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A mathematical analysis of the dynamics of chikungunya virus transmission

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ABSTRACT

In this paper, a deterministic model for the dynamics of chikungunya virus transmission is formulated and analyzed. It is shown that the model has a disease free equilibrium (DFE) and by using the basic reprodution number ($\Re_0$) local stability of DFE is proved when $\Re_0 < 1$. Also, the global stability of DFE is investigated by Lyapunov function and LaSalle Invariance Principle. We show that there exists a unique endemic equilibrium (EE) of the model which is locally asymptotically stable whenever $\Re_0 > 1$ and establish the global stability of the EE when $\Re_0 > 1$, by using Lyapunov function and LaSalle Invariance Principle for a special case. Numerical simulations and sensitivity analysis show that the destruction of breeding sites and reduction of average life spans of vector would be effective prevention to control the outbreak. Controlling of effective contact rates and reducing transmissions probabilities may reduce the disease prevalence.

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1 Intrduction

Chikungunya is an emerging mosqueto-borne viral disease caused by the chikungunya virus (CHIKV). It was first identified during an outbreak in southern Tanzania in 1952 [1, 2]. The term "chikungumya" comes from a word in the Makonde or Kimakonde language of southeast Tanzania and northern Mozambique and means "to become contorted" or "bend over" [1]. Chikungunya virus is transmitted to people by several species of mosquito of the genus *Aedes*, most common genus are *Aedes aegypti* and *Ades albopictus* [1, 3]. They mainly bite during daylight hour [3] and peaks of bitting activity is during early morning and late afternoon [1, 4]. A mosquito becomes infected after biting an infected human and an extrinsic incubation period is between two to four days [5, 6]. After the incubation period mosquitoes can transmit the virus. Mosquitoes remain inefectious for life time [7] and no vertical transmission is yet proved till today [8].
A human is infected after an effective bite of an infected mosquito, the intrinsic incubation period in the human host is usually 1 to 12 days [8, 9, 10] and during this period infected human unable to transmit the virus. After the incubation period most of the infected people develop symptoms. Common symptoms are: fever, severe

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joint pain, headache, rash and some digestive symptoms: abdominal pain, nausea, vomiting or diarrhea may also occur [1, 3, 11]. Pain usually occurs in peripheral joints, such as wrists, ankles, joints of the hands and feet, in some of the larger joints: shoulders, elbows and knees, also may occur in muscles and ligaments [12, 13]. The joint pain may lasts a few days or a few weeks, but in some cases it may persist for a long time (several months, even years) following the acute infection [1, 14, 15] called subacute or chronic phase. After the viraemia period, during the period infected human is infectious, human recovers. But in some cases chikungunya viral antigen was found in a muscle biopsy of a person suffering a recurrent episode of disease three months after initial onset [16]. Additionally, viral antigen and viral RNA were found in macrophages in the synovial joint of a person experiencing a relapse of musculoskeletal disease 18 months after initial infection [17]. So, there is a possibility to transmit virus from human to mosquito after acute phase.

There is no specific preventive vaccine and antiviral medicine to treat the disease [1, 3, 18]. Supportive cares such as rest, drinking water, etc are suggested and symptomatic treatments including the use of nonsteroidal anti-inflammatory drugs such as naproxen, non-aspirin analgesics, such as, paracetamol (acetaminophen) are primarily directed to reduce fever and pain [18]. However, a vaccine exists but it is in an early-stage clinical trial and will not be commercially available in the near future [19].

Chikungunya re-emerges in many countries of Africa, Asia, Europe and America since the first outbreak in Tanzania. In Democratic Republic of the Congo, it was first isolated in 1958, after that it was isolated in 1960 [20]. But in 2000-2001, during an outbreak around 50,000 people became infected [1]. The first outbreak in Asia was in Bangkok, Thailand in 1958 [21]. Other outbreaks include India in 1960 and Srilanka in 1970 [22], Malaysia in 1998-1999 [23], Vietnam in 1975, Indonesia in 1982 [24], Italy in 2007 and France in 2010 [25]. In the last decade, the major outbreak was in Reunion Island in 2005, during this outbreak 2,666,000 people were infected while the total population was 770,000 and around 250 death cases were reported [26]. In India, outbreak was large in 2006-2007 and during this outbreak 1.39 million cases were reported officially in 2006 and 37,683 cases were reported by national authority in 2007 [1]. In American countries, from 2013-2014 around 1,118,763 suspected cases and 24,682 confirmed cases were reported by the Pan American Health Organization (PAHO) regional office [27]. In Bangladesh, the first outbreak of chikungunya was investigated in 2008 [28].

In the last century, compartmental mathematical models have been widely used for studying the epidemiological models, in particular vector borne infectious disease models [29, 30]. A several number of deterministic models have been proposed to study the chikungunya virus [5, 8, 9, 31] and the references therein. A simple deterministic model of the transmission of chikungunya virus between human and mosquitoes has developed by Yakob and Clements [31]. They have fitted the model with real data, estimated the type of basic reproduction number and analyzed the sensitivity of the parameters. Age structured deterministic model also proposed, analyzed theoritically and numerically [32]. Authors in [33], formulate a model that incorporate the dynamics of two circulating viral disease: dengue and chikungunya by considering variable population size and infection in sub-acute and chronic phase but not analyzed the model qualitatively. The spatio-temporal transmission of chikungunya is analysed in [34] and it is shown that the prevention of moving symptomatic individuals is not sufficient mechanisms to control the outbreak, since the presence of asymptomatic individuals spread the disease silently within the population. In [9], a temporal model is proposed to study the outbreak of chikungunya in several cities of Re'union island in 2005. In this model, the existence and the stability of the disease free equilibrium is investigated by using basic reproduction number but the dynamics of the endemic equilibrium are not considered.

In this research article, we develop a new deterministic model to study the transmission of chikungunya virus. In our model, we incorporate a class of infected individuals those are in subacute or chronic phase. Also we consider the treatment to the infected individuals in different classes. The paper is organized as follows: in section 2, the model is formulated and various properties including the boundedness and the positivity of the solutions are analyzed, also the existence and the stability of the equilibrium are investigated by using different techniques in section 3. Numerical simulations and sensitivity analysis are carried out in section 4. In section 5, discussion and conclusion of the study are drawn.

2 Model formulation

The human population is divided into the following six mutually-exclusive classes: susceptible (S_h), exposed (E_h), symptomatically infectious in acute phase (I_{s1}), infected in subacute phase (I_{s2}), asymptomatically infectious (I_a) and recovered (R_h). So, the total population at time t is $N_h(t) = S_h(t)+E_h(t)+I_a(t)+I_{s1}(t)+I_{s2}(t)+R_h(t)$. Similarly, the vector population is divided into the following three classes: susceptible (S_m), exposed

(E_m) and infectious (I_m). Therefore, the total matured mosquito population is $N_m(t) = S_m(t) + E_m(t) + I_m(t)$.

Assume that the requirement rate of human population is constant, π_1, and human population is born as susceptible, thus there is no vertical transmission. Also, suppose that the mosquito requirement rate is constant, π_2. A susceptible human is infected when bitten by an infectious mosquito and goes to the class (E_m). Suppose that the biting rate of each mosquito is b_m per day. Since the number of bites by mosquitoes equals to the total number of bites received by the humans so, we have

$$b_m N_m = b_h(N_h, N_m)N_h \tag{2.1}$$

where, b_h is the rate of bites received by humans. So that

$$N_m = \frac{b_h(N_h, N_m)N_h}{b_m} \tag{2.2}$$

Now, let β_1 be the probability that a bite from an infectious mosquito will lead a host infection. So that, $\beta_1 b_h$ is the effective contact rate between a susceptible human and infectious mosquito. Thus, the rate at which susceptible human acquire infection, after an effective contact with infectious mosquito, is λ_1 and given by

$$\lambda_1 = \frac{\beta_1 b_h(N_h, N_m)I_m}{N_m} \tag{2.3}$$

Thus, equation (2.2) and (2.3) gives

$$\lambda_1 = \frac{b_m \beta_1 I_m}{N_h} \tag{2.4}$$

This infection rate is called force of infection. After an intrinsic incubation period, the time elapsed by the virus from the moment of infection to the beginning of infectiousness, a portion p of populations in the class E_h moves to the asymptomatically infectious class I_a and the remaining portion $(1-p)$ enters into the symptomatically infectious class I_{s1}. After the viremic period, infectious humans of both class class I_a and I_{s1} are recovered and go forward to the class R_h. But some symptomatically infectious individuals go to the sub-acute or chronic phase I_{s2} and symptoms can persist for long time [14, 15]. Moreover, chikungunya virus antigen is found in a muscle biopsy of a person suffering of disease three months after initial onset [16]. So, there is a chance for susceptible mosquitoes to be infected from individuals in class I_{s2}. Let μ_1 be the natural mortality rate of human population.

