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A Nonlinear Malaria Transmission Epidemic Model's Stability Analysis Implementing an Optimized Numerical Approach

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Abstract

Malaria remains one of the major health concerns that has plagued humanity for thousands of years, especially in tropical and subtropical areas. In this study,the dynamics of malaria will be discussed precisely and a detailed SEIHRD (Susceptible - Exposed - Infected - Hospitalized - Recovered - Deceased) model will be proposed that search for the transmission of this vector-borne disease involving the interaction between human hosts and female Anopheles mosquitoes. Key biological and clinical features such as incubation period of both human hosts and mosquitoes,period of infection, hospitalization etc. are integrated in this model. The basic reproduction number R_0 is determined by using next generation matrix method and stability analysis for both disease free equilibrium (DFE) and endemic equilibrium (EE) are discussed thoroughly. Numerical simulation is conducted by using system's dynamics under different observations. MATLAB programming language has been used for the simulation consisting of Runge-Kutta method and algebraic computation. It explores numerous strategies to minimize the effects of malaria by gathering information conducted from simulation. It portrays an important framework about public health and provides a better methodology on modeling modern and effective procedures to control this mosquito-borne disease.

Keywords: Malaria, SEIHRD, Anopheles Mosquitoes,Basic Reproduction Number, Stability

1. Introduction

Malaria is one of the oldest and deadliest infectious diseases ever known to mankind. References to periodic fevers resembling malaria date back to ancient Egyptian and Chinese medical texts[10].Hippocrates (460–370 BC) documented symptoms consistent with malaria, recognizing its association with swampy areas [5].The discovery that mosquitoes were the vectors came much later when Sir Ronald Ross demonstrated the transmission of avian malaria by the *Anopheles* mosquito in 1897,a landmark discovery that revolutionized our understanding of disease transmission [29].In spite of years of medical and research efforts to eradicate malaria,it is still a major health issue worldwide especially in the developing and under-developed countries. According to the World Health Organization (WHO),almost 249 million cases of malaria occurred worldwide in 2022,resulting in approximately 608,000 deaths [41].The vast majority of malaria cases and deaths occurring in the sub-Saharan Africa where children under five years and pregnant women are the most vulnerable groups[35].In total,five species of *Plasmodium* parasites are the reason of malaria in human population: *P. falciparum*, *P. vivax*, *P. malariae*, *P. ovale*, and *P. knowlesi*. *P. falciparum* is the most prevalent and deadly species, accounting for the majority of severe cases and deaths [40].

An infected female *Anopheles* mosquito acting as the biological vector is the carrier of malaria to human through biting. When an infected mosquito bites,sporozoites enter the human blood cells and travels to the human liver where they breed and grow in number. There are over 400 *Anopheles* species but only about 30 are significant vectors of malaria [3].These mosquitoes have features like spotted wings and long palps that are equal in length to the proboscis. Adult *Anopheles* mosquitoes typically rest at a 45-degree angle to the surface with their abdomen sticking up rather than parallel to the resting surface which helps distinguishing them from other genera [9].Female *Anopheles* mosquitoes need blood for egg development. Moreover, the life expectancy and the breeding locations of *Anopheles* species also influence malaria transmission. Like all mosquitoes, *Anopheles* species goes through a thorough metamorphosis having four different stages. These stages are egg, larva, pupa, and adult. Environmental factors including temperature,rainfall and humidity also influence transmission dynamics [17].In infected localities, people often spend a significant proportion of their earnings on preventing and treatment of malaria. The disease also hampers economic development with estimates suggesting that malaria-endemic countries may experience a growth penalty of up to 1.3% of GDP annually [15].

Malaria comes with numerous observable signs and symptoms which depend on the *Plasmodium* parasite and it's load,immunity of host and age as well. One of the most predictable and early symptoms of malaria are fever with chills and stiffness, headache , excessive sweating, body and muscle ache, nausea, loss of appetite, extreme weakness and fatigue. These symptoms normally can be seen within 10-15 days of getting bitten by the infected mosquito. Symptoms can be mild for people who were infected before. In severe cases, particularly with *Plasmodium falciparum* infection,life-threatening complications may arise and can even lead to death. These include cerebral malaria (impaired consciousness and seizures), severe anemia, hypoglycemia, acute respiratory distress syndrome (ARDS), metabolic acidosis, and multi-organ failure [41, 2].

