

A Mathematical Model for a Single Strain Dengue Disease with Control in Aquatic and Adult Phase

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Abstract

Dengue is a mosquito-borne viral infection found in urban and semi-urban areas of the tropical and subtropical regions around the world causes millions of death in every year. Dengue has no proper vaccination; vector control remains the only available strategy to prevent the disease. A mathematical model has been formulated to investigate the control strategies of the disease. The qualitative analysis of the model includes the calculation of basic reproduction number using the next generation operator approach. The method of estimation of the effective reproduction number $R(t)$ for actual epidemic has been explored. The local stability analysis of the disease free and endemic equilibrium points has been studied. It is found that the disease free equilibrium point is stable for $R_0 < 1$, otherwise it is unstable. The endemic equilibrium point exists for $R_0 > 1$. The figure of the basic reproduction number versus control parameters in aquatic and adult phase suggests that if the controls increase basic reproduction number (R_0) becomes less than one consequently the disease die out from the system. In this context, the control in aquatic phase is more effective than adult phase.

Keywords: S-I-R model, basic reproduction number, effective reproduction number, equilibrium, stability.

1. Introduction

Mosquito transmitted diseases are the major problem of public health in the urban and semi-urban area of tropical and subtropical regions around the world, causing millions of death in every year and create a large economic burden [1]. Dengue is one of such mosquito transmitted disease. According to the record of Chinese medical encyclopedia of the Jin Dynasty, we can say that people have known about this disease since 265-420 AD. They think that it is a "Water Poison" associated with flying insects. However the disease dengue was identified and named in 1779. In 1780 dengue epidemic spread to Asia, Africa and North America almost simultaneously. In 1789, Benjamin Rush coined the term "break-bone fever" based on the symptoms of the disease.

The frequency of dengue has developed drastically around the globe in late two decades. Due to various causes the genuine number of dengue cases was underreported. One recent estimate demonstrates 390 million (284-528 million) people are infected by dengue in every year of which 96 million (67-136 million) manifest clinically [2]. Another investigation, of the prevalence of dengue, estimates that 3.9 billion individuals, in 128 nations, are in danger of infection with dengue [2]. The number of dengue cases revealed has expanded from 2.2 million in 2010 to 3.2 million in 2015. Before 1970, just 9 nations had encountered serious dengue infection. The sickness is currently endemic in more than 100 nations in the WHO region of Africa, Americas, Eastern Mediterranean, South-East Asia, and the Western Pacific. However

the most seriously affected regions are the America, South-East Asia and Western Pacific countries. In India the picture will be clear from the following data.

Table 1: Statistics of dengue infection in India [3]:

| Year | Number of Cases Reported | Number of Deaths Reported |
|------|--------------------------|---------------------------|
| 2009 | 15535 | 96 |
| 2010 | 28292 | 110 |
| 2011 | 18860 | 169 |
| 2012 | 50222 | 242 |
| 2013 | 75808 | 193 |
| 2014 | 40571 | 137 |
| 2015 | 99913 | 220 |
| 2016 | 129166 | 245 |
| 2017 | 188401 | 325 |
| 2018 | 101192 | 172 |
| 2019 | 157315 | 166 |

The possible factors for spreading the dengue fever are (i) Unplanned urbanization results in inadequate waste management, drainage system and public health systems (ii) Poor vector control (iii) Ignorance and indifference of the people and government (iii) Climate change and viral evolution. Although initial epidemics were situated in urban zones, expanded dengue spread has included suburban and rural locales in Asia and Latin America. Dengue virus is transmitted to human through the bite of infected female mosquitoes mainly *Aedes aegypti* and to a lesser extent, *Aedes albopictus*. The *Aedes aegypti* mosquito lives in urban areas and breeds generally in man-made water containers like tubs, tyre, cistern etc. Once a mosquito is infected with dengue, it remains infectious throughout its life and transmits dengue virus to the human body during blood feed. People of any age could be infected by dengue and symptoms appear within 3-14 days after infected mosquito bite [2]. DEN-1, DEN-2, DEN-3 and DEN-4 are the four distinct, but closely related, serotypes of the virus dengue [2]. A person recovers from one of the dengue serotype having lifelong immune to that serotype but prone to infection from other three serotypes. About 12 weeks time the person becomes more susceptible to develop dengue hemorrhagic fever or dengue shock syndrome [4].

There is a common saying that prevention is better than cure; this is more applicable when we know that there is no world-wide recommended vaccine for dengue. The prevention and control of dengue is based on the control strategies of mosquito which are divided into three categories physical control, biological control and chemical control. The physical controls includes the following GIS mapping of dengue area, focused and effective surveillance, determination of oviposition sites, community-based control programs and education of prevention strategies. The biological controls are Paratransgenesis and use of *Wolbachia*, genetic modification of vector species, use of sterile insect technique, use of larvivorous fish and crustacean. The chemical control includes the use of insecticides and plant derivatives, use of insect growth regulators, use of pheromone as an attract-and-kill approach. Besides the

above control strategies there are some other types of control efforts like development of immunotherapy and vaccines, development of dengue human infection model etc.