A susceptible mosquito goes to the exposed class when it bites an infectious human in the class I_a and I_{s1}, also in class I_{s2} but the rate may be neglected. The effective contact rate between the susceptible mosquitoes and infectious hosts (I_a and I_{s1}) is $b_m\beta_{12}$, and between S_m and I_{s2} is $b_m b_{22}$, where β_{12} is the probability that a bite leads to infection of the mosquito from the classes I_a and I_{s1}, and β_{22} is that from I_{s2}. Hence, the rate at which mosquitoes acquire infection from both asymptomatically infectious and symptomatically infectious human is given by

$$\lambda_2 = \frac{b_m\beta_{12}(I_a + I_{s1}) + b_m\beta_{22}I_{s2}}{N_h} \tag{2.5}$$

Mosquitoes in the exposed class become infectious after an extrinsic incubation period, the period necessary for the virus to follow a cycle that brings it from the mosquito stomach to its salivary gland. The mortality rate for the classes S_m, E_m and I_m is μ_2. We also assume that there is no vertical transmission. Now, considering all of the above assumtions and using the law in[35], we can construct the following deterministic system of nonlinear

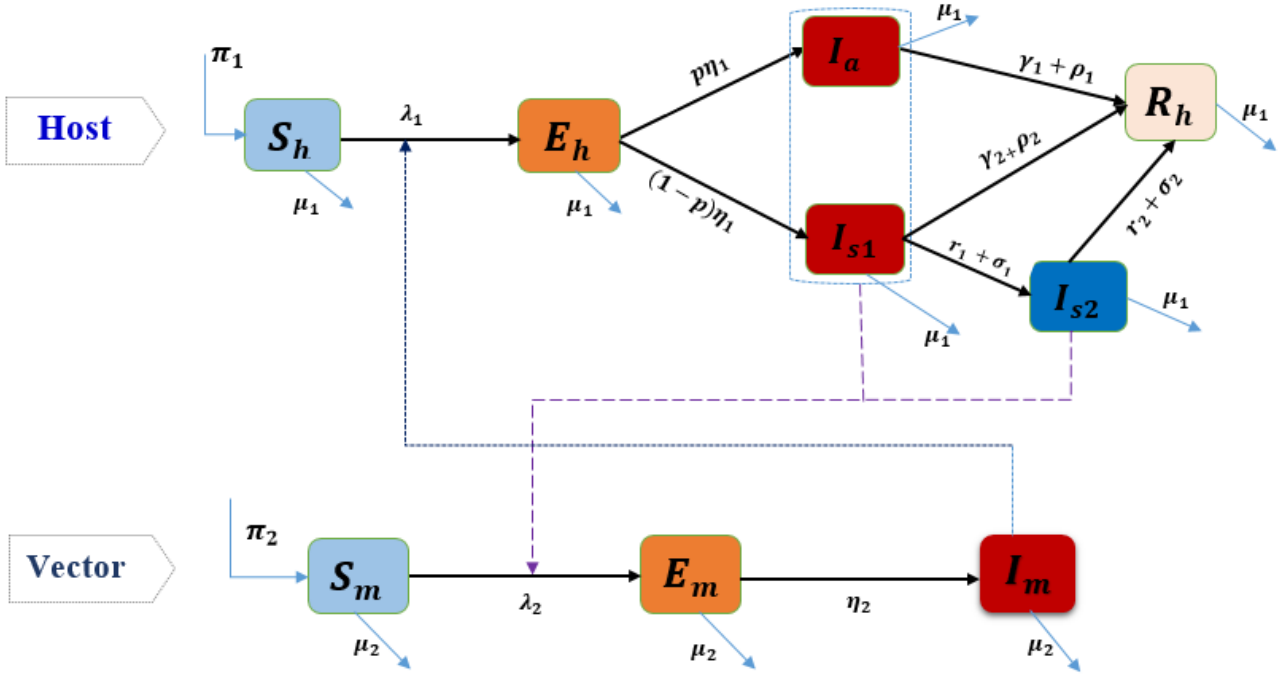


Figure 2.1: ***Schematic diagram for the chikungunya virus transmission.*** *The population of humans is divided into susceptible (S_h), exposed (E_h) to the disease, asymtomatically infectious (I_a), symptomatically infectious (I_{s_1}) in acute phase, infected in sub-acute phase (I_{s_2}) and recovered (R_h) from chikungunya. The mosquitoes population is divided into susceptible (S_m), exposed (E_m) and infectious (I_m). The black heavy arrows indicate the disease transition from one compartment to the other compartment and dashed arrows indicate the contacts between hosts and vector.*

differential equations to describe the transmission dynamics of chikungunya virus:

$$\begin{aligned}
\frac{dS_h}{dt} &= \pi_1 - \lambda_1 S_h - \mu_1 S_h \\
\frac{dE_h}{dt} &= \lambda_1 S_h - \eta_1 E_h - \mu_1 E_h \\
\frac{dI_a}{dt} &= p\eta_1 E_h - \gamma_1 I_a - \rho_1 I_a - \mu_1 I_a \\
\frac{dI_{s1}}{dt} &= (1-p)\eta_1 E_h - \gamma_2 I_{s1} - \rho_2 I_{s1} - r_1 I_{s1} - \sigma_1 I_{s1} - \mu_1 I_s \\
\frac{dI_{s2}}{dt} &= r_1 I_{s1} + \sigma_1 I_{s1} - r_2 I_{s2} - \sigma_2 I_{s2} - \mu_1 I_{s2} \\
\frac{dR_h}{dt} &= \gamma_1 I_a + \rho_1 I_a + \gamma_2 I_{s1} + \rho_2 I_{s1} + r_2 I_{s2} + \sigma_2 I_{s2} - \mu_1 R_h \\
\frac{dS_m}{dt} &= \pi_2 - \lambda_2 S_m - \mu_2 S_m \\
\frac{dE_m}{dt} &= \lambda_2 S_m - \eta_2 E_m - \mu_2 E_m \\
\frac{dI_m}{dt} &= \eta_2 E_m - \mu_2 I_m \\
S_h(0) = S_{h0}, E_h(0) &= E_{h0}, I_a(0) = I_{a0}, I_{s1}(0) = I_{s10}, I_{s2}(0) = I_{s20}, \\
R_h(0) = R_{h0}, S_m(0) &= S_{m0}, E_m(0) = E_{m0}, I_m(0) = I_{m0},
\end{aligned} \tag{2.6}$$

where, λ_1 and λ_2 are given in equation (2.4) and (2.5) respectively.

The schematic diagram of the model is depicted in Figure 2.1, and the description of the associated param-

eters of the model (2.6) is given in Table 2.1.

Table 2.1: The variables and parameters of the model (2.6) with description

Variables and Parameters	Description
S_h	Susceptible human population
E_h	Exposed human population
I_a	Asymptomatically infectious human population
I_{s1}	Symptomatically infectious human population in acute phase
I_{s2}	Infected humans population in sub-acute phase
R_h	Recovered human population
S_m	Susceptible mosquito population
E_m	Exposed mosquito population
I_m	Infectious mosquito population
π_1	Recruitment rate Human population
μ_1	Natural death rate of human population
π_2	Recruitment rate of mosquito population
μ_2	Natural mortality rate of mosquito population
β_1	Transmission probability per bite from I_m to S_h
β_{12}	Transmission probability per bite from both I_a and I_{s1} to S_m
β_{22}	Transmission probability per bite from I_{s2} to S_m
b_m	Daily mosquito biting rate
λ_1	Infection rate for human population
λ_2	Infection rate for mosquito population
η_1	Progression rate of exposed human population
η_2	Progression rate of exposed mosquito population
r_1	Progression rate from I_{s1} to I_{s2}
γ_1	Recovery rate of human population from the class I_a
γ_2	Recovery rate of human population from the class I_{s1}
r_2	Recovery rate of human population from the class I_{s2}
$\rho_1, \rho_2, \sigma_1, \sigma_2$	Treatment rate
p	Fraction of exposed human who do not develop symptoms
$1-p$	Fraction of exposed human who develop symptoms

3 Analysis of the model

3.1 Properties of the model

Here we prove some basic qualitative properties of solutions to the model (2.6), such as positivity and boundedness of the solutions. These properties can shown by the following lemma:

Lemma 1. *The region,* $\Omega = \{(S_h, E_h, I_a, I_{s1}, I_{s2}, R_h, S_m, E_m, I_m) : S_h + E_h + I_a + I_{s1} + I_{s2} + R_h \leq \frac{\pi_1}{\mu_1}, S_m + E_m + I_m \leq \frac{\pi_2}{\mu_2}\} \subset \mathsf{R}^9_+$, *is positively invariant and attracting for the basic model* (2.6).

Proof. The rate of change of the human population and mosquito population are obtained by adding the first six equations and last three equations of the model respectively (2.6) as follows

$$\frac{dN_h(t)}{dt} = \pi_1 - \mu_1 N_h(t)$$

$$\frac{dN_m(t)}{dt} = \pi_2 - \mu_2 N_m(t)$$

Thus, we see that $\frac{dN_h(t)}{dt} < 0$ if $N_h(t) > \pi_1/\mu_1$ and $\frac{dN_m(t)}{dt} < 0$ if $N_m(t) > \pi_2/\mu_2$. Also by using a standard comparison theorem [44], it can be shown that $N_h(t) = N_h(0)e^{-\mu_1 t} + (\pi_1/\mu_1)(1 - e^{-\mu_1 t})$ and $N_m(t) = N_m(0)e^{-\mu_2 t} + (\pi_2/\mu_2)(1 - e^{-\mu_2 t})$. In particular, $N_h(t) < \pi_1/\mu_1$ if $N_h(0) < \pi_1/\mu_1$ and $N_m(t) < \pi_2/\mu_2$ if $N_m(0) < \pi_2/\mu_2$ .Thus, Ω is positively invariant. Further, $N_h(t) > \pi_1/\mu_1$ and $N_m(t) > \pi_2/\mu_2$, then either the

solution enters Ω in finite time, or $N_h(t)$ approaches π_1/μ_1 and $N_m(t)$ approaches π_2/μ_2, and the variables $E_h(t), I_a(t), I_{s1}(t), I_{s2}(t), R_h(t), E_m(t)$ and $I_m(t)$ approach zero. Hence, Ω is attracting. Thus, the model (2.6) is well-posed in Ω epidemiologically and mathematically [30] and it is sufficient to study the dynamics of the model (2.6) in Ω. □

3.2 Equilibrium points and stability analysis

The model (2.6) may have two types of equilibrium, namely disease free equilibrium (*DFE*) and endemic equilibrium (*EE*). At any equilibrium, we set $\frac{dS_h}{dt} = 0, \frac{dE_h}{dt} = 0, \frac{dI_a}{dt} = 0, \frac{dI_{s1}}{dt} = 0, \frac{dI_{s2}}{dt} = 0, \frac{dR_h}{dt} = 0$, and obtain the following relations

$$\begin{aligned}
I_a &= \frac{p\eta_1}{k_5}E_h \\
I_{s1} &= \frac{(1-p)\eta_1}{k_7}E_h \\
I_{s2} &= \frac{\eta_1(1-p)k_1}{k_6k_7}E_h \\
R_h &= \frac{\eta_1(1-p)(k_1k_2k_5 + k_4k_5k_6) + \eta_1pk_3k_6k_7}{\mu_1k_5k_6k_7}E_h \\
S_h &= N_h - \frac{a}{k_5k_6k_7\mu_1}E_h \\
E_h &= \frac{\beta_1b_m\mu_1k_5k_6k_7I_mN_h}{k_5k_6k_7\mu_1(\mu_1+\eta_1)N_h + a\beta_1b_mI_m},
\end{aligned} \tag{3.1}$$

where, $k_1 = \sigma_1 + r_1$, $k_2 = \sigma_2 + r_2$, $k_3 = \gamma_1 + \rho_1$, $k_4 = \gamma_2 + \rho_2$, $k_5 = \gamma_1 + \rho_1 + \mu_1$, $k_6 = r_2 + \sigma_2 + \mu_1$,$k_7 = \gamma_2 + \rho_2 + \sigma_1 + r_1 + \mu_1$,
$a = k_5k_6k_7\mu_1 + k_5k_6\eta_1(1-p)(k_4+\mu_1) + k_1k_5\eta_1(1-p)(k_2+\mu_1+k_6k_7\eta_1p(k_3+\mu_1)$.

