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Modeling the impact of vertical transmission in vectors on the dynamics of dengue fever *

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Abstract. Dengue is a viral illness caused by infection with one of four dengue viruses known as dengue 1, 2, 3 and 4. In this paper we develop a deterministic mathematical model for dengue fever with vertical transmission in female aedes aegypti mosquitoes. The basic reproduction for the model is computed. Sensitivity analysis of the basic reproduction number for the model indicates a direct relationship between basic reproduction number and vertical transmission rate. To reduce or eradicate dengue fever in the community, public health workers should focus on an intervention that destroys most of the aedes aegypti mosquitoes and their eggs. Moreover, numerical simulation shows that, the spread of dengue fever is very fast and therefore needs precautions and immediate and serious attention after epidemic.

Keywords: modeling, vertical transmission, dynamics, dengue fever

1 Introduction

Dengue is a systematic viral infection transmitted to humans through the bites of infected female infective Aedes mosquitoes, most commonly Aedes aegypti, known as the 'dengue mosquito', but sometimes by other mosquito species. Aedes aegypti and Aedes albopictus are the two kinds of mosquitoes that caries four distinct but closely related serotypes of virus known as DEN 1, DEN 2, DEN 3 and DEN4 in which only DEN 2 and DEN 3 are mostly identified in tropical countries. After virus incubation for 4-10 days, an infected mosquito is capable of transmitting the virus for the rest of its life (less than 60 days). Infected humans are the main carriers and multipliers of the virus, serving as a source of the virus for uninfected mosquitoes. Patients who are already infected with the dengue virus can transmit the infection (for 4-12 days) via Aedes mosquitoes after their first symptoms appear. When a person recovers from dengue infection they develop a long-term immunity to that specific virus, but not the other three dengue viruses. If the person becomes infected again with a different dengue virus, there is an increased chance that they may develop a more severe form of the illness known as dengue haemorrhagic fever (DHF). According to [14], dengue cannot be spread from human to human directly.

Although Aedes albopictus can transmit the dengue virus and the presence of the species was detected in Asia in recent years, Aedes aegypti remains the principal vector of dengue virus transmission. The Aedes aegypti mosquito lives in urban habitats and breeds mostly in man-made containers. Unlike other mosquitoes Aedes aegypti is a day-time feeder; its peak biting periods are early in the morning and in the evening before dusk. Female Aedes aegypti bites multiple people during each feeding period.

An additional appealing fact is, the shift of patients' phenomena where dengue fever previously attacked children of primary school age, that now everybody is vulnerable to the fever[16]. The disease is now endemic

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in more than 100 countries of Africa, America, Asia and the Western Pacific with local variations in risk influenced by rainfall, temperature and unplanned rapid urbanization. Dengue fever is regarded as a serious infectious disease that risks about 2.5 billion people all over the world, especially in the tropical countries[7].

The incidence of dengue has grown dramatically around the world in recent decades. The actual numbers of dengue cases are underreported and many cases are misclassified. One recent estimate indicates 390 million dengue infections per year (95% credible interval 284-528 million), of which 96 million (67-136 million) manifest clinically (with any severity of disease)[18]. Another study, of the incidence of dengue, estimates that 3900 million people, in 128 countries, are at risk of infection with dengue viruses[13]. The disease exists in two forms: Dengue Fever or classic dengue and severe dengue or Dengue Haemorrhagic Fever which was first recognized in the 1950s during dengue epidemics in the Philippines and Thailand. Today, severe dengue affects most Asian, West African and Latin American countries and has become a leading cause of hospitalization and death among people in these regions. Furthermore, immunity is acquired only to the serotype contracted and a contact with a second serotype becomes more dangerous. However, cross-immunity to the other serotypes after recovery is only partial and temporary. Subsequent infections by other serotypes increase the risk of developing severe dengue. A person infected by one of the four serotypes will never be infected again by the same serotype (homologus immunity), but he loses immunity to the three other serotypes (heterologus immunity) in about 12 weeks and then becomes more susceptible to developing dengue haemorrhagic fever [3].

