrium from equation (4) is

$$f(\lambda) = (-\mu_h - \gamma_h - \lambda)(-\mu_m - \lambda)[(-r_h - \mu_h - \lambda)(-\mu_m - \lambda) - \beta_m \beta_h {S_h}^0 {S_m}^0]. \tag{5}$$

There are four roots of equation (5) and the first two are $\lambda_1 = -\mu_h - \gamma_h, \lambda_2 = -\mu_m$ which are negative. The other two roots are calculated from the quadratic equation

$$\lambda^2 + \left(r_h + \mu_h + \mu_m\right)\lambda + \left[\mu_m \left(r_h + \mu_h\right) - \beta_m \beta_h S_h^0 S_m^0\right] = 0. \tag{6}$$

The equation (6) can be written as $\lambda^2 + a_1\lambda + a_2 = 0$, where $a_1 = r_h + \mu_h + \mu_m > 0$ and $a_2 = \mu_m \left(r_h + \mu_h\right) - \beta_m \beta_h {S_h}^0 {S_m}^0 = \mu_m \left(r_h + \mu_h\right)\left[1 - R^2\right]$. We observe $a_2 > 0$ if and only if

$1 - R^2 > 0 => R^2 < 1$. Thus for $R^2 < 1$, the coefficients of the equation (6) are positive and by Routh-Hurwitz criteria all the roots are negative.

Thus, all the roots of the characteristic polynomial (5) are negative if $R^2 < 1$. If all the roots of the characteristic polynomial are negative then the DFE is locally asymptotically stable. This concludes that for $R^2 < 1$ the disease free equilibrium is locally asymptotically stable.

### 2.2.2 Local Stability of Endemic Equilibrium

Now the Jacobian matrix (3) at endemic equilibrium point $E_c^*$ is

$$J^* = \begin{bmatrix} -\frac{\beta_m I_m^*}{1+\alpha_1 I_m^*} - \mu_h - \gamma_h & -\gamma_h & 0 & -\frac{\beta_m S_h^*}{(1+\alpha_1 I_m^*)^2} \\ \frac{\beta_m I_m^*}{1+\alpha_1 I_m^*} & -r_h - \mu_h & 0 & \frac{\beta_m S_h^*}{(1+\alpha_1 I_m^*)^2} \\ 0 & -\frac{\beta_h S_m^*}{(1+\alpha_2 I_h^*)^2} & -\frac{\beta_h I_h^*}{1+\alpha_2 I_h^*} - \mu_m & 0 \\ 0 & \frac{\beta_h S_m^*}{(1+\alpha_2 I_h^*)^2} & \frac{\beta_h I_h^*}{1+\alpha_2 I_h^*} & -\mu_m \end{bmatrix}. \quad (7)$$

The trace of the above matrix is

$$\text{trace } (J^*) = -\frac{\beta_m I_m^*}{1+a_1 I_m^*} - \mu_h - \gamma_h - -r_h - \mu_h - \frac{\beta_h I_h^*}{1+a_2 I_h^*} - \mu_m - \mu_m$$

which is negative. The determinant of (7) is

$$\begin{aligned} \det (J^*) \ &= \mu_m \gamma_h \frac{\beta_m I_m^*}{1+\alpha_1 I_m^*} \left( \frac{\beta_h I_h^*}{1+\alpha_2 I_h^*} + \mu_m \right) \\ &\quad + \mu_m (r_h + \mu_h) \left( \frac{\beta_h I_h^*}{1+\alpha_2 I_h^*} + \mu_m \right) \left( \frac{\beta_m I_m^*}{1+\alpha_1 I_m^*} + \mu_h + \gamma_h \right) \\ &\quad - \mu_m (\mu_h + \gamma_h) \frac{\beta_m \beta_h S_m^* S_h^*}{(1+\alpha_1 I_m^*)^2 (1+\alpha_2 I_h^*)^2}. \end{aligned}$$

After some calculations we found the determinant as the following form,

$$\begin{aligned} \det (J^*) \ &= \mu_m \gamma_h \frac{\beta_m I_m^*}{1+\alpha_1 I_m^*} \left( \frac{\beta_h I_h^*}{1+\alpha_2 I_h^*} + \mu_m \right) \\ &\quad + \left( \frac{\beta_m \beta_h I_m^* I_h^*}{(1+\alpha_1 I_m^*)(1+\alpha_2 I_h^*)} + (\mu_h + \gamma_h) \frac{\beta_h I_h^*}{1+\alpha_2 I_h^*} + \mu_m \frac{\beta_m I_m^*}{1+\alpha_1 I_m^*} \right) \frac{\beta_m \beta_h S_m^* S_h^*}{R^2} \\ &\quad + \mu_m \beta_m \beta_h S_m^* S_h^* (\mu_h + \gamma_h) \left[ \frac{1}{R^2} - \frac{1}{(1+\alpha_1 I_m^*)^2 (1+\alpha_2 I_h^*)^2} \right]. \end{aligned}$$

Here the determinant is positive if $R^2 < (1+\alpha_1 I_m^*)^2 (1+\alpha_2 I_h^*)^2$.