Also by setting $\frac{dS_m}{dt} = 0, \frac{dE_m}{dt} = 0, \frac{dI_m}{dt} = 0$, we have the following relations

$$\begin{aligned}
S_m &= \frac{\pi_2}{\lambda_2+\mu_2} \\
E_m &= \frac{\pi_2\lambda_2}{(\eta_2+\mu_2)(\lambda_2+\mu_2)} \\
I_m &= \frac{\pi_2\eta_2\lambda_2}{\mu_2(\eta_2+\mu_2)(\lambda_2+\mu_2)}
\end{aligned} \tag{3.2}$$

3.3 Stability of disease free equilibrium (*DFE*)

DFE of the model (2.6) is given by

$$\bar{E}_0 = (S_h, E_h, I_a, I_{s1}, I_{s2}, R_h, S_m, E_m, I_m) = \left(\frac{\pi_1}{\mu_1}, 0, 0, 0, 0, 0, \frac{\pi_2}{\mu_2}, 0, 0\right)$$

3.3.1 Local stability of *DFE*

We investigate the local stability of the *DFE* by using the next generation matrix of the system (2.6). Now, we calculate the basic reproduction number of the model (2.6) according to [49]. Consider the compartments

which are related to the infection to obtain the following subsystem

$$\begin{aligned}
\frac{dE_h}{dt} &= \lambda_1 S_h - \eta_1 E_h - \mu_1 E_h \\
\frac{dI_a}{dt} &= p\eta_1 E_h - \gamma_1 I_a - \rho_1 I_a - \mu_1 I_a \\
\frac{dI_{s1}}{dt} &= (1-p)\eta_1 E_h - \gamma_2 I_{s1} - \rho_2 I_{s1} - r_1 I_{s1} - \sigma_1 I_{s1} - \mu_1 I_{s1} \\
\frac{dI_{s2}}{dt} &= r_1 I_{s1} + \sigma_1 I_{s1} - r_2 I_{s2} - \sigma_2 I_{s2} - \mu_1 I_{s2} \\
\frac{dE_m}{dt} &= \lambda_2 S_m - \eta_2 E_m - \mu_2 E_m \\
\frac{dI_m}{dt} &= \eta_2 E_m - \mu_2 I_m.
\end{aligned} \tag{3.3}$$

From the subsystem (3.3), we find the following transmission matrix F (associated with new infection terms) and transition matrix V (considering transferred terms):

$$F = \begin{bmatrix}
0 & 0 & 0 & 0 & 0 & \frac{{}_1 b_m S_h}{N_h} \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & \frac{{}_{12} b_m S_m}{N_h} & \frac{{}_{12} b_m S_m}{N_h} & \frac{{}_{22} b_m S_m}{N_h} & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0
\end{bmatrix}$$

and

$$V = \begin{bmatrix}
\eta_1 + \mu_1 & 0 & 0 & 0 & 0 & 0 \\
-p\eta_1 & k_5 & 0 & 0 & 0 & 0 \\
-(1-p)\eta_1 & 0 & k_7 & 0 & 0 & 0 \\
0 & 0 & -k_1 & k_6 & 0 & 0 \\
0 & 0 & 0 & 0 & \eta_2 + \mu_2 & 0 \\
0 & 0 & 0 & 0 & -\eta_2 & \mu_2
\end{bmatrix},$$

where, $k_1 = r_1 + \sigma_1, k_5 = \gamma_1 + \rho_1 + \mu_1, k_6 = r_2 + \sigma_2 + \mu_1, k_7 = \gamma_2 + \rho_2 + r_1 + \sigma_1 + \mu_1$.
The basic reproduction number [49] is the spectral radius of the matrix FV^{-1}, that is, $\Re_0 = \rho(FV^{-1})$.

Now, the eigenvalues of FV^{-1} are
$0, 0, 0, 0, \frac{b_m \sqrt{{}_1{}_1{}_2 f\ {}_{12} k_5 k_6 (1-p) + {}_{12} k_6 k_7 p + {}_{22} k_1 k_5 (1-p) g S_h S_m}}{\sqrt{k_5 k_6 k_7\ {}_2(\ {}_1+\ {}_1)(\ {}_2+\ {}_2) N_h}}$ and $-\frac{b_m \sqrt{{}_1{}_1{}_2 f\ {}_{12} k_5 k_6 (1-p) + {}_{12} k_6 k_7 p + {}_{22} k_1 k_5 (1-p) g S_h S_m}}{\sqrt{k_5 k_6 k_7\ {}_2(\ {}_1+\ {}_1)(\ {}_2+\ {}_2)} N_h}$.
Therefore, it follows that the basic reproduction number of the model is

$$\Re_0 = \frac{b_m \sqrt{\beta_1 \eta_1 \eta_2 \{\beta_{12} k_5 k_6 (1-p) + \beta_{12} k_6 k_7 p + \beta_{22} k_1 k_5 (1-p)\} S_m S_h}}{\sqrt{k_5 k_6 k_7 \mu_2 (\mu_1 + \eta_1)(\mu_2 + \eta_2)} N_h}$$

According to [49], we know that if $\Re_0 < 1$ then the *DFE* is locally asymptotically stable, that is the disease will not persist in the community; whereas if $\Re_0 > 1$, then it is unstable and the disease will be spread out. Thus we have the following result.

Theorem 1. *The DFE, $\bar{E}_0$, of the model (2.6) is locally asymptotically stable if $\Re_0 < 1$ and unstable if $\Re_0 > 1$.*

The basic reproduction number, $\Re_0$, is the average number of new cases produced by a single infected individual in a population that is totally susceptible. Thus, from the above Theorem (1), chikungunya will be eliminated from the community when $\Re_0 < 1$.

3.3.2 Global stability of *DFE*

Before to prove the global stability of *DFE*, we consider the region,

$$\Omega = \{(S_h, E_h, I_a, I_{s1}, I_{s2}, R_h, S_m, E_m, I_m) \in \Omega : S_h \le S_h, S_m \le S_m\}$$

and prove the following lemma:

Lemma 2. *The region* Ω *is positively invariant and attracting for the model* (2.6).

Proof. From the first equation of the model (2.6), where, $S_h = \pi_1/\mu_1$, it follows that

$$\begin{aligned}\frac{dS_h}{dt} &= \pi_1 - \lambda_1 S_h(t) - \mu_1 S_h(t)\\ &\leq \pi_1 - \mu_1 S_h(t)\\ &= \mu_1[\pi_1/\mu_1 - S_h(t)]\\ &= \mu_1[S_h - S_h(t)]\end{aligned}$$

Hence, we have

$$S_h(t) \leq S_h - [S_h - S_h(0)]e^{\ {}_1 t}$$

Thus, if $N_h(t) \leq \pi_1/\mu_1$ and $S_h(0) \leq \pi_1/\mu_1$, then it follows that either $S_h(t) \to S_h$ as $t \to \infty$, or after finite time $S_h(t) \leq S_h$, since $\frac{dS_h}{dt} < 0$ for $S_h(t) > S_h$.

Finally, it follows from the seventh equation of the model (2.6), where, $S_m = \pi_2/\mu_2$, it follows that

$$\begin{aligned}\frac{dS_m}{dt} &= \pi_2 - \lambda_2 S_m(t) - \mu_2 S_m(t)\\ &\leq \pi_2 - \mu_2 S_m(t)\\ &= \mu_2[\pi_2/\mu_2 - S_m(t)]\\ &= \mu_2[S_m - S_m(t)]\end{aligned}$$

Thus,

$$S_m(t) \leq S_m - [S_m - S_m(0)]e^{\ {}_2 t}$$

Hence, if $S_m(0) \leq \pi_2/\mu_2$, then either $S_m(t)$ approaches S_m asymptotically, or after some finite time $S_m(t) \leq S_m$, since $\frac{dS_m}{dt} < 0$, if $S_m(t) > S_m$. Therefore, the region Ω is positively invariant and attracts all solutions of the model (2.6) in R^9_+. □

Now, we claim the following:

Theorem 2. *The DFE,* $\bar{E}_0$*, of the model* (2.6) *is globally asymptotically stable in the region* Ω *if* $\Re_0 < 1$.