In order to understand and control mosquito borne disease a number of mathematical model can be found in the literature [5, 6, 7, 8]. In this article, I consider an integer order system of ODE for investigation of control strategies. I use a mathematical model to analyze the dengue outbreak. My focus is on the calculations of the basic reproduction number R_0 of actual epidemics as well as on the calculation of effective reproduction number $R(t)$, since the basic reproduction number may change during the epidemic. The local stability of the system is also analyzed. My aim is to estimate the role of vector control in the reduction of the intensity and duration of epidemics.

The work is organized as follows. In section-2, I formulate the model. In section-3, I have determined the disease-free equilibrium and calculate the expression for R_0 for the epidemic. In this section, I also calculate the expression for effective reproduction number $R(t)$ and estimate R_0 for actual epidemic. In section-4, the local stabilities for both the disease-free and endemic steady state have been discussed. In section-5, I calculate the numerical values of the equilibrium points, characteristic roots of the respective equilibrium points and basic reproduction number using the force of infection. Finally, in section-6 I have drawn valuable conclusions.

2. Model Formation

I assume uniform mixing between the human and the mosquito population, i.e., each mosquito bite has equal probability of transmitting the disease to susceptible human (or become infected by biting an infected human). Here the total mosquito population M is divided into three mutually exclusive sub-populations; namely aquatic (M_a), susceptible (M_s) and infectious (M_i). The parameters are the intrinsic oviposition rate δ , the per capita mortality rate of adult female μ_m and the per capita mortality rate of mosquitoes in aquatic forms μ_a . The per capita rate at which mosquitoes emerge from the aquatic phase and become female adults is a . The remaining parameters are the carrying capacity C , the control efforts, modeled by additional mortality rates applied to the aquatic and terrestrial phase respectively c_a and c_m . The human population, H is assumed to be constant with per capita mortality rate given by μ_h and it is divided into three sub-populations such as susceptible (H_s), infective (H_i), recovered individuals (H_r). The per capita biting rate of mosquito b is the average number of bites per mosquito per day, while the transmission probability is the probability that an infection bite produced a new case in a susceptible numbers of other species. The transmission probabilities from the infected mosquito to the susceptible human and the infected human to the susceptible mosquito are denoted by β_h and β_m respectively. Assuming that bMH is the average number of bites that a human received. Therefore, two infection rates, one is arising from the infected mosquito to the susceptible human, and the other one is arising from the infected human to the susceptible mosquito are defined as, $\beta_h M_i H_s$ and $\beta_m H_i M_s$. α_h is the per capita human recovery rate. I have assumed that once a mosquito is infected with dengue remain infectious during their entire life. On the basis of the above assumption, we can formulate the following model.

$$\frac{dM_a}{dt} = K\delta M_a \left(1 - \frac{M_a}{C}\right) - (\gamma_m + \mu_a + c_a)M_a$$

$$\begin{aligned}
 \frac{dM_s}{dt} &= \gamma_m M_a - \frac{b\beta_m M_s H_i}{H} - (\mu_m + c_m) M_s \\
 \frac{dM_i}{dt} &= \frac{b\beta_m M_s H_i}{H} - (\mu_m + c_m) M_i \\
 \frac{dH_s}{dt} &= \mu_h (H_r - H_i) - \frac{b\beta_h M_i H_s}{H} \dots\dots\dots (1) \\
 \frac{dH_i}{dt} &= \frac{b\beta_h M_i H_s}{H} - (\mu_h + \alpha_h) H_i \\
 \frac{dH_r}{dt} &= \alpha_h H_i - \mu_h H_r
 \end{aligned}$$

All of the parameters are constant and positive. Moreover, most of the parameter values considered here has been estimated at [5, 8]. In particular, higher temperatures increase the mosquito's survival and oviposition rate and accelerate its reaching to the adult phase [9]. I adopted a linear interpolation to evaluate mosquito parameters. In short, the parameter range is summarized in Table 2.

Table 2: Parameters used in model (1), biological description and range of values

| Parameter symbol | Biological meaning | Estimated range |
|------------------|---|---------------------------|
| K | Sex ratio of mosquito | 0-1 |
| δ | Average oviposition rate of mosquito | 0-11.2 day^{-1} |
| μ_m | Average mortality rate of adult mosquito | 0.02-0.09 day^{-1} |
| μ_a | Average mortality rate of mosquito in aquatic | 0.01-0.47 day^{-1} |
| γ_m | Average transmission rate from aquatic to terrestrial | 0-0.19 day^{-1} |
| μ_h | Death rate of human | 0.0143-0.0167 $year^{-1}$ |
| α_h | Human recovery rate | 0.083-0.25 day^{-1} |
| C | Carrying capacity of mosquito in aquatic phase | 200000000 |
| b | Average bit per mosquito | 0-1 day^{-1} |
| β_m | Transmission probability from human to mosquito | 0-1 |
| β_h | Transmission probability from mosquito to human | 0-1 |
| c_a | Control effort rates in aquatic phase | 0-1 day^{-1} |

| | | |
|-------|---|------------------------|
| c_m | Control effort rates in terrestrial phase | $0-1 \text{ day}^{-1}$ |
|-------|---|------------------------|

3. Basic Reproduction Number

Mathematical measurement of epidemics is done by calculating basic reproduction number [10, 11] R_0 which is the average number of secondary infections produced when one infected individual is introduced into a host virgin population. Now a system affecting some disease fails into epidemic when $R_0 > 1$. R_0 is undoubtedly the most important threshold value to determine the nature of an epidemic. It carries the information about the persistence of disease [12, 13]. In this section, I shall determine the basic reproduction number using next generation matrix method [11, 14] and effective reproduction number with the method proposed by Wallinga and Lipsitch [15].