The immune response to parasitic diseases is a complex interplay between the innate and adaptive immune system. Body's first line of defense against pathogens and foreign substances is the innate immune response. Innate defenses include the activity of macrophages, natural killer (NK) cells and dendritic cells which recognize infected red blood cells and help contain early infection. The adaptive immune response is a defense mechanism that develops after exposure to a pathogen or vaccination. Genetic factors can significantly influence an individual's susceptibility to parasitic infections.

These factors involve specific gene variations that can affect immune responses,red blood cell structures and other cellular mechanisms. For example,the sickle cell trait (heterozygous HBAS genotype) confers significant protection against severe *P. falciparum* malaria. Other genetic polymorphisms such as G6PD deficiency and the absence of Duffy antigen (protecting against *P. vivax*) demonstrate the evolutionary pressure exerted by malaria on human population [39].Despite these defenses, parasites employ diverse strategies to evade the host immune system including hiding,becoming invisible,changing their identity and actively manipulating the immune response .These mechanisms allow parasites to survive and persist within the host often leading to chronic infections.

This study combines compartmental equations with numerical simulation to explore various disease dynamics under different conditions. It provides mathematical framework to analyze the outbreak patterns, assuring the control measures.

2. Model Formulation and Analysis

In this section, properties of the model and stability in different equilibria have been attained following by the SEIHRD model and the dynamic equations of the model. It consists of both host (human) population having six distinct classes and vector (mosquito) population with three distinct classes. Human population consist of susceptible (S_h), exposed (E_h), infectious (I_h), hospitalized (H_h), recovered (R_h), and deceased (D_h) classes. Hence, total living population of human is N_h=S_h+E_h+I_h+H_h+R_h and the total living mosquito population is N_m=S_m+E_m+I_m.

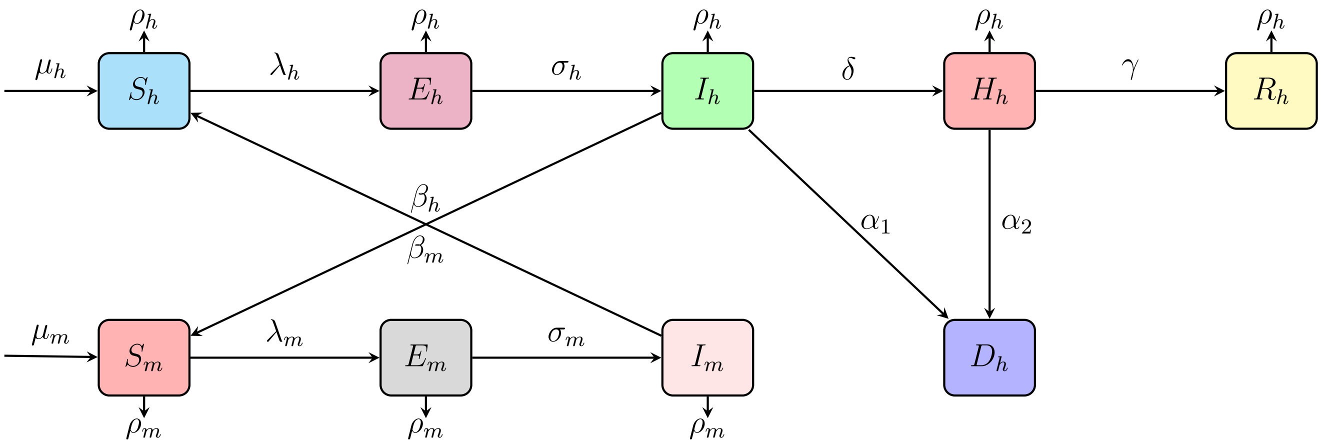

Figure 1: SEIHRD malaria transmission model with natural death/removal shown as outgoing flows.