Proof. We prove the theorem by using Lyapunov function [40, 45, 46] and LaSalle Invariance Principle [47]. Consider the following Lyapunov function

$$\mathcal{F} = f_1 E_h + f_2 I_a + f_3 I_{s1} + f_4 I_{s2} + f_5 E_m + f_6 I_m$$

where,

$$f_1 = \frac{\mu_2 N_h \Re_0}{b_m \beta_1 S_h}, f_2 = \frac{b_m \beta_{12} \eta_2 S_m}{k_5 N_h(\mu_2+\eta_2)\Re_0}, f_3 = \frac{b_m \eta_2 S_m(\beta_{12}k_6 + \beta_{22}k_1)}{k_6 k_7 N_h(\mu_2+\eta_2)\Re_0},$$

$$f_4 = \frac{b_m \eta_2 \beta_{22} S_m}{k_6 N_h(\mu_2+\eta_2)\Re_0}, f_5 = \frac{\eta_2}{\mu_2+\eta_2}, f_6 = 1$$

Now, the time derivative of the Lyapunov function is given by

$$\begin{aligned}\dot{\mathcal{F}} &= f_1\dot{E_h} + f_2\dot{I_a} + f_3\dot{I_{s1}} + f_4\dot{I_{s2}} + f_5\dot{E_m} + f_6\dot{I_m}\\ &= \frac{\mu_2 N_h \Re_0}{b_m \beta_1 S_h}[\lambda_1 S_h - \eta_1 E_h - \mu_1 E_h] + \frac{b_m \beta_{12}\eta_2 S_m}{k_5 N_h(\mu_2+\eta_2)\Re_0}[p\eta_1 E_h - k_5 I_a]\\ &+ \frac{b_m \eta_2 S_m(\beta_{12}k_6+\beta_{22}k_1)}{k_6 k_7 N_h(\mu_2+\eta_2)\Re_0}[(1-p)\eta_1 E_h - k_7 I_{s1}] + \frac{b_m \eta_2 \beta_{22} S_m}{k_6 N_h(\mu_2+\eta_2)\Re_0}[k_1 I_{s1} - k_6 I_{s2}]\\ &+ \frac{\eta_2}{\mu_2+\eta_2}[\lambda_2 S_m - \eta_2 E_m - \mu_2 E_m] + \eta_2 E_m - \mu_2 I_m\\ &= \frac{b_m \eta_2 \beta_{12} S_m}{N_h(\mu_2+\eta_2)\Re_0}(\Re_0 - 1)I_a + \frac{b_m \eta_2 \beta_{12} S_m}{N_h(\mu_2+\eta_2)\Re_0}(\Re_0 - 1)I_{s1}\\ &+ \frac{b_m \eta_2 \beta_{12} S_m}{N_h(\mu_2+\eta_2)\Re_0}(\Re_0 - 1)I_{s2} + \mu_2(\Re_0 - 1)I_m\end{aligned}$$

Thus, $\dot{\mathcal{F}} < 0$ if $\Re_0 < 1$ and $\dot{\mathcal{F}} = 0$ if and only if $I_a = I_{s1} = I_{s2} = I_m = 0$. It follows, from the LaSalle Invariance Principle [47] , that $E_h \to 0, I_a \to 0, I_{s1} \to 0, I_{s2} \to 0, E_m \to 0$ and $I_m \to 0$ as $t \to \infty$. That is, the disease will be eliminated. Now, from the first and seventh equations of the model with $E_h = I_a = I_{s1} = I_{s2} = R_h = E_m = I_m = 0$, it follows that $S_h \to S_h$ and $S_m \to S_m$ as $t \to \infty$.
Thus, $\lim_{t\,!\,1}(S_h, E_h, I_a, I_{s1}, I_{s2}, R_h, S_m, E_m, I_m) = (S_h, 0, 0, 0, 0, 0, S_m, 0, 0) = \bar{E}_0$ for $\Re_0 \leq 1$. Therefore, the *DFE*, $\bar{E}_0$, is *GAS* in Ω if $\Re_0 \leq 1$. □

Epidemiological significance of the above theorem is that the chikungunya disease will be eliminated permanently from the community if we can reduce the threshold quantity ($\Re_0$) to less than one. The convergence of the total number of infected human population and mosquito population is shown in Figure 3.1 whenever the quantity $\Re_0 < 1$.

3.4 Stability of Endemic Equilibrium (*EE*)

3.4.1 Existence of Endemic Equilibrium

In this section, we find the condition for the existence of endemic equilibrium. Let the endemic equilibrium point of the model (2.6) be

$$\bar{E}_1 = (S_h\ , E_h\ , I_a\ , I_{s1}, I_{s2}, R_h\ , S_m\ , E_m\ , I_m\ ),$$

and at the *EE*, the expressions in (3.1) and (3.2) become

$$\begin{aligned}
S_h &= \frac{\pi_1}{\lambda_1\ + \mu_1} \\
E_h &= \frac{\pi_1\lambda_1}{(\lambda_1\ + \mu_1)(\eta_1 + \mu_1)} \\
I_a &= \frac{p\pi_1\eta_1\lambda_1}{k_5(\lambda_1\ + \mu_1)(\eta_1 + \mu_1)} \\
I_{s1} &= \frac{(1-p)\pi_1\eta_1\lambda_1}{k_7(\lambda_1\ + \mu_1)(\eta_1 + \mu_1)} \\
I_{s2} &= \frac{(1-p)\pi_1\eta_1\lambda_1\ k_1}{k_6k_7(\lambda_1\ + \mu_1)(\eta_1 + \mu_1)} \\
R_h &= \frac{\pi_1\eta_1\lambda_1\ [k_5(1-p)(k_1k_2 + k_4k_6) + pk_3k_6k_7]}{k_5k_6k_7\mu_1(\lambda_1\ + \mu_1)(\eta_1 + \mu_1)}
\end{aligned}$$

$$\begin{aligned}
S_m &= \frac{\pi_2}{\lambda_2\ + \mu_2} \\
E_m &= \frac{\pi_2\lambda_2}{(\lambda_2\ + \mu_2)(\eta_2 + \mu_2)} \\
I_m &= \frac{\pi_2\eta_2\lambda_2}{\mu_2(\lambda_2\ + \mu_2)(\eta_2 + \mu_2)}
\end{aligned}$$

The forces of infection at the *EE* state are

$$\lambda_1\ = \frac{\beta_1 b_m I_m}{N_h}$$

and

$$\lambda_2\ = \frac{\beta_{12} b_m (I_a\ + I_{s1}) + b_m\beta_{22}I_{s2}}{N_h}$$

Now, substituting the expression of $I_a\ , I_{s1}, I_{s2}$ in the expression of λ_2 , we have

$$\begin{aligned}
\lambda_2\ &= \frac{b_m\beta_{12}I_a\ + b_m\beta_{12}I_{s1} + b_m\beta_{22}I_{s2}}{N_h} \\
&= \frac{b_m\pi_1\eta_1\lambda_1\ [k_5(1-p)(k_1\beta_{22} + k_6\beta_{12}) + pk_6k_7\beta_{12}]}{k_5k_6k_7(\lambda_1\ + \mu_1)(\eta_1 + \mu_1)N_h}
\end{aligned}$$

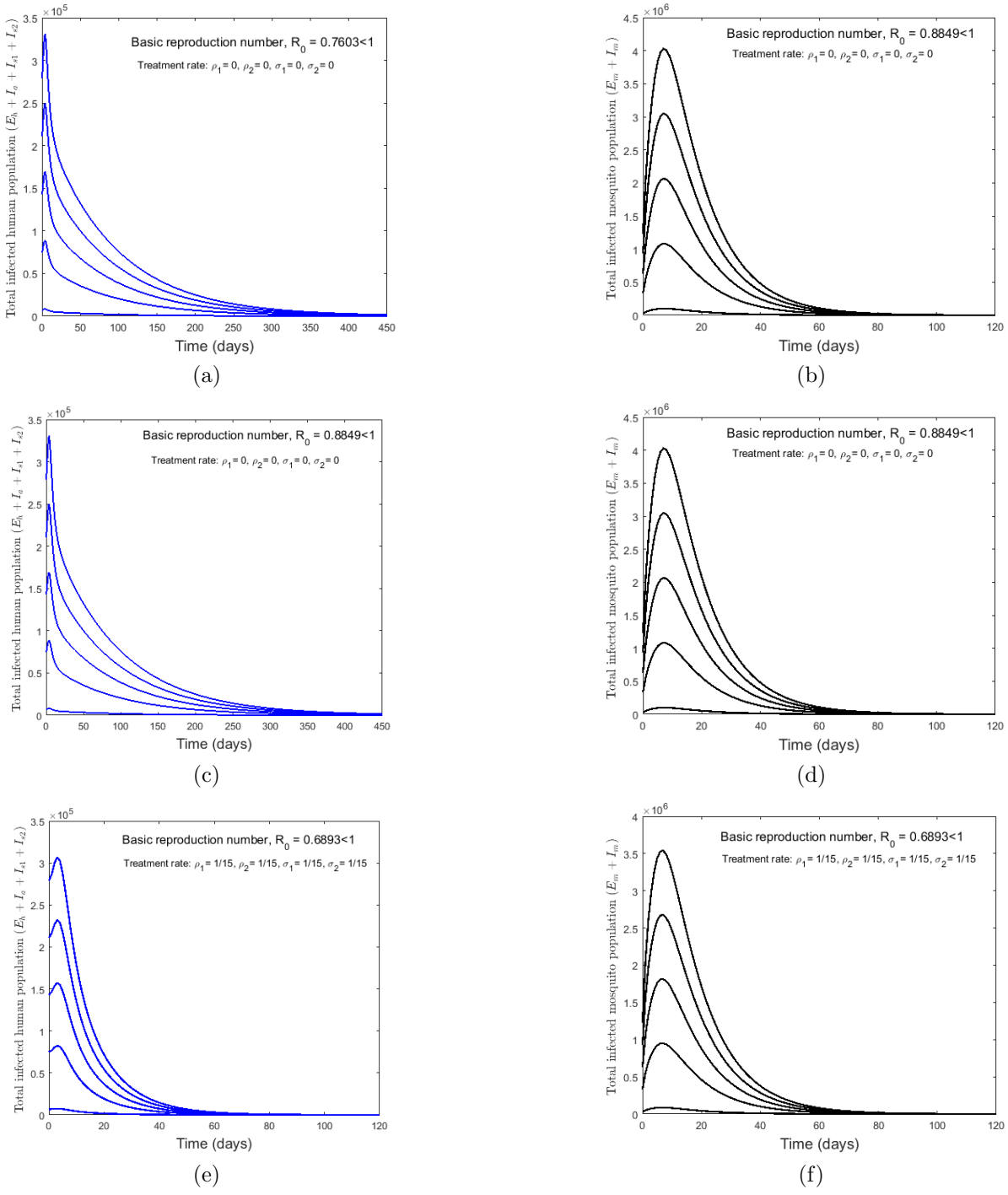


Figure 3.1: Simulations of the model (2.6) showing the total number of infected human population ($E_h + I_a + I_{s1} + I_{s2}$) as a function of time when $\Re_0 < 1$, using various initial conditions. Parameter values used as given in Table 4.2 with $\pi_1 = 1.2, \pi_2 = 3800$; (a) $\beta_{22} = 0$, without treatment; (c) $\beta_{22} = 0.005$, without treatment; (e) $\beta_{22} = 0.005$, with treatment. And (b), (d), (f) depict the corresponding total number of infected mosquito population ($E_m + I_m$).