For the matrix (7), the trace is negative for any positive parameter. The determinant will be positive if the above condition on reproduction number holds. If trace is negative and

determinant is positive then all the eigenvalues of the matrix (7) are negative and accordingly the endemic equilibrium is locally asymptotically stable. Hence, this concludes the endemic equilibrium is locally asymptotically stable if the above condition on reproduction number is satisfied.

### 2.2.3 Global Stability of Disease Free Equilibrium

The global stability for DFE of the model (2) has been studied using Lyapunov function. The method of matrix-theoretic [19-21] is used to construct Lyapunov function. We consider

$$f(x,y) = (F-V)\,x - \digamma(x,y) - \vee(x,y),$$

where

$$\digamma(x,y) = \begin{pmatrix} \beta_m \dfrac{S_h I_m}{1+\alpha_1 I_m} \\ \beta_h \dfrac{S_m I_h}{1+\alpha_2 I_h} \end{pmatrix},\ \vee(x,y) = \begin{pmatrix} (r_h+\mu_h) I_h + u \\ \mu_m I_m \end{pmatrix},$$

$$F = \begin{bmatrix} 0 & \beta_m S_h^0 \\ \beta_h S_m^0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} r_h+\mu_h & 0 \\ 0 & \mu_m \end{bmatrix}.$$

The disease compartment can be written as, $x' = (F-V)\,x - f(x,y)$ and the left eigenvector of the nonnegative matrix $V^{-1}F$ is

$$\omega^T = \left( \frac{r_h+\mu_h}{\beta_m {S_h}^0} R, 1 \right).$$

After some calculations we have

$$f(x,y) = \begin{bmatrix} \dfrac{\alpha_1 \beta_m S_h^0 I_m^2 + \beta_m I_m (S_h^0 - S_h)}{1+\alpha_1 I_m} + u \\ \dfrac{\alpha_2 \beta_h S_m^0 I_h^2 + \beta_h I_h (S_m^0 - S_m)}{1+\alpha_2 I_h} \end{bmatrix}.$$

Note that, $f(x,y) \geq 0$ in $\Gamma$ if $S_h \leq S_h^0$, $S_m \leq S_m^0$, and $f(0,y_0) = 0$. Since $F \geq 0$, $V^{-1} \geq 0$ and $f(x,y) \geq 0$, by theorem [21], $Q = \omega^T V^{-1} x$ is the Lyapunov function. Straightforward calculation gives

$$Q = \frac{R}{\beta_m S_h^0} I_h + \frac{I_m}{\mu_m}.$$

The following theorem is required to establish for global stability of DFE.

**Theorem 1** *The disease free equilibrium of the model (2) is globally asymptotically stable in* $\Gamma$ *if* $R \leq 1$.

**Proof** The Lyapunov function is

$$Q = \frac{R}{\beta_m {S_h}^0} I_h + \frac{I_m}{\mu_m}$$

with $R < 1$ and $f(x, y) \geq 0$. Differentiating $Q$ along solutions of (2) gives

$$\begin{aligned}
Q' &= \omega^T V^{-1} x' \\
&= \omega^T V^{-1} (F - V) x - \omega^T V^{-1} f(x, y) \\
&= \frac{R}{\beta_m S_h^0} \left( \beta_m \frac{S_h I_m}{1 + \alpha_1 I_m} - r_h I_h - \mu_h I_h - u \right) + \frac{1}{\mu_m} \left( \beta_h \frac{S_m I_h}{1 + \alpha_2 I_h} - \mu_m I_m \right) \\
&= (R - 1) \omega^T x - \omega^T V^{-1} f(x, y) \\
&= (R - 1) \left( \frac{r_h + \mu_h}{\beta_m S_h^0} R I_h + I_m \right) - \frac{R}{\beta_m S_h^0} \left[ \frac{\alpha_1 \beta_m S_h^0 I_m^2 + \beta_m I_m (S_h^0 - S_h)}{1 + \alpha_1 I_m} + u \right] \\
&\quad - \frac{1}{\mu_m} \left[ \frac{\alpha_2 \beta_h S_m^0 I_h^2 + \beta_h I_h (S_m^0 - S_m)}{1 + \alpha_2 I_h} \right].
\end{aligned}$$