The following table presents the parameters and variables with description:

Parameters	**Description**
S_h	Susceptible class for human population
E_h	Exposed class for human population
I_h	Infected class for human population
H_h	Hospitalized class for human population
R_h	Recovery class for human population
D_h	Deceased class for human population
S_m	Susceptible class for mosquito population
E_m	Exposed class for mosquito population
I_m	Infected class for mosquito population
μ_h	Required human rate
λ_h	Rate at which susceptible human gets exposed to the infection
β_h	Transmission rate from infectious mosquitoes to susceptible humans
β_m	Transmission rate from infectious humans to susceptible mosquitoes
σ_h	Rate at which exposed humnans become infectious
δ	Hospitalization rate of infectious humans
α_1	Malaria induced death rate from infectious humans(before hospitalization)
α_2	Malaria induced death rate from hospitalized humans
γ	Recovery rate from hospitalization
μ_m	Required mosquito rate
λ_m	Rate at which susceptible mosquitoes get exposed to the infection
σ_m	Rate at which exposed mosquitoes become infectious
ρ_h	Natural death rate of humans
ρ_m	Natural death rate of mosquitoes
b	mosquito bite per day
N_h	Total human population
N_m	Total mosquito population

Table 1: Parameters and Variables with Descriptions

2.1 Dynamic Equations of the SEIHRD Model for Human and Mosquito Population

$$\begin{aligned}
\frac{dS_h}{dt} &= \mu_h - \lambda_h S_h - \rho_h S_h \\
\frac{dE_h}{dt} &= \lambda_h S_h - \sigma_h E_h - \rho_h E_h \\
\frac{dI_h}{dt} &= \sigma_h E_h - \delta I_h - \alpha_1 I_h - \rho_h I_h \\
\frac{dH_h}{dt} &= \delta I_h - \gamma H_h - \alpha_2 H_h - \rho_h H_h \\
\frac{dR_h}{dt} &= \gamma H_h - \rho_h R_h \\
\frac{dD_h}{dt} &= \alpha_1 I_h + \alpha_2 H_h - \rho_h D_h \\
\frac{dS_m}{dt} &= \mu_m - \lambda_m S_m - \rho_m S_m \\
\frac{dE_m}{dt} &= \lambda_m S_m - \sigma_m E_m - \rho_m E_m \\
\frac{dI_m}{dt} &= \sigma_m E_m - \rho_m I_m
\end{aligned} \tag{1}$$

2.2 Properties of the Model

In this part, some basic qualitative properties of solutions of the model (1) are proven.The properties can be discussed by the following theorem:

Theorem: For all time t, the model (1) has positive variables. With positive initial data,the solution of the model will stay positive for all time t>0.

Proof: Let at initial points, $S_h(0) \geq 0,\ E_h(0) \geq 0,\ I_h(0) \geq 0,\ H_h(0) \geq 0,\ R_h(0) \geq 0,\ D_h(0) \geq 0,\ S_m(0) \geq 0,\ E_m(0) \geq 0,\ I_m(0) \geq 0.$

From the first equation,

$$\begin{aligned}
\frac{dS_h}{dt} &= \mu_h - \lambda_h S_h - \rho_h S_h \\
&\geq S_h(-\lambda_h - \rho_h)
\end{aligned}$$

Hence, $S_h(t) \geq S_h(0)e^{-(\lambda_h+\rho_h)t} > 0$

Again,

$$\begin{aligned}
\frac{dS_m}{dt} &= \mu_m - \lambda_m S_m - \rho_m S_m \\
&\geq S_m(-\lambda_m - \rho_m)
\end{aligned}$$

So, $S_m(t) \geq S_m(0)e^{-(\lambda_m-\rho_m)t} > 0$

Similarly, all the compartments of the model (1) are greater than zero or positive for all time t>0.

2.3 Equilibrium Point and Stability Analysis

The model (1) has two types of equilibria. These are disease free equilibrium (DFE) and endemic equilibrium (EE) .The equilibrium is obtained by setting the right side of equations in (1) to zero.