According to [15], dengue should be suspected when a high fever (40C/104F) is accompanied by severe headache, pain behind the eyes, muscle and joint pains, nausea, vomiting, swollen glands or rash. Symptoms usually last for 2-7 days, after an incubation period of 4-10 days after the bite from an infected mosquito. Severe dengue is a potentially deadly complication due to plasma leaking, fluid accumulation, respiratory distress, severe bleeding, or organ impairment. Warning signs occur 3-7 days after the first symptoms in conjunction with a decrease in temperature (below 38°/100°F) and include: severe abdominal pain, persistent vomiting, rapid breathing, bleeding gums, fatigue, restlessness and blood in vomit. The next 24-48 hours of the critical stage can be lethal; proper medical care is needed to avoid complications and risk of death.

Severe dengue is a potentially deadly complication due to plasma leaking, fluid accumulation, respiratory distress, severe bleeding, or organ impairment. Warning signs occur 3-7 days after the first symptoms in conjunction with a decrease in temperature (below 38°/100°F) and include: severe abdominal pain, persistent vomiting, rapid breathing, bleeding gums, fatigue, restlessness and blood in vomit. The next 24-48 hours of the critical stage can be lethal; proper medical care is needed to avoid complications and risk of death. Currently, there are no licensed or specific therapeutics and substantial vector control efforts have not stopped its rapid emergence and global spread.

In 2012, an outbreak of dengue on the Madeira Islands of Portugal resulted in over 2000 cases and imported cases were detected in mainland Portugal and 10 other countries in Europe. In 2013, cases occurred in Florida (United States of America) and Yunnan province of China. In 2014, trends indicated increases in the number of cases in the People's Republic of China, the Cook Islands, Fiji, Malaysia,Vanuatu and Tanzania. Dengue was also reported in Japan after a lapse of over 70 years. In 2015 an increase in the number of cases was reported in Brazil and several neighbouring countries and about 2.5% of those affected die.

Modelling is used to quantify uncertainty due to different gaps in our knowledge to help identify research priorities. The influence for the use of mathematical modelling in theory and practice of disease management and control have increased due to the fact that, the approach helps in figuring out decisions that are of significant importance on the outcomes and provide comprehensive examinations that enter into decisions in a way that gives quick approach and control of the disease with main interest of developing more effective public health interventions[6, 11, 12].

Nuraini, N.[10] formulated a mathematical model for dengue internal transmission process human and found that the virus free equilibrium is locally asymptomatically stable if the basic reproduction number is less than unit. [19] developed a mathematical model taking into account the hospitalized compartment to present transmission of mosquitoes (Aedes aegypti) virus and its existence in human population. Their findings show that, equilibria can be controlled by basic reproduction number of the disease transmission. Massawe [8] developed SITR (susceptible, infected, treated, recovery) and ASI (aquatic, susceptible, infected) epidemic model

to describe the interaction between human and dengue fever mosquito populations. Their results showed that, dengue fever eradication is possible if daily maturation of Aedes mosquitos' larvae to adult can be reduced.

Dengue fever is reported to be one of the most challenging diseases in human mind. Although the disease has been known for decades, many nations are caught unprepared to handle large outbreaks. To date, there is no specific treatment for dengue fever and the main prevention method is to combat the vector mosquitoes through different non pharmaceutical measures, including the destruction of the vectors breeding sites. Therefore, the objective of this study is to develop a deterministic mathematical model of dengue fever transmission which incorporates vertical transmission in vector mosquitoes. The Aedes eggs population is included in order to determine the effect of vertical transmission in the initial spread of dengue fever. The study focuses on the analysis of the model by computing the basic reproduction number, which is the initial transmission of the disease. In addition, the sensitivity of each parameter involved in the basic reproduction number is studied. The sensitivity indices aid in determining which parameter is of importance to measure accurately and study any variations.