This follows that $Q' \leq 0$ if $R \leq 1$. If $R = 1$ then $Q' = 0$ if and only if $I_h = I_m = 0$ ($u = 0$ for $I_h = 0$). Therefore, every solution trajectory of equations of model (2) converges to the largest compact invariant set $M = (S_h^0, I_h, S_m^0, I_m)$, and the only point in $M$ is the disease free equilibrium. Then by LaSalle's invariant principle [22], $E_c^0$ is globally asymptotically stable in $\Gamma$ if $R \leq 1$. That is every solution trajectory of equations in the model (2) approaches to $E_c^0$ as $t \to \infty$. □

## 3 Data Collection

To find the numerical simulations for the model, data are collected for chikungunya disease from several medical and health centers in Bangladesh, from the Institute of Epidemiology Disease Control and Research (IEDCR), Bangladesh, and from Directorate General of Health Services, Bangladesh. For the transmission of chikungunya disease we have collected real data of the infected people during the time of 12-05-2017 to 28-09-2017, which is approximately 20 weeks. During that time intervals, the transmission of a mosquito borne disease like chikungunya is usually high. In the year 2017, the chikungunya transmission abruptly increased in comparison with the previous years and become a serious health issue throughout the country. Table 3 gives the particulars of the data during this interval from 12-05-2017 to 28-09-2017.

Besides these the information of 185 possible patients from 17 different cities except Dhaka city are send to IEDCR and after testing at IEDCR, among them 52 patients are detected as an infected due to chikungunya. In summary we have collected a data of 20 weeks in which a total of 13866 patients are found to be infected in different medical and health centers [23]. Point to be noted that no patient were found to be dead in any medical or health center due to the cause of chikungunya disease. These data sets are used to perform the numerical simulations of the transmission of chikungunya disease.

## 4 Numerical Simulations

In this section, numerical simulations of chikungunya (2) model have been discussed. To simulate the compartments of the model we have used MATLAB program and developed the code. We have estimated the parameter values for simulation of chikungunya model (2) from

Table 3: Data of Chikungunya Infected Patient

| **Name of the Hospital** | **Number of Chikungunya Patient** |
|---|---|
| Dhaka Medical College Hospital | 4864 |
| Mitford Hospital | 2348 |
| Shaheed Suhrawardy Medical College & Hospital | 2558 |
| Shaheed Monsur Ali Medical College and Hospital | 9 |
| Mugda Medical College and Hospital | 131 |
| Dhaka Shishu Hospital | 93 |
| United Hospital | 522 |
| Apollo Hospitals Dhaka | 197 |
| Delta Hospital Ltd. | 255 |
| Other private hospital/Doctor | 547 |
| IEDCR | 2290 |
| Total | 13814 |

data which are discussed in the above section. The estimation process has been carried out by following [25]. However, since the real data for the mosquito population is not available, we have only estimated the parameters associated with human rates. We have assumed the mosquito infection rate as equal as human infection rate. Due to the involvement of massive number of mosquito population, we have assumed the mosquito birth and death rate as hundred times as human birth and death rate. Based on the estimations and assumptions, the parameter values are as follows:

$$\begin{aligned}&\beta_h = 0.000435146(person \cdot week)^{-1},\ \beta_m = 0.000435146\,(person \cdot week)^{-1},\\ &\lambda_h = 0.00036154\ person{\cdot}week^{-1},\ \mu_h = 0.00010385\ week^{-1},\ \lambda_m = 0.036154\ person{\cdot}week^{-1},\\ &\mu_m = 0.010385\ week^{-1},\ \alpha_1 = 0.065\,person^{-1},\ \alpha_2 = 6.5\,person^{-1}, r_h = 0.0514668\ week^{-1},\\ &\gamma_h = 0.0192308\ week^{-1},\ u = 0.0005\,person{\cdot}week^{-1}\end{aligned} \tag{8}$$

The approximate population of Bangladesh in 2017 was 164 million and at the beginning of the time period of the data (12-05-2017) there was 7 infected found countrywide. As a consequence, we have assumed the initial conditions for human individual as $S_h(0) = 164$ and $I_h(0) = 7\times10^{-6}$. However, the real data for mosquito is not available and we have assumed the initial susceptible mosquito is as same as initial human and initial infected mosquito as 2.45 million. That is, the initial condition for mosquito individual as $S_m(0) = 164$ and $I_m(0) = 2.45$ Using the parameter (8) and above mentioned initial conditions, we have generated the time series of the compartments of model (2) which is shown in the Figure 2.