At any equilibrium,

$$\frac{dS_h}{dt} = \frac{dE_h}{dt} = \frac{dI_h}{dt} = \frac{dH_h}{dt} = \frac{dR_h}{dt} = \frac{dD_h}{dt} = \frac{dS_m}{dt} = \frac{dE_M}{dt} = \frac{dI_m}{dt} = 0$$

hence for human,

$$\begin{aligned}
\mu_h - \lambda_h S_h - \rho_h S_h = 0\\
\lambda_h S_h - \sigma_h E_h - \rho_h E_h = 0\\
\sigma_h E_h - \delta I_h - \alpha_1 I_h - \rho_h I_h = 0\\
\delta I_h - \gamma H_h - \alpha_2 H_h - \rho_h H_h = 0\\
\gamma H_h - \rho_h R_h = 0\\
\alpha_1 I_h + \alpha_2 H_h - \rho_h D_h = 0
\end{aligned}$$

Now, solving the above equation,

$$\begin{aligned}
S_h &= \frac{\mu_h}{\rho_h + \lambda_h}\\
E_h &= \frac{\lambda_h \mu_h}{(\sigma_h + \rho_h)(\rho_h + \lambda_h)}\\
I_h &= \frac{\sigma_h \lambda_h \mu_h}{(\delta + \rho_h + \alpha_1)(\sigma_h + \rho_h)(\rho_h + \lambda_h)}\\
H_h &= \frac{\delta \sigma_h \lambda_h \mu_h}{(\gamma + \rho_h + \alpha_2)(\delta + \rho_h + \alpha_1)(\sigma_h + \rho_h)(\rho_h + \lambda_h)}\\
R_h &= \frac{\gamma \delta \sigma_h \lambda_h \mu_h}{(\gamma + \rho_h + \alpha_2)(\delta + \rho_h + \alpha_1)(\sigma_h + \rho_h)(\rho_h + \lambda_h)}\\
D_h &= \frac{\sigma_h \lambda_h \mu_h}{\rho_h(\rho_h + \lambda_h)(\rho_h + \sigma_h)(\delta + \rho_h + \alpha_1)}[\frac{\delta \alpha_2}{(\gamma + \rho_h + \alpha_2)} + \alpha_1]
\end{aligned} \tag{2}$$

and for mosquito,

$$\mu_m - \lambda_m S_m - \rho_m S_m = 0$$
$$\lambda_m S_m - \sigma_m E_m - \rho_m E_m = 0$$
$$\sigma_m E_m - \rho_m I_m = 0$$

After solving the above equations,

$$S_m = \frac{\mu_m}{\lambda_m + \rho_m}$$
$$E_m = \frac{\lambda_m \mu_m}{(\sigma_m + \rho_m)(\lambda_m + \rho_m)} \tag{3}$$
$$I_m = \frac{\sigma_m \lambda_m \mu_m}{\rho_m(\sigma_m + \rho_m)(\lambda_m + \rho_m)}$$

2.4 Stability for Disease Free Equilibrium (DFE)

For DFE all the other compartments are zero except for S_h and S_m. Thus
$E_h^* = I_h^* = H_h^* = R_h^* = D_h^* = E_m^* = I_m^* = 0$
So, DFE is given by

$$\overline{E_0} = (S_h^*, E_h^*, I_h^*, H_h^*, R_h^*, D_h^*, S_m^*, E_m^*, I_m^*) = (\frac{\mu_h}{\rho_h}, 0, 0, 0, 0, 0, \frac{\mu_m}{\rho_m}, 0, 0)$$

2.4.1 Local Stability of DFE

In this section, the local stability of the DFE can be found by using the next generation matrix of the system (1) and then the calculation of the basic reproduction number of the given model will be attained.