Modelling the impact of vertical transmission in vectors on the dynamics of dengue fever is important because it will help health personnel and policy makers to plan appropriately for preventive measures in the early stages of disease outbreak. The preventive measures may include the destruction of mosquito breeding sites, application of insecticides, or vaccination strategies or both in order to avoid other possible transmission. The study will also help health personnel and policy maker involved in disease control strategies to quantify the effect of vectorial capacity on dengue fever transmission and set appropriate strategies for disease control. Moreover, the study will create awareness to people on the dynamics of dengue fever and how to prevent themselves from any possible ways which may increase breeding sites for mosquitoes and vectorial capacity.

This paper has significant contribution to the existing body of knowledge about the dengue fever. The analytical techniques involved in this paper can be applied by other researcher to develop other models. The study will also serve as baseline for further studies on the effect of vertical transmission in dengue fever dynamics and the vectorial capacity.

2 Model formulation

In this section, we formulate a vector-host epidemic model for the spread of dengue fever in the human and mosquito populations with the total populations at time t given by $N_h(t)$ and $N_v(t)$, respectively. The human (host) population is divided into susceptible, $S_h(t)$, exposed, $E_h(t)$, infected, $I_h(t)$ and recovered, $R_h(t)$ individuals. The mosquito (vector) population at time t is divided into susceptible, $S_v(t)$, exposed, $E_v(t)$, and infective, $I_v(t)$ class. The incubating classes, $E_h(t)$ and $E_v(t)$, reflect the viral intrinsic and extrinsic incubation periods $1/\alpha$ days and $1/\varepsilon$ days respectively. The extrinsic incubation period is the time necessary for virus to follow a cycle that brings it from the mosquito's stomach to its salivary gland and it varies depending on the factors like temperature. For humans, the intrinsic incubation period or latent period is the period from the point of infection to the beginning of infectiousness[5]. The vector component of the model does not include recovered class as mosquitoes do not recover from infection due to their relatively short life-cycle. In order to determine the effect of vertical transmission in the initial transmission of Dengue fever, we include both infected and uninfected eggs in the model. The infected eggs may hatch into either an infected mosquito or uninfected ones[5].

In this study we assume that the mixing of mosquito and human populations is homogeneous and that the development of dengue starts when the infectious female Aedes mosquito bites the human host. Infected human host may recover with temporary immunity while mosquitoes never recover from infection due to their relatively short life-cycle. Figure 1shows the compartmental diagram which has been created based on the made assumptions. The parameters indicated in Fig. 1 are described in Tab. 1.