Also, we have

$$\begin{aligned}\lambda_1 &= \frac{b_m \beta_1 I_m}{N_h} \\ &= \frac{b_m^2 \pi_1 \pi_2 \eta_1 \eta_2 \beta_1 \lambda_1 \ [k_5(1-p)(k_1\beta_{22} + k_6\beta_{12}) + pk_6k_7\beta_{12}]/[\mu_2(\mu_2+\eta_2)N_h \ ]}{b_m \pi_1 \eta_1 \lambda_1 \ [k_5(1-p)(k_1\beta_{22} + k_6\beta_{12}) + pk_6k_7\beta_{12}] + k_5k_6k_7\mu_2(\lambda_1 \ + \mu_1)(\eta_1+\mu_1)N_h}\end{aligned}$$

From where, we find the following quadratic equation

$$c_1(\lambda_1\ )^2 - c_2(\lambda_1\ ) = 0,$$

where,

$$c_1 = \mu_2(\mu_2+\eta_2)N_h\ [\beta_{12}b_m k_6 k_7 \eta_1 p\pi_1 + \pi_1\eta_1 b_m k_5(1-p)(k_1\beta_{22}+k_6\beta_{12}) + k_5k_6\mu_2(\mu_1+\eta_1)N_h\ ]$$

and

$$\begin{aligned} c_2 &= b_m^2\pi_1\pi_2\eta_1\eta_2\beta_1[k_5(1-p)(k_1\beta_{22}+k_6\beta_{12}) + pk_6k_7\beta_{12}] - k_5k_6k_7\mu_1\mu_2^2(\mu_1+\eta_1)(\mu_2+\eta_2)(N_h\ )^2 \\ &= k_5k_6k_7\mu_1\mu_2^2(\mu_1+\eta_1)(\mu_2+\eta_2)(N_h\ )^2 \\ &\quad \left[\frac{b_m^2\pi_1\pi_2\eta_1\eta_2\beta_1[k_5(1-p)(k_1\beta_{22}+k_6\beta_{12}) + pk_6k_7\beta_{12}]}{k_5k_6k_7\mu_1\mu_2^2(\mu_1+\eta_1)(\mu_2+\eta_2)(N_h\ )^2} - 1\right] \\ &= k_5k_6k_7\mu_1\mu_2^2(\mu_1+\eta_1)(\mu_2+\eta_2)N_h^2(\Re_0^2-1) \end{aligned}$$

Therefore, the quadratic equation has a positive real root if $\Re_0^2 > 1$.

According to the above result, we claim the following:

Lemma 3. *The model (2.6) has a unique endemic equilibrium whenever $\Re_0 > 1$, and no positive equilibrium otherwise.*

3.4.2 Local Stability of *EE*

Using $N_h = N_h\ $, $N_m = N_m\ $ and the definition $S_h = N_h\ - E_h - I_a - I_{s1} - I_{s2} - R_h$ and $S_m = N_m\ - E_m - I_m$, we have the following reduced system

$$\begin{aligned} \frac{dE_h}{dt} &= \frac{b_m\beta_1 I_m}{N_h}(N_h\ - E_h - I_a - I_{s1} - I_{s2} - R_h) - \eta_1 E_h - \mu_1 E_h \\ \frac{dI_a}{dt} &= p\eta_1 E_h - k_5 I_a \\ \frac{dI_{s1}}{dt} &= (1-p)\eta_1 E_h - k_7 I_{s1} \\ \frac{dI_{s2}}{dt} &= k_1 I_{s1} - k_6 I_{s2} \\ \frac{dR_h}{dt} &= k_3 I_a + k_4 I_{s1} + k_2 I_{s2} - \mu_1 R_h \\ \frac{dE_m}{dt} &= \frac{b_m\beta_{12}I_a + b_m\beta_{12}I_{s1} + b_m\beta_{22}I_{s2}}{N_h}(N_m\ - E_m - I_m) - \eta_2 E_m - \mu_2 E_m \\ \frac{dI_m}{dt} &= \eta_2 E_m - \mu_2 I_m \end{aligned} \tag{3.4}$$

It is easy to show that the system (3.4) has a unique *EE* of the form $\bar{E}_1 = (E_h\ , I_a\ , I_{s1}, I_{s2}, R_h\ , E_m, I_m\ )$. Now, we prove the local stability of *EE* following the method given in [42], which is based on using Krasnoselskii sub-linearity trick [48]. Assume that the reduced system has a solution of the form

$$\bar{Z}(t) = \bar{Z}_0 e^{\ wt} \tag{3.5}$$

with $\bar{Z}_0 = (Z_1, Z_2, Z_3, Z_4, Z_5, Z_6, Z_7)$ and $w, Z_i \in \mathsf{C}(i = 1, 2, ..., 7)$, where, C denotes the set of complex numbers. Substituting a solution of the form (3.5) into the linearized system of (3.4) around the endemic equilibrium, we have the following system of linear equations

$$
\begin{aligned}
wZ_1 &= -\left(\frac{b_m\beta_1 I_m}{N_h}+\eta_1+\mu_1\right)Z_1-\frac{b_m\beta_1 I_m}{N_h}Z_2-\frac{b_m\beta_1 I_m}{N_h}Z_3-\frac{b_m\beta_1 I_m}{N_h}Z_4\\
&\quad-\frac{b_m\beta_1 I_m}{N_h}Z_5+\frac{b_m\beta_1(N_h\ -N_h\ -I_{s1}\ -I_{s2}\ -R\ \ )}{N_h}Z_7\\
wZ_2 &= p\eta_1 Z_1-k_5Z_2\\
wZ_3 &= (1-p)\eta_1 Z_1-k_7Z_3\\
wZ_4 &= k_1Z_3-k_6Z_4\\
wZ_5 &= k_3Z_2+k_4Z_3+k_2Z_4-\mu_1Z_5\\
wZ_6 &= \frac{b_m\beta_{12}(N_m\ -E_m\ -I_m\ )}{N_h}Z_2+\frac{b_m\beta_{12}(N_m\ -E_m\ -I_m\ )}{N_h}Z_3\\
&\quad+\frac{b_m\beta_{22}(N_m\ -E_m\ -I_m\ )}{N_h}Z_4-\left(\frac{b_m\beta_{12}I_a\ +b_m\beta_{12}I_{s1}+b_m\beta_{22}I_{s2}}{N_h}+\eta_2+\mu_2\right)Z_6\\
&\quad-\frac{b_m\beta_{12}I_a\ +b_m\beta_{12}I_{s1}+b_m\beta_{22}I_{s2}}{N_h}Z_7\\
wZ_7 &= \eta_2Z_6-\mu_2Z_7
\end{aligned}
\tag{3.6}
$$

Solving the second, third and fourth equations for Z_2, Z_3, Z_4 in terms of Z_1 and substituting into the first equation, solving sixth equation for Z_7 substituting into the fifth equation and simplifying we obtain, the following equivalent system

$$
\begin{aligned}
&\left[1+\frac{1}{\eta_1+\mu_1}\left(w+\frac{b_m\beta_1 I_m}{N_h}\left[1+\frac{p\eta_1}{w+k_5}+\frac{(1-p)\eta_1}{w+k_7}+\frac{k_1(1-p)\eta_1}{(w+k_6)(w+k_7)}\right]\right)\right]Z_1\\
&=-\frac{b_m\beta_1 I_m}{N_h}Z_5+\frac{b_m\beta_1(N_h\ -N_h\ -I_{s1}\ -I_{s2}\ -R_h\ )}{N_h}Z_7\\
&\left(1+\frac{w}{k_5}\right)Z_2=\frac{p\eta_1}{k_5}Z_1\\
&\left(1+\frac{w}{k_7}\right)Z_3=\frac{(1-p)\eta_1}{k_7}Z_1\\
&\left(1+\frac{w}{k_6}\right)Z_4=\frac{k_1}{k_6}Z_3\\
&\left(1+\frac{w}{\mu_1}\right)Z_5=\frac{k_3}{\mu_1}Z_2+\frac{k_4}{\mu_1}Z_3+\frac{k_2}{\mu_1}Z_4\\
&Z_6+\frac{1}{\eta_2+\mu_2}\left(w+\frac{b_m\beta_{12}I_a\ +b_m\beta_{12}I_{s1}+b_m\beta_{22}I_{s2}}{N_h}\right)Z_6\\
&+\frac{1}{\eta_2+\mu_2}\left(\frac{\eta_2}{w+\mu_2}\frac{b_m\beta_{12}I_a\ +b_m\beta_{12}I_{s1}+b_m\beta_{22}I_{s2}}{N_h}\right)Z_6\\
&=\frac{b_m\beta_{12}(N_m\ -E_m\ -I_m\ )}{N_h}Z_2+\frac{b_m\beta_{12}(N_m\ -E_m\ -I_m\ )}{N_h}Z_3\\
&+\frac{b_m\beta_{22}(N_m\ -E_m\ -I_m\ )}{N_h}Z_4\\
&\left(1+\frac{w}{\mu_2}\right)Z_7=\frac{\eta_2}{\mu_2}Z_6
\end{aligned}
\tag{3.7}
$$