3.1 Disease Free Equilibrium

System (1) can be reduced by the following conservation relation $H_r = H - (H_s + H_i)$. Then the system (1) can be rewritten as

$$\left. \begin{aligned} \frac{dM_a}{dt} &= K\delta M_a \left(1 - \frac{M_a}{C}\right) - (\gamma_m + \mu_a + c_a)M_a \\ \frac{dM_s}{dt} &= \gamma_m M_a - \frac{b\beta_m M_s H_i}{H} - (\mu_m + c_m)M_s \\ \frac{dM_i}{dt} &= \frac{b\beta_m M_s H_i}{H} - (\mu_m + c_m)M_i \\ \frac{dH_s}{dt} &= \mu_h(H - H_s) - \frac{b\beta_h M_i H_s}{H} \\ \frac{dH_i}{dt} &= \frac{b\beta_h M_i H_s}{H} - (\mu_h + \alpha_h)H_i \end{aligned} \right\} \dots\dots\dots(2)$$

In order to determine the equilibrium points of the system (2) and expression for R_0 at the beginning of the epidemic, we solve the following algebraic system corresponding to $H_i = 0$.

$$\left. \begin{aligned} K\delta M_a \left(1 - \frac{M_a}{C}\right) - (\gamma_m + \mu_a + c_a)M_a &= 0 \\ \gamma_m M_a - \frac{b\beta_m M_s H_i}{H} - (\mu_m + c_m)M_s &= 0 \\ \frac{b\beta_m M_s H_i}{H} - (\mu_m + c_m)M_i &= 0 \\ \mu_h(H - H_s) - \frac{b\beta_h M_i H_s}{H} &= 0 \\ \frac{b\beta_h M_i H_s}{H} - (\mu_h + \alpha_h)H_i &= 0 \end{aligned} \right\} \dots\dots\dots(3)$$

which gives, $M_a = C \left(1 - \frac{\gamma_m + \mu_a + c_a}{K\delta}\right) = \bar{M}_a$, $M_s = \frac{\gamma_m C}{\mu_m + c_m} \left(1 - \frac{\gamma_m + \mu_a + c_a}{K\delta}\right) = \bar{M}_s$, $H_i = 0$; $H_s = H$. Therefore the DFE point is $E_0 = (\bar{M}_a, \bar{M}_s, 0, H, 0)$ provided $\gamma_m + \mu_a + c_a < K\delta$.

3.2 Method of Next Generation Matrix

Using the next generation matrix method [11, 14], I have derived the expression for R_0 , associated to the disease free equilibrium $E_0(M_a, M_s, 0, H, 0)$. Consider the 3rd and 5th equations of (2)

$$\frac{dM_i}{dt} = \frac{b\beta_m M_s H_i}{H} - (\mu_m + c_m)M_i$$

$$\frac{dH_i}{dt} = \frac{b\beta_h M_i H_s}{H} - (\mu_h + \alpha_h)H_i$$

The 1st term in each of the equations represents the new infection and the 2nd term represents the infection out. Here the vector ϕ is the rate of new infections entered in each class and v is the difference of transfer out, from the compartment and in, into the compartment. Therefore

$$\phi = \begin{bmatrix} \frac{b\beta_m M_s H_i}{H} \\ \frac{b\beta_h M_i H_s}{H} \end{bmatrix}, v = \begin{bmatrix} (\mu_m + c_m)M_i \\ (\mu_h + \alpha_h)H_i \end{bmatrix}. \text{ Let } I = \begin{bmatrix} M_i \\ H_i \end{bmatrix} \text{ and } \frac{dI}{dt} = \phi - v$$

The corresponding Jacobean matrices at disease-free equilibrium E_0 are computed as

$$F = \frac{\partial \phi(E_0)}{\partial x_i} = \begin{bmatrix} 0 & \frac{b\beta_m \bar{M}_s}{H} \\ b\beta_h & 0 \end{bmatrix} \text{ and } V = \frac{\partial v(E_0)}{\partial x_i} = \begin{bmatrix} (\mu_m + c_m) & 0 \\ 0 & (\mu_h + \alpha_h) \end{bmatrix}$$

where, $x_1 = M_i$ and $x_2 = H_i$. The basic reproduction number R_0 is the spectral radius of the next generation matrix FV^{-1} .