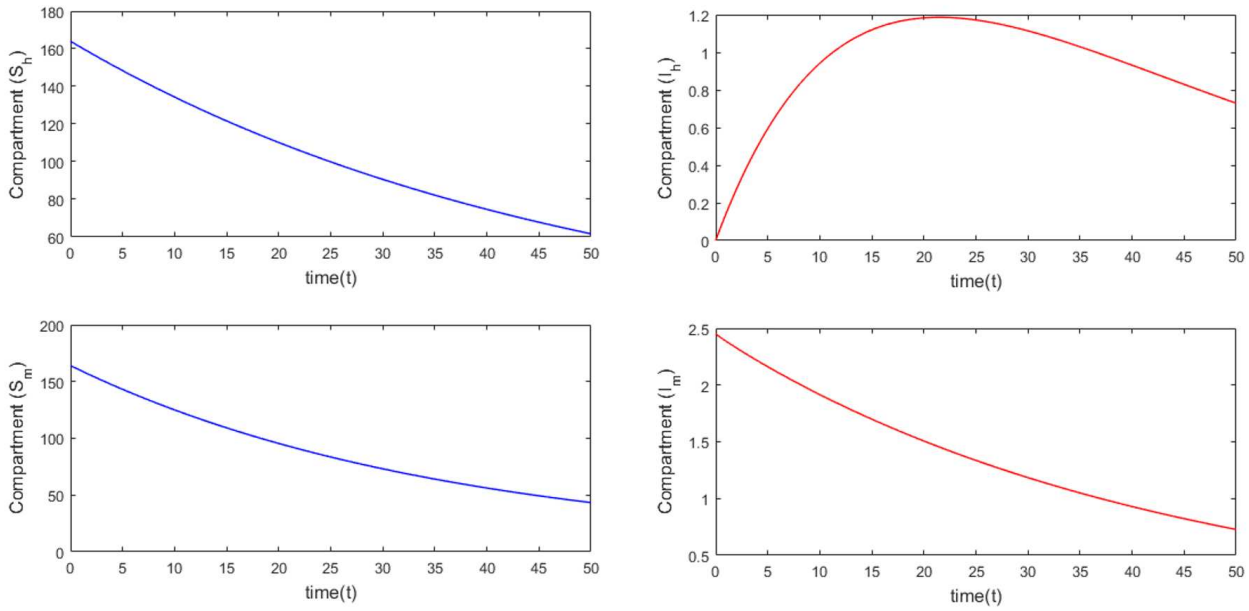


Figure 2: Time Series of the Compartments of the Model (2)

From Figure 2, it has been observed that the susceptible populations for both human and mosquito are decreasing but never vanishes. As we have assumed in our model that there is no permanent immunity from this disease and the recovered populations become again susceptible, therefore, this compartment gives a positive value. Furthermore we observe that the infection is still present among the population after a time of 50 weeks. The infected human population gradually increases and becomes maximum at the time of 20 weeks and afterword it gradually decreases. From the data set (discussed in the above section) we observe the data are collected for 20 weeks during the time period from May to August. This interval of time is the peak time for the transmission of chikungunya and we found the maximum infection occurs during this time period. The simulation matches the real phenomenon and that's why we found the maximum infected population in the time of 20 weeks. Using these data from the model (2), we found the value $R = 0.06546$ which is less than one. Since $R < 1$, so according to the theory the infection will die out from the population in a long term. As we see in the simulation that the infected compartments are decreasing and it will become zero in a long term which matches the theoretical result.

Now we have changed the parameters in such a way that the reproduction number increases and becomes greater than one. For this we have just changed the death rate of mosquito population ($\mu_m$) and keep other parameters of (8) are same. The new set of parameter is given below.

$$\begin{aligned}
&\beta_h = 0.000435146(person \cdot week)^{-1}, \beta_m = 0.000435146(person \cdot week)^{-1},\\
&\lambda_h = 0.00036154\ person{\cdot}week^{-1}, \mu_h = 0.00010385\ week^{-1}, \lambda_m = 0.036154\ person{\cdot}week^{-1},\\
&\mu_m = 0.00010385\ week^{-1},\ \alpha_1 = 0.065\ person^{-1},\ \alpha_2 = 6.5\ person^{-1}, r_h = 0.0514668\ week^{-1},\\
&\gamma_h = 0.0192308\ week^{-1},\ u = 0.0005\ person{\cdot}week^{-1}
\end{aligned} \tag{9}$$

We now simulate the infected compartments of the model for a long term of time. Using these set of parameters (9) we found the reproduction number is $R = 6.5461$, which is greater

than one. The time series for a long interval of time of infected compartments of model (2) for both parameter set (8) and (9) is shown in the Figure 3.