Considering the compartments related to the infection,

$$\frac{dE_h}{dt} = \lambda_h S_h - \sigma_h E_h - \rho_h E_h$$
$$\frac{dI_h}{dt} = \sigma_h E_h - \delta I_h - \alpha_1 I_h - \rho_h I_h$$
$$\frac{dH_h}{dt} = \delta I_h - \gamma H_h - \alpha_2 H_h - \rho_h H_h$$
$$\frac{dE_m}{dt} = \lambda_m S_m - \sigma_m E_m - \rho_m E_m$$
$$\frac{dI_m}{dt} = \sigma_m E_m - \rho_m I_m$$

From the above equations, transmission matrix F(associated with new infection terms) and transition matrix V(considering transferred terms) are attained.

Also,from the model (1),

$$\lambda_h = \frac{\beta_h I_m}{N_m}$$
$$\lambda_m = \frac{\beta_m I_h}{N_h}$$

Now,

$$f = \begin{bmatrix} E_h \\ I_h \\ H_h \\ E_m \\ I_m \end{bmatrix}$$

After calculating E_h and E_m,

$$f = \begin{bmatrix} \frac{\beta_h I_m}{N_m} S_h \\ 0 \\ 0 \\ \frac{\beta_m I_h}{N_h} S_m \\ 0 \end{bmatrix}$$

So,

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\beta_h}{N_m} S_h \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_m}{N_h} S_m & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

and

$$v = \begin{bmatrix} E_h(\sigma_h + \rho_h) \\ -E_h\sigma_h + I_h(\delta + \rho_h + \alpha_1) \\ -\delta I_h + H_h(\gamma + \rho_h + \alpha_2) \\ E_m(\sigma_m + \rho_2) \\ -E_m\sigma_m + \rho_m I_m \end{bmatrix}$$

Hence,

$$V = \begin{bmatrix} \sigma_h + \rho_h & 0 & 0 & 0 & 0 \\ -\sigma_h & \delta + \rho_h + \alpha_1 & 0 & 0 & 0 \\ 0 & -\delta & \gamma + \rho_h + \alpha_2 & 0 & 0 \\ 0 & 0 & 0 & -\sigma_m + \rho_m & 0 \\ 0 & 0 & 0 & -\sigma_m & \rho_m \end{bmatrix}$$

Here,let,

$a = \delta + \rho_h + \alpha_1$
$b = \gamma + \rho_h + \alpha_2$

So,V becomes

$$V = \begin{bmatrix} \sigma_h + \rho_h & 0 & 0 & 0 & 0 \\ -\sigma_h & a & 0 & 0 & 0 \\ 0 & -\delta & b & 0 & 0 \\ 0 & 0 & 0 & -\sigma_m + \rho_m & 0 \\ 0 & 0 & 0 & -\sigma_m & \rho_m \end{bmatrix}$$

Now the inverse of the transition matrix is

$$V^{-1} = \begin{bmatrix} \frac{1}{\sigma_h+\rho_h} & 0 & 0 & 0 & 0 \\ \frac{\sigma_h}{a(\sigma_h+\rho_h)} & \frac{1}{a} & 0 & 0 & 0 \\ \frac{\delta\sigma_h}{ab(\sigma_h+\rho_h)} & \frac{\delta}{ab} & \frac{1}{b} & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{\sigma_m+\rho_m} & 0 \\ 0 & 0 & 0 & \frac{\sigma_m}{\rho_m(\sigma_m+\rho_m)} & \frac{1}{\rho_m} \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} 0 & 0 & 0 & \frac{S_h\beta_h\sigma_m}{N_m\rho_m(\sigma_m+\rho_m)} & \frac{S_h\beta_h}{N_m\rho_m} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{S_m\beta_m\sigma_h}{aN_h(\sigma_h+\rho_h)} & \frac{S_m\beta_m}{aN_h} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

and the eigenvalues of FV^{-1} are

$$0, 0, 0, \sqrt{\frac{S_hS_m\beta_h\beta_m\sigma_h\sigma_m}{N_hN_m\rho_m(\sigma_h+\rho_h)(\sigma_m+\rho_m)(\delta+\rho_h+\alpha_1)}}, -\sqrt{\frac{S_hS_m\beta_h\beta_m\sigma_h\sigma_m}{N_hN_m\rho_m(\sigma_h+\rho_h)(\sigma_m+\rho_m)(\delta+\rho_h+\alpha_1)}}$$