$$\begin{aligned} R_0 = \rho(FV^{-1}) &= \rho \left(\begin{bmatrix} 0 & \frac{b\beta_m \bar{M}_s}{H} \\ b\beta_h & 0 \end{bmatrix} \times \frac{1}{(\mu_m + c_m)(\mu_h + \alpha_h)} \begin{bmatrix} (\mu_h + \alpha_h) & 0 \\ 0 & (\mu_m + c_m) \end{bmatrix} \right) \\ &= \rho \left(\frac{1}{(\mu_m + c_m)(\mu_h + \alpha_h)} \begin{bmatrix} 0 & \frac{b\beta_m \bar{M}_s}{H}(\mu_m + c_m) \\ b\beta_h(\mu_h + \alpha_h) & 0 \end{bmatrix} \right) \\ &= \sqrt{\frac{b^2 \beta_h \beta_m \bar{M}_s}{H(\mu_m + c_m)(\mu_h + \alpha_h)}} \dots\dots\dots(4) \end{aligned}$$

3.3 Estimating R_0 for Actual Epidemics

There are quite good number of methods exist in the literature by which one can evaluate R_0 using incidence data [16]. In this article, we estimate R_0 from the initial growth phase of the epidemics. Following [5, 8] we suppose that at the initial phase of epidemic, the cumulative number of cases, $c(t)$ varies as $\exp(\Lambda t)$, where Λ is the force of infection which can be evaluated. With these assumptions, the time evolution of the infected host and vector for the initial phase of epidemic has the following form

$$\left. \begin{aligned} H_i &\sim H_{i0} \exp(\Lambda t) \\ M_i &\sim M_{i0} \exp(\Lambda t) \end{aligned} \right\} \dots\dots\dots(5)$$

where H_{i0} and M_{i0} are constant, evaluated from the given data. Further at the earlier phase of an epidemic, the number of infected hosts and vectors can be assumed to be very small and therefore, the expression of the susceptible human and vectors from the model (2) are given by

$$M_s = \bar{M}_s, H_s = H.$$

$$\begin{aligned} \text{Now, } H_{i0} \Lambda \exp(\Lambda t) &= \frac{b\beta_h M_{i0} H_s}{H} \exp(\Lambda t) - (\mu_h + \alpha_h) H_{i0} \exp(\Lambda t) \\ &\Rightarrow \left[\frac{\Lambda}{(\mu_h + \alpha_h)} + 1 \right] H_{i0} = \frac{b\beta_h}{(\mu_h + \alpha_h)} M_{i0} \end{aligned}$$

$$\begin{aligned} \text{and } M_{i0} \Lambda \exp(\Lambda t) &= \frac{b\beta_m M_s H_{i0}}{H} \exp(\Lambda t) - (\mu_m + c_m) M_{i0} \exp(\Lambda t) \\ &\Rightarrow \left[\frac{\Lambda}{(\mu_m + c_m)} + 1 \right] M_{i0} = \frac{b\beta_m \bar{M}_s}{H(\mu_m + c_m)} H_{i0} \end{aligned}$$

$$\text{Therefore } \left[\frac{\Lambda}{(\mu_h + \alpha_h)} + 1 \right] \left[\frac{\Lambda}{(\mu_m + c_m)} + 1 \right] = \frac{b\beta_h}{(\mu_h + \alpha_h)} \times \frac{b\beta_m \bar{M}_s}{H(\mu_m + c_m)} = R_0^2$$

$$\Rightarrow R_0^2 = \left[\frac{\Lambda}{(\mu_h + \alpha_h)} + 1 \right] \left[\frac{\Lambda}{(\mu_m + c_m)} + 1 \right] \dots\dots\dots(6)$$

3.4 Effective Reproduction Number R(t)

When an epidemic starts in a partially susceptible population, the control measure of the disease should be taken on the basis of the effective reproductive number or time-varying reproduction number $R(t)$ because the value of $R(t)$ provides information about the severity of the disease over different time. The effective reproductive number $R(t)$ is defined as the number of secondary infections that arise from a typical primary case with a symptom onset at the week t [15]. The estimate provides useful information about the intervention strategies to be needed for controlling the outbreak. There are several technique for estimation of $R(t)$ [17]. Here I have estimated $R(t)$ from the given data using the renewal equation of birth process [15, 8].

$$F(t) = \frac{b(t)}{\int_{a=0}^{\infty} b(t-a)g(a)da} \dots\dots\dots(7)$$

where, $b(t)$ represents the number of new cases at the day t and $g(a)$ is the generation interval distribution for the disease, which is defined as the probability distribution function of the time. The rates of leaving infection classes, $s_1 = \mu_m + c_m$ and $s_2 = \mu_h + \alpha_h$ are constant quantities. Therefore the generation interval distribution is the convolution of two exponential distributions $s_1 e^{-s_1 t}$ and $s_2 e^{-s_2 t}$ with a mean

$$T_c = \frac{1}{s_1} + \frac{1}{s_2}.$$

Following [18] the explicit expression of the density function is given by

$$g(t) = \sum_{i=1}^2 \frac{s_1 s_2 \exp(-s_i t)}{\prod_{j=1, j \neq i}^2 (s_j - s_i)} \dots\dots\dots(8)$$

The above expression is valid when the force of infection Λ satisfies $\Lambda > \min(-s_1, -s_2)$ [16]. Also we have $\int_0^{\infty} g(t)dt = 1$. Substituting $g(t)$ into the equation (7) and using the epidemiological data we can compute $F(t)$ and since the number of human secondary cases derived from a human primary case is equal to $R^2(t)$, then $R(t) = \sqrt{F(t)}$.