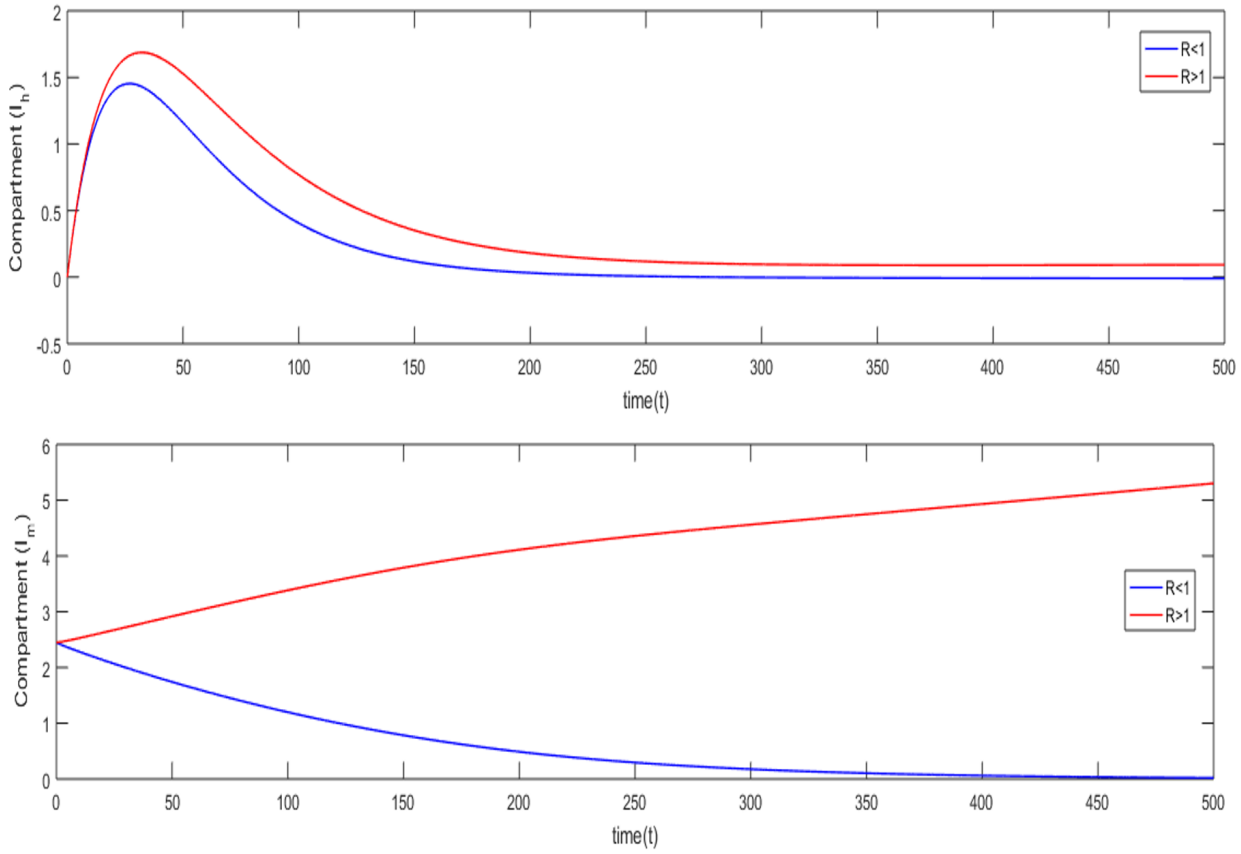


Figure 3: Time Series of the Infected Compartments of the Chikungunya Transmission Model for $R < 1$ and $R > 1$

From Figure 3, it has been observed that, the infected population highly increases when $R > 1$. According to the theoretical result it has been known that in a long time of interval the infection will be present among the population for $R > 1$ and dies out for $R < 1$. From the Figure 4, we have observed the similar phenomenon. The curve for the infected population for $R > 1$ is much higher than the case for $R < 1$. This figure indicates that the infection reaches at zero for long interval of time. We found the infected compartments both for the human and the mosquito populations are high throughout the period of time. If the infected mosquito presents then the infection will be spread through the human populations and it will be an epidemic scenario throughout the year. The phase portrait of infected human population over susceptible human population for both of the cases $R > 1$ and $R < 1$ is given in the Figure 4.