Adding the first and fifth equation of the system (3.7), and moving all the negative terms to their respective left-hand side to give

$$\begin{aligned}
[1+F_1(w)]\,Z_1 + [1+F_5(w)]Z_5 &= (M\bar{Z})_1 + (M\bar{Z})_5\\
[1+F_2(w)]Z_2 &= (M\bar{Z})_2\\
[1+F_3(w)]Z_3 &= (M\bar{Z})_3\\
[1+F_4(w)]Z_4 &= (M\bar{Z})_4\\
[1+F_6(w)]Z_6 &= (M\bar{Z})_6\\
[1+F_7(w)]Z_7 &= (M\bar{Z})_7,
\end{aligned} \tag{3.8}$$

where,

$$\begin{aligned}
F_1(w) &= \frac{1}{\eta_1+\mu_1}\left(w + \frac{b_m\beta_1 I_m}{N_h}\left[1 + \frac{p\eta_1}{w+k_5} + \frac{(1-p)\eta_1}{w+k_7} + \frac{k_1(1-p)\eta_1}{(w+k_6)(w+k_7)}\right]\right)\\
F_2(w) &= \frac{w}{k_5}\\
F_3(w) &= \frac{w}{k_7}\\
F_4(w) &= \frac{w}{k_6}\\
F_5(w) &= \frac{w}{\mu_1} + \frac{b_m\beta_1 I_m}{(\eta_1+\mu_1)N_h}\\
F_6(w) &= \frac{1}{\eta_2+\mu_2}\left(w + \frac{b_m\beta_{12}I_a + b_m\beta_{12}I_{s1} + b_m\beta_{22}I_{s2}}{N_h}\right)\\
&\quad + \frac{\eta_2}{(w+\mu_2)(\eta_2+\mu_2)}\frac{b_m\beta_{12}I_a + b_m\beta_{12}I_{s1} + b_m\beta_{22}I_{s2}}{N_h}\\
F_7(w) &= \frac{w}{\mu_2},
\end{aligned}$$

and M is the matrix

$$M = \begin{bmatrix}
0 & 0 & 0 & 0 & 0 & 0 & M_h\\
\frac{p}{k_5}\eta_1 & 0 & 0 & 0 & 0 & 0 & 0\\
\frac{(1-p)}{k_5}\eta_1 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & \frac{k_1}{k_6} & 0 & 0 & 0 & 0\\
0 & \frac{k_3}{1} & \frac{k_4}{1} & \frac{k_2}{1} & 0 & 0 & 0\\
0 & M_m & M_m & M_m & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & \frac{n_2}{2} & 0
\end{bmatrix},$$

where, $M_h = \frac{b_m\ \ 1(N_h\ \ N_h\ \ I_{s1}\ \ I_{s2}\ \ R_h\ )}{N_h}$
and $M_m = \frac{b_m\ \ 12(N_m\ \ E_m\ \ I_m\ )}{N_h}$.

The notation $(M\bar{Z})_i$ $(i = 1, 2, ..., 7)$ denotes the ith coordinate of the vector $M(\bar{Z})$. Note that the matrix M has non-negative entries, and the equilibrium $\bar{E}_1$ satisfies $\bar{E}_1 = M\bar{E}_1$. Moreover, since the coordinates of the equilibrium $\bar{E}_1$ are all positive, it follows then that if $\bar{Z}$ is a solution of (3.8), then it is possible to find a minimal positive real number s, such that

$$|\bar{Z}| \le s\bar{E}_1,$$

where, $|\bar{Z}| = (|Z_1|, |Z_2|, ..., |Z_7|)$ and $|.|$ is the norm in C.

Now, our goal is to show that $Re(w) < 0$. Assume the contrary (i.e., $Re(w) \ge 0$), we consider two cases: $w = 0$ and $w \ne 0$. For the first case $(w = 0)$, the system (3.8) is a homogeneous linear system in the variables $Z_i (i = 1, 2, ..., 7)$. The determinant of this system is given by

$$\begin{aligned}
\Delta &= \frac{\beta_1 b_m I_m\,(\lambda\quad + \mu_2)[k_5k_6k_7(\mu_1+\eta_1) + \beta_1 b_m I_m\,\{k_6k_7(p\eta_1+k_5) + k_5\eta_1(1-p)(k_1+k_6)\}]}{k_5k_6k_7\mu_2(\mu_1+\eta_1)^2N_h}\\
&\quad + \frac{(\mu_1+\eta_1)N_h\ [k_5k_6k_7(\mu_1+\eta_1) + \beta_1 b_m I_m\,\{k_6k_7(p\eta_1+k_5) + k_5\eta_1(1-p)(k_1+k_6)\}]}{k_5k_6k_7\mu_2(\mu_1+\eta_1)^2N_h} > 0
\end{aligned}$$

Thus, the system (3.8) has only trivial solution $\bar{Z} = 0$ for $w = 0$.

Now, we assume that $w \neq 0$ and $Re(w) > 0$. It is easy to see that in this case $|1+F_i(w)| > 1$ for $i = 1, 2, ..., 7$ and we define $F(w) = min\{|1 + F_i(w)|, i = 1, 2, ..., 7\}$, then $F(w) > 1$. Therefore, $\frac{s}{F(w)} < s$. Since s is the minimal positive real number such that $|\bar{Z}| \leq s\bar{E}_1$,so

$$|\bar{Z}| > \frac{s}{F(w)}\bar{E}_1.$$

Taking norms on both sides of (3.8) and using the fact that M is non-negative, we obtain the following inequality

$$F(w)\bar{Z} \leq M\bar{Z} \leq s(M\bar{E}_1) \leq s\bar{E}_1.$$

Thus, it follows that $\bar{Z} \leq \frac{s}{F(w)}\bar{E}_1$, which is a contradiction. Therefore, $Re(w) < 0$ and this proves that the EE, $\bar{E}_1$, is locally asymptotically stable for $\Re_0 > 1$.

3.4.3 Global Stability of *EE*

The global stability of EE of the model (2.6) is considered for a special case, where the ratio $E_h\ /E_h$ equals to $E_m\ /E_m$. From the first six equations of the model (2.6), we see that

$$\begin{aligned} \frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dI_a}{dt} + \frac{dI_{s1}}{dt} + \frac{dI_{s2}}{dt} + \frac{dR_h}{dt} &= \pi_1 - \mu_1 S_h - \mu_1 E_h - \mu_1 I_a - \mu_1 I_{s1} - \mu_1 I_{s2} - \mu_1 R_h \\ or, \frac{dN_h}{dt} &= \pi_1 - \mu_1 N_h, \end{aligned}$$

So, we have $N_h(t) \leq \frac{\pi_1}{\mu_1}$ for all $t \geq 0$. To prove the global stability, we consider $N_h = \frac{\pi_1}{\mu_1}$ for all $t > 0$. It is easy to see that the above assumptions have no effect on the basic reproduction number $\Re_0$ and the existence of unique EE for $\Re_0 > 1$. Now, we claim the following result.

Theorem 3. *The unique EE, $\bar{E}_1$, of the model (2.6), is globally asymptotically stable in Ω for a special case, $E_h\ /E_h = E_m\ /E_m$, whenever $\Re_0 > 1$.*

Proof. Consider the non-linear Lyapunov function

$$\begin{aligned} \mathcal{F} = &\left(S_h - S_h\ - S_h\ ln\frac{S_h}{S_h}\right) + \left(E_h - E_h\ - E_h\ ln\frac{E_h}{E_h}\right) \\ &+ \frac{b_m\beta_{12}S_m}{k_5N_h}\left(I_a - I_a\ - I_a\ ln\frac{I_a}{I_a}\right) + \frac{b_m\beta_{12}S_m}{k_7N_h}\left(I_{s1} - I_{s1}\ - I_{s1}ln\frac{I_{s1}}{I_{s1}}\right) \\ &+ \frac{b_m\beta_{22}k_1S_m}{k_6k_7N_h}\left(I_{s1} - I_{s1}\ - I_{s1}ln\frac{I_{s1}}{I_{s1}}\right) + \frac{b_m\beta_{22}S_m}{k_6N_h}\left(I_{s2} - I_{s2}\ - I_{s2}ln\frac{I_{s2}}{I_{s2}}\right) \\ &+ \left(S_m - S_m\ - S_m\ ln\frac{S_m}{S_m}\right) + \left(E_m - E_m\ - E_m\ ln\frac{E_m}{E_m}\right) \\ &+ \frac{b_m\beta_1S_h}{\mu_2N_h}\left(I_m - I_m\ - I_m\ ln\frac{I_m}{I_m}\right) \end{aligned}$$

and the Lyapunov derivative is given by

$$\begin{aligned} \dot{\mathcal{F}} = &\left(1 - \frac{S_h}{S_h}\right)\dot{S}_h + \left(1 - \frac{E_h}{E_h}\right)\dot{E}_m + \frac{b_m\beta_{12}S_m}{k_5N_h}\left(1 - \frac{I_a}{I_a}\right)\dot{I}_a \\ &+ \left(\frac{b_m\beta_{12}S_m}{k_7N_h} + \frac{b_m\beta_{22}k_1S_m}{k_6k_7N_h}\right)\left(1 - \frac{I_{s1}}{I_{s1}}\right)\dot{I}_{s1} \\ &+ \frac{b_m\beta_{22}S_m}{k_6N_h}\left(1 - \frac{I_{s2}}{I_{s2}}\right)\dot{I}_{s2} + \left(1 - \frac{S_m}{S_m}\right)\dot{S}_m \\ &+ \left(1 - \frac{E_m}{E_m}\right)\dot{E}_m + \frac{b_m\beta_1S_h}{\mu_2N_h}\left(1 - \frac{I_m}{I_m}\right)\dot{I}_m \end{aligned} \tag{3.9}$$

For further simplification, we use the following relations

$$\pi_1 = \frac{b_m \beta_1 S_h \ I_m}{N_h \ + \mu_1 S_h}$$
$$\pi_2 = \frac{b_m \beta_{12} S_m \, I_a}{N_h} + \frac{b_m \beta_{12} S_m \, I_{s1}}{N_h} + \frac{b_m \beta_{22} S_m \, I_{s2}}{N_h} + \mu_2 S_m$$
$$(\eta_1 + \mu_1) E_h \ = \frac{b_m \beta_1 S_h \ I_m}{N_h}$$
$$(\eta_2 + \mu_2) E_m \ = \frac{b_m \beta_{12} S_m \, I_a}{N_h} + \frac{b_m \beta_{12} S_m \, I_{s1}}{N_h} + \frac{b_m \beta_{22} S_m \, I_{s2}}{N_h}$$
$$\frac{p\eta_1}{k_5} = \frac{I_a}{E_h}$$
$$\frac{(1-p)\eta_1}{k_7} = \frac{I_{s1}}{E_h}$$
$$\frac{(1-p)\eta_1 k_1}{k_6 k_7} = \frac{I_{s2}}{E_h}$$
$$\frac{k_1}{k_6} = \frac{I_{s2}}{I_{s1}}$$
$$\frac{\eta_2}{\mu_2} = \frac{I_m}{E_m}$$