4. Local Stability Analysis

Equilibrium points of a dynamical system are the state of the system when there is no change of the variables. It is important to investigate the local stability of the equilibrium points to predict about the system. In this section the local stability of the disease free equilibrium point and endemic equilibrium point has been investigated.

4.1 Local Stability Analysis of Disease Free Equilibrium

Theorem: The disease free equilibrium $E_0 = (\bar{M}_a, \bar{M}_s, 0, H, 0)$ of the system corresponding to the model (2) is locally asymptotically stable if $R_0 < 1$ otherwise it is unstable.

Proof: The Jacobean matrix of the model (2) at E_0 is

$$J_{E_0} = \begin{bmatrix} -M_0 & 0 & 0 & 0 & 0 \\ \gamma_m & -\frac{b\beta_m H_i}{H} - Q & 0 & 0 & -\frac{b\beta_m M_s}{H} \\ 0 & \frac{b\beta_m H_i}{H} & -Q & 0 & \frac{b\beta_m M_s}{H} \\ 0 & 0 & -\frac{b\beta_h H_s}{H} & -\mu_h - \frac{b\beta_h M_i}{H} & 0 \\ 0 & 0 & \frac{b\beta_h H_s}{H} & \frac{b\beta_h M_i}{H} & -(\mu_h + \alpha_h) \end{bmatrix}_{E_0}$$

where $P = (\gamma_m + \mu_a + c_a)$, $M_0 = \left(-K\delta + P + \frac{2K\delta M_a}{c}\right)|_{E_0}$ and $Q = (\mu_m + c_m)$

The Eigen values of J_{E_0} are the roots of the characteristic equation

$$\begin{vmatrix} -M_0 - \lambda & 0 & 0 & 0 & -\frac{b\beta_m \bar{M}_s}{H} \\ \gamma_m & -Q - \lambda & 0 & 0 & \frac{b\beta_m \bar{M}_s}{H} \\ 0 & 0 & -Q - \lambda & 0 & \frac{b\beta_m \bar{M}_s}{H} \\ 0 & 0 & -b\beta_h & -\mu_h - \lambda & 0 \\ 0 & 0 & b\beta_h & 0 & -(\mu_h + \alpha_h) - \lambda \end{vmatrix} = 0$$

$$\Rightarrow (\lambda + M_0)(Q + \lambda)(\mu_h + \lambda)(\lambda^2 + \lambda\sigma_1 + \sigma_2) = 0$$

Where $\sigma_1 = (\mu_m + c_m) + (\mu_h + \alpha_h)$ and $\sigma_2 = (\mu_m + c_m)(\mu_h + \alpha_h)(1 - R_0^2)$.

The eigenvalues of J_{E_0} are the following.

$$\lambda_1 = -M_0 = -K\delta + P + \frac{2K\delta \bar{M}_a}{c} = (\gamma_m + \mu_a + c_a) - K\delta,$$

$$\lambda_2 = -(\mu_m + c_m), \quad \lambda_3 = -\mu_h.$$

Other two eigenvalues are obtained from $\lambda^2 + \lambda\sigma_1 + \sigma_2 = 0$.

$$\lambda_4 = \frac{1}{2}(-\sigma_1 + \sqrt{\sigma_1^2 - 4\sigma_2}) \text{ and } \lambda_5 = \frac{1}{2}(-\sigma_1 - \sqrt{\sigma_1^2 - 4\sigma_2}).$$

Now, $\sigma_1^2 - 4\sigma_2 = \{(D_m + c_m) + (\mu_h + \alpha_h)\}^2 - 4(\mu_m + c_m)(\mu_h + \alpha_h)(1 - R_0^2)$.

If $R_0 > 1$, then $(1 - R_0^2) < 0$ therefore $\sqrt{\sigma_1^2 - 4\sigma_2} > \sigma_1$. In this case $\lambda_4 > 0$ so the system is unstable.

If $R_0 < 1$, then $(1 - R_0^2) > 0$ therefore $\sqrt{\sigma_1^2 - 4\sigma_2} < \sigma_1$. In this case the real part of all the eigen values are negative so the system is asymptotically stable.

4.2 Endemic Equilibrium

Let $E^* = (M_a^*, M_s^*, M_i^*, H_s^*, H_i^*)$ be any arbitrary positive endemic equilibrium of the model (3).

Let us define

$$\left. \begin{aligned} \lambda_1^* &= \frac{b\beta_h M_i^*}{H} \\ \text{and } \lambda_2^* &= \frac{b\beta_m H_i^*}{H} \end{aligned} \right\} \dots\dots\dots(9)$$

Substituting the value of λ_1^* and λ_2^* in (3) we obtain the following endemic equilibrium in terms of λ_1^* and λ_2^* .

$$\left. \begin{aligned} H_s^* &= \frac{\mu_h H}{\mu_h + \lambda_1^*}, H_i^* = \frac{\lambda_1^* H_s^*}{\mu_h + \alpha_h}, M_s^* = \frac{\gamma_m M_a^*}{\lambda_2^* + \mu_m + c_m}, \\ M_i^* &= \frac{\lambda_2^* M_s^*}{\mu_m + c_m} \text{ and } M_a^* = \frac{c\{K\delta - (\mu_m + \mu_a + c_a)\}}{\bar{A}\delta} \end{aligned} \right\} \dots\dots\dots(10)$$