From Figure 4, we have observed that infected human population for the transmission of chikungunya is also higher when $R > 1$. In our proposed model (2), we have used a treatment function $T(I_h)$. According to the definition, the value of the treatment function is considered as a constant $(u)$ in case of the presence of the infection. On the other hand, the value of the function is zero when there is no infective case. To see the effect of treatment function over infected human population, we have varied the value of $u$ from 0.0005 to 0.0125 and keep the other parameters of (8) as same as before. Figure 5 gives the simulation result.

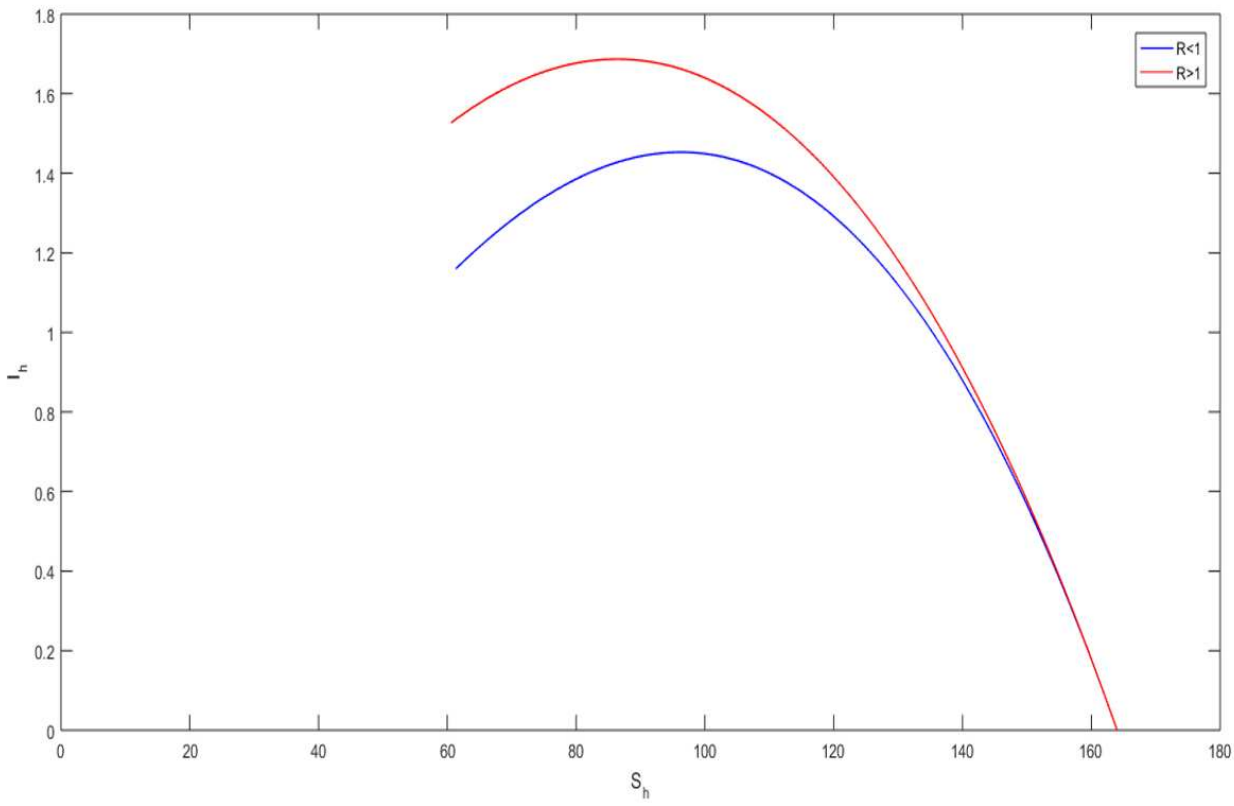


Figure 4: Phase Portrait of Infected Human Population over Susceptible Human Population for Chikungunya Transmission

From Figure 5, it can be observed that the human infection increases for the low value $u$ and decreases for a higher value of $u$. This indicates that when more people are infected by Chikungunya and obtained treatment in hospitals or medical centers there is less chance of occurring infection of others susceptible people through these infected individuals, and in this way new infection could be decreased or even stopped. However, if there is no adequate treatment facilities available and majority of the infected individuals are untreated the infection must be increased. Sometimes the infected persons are not conscious of the disease and do not take proper treatment while infected by the disease. Moreover, in some cities substantial amount of treatment facilities are not available that results a scenario of high infection. An infected human can spread the disease by infecting a susceptible mosquito if the individual is not aware of the disease and do not take adequate treatment.