Hence basic reproduction number of a model is

$$R_0 = \sqrt{\frac{S_hS_m\beta_h\beta_m\sigma_h\sigma_m}{N_hN_m\rho_m(\sigma_h+\rho_h)(\sigma_m+\rho_m)(\delta+\rho_h+\alpha_1)}}$$

If $R_0 < 1$ then the DFE is locally asymptotically stable that is the disease will not persist in the community. Whereas if $R_0 > 1$ then it is unstable and the disease will be spread out [31, 11, 22, 27, 26].

2.4.2 Global Stability of DFE

Before working on the global stability of the DFE, it is necessary to prove the following lemma :

Lemma: The region of the model (1) is positively invariant and attracting for the model (1).

Proof: From the first equation of the model (1), where $S_h^* = \frac{\mu_h}{\rho_h}$;

$$\frac{dS_h}{dt} = \mu_h - \lambda_h S_h(t) - \rho_h S_h(t)$$
$$\leq \mu_h - \rho_h S_h(t)$$
$$= \rho_h \left[\frac{\mu_h}{\rho_h} - S_h(t)\right]$$
$$= \rho_h[S_h^* - S_h(t)]$$

Thus,

$$S_h(t) \leq S_h^* - [S_h^* - S_h(0)]e^{-\rho_h t}.$$

Therefore, if $N_h(t) \leq \frac{\mu_h}{\rho_h}$ and $S_h(0) \leq \frac{\mu_h}{\rho_h}$, then either $S_h(t) \to S_h$ as $t \to \infty$ or after finite time t, $S_h(t) \leq S_h^*$ since

$$\frac{dS_h}{dt} < 0 \quad \text{for} \quad S_h(t) > S_h^*.$$

Similarly, from the seventh equation of (1) where $S_m^* = \frac{\mu_m}{\rho_m}$,

$$\frac{dS_m}{dt} = \mu_m - \lambda_m S_m(t) - \rho_m S_m(t)$$
$$\leq \mu_m - \rho_m S_m(t)$$
$$= \rho_m \left[\frac{\mu_m}{\rho_m} - S_m(t)\right]$$
$$= \rho_m[S_m^* - S_m(t)]$$

Therefore, if $N_m(t) \leq \frac{\mu_m}{\rho_m}$ and $S_m(0) \leq \frac{\mu_m}{\rho_m}$, then either $S_m(t) \to S_m$ as $t \to \infty$ or after finite time t, $S_m(t) \leq S_m^*$ since

$$\frac{dS_m}{dt} < 0 \quad \text{for} \quad S_m(t) > S_m^*.$$

Therefore,the region of the model (1) is positively invariant, attracting all the solutions i.e. globally asymptotic.

Now the following theorem can be claimed:

Theorem: The DFE of the model is globally asymptotically stable if $R_0 < 1$.

Proof: Construct a Lyapunov function[32],

$$V = aE_h + bI_h + cH_h + dE_m + eI_m$$

having $a, b, c, d, e > 0$ such that $\dot{V} \leq 0$ if $R_0 < 1$.

Now, assume $S_h \approx 1$ and $S_m \approx 1$.

$$\dot{V} = a\dot{E}_h + b\dot{I}_h + c\dot{H}_h + d\dot{E}_m + e\dot{I}_m \tag{4}$$

$$\begin{aligned}\dot{V} =& a\Big[\lambda_h - (\sigma_h + \rho_h)E_h\Big] + b\Big[\sigma_h E_h - (\delta + \rho_h + \alpha_1)I_h]\Big) + c\Big[\delta I_h - (\rho_h + \gamma + \alpha_2)H_h\Big]\\ &+ d\Big[\lambda_m - (\sigma_m + \rho_m)E_m\Big] + e\Big(\sigma_m E_m - \rho_m I_m\Big).\end{aligned}$$