Substituting the value of M_i and H_i from (10) in (9) and simplifying we obtain

$$\left. \begin{aligned} \lambda_1^* &= \frac{b\beta_h \lambda_2^* \gamma_m M_a^*}{H(\mu_m + c_m)(\lambda_2^* + \mu_m + c_m)} \\ \text{and } \lambda_2^* &= \frac{\bar{A}\beta_m \lambda_1^* \mu_h}{(\mu_h + \alpha_h)(\mu_h + \lambda_1^*)} \end{aligned} \right\} \dots\dots\dots(11)$$

Using the expression of R_0 from (4) we obtain the following expression for λ_1^* and λ_2^* in terms of R_0 .

$$\left. \begin{aligned} (11) \Rightarrow \lambda_1^* &= \frac{\mu_h(\mu_m + c_m)(\mu_h + \alpha_h)(R_0^2 - 1)}{b\beta_m \mu_h + (\mu_m + c_m)(\mu_h + \alpha_h)} \\ \text{and } \lambda_2^* &= \frac{b\beta_m \mu_h(\mu_m + c_m)(R_0^2 - 1)}{b\beta_m \mu_h + (\mu_m + c_m)(\mu_h + \alpha_h)R_0^2} \end{aligned} \right\} \dots\dots\dots(12)$$

Using the relation (12) we can evaluate E^* in terms of R_0 . Therefore, if $R_0 > 1$ then, $(R_0^2 - 1) > 0$ so from (12) λ_1^* and λ_2^* are positive consequently there exists a unique positive endemic equilibrium.

Theorem: The positive endemic equilibrium, $E^* = (M_a^*, M_s^*, M_i^*, H_s^*, H_i^*)$ of the system (2) is unique if $R_0 > 1$.

4.3 Local Stability Analysis of Endemic Equilibrium

The Jacobean matrix J of the system (2) evaluated at the endemic equilibrium point $E^* = (M_a^*, M_s^*, M_i^*, H_s^*, H_i^*)$ is given by

$$J_{E^*} = \begin{bmatrix} -M_0 & 0 & 0 & 0 & 0 \\ \gamma_m & -\frac{b\beta_m H_i}{H} - Q & 0 & 0 & -\frac{b\beta_m M_s^*}{H} \\ 0 & \frac{b\beta_m H_i}{H} & -Q & 0 & \frac{b\beta_m M_s^*}{H} \\ 0 & 0 & -\frac{\beta_h H_s^*}{H} & -\mu_h - \frac{b\beta_h M_i^*}{H} & 0 \\ 0 & 0 & \frac{b\beta_h H_s^*}{H} & \frac{b\beta_h M_i^*}{H} & -(\mu_h + \alpha_h) \end{bmatrix}$$

Where $P = (\gamma_m + \mu_a + c_a)$ and $M_0 = -K\delta + P + \frac{2K\delta M_a^*}{c}$.

The eigenvalues of J_{E_0} are the roots of the characteristic equation

$$\begin{vmatrix} -M_0 - l & 0 & 0 & 0 & 0 \\ \gamma_m & -M_1 - l & 0 & 0 & -M_2 \\ 0 & M_1 - M_3 & -M_3 - l & 0 & M_2 \\ 0 & 0 & -M_4 & -M_5 - l & 0 \\ 0 & 0 & M_4 & M_5 - \mu_h & -M_6 - l \end{vmatrix} = 0$$

$$\Rightarrow (M_0 + l)(M_3 + l)(l^3 + a_1 l^2 + a_2 l + a_3) = 0 \dots\dots\dots(13)$$

where $a_1 = M_1 + M_5 + M_6$, $a_2 = M_1 M_5 + M_1 M_6 + M_5 M_6 - M_2 M_4$, $a_3 = M_1 M_5 M_6 - M_2 M_4 \mu_h$ and

$$M_1 = \frac{b\beta_m H_i}{H} + (\mu_m + c_m), M_2 = \frac{b\beta_m M_s^*}{H}, M_3 = (\mu_m + c_m), M_4 = \frac{b\beta_h H_s^*}{H}, M_5 = \mu_h + \frac{b\beta_h M_i^*}{H}, M_6 = (\mu_h + \alpha_h).$$

According to Routh-Hurwitz criterion the real part of the roots of the cubic equation $l^3 + a_1l^2 + a_2l + a_3 = 0$ is negative if $a_1 > 0, a_2 > 0, a_3 > 0$ and $a_1a_2 > a_3$. Therefore the endemic equilibrium point E^* of the system (2) is locally asymptotically stable if $K\delta > P, a_1 > 0, a_2 > 0, a_3 > 0$ and $a_1a_2 > a_3$.