## 5 Conclusion

We have presented here a mathematical model to explore the dynamics of chikungunya disease in Bangladesh. The local and global stability analyses of the model are shown. We have collected real data from several health institutions in Bangladesh and performed the numerical simulations. It has been observed the infection still presents among the population after a time of 50 weeks in the case of chikungunya. The infected human population gradually increases and becomes maximum at the time of 20 weeks and then it gradually decreases. The simulation

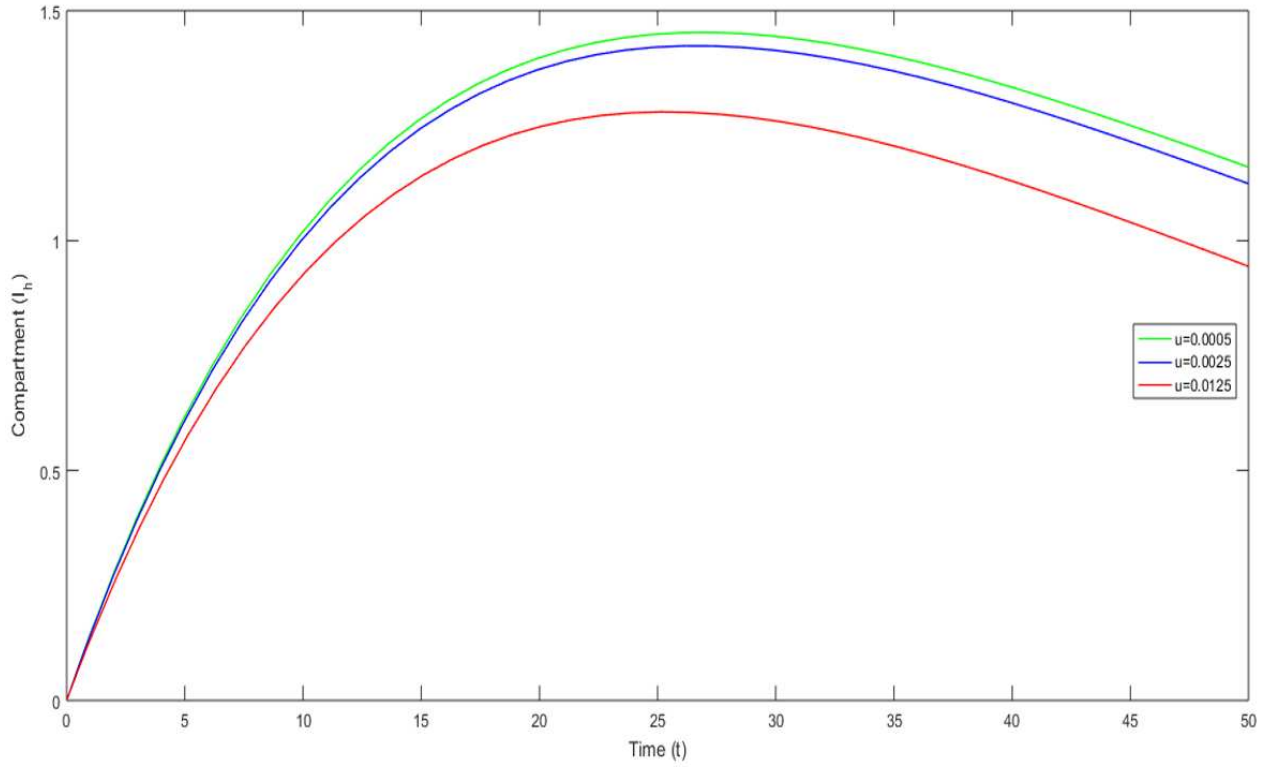


Figure 5: Effect of Treatment Function over Infected Human Population for Chikungunya Transmission

matches the real phenomenon and we found the maximum infected population in the time of 20 weeks. When $R < 1$ , the simulation result shows the infection dies out from the population in the long term, and it supports the theoretical result. In our proposed model, we have used a treatment function. Through our simulations we have observed that there is a great effect of the treatment function over the infected human population. To better modeling treatment parameter is required to investigate the dynamics and control of the disease. Furthermore, we have drawn phase portrait between susceptible human populations versus infected human populations and observed that infected human population is higher when $R > 1$, and lower when $R < 1$. These numerical simulations also support our theoretical results. From the numerical simulations, we have also observed that when the value of transmission parameters decreases infected cases decreases and vice versa. So it can be concluded that to lessen the chikungunya disease diffusion, it is necessary to reduce the transmission parameters. For instance, to avoid mosquito bites, people may wear the cloth covering in most of the body and could use mosquito repellent and spray as well. Any act which influences the reduction of the bites of infected mosquito will affect the reproduction number $R$. As we observe the diseases die out in the long term when the reproduction number is less than one. So the government should take necessary initiatives of killing the mosquito and not to grow these types of mosquito beforehand. In these cases, the transmission of virus will also be reduced or completely eradicated. Lastly, we have observed that treatment function has a great effect on disease dynamics. For a higher value of the treatment function, the infectious decrease and vice versa. Therefore to control the disease outbreak adequate facilities of hospitals and medical centers are needed to be ensured, as well

as proper treatment and awareness are required.