Now, from equation (3.9), we obtain the following simplified form

$$\begin{aligned}
\dot{\mathcal{F}} = {} & \frac{b_m \beta_1}{N_h} \left(3 S_h \ I_m \ - \frac{(S_h \ )^2}{S_h} I_m \ - S_h \ \frac{E_m}{E_m} \frac{(I_m \ )^2}{I_m} - S_h \ I_m \ \frac{E_h}{E_h} - S_h I_m \frac{E_h}{E_h} + S_h \ I_m \ \frac{E_m}{E_m} \right) \\
& + \frac{b_m \beta_{12}}{N_h} \left(3 S_m \, I_a \ - \frac{(S_m \ )^2}{S_m} I_a \ - S_m \ \frac{E_h}{E_h} \frac{(I_a \ )^2}{I_a} - S_m \, I_a \ \frac{E_m}{E_m} - S_m I_a \frac{E_m}{E_m} + S_m \, I_a \ \frac{E_h}{E_h} \right) \\
& + \frac{b_m \beta_{12}}{N_h} \left(3 S_m \, I_{s1} - \frac{(S_m \ )^2}{S_m} I_{s1} - S_m \ \frac{E_h}{E_h} \frac{(I_{s1})^2}{I_{s1}} - S_m \, I_{s1} \frac{E_m}{E_m} - S_m I_{s1} \frac{E_m}{E_m} + S_m \, I_{s1} \frac{E_h}{E_h} \right) \\
& + \frac{b_m \beta_{22}}{N_h} \left(4 S_m \, I_{s2} - S_m \, I_{s2} \frac{E_h}{E_h} \frac{I_{s1}}{I_{s2}} - \frac{(S_m \ )^2}{S_m} I_{s2} - S_m \, \frac{I_{s1}}{I_{s1}} \frac{(I_{s2})^2}{I_{s2}} - S_m \, I_{s2} \frac{E_m}{E_m} - S_m I_{s2} \frac{E_m}{E_m} \right) \\
& + \frac{b_m \beta_{22}}{N_h} S_m \, I_{s2} \frac{E_h}{E_h} + \mu_1 S_h \ \left(2 - \frac{S_h}{S_h} - \frac{S_h}{S_h} \right) + \mu_2 S_m \ \left(2 - \frac{S_m}{S_m} - \frac{S_m}{S_m} \right) \\
= {} & \frac{b_m \beta_1 S_h \ I_m}{N_h} \left(3 - \frac{S_h}{S_h} - \frac{E_m}{E_m} \frac{I_m}{I_m} - \frac{S_h}{S_h} \frac{I_m}{I_m} \frac{E_m}{E_m} \right) \\
& + \frac{b_m \beta_{12} S_m \, I_a}{N_h} \left(3 - \frac{S_m}{S_m} - \frac{E_h}{E_h} \frac{I_a}{I_a} - \frac{S_m}{S_m} \frac{I_a}{I_a} \frac{E_h}{E_h} \right) \\
& + \frac{b_m \beta_{12} S_m \, I_{s1}}{N_h} \left(3 - \frac{S_m}{S_m} - \frac{E_h}{E_h} \frac{I_{s1}}{I_{s1}} - \frac{S_m}{S_m} \frac{I_{s1}}{I_{s1}} \frac{E_h}{E_h} \right) \\
& + \frac{b_m \beta_{22} S_m \, I_{s2}}{N_h} \left(4 - \frac{S_m}{S_m} - \frac{E_h}{E_h} \frac{I_{s1}}{I_{s1}} - \frac{I_{s1}}{I_{s1}} \frac{I_{s2}}{I_{s2}} - \frac{S_m}{S_m} \frac{I_{s2}}{I_{s2}} \frac{E_h}{E_h} \right) \\
& + \mu_1 S_h \ \left(2 - \frac{S_h}{S_h} - \frac{S_h}{S_h} \right) + \mu_2 S_m \ \left(2 - \frac{S_m}{S_m} - \frac{S_m}{S_m} \right)
\end{aligned}$$

Since the geometric mean does not exceed the arithmetic mean, it follows that

$$2 - \frac{S_h}{S_h} - \frac{S_h}{S_h} \leq 0, \ 2 - \frac{S_m}{S_m} - \frac{S_m}{S_m} \leq 0 \ 3 - \frac{S_h}{S_h} - \frac{E_m}{E_m} \frac{I_m}{I_m} - \frac{S_h}{S_h} \frac{I_m}{I_m} \frac{E_m}{E_m} \leq 0$$
$$3 - \frac{S_m}{S_m} - \frac{E_h}{E_h} \frac{I_a}{I_a} - \frac{S_m}{S_m} \frac{I_a}{I_a} \frac{E_h}{E_h} \leq 0 \ 3 - \frac{S_m}{S_m} - \frac{E_h}{E_h} \frac{I_{s1}}{I_{s1}} - \frac{S_m}{S_m} \frac{I_{s1}}{I_{s1}} \frac{E_h}{E_h} \leq 0$$
$$4 - \frac{S_m}{S_m} - \frac{E_h}{E_h} \frac{I_{s1}}{I_{s1}} - \frac{I_{s1}}{I_{s1}} \frac{I_{s2}}{I_{s2}} - \frac{S_m}{S_m} \frac{I_{s2}}{I_{s2}} \frac{E_h}{E_h} \leq 0$$

Therefore, we have $\dot{\mathcal{F}} \leq 0$ for $\Re_0 > 1$. Thus, by the lyapunov function $\mathcal{F}$, and the LaSalle Invariance Principle [47], every solution to the equations in the model (2.6) approaches $\bar{E_1}$ as $t \to \infty$ for $\Re_0 > 1$. □

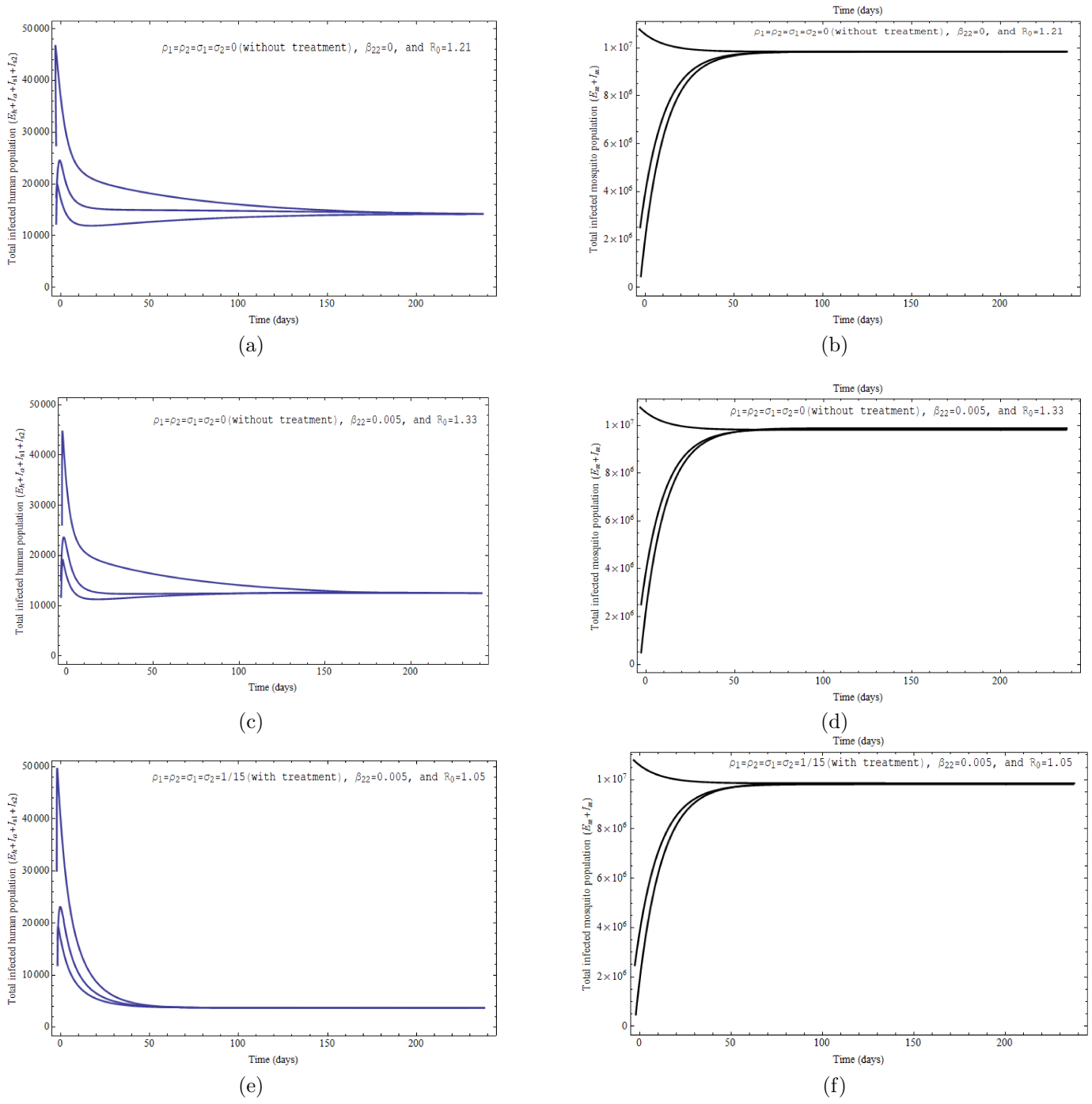


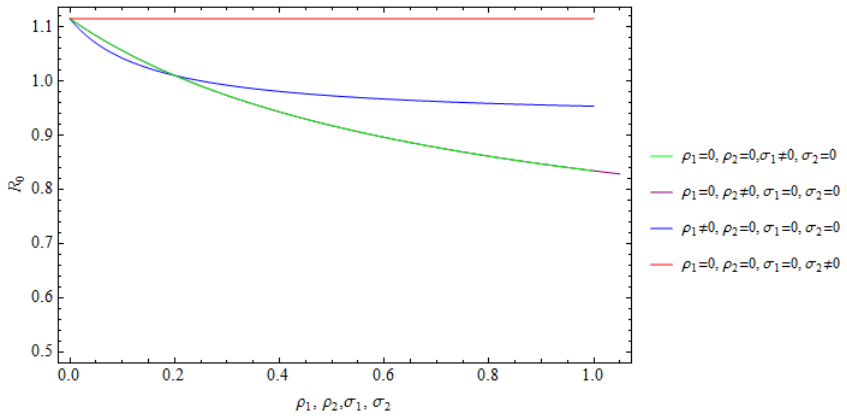
Figure 3.2: Simulations of the model (2.6) showing the total number of infected human population ($E_h + I_a + I_{s1} + I_{s2}$) as a function of time when $\Re_0 > 1$, using various initial conditions. Parameter values used as given in Table 4.2 with $\pi_1 = 332.5; \pi_2 = 75500; b_m = 0.6; p = 0.15; \beta_{12} = 0.35;$ (a) $\beta_{22} = 0$, without treatment, and $\Re_0 = 1.21;$ (c) $\beta_{22} = 0.005$, without treatment, and $\Re_0 = 1.33;$ (e) $\beta_{22} = 0.005$, with treatment, and $\Re_0 = 1.05$. And (b), (d), (f) depict the corresponding total number infected mosquito population ($E_m + I_m$).