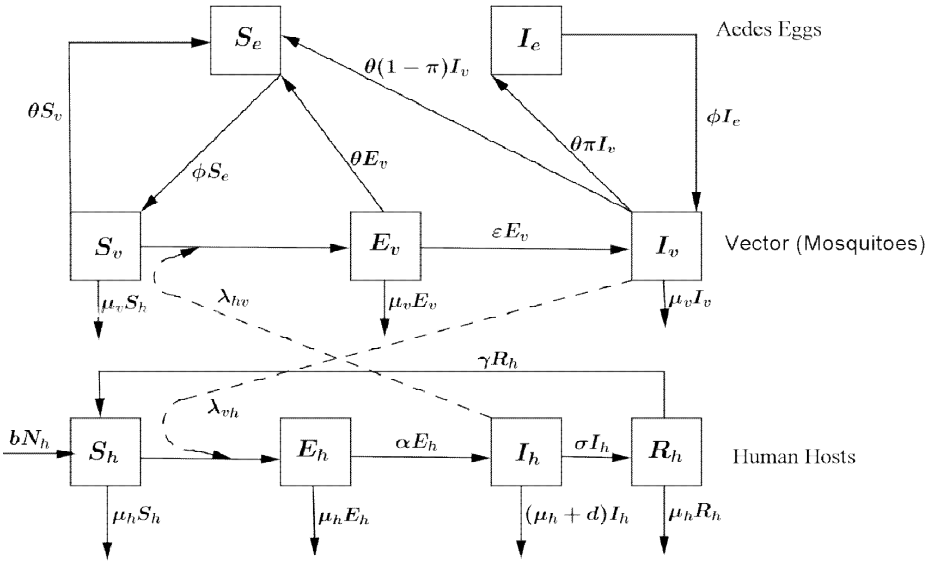

Fig. 1: The compartmental diagram for the dengue model

Table 1: Model parameters and their descriptions

Parameter	Value	Source	Description
b	0.0001	Assumed	Daily human recruitment rate
λ_{vh}	0.75	[3]	Effective contact rate (human to vector)
λ_{hv}	0.375	[3]	Effective contact rate (vector to human)
θ	0.005	[5]	Number of Aedes eggs laid per day
π	0.05	[1]	Vertical transmission rate in Aedes mosquitoes
ϕ	0.19day^{-1}	[2]	Development rate of Aedes mosquitoes
μ_v	0.03day^{-1}	[2]	Per capita natural death rate of mosquitoes
μ_h	1/365/60 day^{-1}	[5]	Per capita natural death rate of humans
$1/\varepsilon$	10days	[19]	Incubation period of mosquitoes
γ	0.1	[17]	Rate at which recovered individuals become susceptible
σ	1/365/60 day^{-1}	[2]	Recovery rate of humans
$1/\alpha$	10days	[19]	Incubation period of humans
d	0.01	[19]	Disease-induced mortality rate of humans

3 Model equations

Based on the assumptions and the inter-relations between the variables and the parameters as shown in the model compartments in Fig. 1, the effect of vertical transmission on Dengue transmission dynamics can be described by the following ordinary differential equations:

Human population

$$\frac{dS_h}{dt} = bN_h + \gamma R_h - \left(\mu_h + \lambda_{vh}\frac{I_v}{N_v}\right) S_h$$
$$\frac{dE_h}{dt} = \lambda_{vh}\frac{I_v}{N_v} S_h - (\alpha + \mu_h)\, E_h$$
$$\frac{dI_h}{dt} = \alpha E_h - (\sigma + d + \mu_h)\, I_h$$
$$\frac{dR_h}{dt} = \sigma I_h - (\gamma + \mu_h)\, R_h$$

Vector Population

$$\begin{aligned}
\frac{dS_v}{dt} &= \phi S_e - \left(\mu_v + \lambda_{hv}\frac{I_h}{N_h}\right) S_v \\
\frac{dE_v}{dt} &= \lambda_{hv}\frac{I_h}{N_h} S_v - (\varepsilon + \mu_v)\, E_v \\
\frac{dI_v}{dt} &= \varepsilon E_v + \phi I_e - \mu_v I_v
\end{aligned} \tag{1}$$

Eggs Population

$$\begin{aligned}
\frac{dS_e}{dt} &= \theta N_v - \theta\pi I_v - \phi S_e \\
\frac{dI_e}{dt} &= \theta\pi I_v - \phi I_e
\end{aligned}$$

with initial conditions:

$$S_h(0) \geq 0, E_h(0) \geq 0, I_h(0) \geq 0, S_v(0) \geq 0, E_v(0) \geq 0, I_v(0) \geq 0, S_e(0) \geq 0 \text{ and } I_e(0) \geq 0$$

In terms of proportions, the model Eq. (1) can be written as

$$\begin{aligned}
\frac{ds_h}{dt} &= b + \gamma r_h - (\mu_h + \lambda_{vh} i_v) s_h \\
\frac{de_h}{dt} &= \lambda_{vh} i_v s_h - (\alpha + \mu_h) e_h \\
\frac{di_h}{dt} &= \alpha e_h - (\sigma + d + \mu_h) i_h \\
\frac{dr_h}{dt} &= \sigma i_h - (\gamma + \mu_h) r_h \\
\frac{ds_v}{dt} &= \varphi s_e - (\mu_v + \lambda_{hv} i_h) s_v \\
\frac{de_v}{dt} &= \lambda_{hv} i_h s_v - (\varepsilon + \mu_v) e_v \\
\frac{di_v}{dt} &= \varepsilon e_v + \phi i_e - \mu_v i_v \\
\frac{ds_e}{dt} &= \theta - \theta\pi i_v - \phi s_e \\
\frac{di_e}{dt} &= \theta\pi i_v - \phi i_e
\end{aligned} \tag{2}$$