## Appendix A. Basic reproduction number

To find this parameter from our proposed model, we have used the next generation matrix method. This method is introduced by Diekmann *et al.*, 1990 [16]. According to this method we need to identify the infection classes of our proposed model. The model (2) has two infection classes which are

$$\begin{aligned} \frac{dI_h}{dt} &= \beta_m \frac{S_h I_m}{1 + a_1 I_m} - r_h I_h - \mu_h I_h - u, \\ \frac{dI_m}{dt} &= \beta_h \frac{S_m I_h}{1 + a_2 I_h} - \mu_m I_m. \end{aligned} \tag{10}$$

Let us consider, $x^T = (I_h, I_m)$ and $y^T = (S_h, S_m)$ be the disease and non-disease compartment respectively and further consider $\digamma_i$ be the rate of appearance of new infection in the $i$th disease compartment and let $\vee_i = \vee_i^- - \vee_i^+$, where $\vee_i^+$ is the rate of transfer of individuals into compartment $i$ and $\vee_i^-$ is the rate of transfer of individuals out of the $i$th compartment then (10) can be written as

$$\frac{dx_i}{dt} = \digamma_i(x, y) - \vee_i(x, y), \tag{11}$$

$$\text{where } \digamma_i(x, y) = \begin{pmatrix} \beta_m \dfrac{S_h I_m}{1 + \alpha_1 I_m} \\ \beta_h \dfrac{S_m I_h}{1 + \alpha_2 I_h} \end{pmatrix} \tag{12}$$

$$\text{and } \vee_i(x, y) = \begin{pmatrix} (r_h + \mu_h) I_h + u \\ \mu_m I_m \end{pmatrix}. \tag{13}$$

From the matrices of partial derivatives of $\digamma_i(x, y)$ and $\vee_i(x, y)$ at DFE, one can form the next generation matrix (operator) $FV^{-1}$. That is,

$$F = \left[ \frac{\partial \digamma_i(0, y_0)}{\partial x_j} \right] \text{ and } V = \left[ \frac{\partial \vee_i(0, y_0)}{\partial x_j} \right], \tag{14}$$

where $i, j = 1, 2, ..., m$ and $m$ is the number of infection class.

Using (12) and (13) in (14) we get

$$F = \begin{bmatrix} 0 & \beta_m S_h^0 \\ \beta_h S_m^0 & 0 \end{bmatrix}, \tag{15}$$

$$V = \begin{bmatrix} r_h + \mu_h & 0 \\ 0 & \mu_m \end{bmatrix}. \tag{16}$$

The next generation matrix is defined as

$$K = F * V^{-1} = \begin{bmatrix} 0 & \beta_m S_h^0 \\ \beta_h S_m^0 & 0 \end{bmatrix} * \begin{bmatrix} \dfrac{1}{r_h + \mu_h} & 0 \\ 0 & \dfrac{1}{\mu_m} \end{bmatrix} = \begin{bmatrix} 0 & \dfrac{\beta_m S_h^0}{\mu_m} \\ \dfrac{\beta_h S_m^0}{r_h + \mu_h} & 0 \end{bmatrix}.$$

To find the dominant eigenvalue, we need to find out the characteristic equation of the next generation matrix. The characteristic equation of the matrix is

$$\lambda^2 - (0+0)\lambda + \left(0 - \frac{\beta_m S_h^0}{\mu_m} \cdot \frac{\beta_h S_m^0}{r_h + \mu_h}\right) = 0.$$

Solving the value of $\lambda$ we get

$$\lambda = \sqrt{\frac{\beta_m \beta_h S_h^0 S_m^0}{\mu_m (r_h + \mu_h)}}.$$

Thus the spectral radius (dominant eigenvalue) of the matrix is

$$R = \sqrt{\frac{\beta_m \beta_h S_h^0 S_m^0}{\mu_m (r_h + \mu_h)}}$$

which is the basic reproduction number for our proposed model (2).

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