5. Numerical Analysis:

The model (2) is a system of non-linear ordinary differential equations whose analytical solution is hardly possible. So a suitable numerical technique must be used to solve the system of equations and the expressions used in our above theoretical study. Let us take a help of MATLAB to get the numerical solution on the basis of estimated parameter values in the table 2. I investigate numerically the value of the endemic equilibrium point and its stability with the parameters $\delta = 10, \mu_m = 0.05, \mu_a = 0.3, \gamma_m = 0.1, \mu_h = 0.16/365, \alpha_h = 0.1, K = 0.5, C = 200000000, b = 1, \beta_m = 0.75, \beta_h = 0.75, c_a = 0.5, c_m = 0.5, H = 100000000$. $E^* = (164000000, 29806000, 11964, 0.0051, 0.000004)$ and eigenvalues of the Jacobian matrix J_{E^*} are $\lambda_1 = -4.1000; \lambda_2 = -0.5500; \lambda_3 = -574900; \lambda_4 = 611.8972; \lambda_5 = 0.000046$. Therefore the endemic equilibrium point is unstable. In this case $R_0 = 1.7439$ obtained from the expression (4). Figure 3 showing the level of control effort of mosquito to drop down the basic reproduction number below unity. So in this case control mechanisms should be applied to the mosquito population, mostly in places where high number of dengue hemorrhagic case was notified. Intensity of control strategies must be according to the value of effective reproduction number $R(t)$. However we may calculate this effective reproduction number $R(t)$ for any dengue affected region if the data of new dengue infection is available (figure 2). Suppose the dengue situation in some city is given by the total number of new dengue infected people admitted in all the hospital of the city per week.

Table 3: Hypothetical dengue data

| | | | | | | | | | | | | |
|-----------|------|------|------|------|-----|------|------|------|------|------|------|------|
| week | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
| New cases | 50 | 100 | 120 | 250 | 500 | 1000 | 1500 | 2500 | 3000 | 2600 | 2400 | 3100 |
| week | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 |
| New cases | 2700 | 2400 | 2000 | 1000 | 900 | 500 | 200 | 100 | 50 | 20 | 10 | 2 |

From this data of the table 3, it is found that correlation coefficient $R = 0.9883$ (figure 1) and the force of infection $\Lambda = 0.38$ using (5). Substituting the value of Λ in the equation (6) we can obtain the value of the basic reproduction number $R_0 = 2.106704$, which is nearly equal to the value of R_0 obtained from the expression (4). Here I investigate numerically the value of the disease free equilibrium point and its stability with the parameter values $\delta = 10, \mu_m = 0.05, \mu_a = 0.3, \gamma_m = 0.1, \mu_h = 0.1/365, \alpha_h = 0.1, K = 0.5, C = 200000000, b = 0.5, \beta_m = 0.75, \beta_h = 0.75, c_a = 0.5, c_m = 0.5, H = 10000000$. The disease free equilibrium point is $E_0 = 10^8 (1.64, 0.2982, 0, 1.0, 0)$ and eigenvalues of the Jacobian matrix J_{E_0} are $\lambda_1 = -4.1; \lambda_2 = -0.55; \lambda_3 = -0.00027397; \lambda_4 = -0.021; \lambda_5 = -0.6293$. Therefore the disease free equilibrium point is asymptotically stable. In this case $R_0 = 0.872$ obtained from the expression (4).

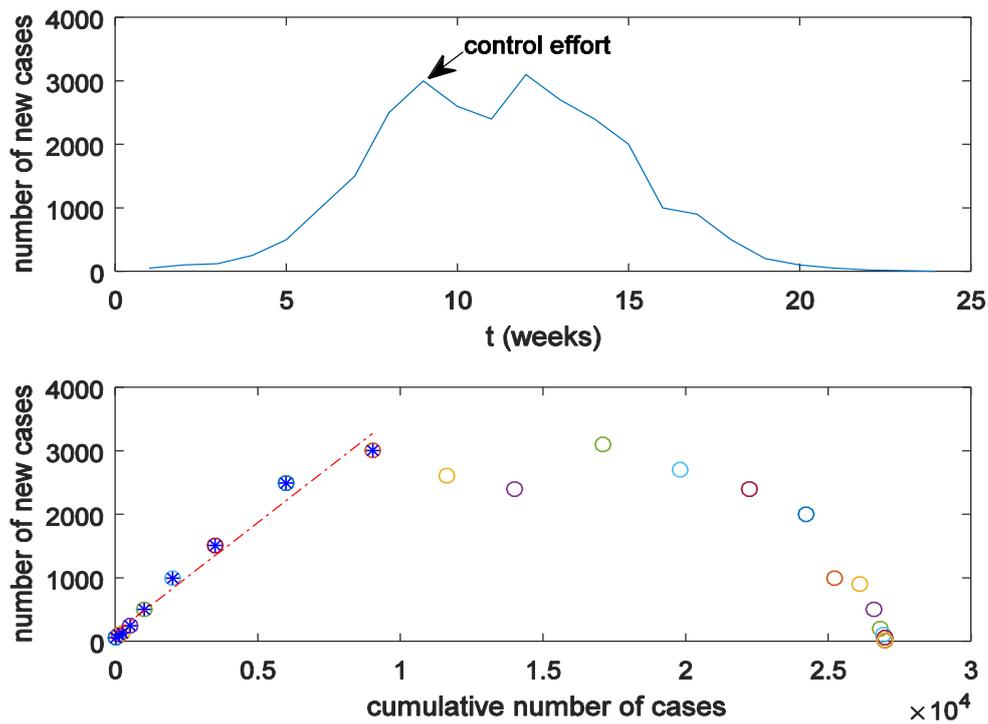


Figure 1: First figure is the time series of the hypothetical weekly dengue data. Second figure includes the scattered diagram of the weekly number of new cases against the cumulative number of cases and the list square straight line fit to obtained \ddot{E} .