Here, from the above equation,clearly $a\lambda_h$ and $d\lambda_m$ increase the infection.
to eliminate them, let,

$$a = \frac{e\rho_m N_m}{\beta_h} \implies a\beta_h = e\rho_m N_m$$

$$d = \frac{b(\delta + \rho_h + \alpha_1)N_h}{\beta_m} \implies d\beta_m = b(\delta + \rho_h + \alpha_1)N_h$$

putting the value in equation (4),

$$\begin{aligned}\dot{V} =& a\Big[\frac{\beta_h I_m}{N_m} - (\sigma_h + \rho_h)E_h\Big] + b\Big[\sigma_h E_h - (\delta + \rho_h + \alpha_1)I_h\Big] + c\Big[\delta I_h - (\rho_h + \gamma + \alpha_2)H_h\Big]\\ &+ d\Big[\frac{\beta_m I_h}{N_h} - (\sigma_m + \rho_m)E_m\Big] + e\Big(\sigma_m E_m - \rho_m I_m\Big)\\ &= -[a(\sigma_h + \rho_h) - b\sigma_h]E_h - [d(\sigma_m + \rho_m) - e\sigma_m]E_m\end{aligned}$$

From above $\dot{V} < 0$ if $R_0 < 1$ and $\dot{V} = 0$ if and only if $I_h = I_m = 0$.

From Lasalle's invariance principle[24] $E_h \to 0$, $E_m \to 0$ as $t \to \infty$ that indicates the elimination of the disease.

Again,

$$E_h = I_h = H_h = R_h = D_h = E_m = I_m = 0$$

then

$$S_h \to S_h^*, S_m \to S_m^*$$

as $t \to \infty$

$$\lim_{t\to\infty}(S_h, E_h, I_h, H_h, R_h, D_h, S_m, E_m, I_m) = (S_h^*, 0, 0, 0, 0, 0, S_m^*, 0, 0) = \bar{E}_0$$

Therefore, for the condition $R_0 < 1$, the DEF is globally asymptotically stable.

2.5 Stability of Endemic Equilibrium(EE)

2.5.1 Existence of Endemic Equilibrium

In this section, to find the condition for the existence of endemic equilibrium, let the endemic equilibrium point of the model (1) be

$$\overline{E_1} = (S_h^{**}, E_h^{**}, I_h^{**}, H_h^{**}, R_h^{**}, D_h^{**}, S_m^{**}, E_m^{**}, I_m^{**})$$

and at EE, equations of (2) and (3) become

$$\frac{dE_h}{dt} = 0$$

so,

$$E_h = \frac{\lambda_h S_h}{\sigma_h + \rho_h}$$

Similarly,

$$\frac{dI_h}{dt} = 0$$

Therefore,

$$\begin{aligned} I_h &= \frac{\sigma_h \lambda_h S_h}{(\delta + \rho_h + \alpha_1)(\sigma_h + \rho_h)} \\ &= \frac{\sigma_m \beta_h I_m S_h}{(\delta + \rho_h + \alpha_1)(\sigma_h + \rho_h) N_m} \\ &= K_1 S_h I_m \end{aligned}$$

where $K_1 = \frac{\sigma_m \beta_h}{(\delta+\rho_h+\alpha_1)(\sigma_h+\rho_h)N_m}$

Again,

$$\frac{dE_m}{dt} = 0$$

so,

$$E_m = \frac{\lambda_m S_m}{\sigma_m + \rho_m}$$

Likewise,

$$\frac{dI_m}{dt} = 0$$

$$\begin{aligned} I_m &= \frac{\sigma_m \; E_m}{\rho_m} \\ &= \frac{\sigma_m \lambda_m S_m}{\rho_m(\sigma_m + \rho_m)} \\ &= \frac{\sigma_m \beta_m S_m}{N_h \rho_m(\sigma_m + \rho_m)} S_h I_m K_1 \\ &= S_h S_m I_m K_2 \end{aligned}$$

where,

$$K_2 = \frac{\alpha_m \beta_m K_1}{N_h \rho_m(\sigma_m + \rho_m)}$$

Therefore,

$$I_m(1 - K_2 S_m S_h) = 0$$

from here, two solutions are,

$I_m = 0$ which is a DFE.