4 Numerical simulations

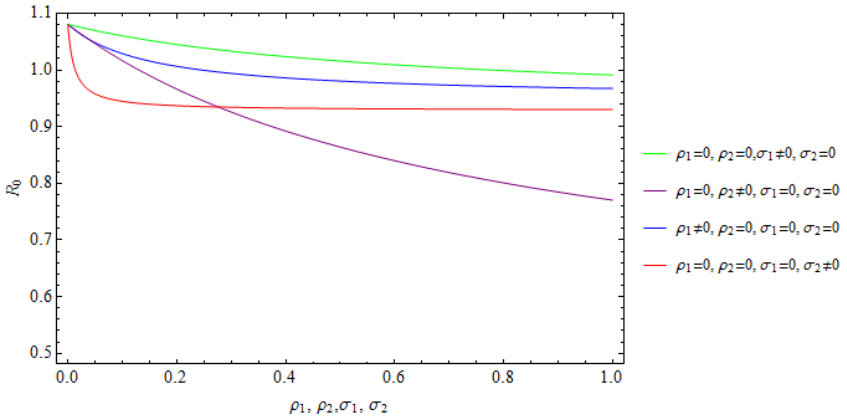
Simulations of the model (2.6) presented in Figure 3.1 and Figure 3.2 show that the DFE and EE are stable for different parameter values with the threshold quantity $\Re_0 < 1$ and when $\Re_0 > 1$ respectively. Therefore, if the disease burden reduces $\Re_0$ to less one then the infected population will be eliminated and in contrast, when $\Re_0 > 1$ the disease will persist in the population. So, some measure should be taken to alleviate or eliminate the disease. Now, we investigate the effect of treatment of different population class and also analyse the sensitivity of the decease burden to the various parameters in the following two subsections.

4.1 Efficacy of treatment

Medicine of chikungunya is not available commercially. An experimental vaccine in an early stage of clinical trial. But we are interested to analyze effectiveness of treatment of the infected individuals by determining the impact of treatments on the threshold number $\Re_0$. Figure 4.1 depicts the profile of $\Re_0$ as a function of treatment rates ρ_1, ρ_2, σ_1, and σ_2. When $\beta_{22} = 0$, Figure 4.1(a) shows that the treatment of symptomatically infected individuals and asymptomatically infected individuals reduces the quantity $\Re_0$ to less than unity but the treatment in class I_{s1} is more effective and that of has no impact to infected individuals in sub acute phase. If the infected individuals at subacute phase are considered as infectious, Figure 4.1(b) reveals that the treatment of individuals in this class has a significant impact to reduce the disease burden.



(a)



(b)

Figure 4.1: Reproduction number $\Re_0$ as a function of treatment rates ρ_1 or ρ_2 or σ_1 or σ_2. Parameter values used as given in Table 4.2 with (a) $\pi_1 = 2.2, \pi_2 = 15000, \beta_{22} = 0$, and (b) $\pi_1 = 1.8, \pi_2 = 8500, \beta_{22} = 0.005$.

4.2 Sensitivity Analysis

Sensitivity analysis is performed on the parameters of a model to determine which of the parameters play vital role in the dynamics of the model [36]. We calculate the sensitivity indices of the basic reproduction number ($\Re_0$) to the parameters in the model (2.6), to determine which of the parameter have high impact on $\Re_0$, and consequently to the disease transmission. We follow the approach as in [37]. The normalized sensitivity indices of $\Re_0$ on parameter P_i is given by

$$I_{P_i}^{\Re_0} = \frac{\partial \Re_0}{\partial P_i} \frac{P_i}{\Re_0}.$$

Sensitivity indices of $\Re_0$ are calculated at the parameter values as in Table 4.2 and the results are presented in Table 4.1, where second column corresponds to the case when $\rho_1 = 0, \rho_2 = 0, \sigma_1 = 0, \sigma_2 = 0$, and the third column for $\rho_1 = 1/15, \rho_2 = 1/15, \sigma_1 = 1/15, \sigma_2 = 1/15$.
In general, when one of the parameters with positive sign increases (or decreases) while the other parameters are constant, the value of $\Re_0$ increases (or decreases). For example, increasing β_1 by 10% increases $0.5 \times 10\%$ of $\Re_0$. Table 4.1 shows that the most important cruicial parameters are mosquito mortality rate (μ_2), mosquito biting rate (b_m). Other important parameters are disease transmission probability rate from infectious mosquito to susceptible human (β_1), human recruitment rate (π_1), mosquito recruitment rate (π_2), human mortality rate (μ_1), disease transmission probability rate from infectious human to susceptible mosquito (β_{12}) and (β_{22}), and recovery rates (γ_2, γ_1 and r_2).

Table 4.1: Sensitivity indices of $\Re_0$ to parameters for the model (2.6), evaluated at the parameter values given in Table 4.2.

Parameter	Sensitivity Indices without Treatment ($\rho_1 = \rho_2 = \sigma_1 = \sigma_2 = 0$)	Sensitivity Indices with Treatment ($\rho_1 = \rho_2 = \sigma_1 = \sigma_2 = 1/15$)
π_1	-0.5	-0.5
π_2	0.5	0.5
μ_1	0.49942	0.49988
μ_2	-1.06667	-1.06667
b_m	1.00002	1.00002
p	0.0706	0.08623
η_1	0.00006	0.00006
η_2	0.06667	0.06667
β_1	0.5	0.5
β_{12}	0.36905	0.46907
β_{22}	0.13095	0.03093
γ_1	-0.11351	-0.08699
γ_2	-0.19322	-0.150963
r_1	-0.06227	-0.12589
r_2	-0.13049	-0.00442
ρ_1	...	-0.04059
ρ_2	...	-0.03522
σ_1	...	-0.02937
σ_2	...	-0.02649

5 Conclusion

In this research, a new deterministic model for the dynamics of chikungunya virus transmission is formulated and analyzed rigorously. It has been shown that the model is mathematically and epidemiologically well-posed and it has a locally-asymptotically stable disease free equilibrium (DFE) when the basic reproduction number $\Re_0$, which is derived by next generation matrix method, is less than unity. The DFE is also globally-asymptotically stable, which is established by Lyapunov function and LaSalle Invariance Principle whenever $\Re_0 < 1$. Also, when $\Re_0 > 1$, there exists a unique endemic equilibrium (EE) of the model. Local stability of the EE is shown by sublinearity trick when $\Re_0 > 1$ and the global stability of EE is proved by using nonlinear Lyapunov function and LaSalle Invariance Principle for a special case whenever the threshold, $\Re_0$ is greater

Table 4.2: Value of the parameters of the model (2.6) for simulation. Most of the parameter values are obtained from entomologists researches on *Aedes sp.* and chikungunya.

Parameters	Base Line Value	Range	Reference
π_1	1.2	$Variable$	—
π_2	1800	$Variable$	—
$1/\mu_1$	70×365		[43]
$1/\mu_2$	13	$[7, 42]$	[9, 38]
b_m	0.4	$[0.1, 1]$	[9, 38]
β_1	0.35	$[0.001, 0.8]$	[9, 38, 6]
β_{12}	0.35	$[0.005, 0.9]$	[9, 38, 6]
β_{22}	0.005		[6]
$1/\eta_1$	3	$[1, 12]$	[9, 10]
$1/\eta_2$	2	$[2, 4]$	[5]
$1/\gamma_1$	7	$[3, 7]$	[41]
$1/\gamma_2$	0.5×7	$0.5/\gamma_1$	[10]
$1/r_1$	0.5×7	$0.5/\gamma_1$	[10]
$1/r_2$	90	$[14, 280]$	[10]
$\rho_1, \rho_2, \sigma_1, \sigma_2$	1/15		[19]
p	0.1	$[0.03, 0.28]$	[39]

than unity. Numerical simulations of the model for different parameter values and initial conditions verify these mathematical analysis for both the cases $\Re_0 < 1$ and $\Re_0 > 1$. Further, simulation of the model has been suggested that the treatment of symptomatically infected individuals and asymptomatically infected individuals can reduce the disease burden but the treatment of infected individuals in acute phase is more effective and that of has no impact of infected individuals in sub acute phase when they are not infectious. Also the sensitivity analysis has been revealed that the most effective parameters are mosquito mortality rate and average biting rate . So, the reduction of average life spans of mosquito and prevention against the effective contact with infectious mosquitoes would be effective control strategy. So, during the outbreak of the disease, insects repellents can be used to prevent the mosquito bites. Also the mosquito bed nets, which are available in market, can protect from mosquito biting. Transmission probability rates are also important parameters, reducing the value of these parameters can curtail the disease burden. Human and mosquito recruitment rates have great significance on controlling the disease so destruction of breeding sites of mosquito may be better prevention to the disease.

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