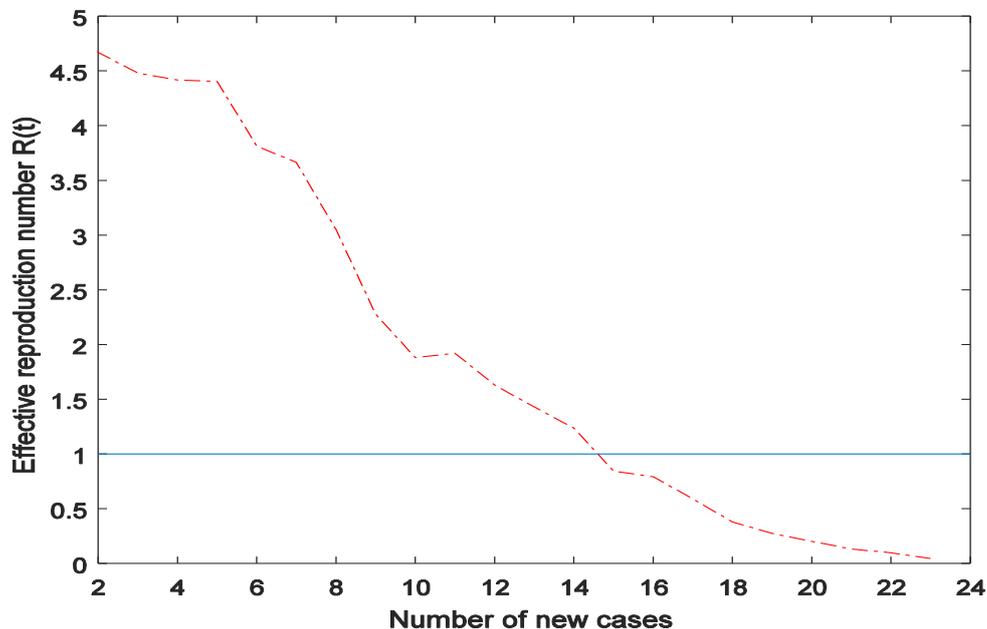


Figure 2: Value of R_0 decreasing more rapidly when control effort is paid on aquatic phase.

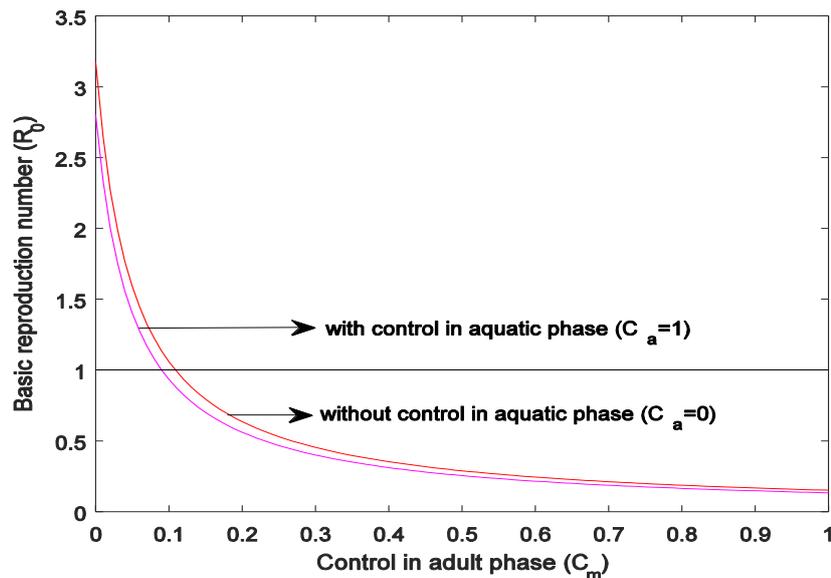


Figure 3: Value of R_0 decreasing more rapidly when control effort is paid on aquatic phase.

6. Conclusions:

Here I couple an S-I-R model for human population and an S-I model of mosquito population. The estimated parameter values from [8] have been used for the numerical analysis of the model. The basic reproduction number has been calculated analytically using the next generation matrix method. Then I estimation of the basic reproduction number R_0 and effective reproduction number $R(t)$ for actual epidemic using data. After that I discuss the stability of disease free and endemic equilibrium points. It is found that the disease free equilibrium point is stable for $R_0 < 1$, otherwise it is unstable. The endemic equilibrium point exists for $R_0 > 1$. In the figure 3, I have plotted the basic reproduction number with respect to control parameters in aquatic and adult phase and observed that if the controls increase the basic reproduction number (R_0) decreases and become less than one. The basic reproduction number has been evaluated using the expression (4) and verified with the basic reproduction number (6) in terms of force of infection which is obtained analyzing the data. I have discuss the method for estimating the effective reproduction number from a given data which determine the intensity of control effort.

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