4 Disease free equilibrium (DFE), E_0

The disease free equilibrium of the model (2) is obtained by setting the LHS of (2) equals zero and $e_h = e_v = i_h = i_v = 0$. Further computation gives:

$$s_h = \frac{b}{\mu_h},\ s_v = \frac{\varphi}{\mu_v},\ \text{and} s_e = \frac{\theta}{\varphi}. \tag{3}$$

Therefore, the disease free equilibrium (DFE) of the model (2) is given by:

$$E_0 = \left(\frac{b}{\mu_h}, \frac{\varphi}{\mu_v}, \frac{\theta}{\varphi}, 0, 0, 0, 0, 0, 0\right).$$

5 The basic reproduction number, R_0

The basic reproduction number, R_0 is defined as the effective number of secondary infections caused by typical infected individual during his entire period of infectiousness[4]. This definition is given for the models that represent spread of infection in a population. We calculate the basic reproduction number by using the next generation operator method on the system (2).

The basic reproduction number is obtained by taking the largest (dominant) eigenvalue (spectral radius) of $\mathbf{FV}^{-1} = \left[\frac{\partial \mathcal{F}_i(E_0)}{\partial x_j}\right]\left[\frac{\partial \mathcal{V}_i(E_0)}{\partial x_j}\right]^{-1}$.

where $\mathcal{F}_i$ is the rate of appearance of new infection in compartment i, $\mathcal{V}_i$ is the transfer of infections from one compartment to another and E_0 is the disease-free equilibrium.

From system (2), we derive $\mathcal{F}_i$ and $\mathcal{V}_i$ as

$$\mathcal{F}_i = \begin{bmatrix} \mathcal{F}_1 \\ \mathcal{F}_2 \\ \mathcal{F}_3 \\ \mathcal{F}_4 \\ \mathcal{F}_5 \end{bmatrix} = \begin{bmatrix} \lambda_{vh} i_v s_h \\ 0 \\ \lambda_{hv} i_h s_v \\ 0 \\ 0 \end{bmatrix}$$

and

$$\mathcal{V}_i = \begin{bmatrix} \mathcal{V}_1 \\ \mathcal{V}_2 \\ \mathcal{V}_3 \\ \mathcal{V}_4 \\ \mathcal{V}_5 \end{bmatrix} = \begin{bmatrix} (\alpha + \mu_h) e_h \\ -\alpha e_h + (\sigma + d + \mu_h) i_h \\ (\varepsilon + \mu_v) e_v \\ -\varepsilon e_v - \phi i_e + \mu_v i_v \\ -\pi\theta i_v + \phi i_e \end{bmatrix}.$$

Using the linearization method, the associated matrix at DFE for F is given by:

$$\mathbf{F} = \begin{bmatrix} 0 & 0 & 0 & \lambda_{vh} s_h & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \lambda_{hv} s_v & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

On the other hand, the matrix $\mathbf{V}$ for the transfer of individuals out of the compartment i is given by;

$$\mathbf{V} == \begin{bmatrix} (\alpha + \mu_h) & 0 & 0 & 0 & 0 \\ -\alpha & (\sigma + d + \mu_h) & 0 & 0 & 0 \\ 0 & 0 & (\varepsilon + \mu_v) & 0 & 0 \\ 0 & -\varepsilon & 0 & \mu_v & -\phi \\ 0 & 0 & 0 & -\pi\theta & \phi \end{bmatrix}. \tag{4}$$

Finding the inverse of $\mathbf{V}$, we get:

$$\mathbf{V}^{-1} = \begin{bmatrix} \frac{1}{\alpha+\mu_h} & 0 & 0 & 0 & 0 \\ \frac{\alpha}{(\alpha+\mu_h)(\sigma+d+\mu_h)} & \frac{1}{(\sigma+d+\mu_h)} & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{(\varepsilon+\mu_v)} & 0 & 0 \\ 0 & 0 & \frac{\varepsilon}{(\varepsilon+\mu_v)(\mu_v-\pi\theta)} & \frac{1}{\mu_v-\pi\theta} & \frac{1}{\mu_v-\pi\theta} \\ 0 & 0 & \frac{\varepsilon\pi\theta}{\phi(\varepsilon+\mu_v)(\mu_v-\pi\theta)} & \frac{\pi\theta}{\phi(\mu_v-\pi\theta)} & \frac{\mu_v}{\phi(\mu_v-\pi\theta)} \end{bmatrix}.$$

For simplicity, $\mathbf{FV}^{-1}$ can be written as:

$$\mathbf{FV}^{-1} = \begin{bmatrix} 0 & 0 & \frac{\varepsilon\lambda_{vh}s_h}{(\varepsilon+\mu_v)(\mu_v-\pi\theta)} & \frac{\lambda_{vh}s_h}{(\mu_v-\pi\theta)} & \frac{\lambda_{vh}s_h}{(\mu_v-\pi\theta)} \\ \frac{\alpha\lambda_{hv}s_v}{(\alpha+\mu_h)(\sigma+d+\mu_h)} & \frac{\lambda_{hv}s_v}{(\sigma+d+\mu_h)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

Thus,

$$R_0 = \sqrt{\frac{\alpha\varepsilon\lambda_{vh}\lambda_{hv}s_v s_h}{(\alpha+\mu_h)(\sigma+d+\mu_h)(\varepsilon+\mu_v)(\mu_v-\pi\theta)}}. \tag{5}$$

Substituting (3) in (4) we have:

$$R_0 = \sqrt{\frac{b\phi\alpha\varepsilon\lambda_{vh}\lambda_{hv}}{\mu_v\mu_h(\alpha+\mu_h)(\sigma+d+\mu_h)(\varepsilon+\mu_v)(\mu_v-\pi\theta)}}. \tag{6}$$

When there is no vertical transmission, $\pi = 0$ we have:

$$R_0 = \sqrt{\frac{b\phi\alpha\varepsilon\lambda_{vh}\lambda_{hv}}{{\mu_v}^2\mu_h(\alpha+\mu_h)(\sigma+d+\mu_h)(\varepsilon+\mu_v)}}. \tag{7}$$

Eq. (5) and (6) shows the basic reproduction number increases as vertical transmission increases.

6 Sensitivity analysis

To find out how best we can do in order to reduce human mortality and morbidity due to dengue fever, it is essential to know the relative importance of the different factors responsible for its transmission and prevalence. We use sensitivity analysis to determine the robustness of model predictions to parameter values and discover parameters that have a high impact on basic reproduction number, R_0 that should be targeted by intervention strategies. In this section, we calculate the sensitivity indices of the reproductive number, R_0 to the parameters in model (2). The sensitivity indices tell us how vital each parameter is to disease transmission and prevalence. The explicit expression of R_0 is given by the Eq. (6). Since R_0 depends only on twelve parameters, we derive an analytical expression for its sensitivity to each parameter using the normalized forward sensitivity index[9] as follows:

$$\Upsilon_b^{R_0} = \frac{\partial R_0}{\partial b} \times \frac{b}{R_0} = +0.5$$

$$\Upsilon_{\mu_v}^{R_0} = \frac{\partial R_0}{\partial \mu_v} \times \frac{\mu_v}{R_0} = -1.15$$

$$\Upsilon_\pi^{R_0} = \frac{\partial R_0}{\partial \pi} \times \frac{\pi}{R_0} = +0.045.$$

In a similar manner we compute the sensitivity indices for all parameters used in Eq. (6). Tab. 2 shows the sensitivity indices of R_0 with respect to the twelve parameters.

Tab. 2, shows that, the most sensitive parameters are per capita natural death rate of mosquitoes, μ_v, daily human recruitment rate, b, effective contact rate (human to vector), λ_{hv}, effective contact rate (vector to human), λ_{vh}, and per capita natural death rate of human, μ_h. For instance, $\Upsilon_{\mu_v}^{R_0} = -1.147$, means increasing or decreasing of μ_v by 10% decreases or increases R_0 by 11.47%. Similarly, $\Upsilon_\pi^{R_0} = +0.045$ means that, increasing or decreasing of π by 10% increases or decreases R_0 by 0.45%. Thus, to reduce or eradicate dengue fever in the community, we should focus on an intervention that kills most of the *aedes aegypti* mosquitoes and their eggs.

7 Numerical results and discussion

This section presents numerical simulations for model system (2) for the purpose of verifying some of the analytical results. The parameters values used in our computations are mainly from literature as well as estimation in order to have more realistic simulation results. The parameter values and their respective sources are in Tab. 1 and Fig. 2 illustrates the variations in human subpopulations as time increases. .

Table 2: Sensitivity indices of R_0 with respect to each parameter

Parameter	Parameter sensitivity index
b	+0.5
λ_{hv}	$+0.5$
λ_{vh}	$+0.5$
ϕ	$+0.5$
α	-0.49
ε	-0.49
d	-0.033
π	$+0.045$
θ	$+0.045$
μ_v	-1.147
μ_h	-0.5
σ	-0.47

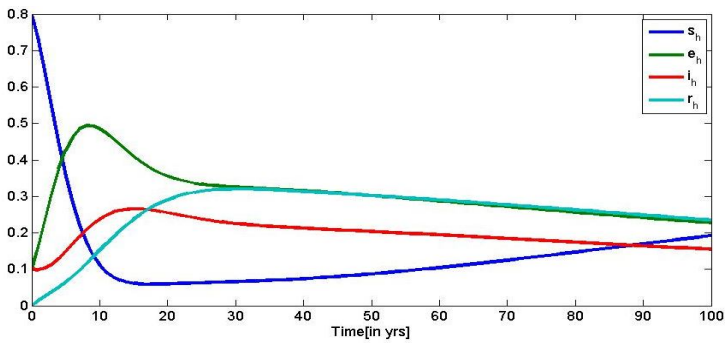

Fig. 2: Time series of human subpopulations

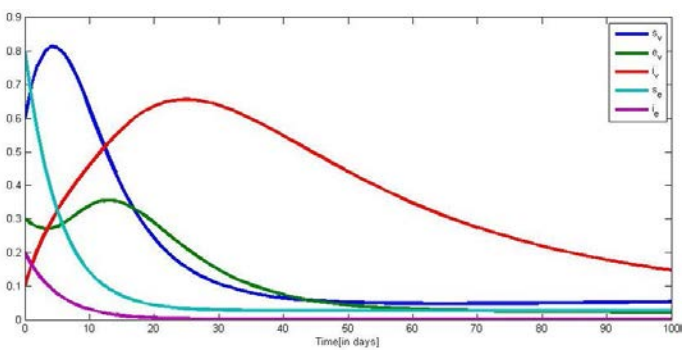

Fig. 3: Time series of vector subpopulations

Fig. 2 shows that susceptible people decreases rapidly due to dengue epidemic while the exposed as well as the infectious people initially increases with time but after sometime these subpopulation decreases with time. The increase is due high dengue transmission rate and the decrease is due to different interventions like treatment and other precautions taken by the society. The graph for recovered group initially increases because high treatment rate as most the dengue cases need immediate attention and the decrease is due to decrease in dengue cases.

Furthermore, Fig. 3 shows variations in vector subpopulations as time increases.

Fig. 3 shows that susceptible *aedes egypti* mosquitoes decrease rapidly due to dengue epidemic while the infectious mosquitoes initially increase with time but after sometime these subpopulation decreases with time. The increase is due high dengue transmission rate from infected human and the decrease is due to different interventions and precautions taken by human and the short lifespan for mosquitoes. Susceptible eggs decrease due to vertical transmission in infected *aedes aegypti* mosquitoes.

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