and

$$\begin{aligned}(1 - K_2 S_m S_h) &= 0\\ \implies K_2 S_m S_h &= 1\end{aligned}$$

We know,

$$\begin{aligned}N_h &= S_h + E_h + I_h + \dots\\ \implies S_h &= N_h - E_h - I_h - \dots\\ &= \frac{\mu_h}{\rho_h} - E_h - I_h\\ &\approx S_h^{**} - cI_m\end{aligned}$$

Similarly,

$$S_m \approx S_m^{**} - dI_m$$

Hence the equation becomes,

$$\begin{aligned}K_2(S_h^{**} - cI_m)(S_m^{**} - dI_m) &= 1\\ \implies K_2 cd I_m^2 - K_2(cS_m^{**} + dS_h^{**})I_m + (K_2 S_h^{**} S_m^{**} - 1) &= 0\end{aligned}$$

This can be expressed as,

$$AI_m^2 + BI_m + C = 0$$

To have a existence of EE, discriminant must be greater than zero i.e. roots must be positive.

Here,

$$\begin{aligned}C = K_2 S_h^{**} S_m^{**} - 1 &= \frac{\sigma_h \sigma_m \beta_h \beta_m S_h^{**} S_m^{**}}{N_h N_m \rho_m(\sigma_m + \rho_m)(\sigma_h + \rho_h)(\delta + \rho_h + \alpha_1)} - 1\\ &= R_0^2 - 1\end{aligned}$$

So, if $R_0^2 > 1$ then $C > 0$. Hence the system can have a positive root which means EE exists and if $R_0^2 \leq 1$ then $C \leq 0$. Thus EE does not exist[20].

Since $R_0^2 = C + 1$, hence $R_0^2 > 1$. This concludes $C > 0$. So, $I_m^{**} > 0$ and hence EE exists.

2.5.2 Local Stability of EE

Theorem: Whenever the basic reproduction number $R_0 > 1$, the endemic equilibrium (EE) of the SEIHRD model with a mosquito vector is locally asymptotically stable [13].

Proof: Consider the SEIHRD model extended with human and mosquito compartments:

Human population:

$$\frac{dE_h}{dt} = \lambda_h S_h - (\sigma_h + \rho_h)E_h,$$
$$\frac{dI_h}{dt} = \sigma_h E_h - (\delta + \rho_h + \alpha_1)I_h,$$
$$\frac{dH_h}{dt} = \delta I_h - (\gamma + \rho_h + \alpha_2)H_h,$$

Mosquito population:

$$\frac{dE_m}{dt} = \lambda_m S_m - (\sigma_m + \rho_m)E_m,$$
$$\frac{dI_m}{dt} = \sigma_m E_m - \rho_m I_m.$$

At endemic equilibrium, linearizing the infected subsystem,

$$F = (E_h^{**}, I_h^{**}, H_h^{**}, E_m^{**}, I_m^{**})^T$$

Here,

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\beta_h S_h^{**}}{N_m} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_m S_m^{**}}{N_h} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} \sigma_h + \rho_h & 0 & 0 & 0 & 0 \\ -\sigma_h & \delta + \rho_h + \alpha_1 & 0 & 0 & 0 \\ 0 & -\delta & \gamma + \rho_h + \alpha_2 & 0 & 0 \\ 0 & 0 & 0 & -\sigma_m + \rho_m & 0 \\ 0 & 0 & 0 & -\sigma_m & \rho_m \end{bmatrix}.$$

The next generation matrix is $K = FV^{-1}$ [12] and the basic reproduction number is $R_0 = \rho(K)$.

where

$$K = \begin{bmatrix} 0 & 0 & 0 & \frac{\sigma_m \beta_h S_h^{**}}{N_m \rho_m (\sigma_m + \rho_m)} & \frac{\beta_h S_h^{**}}{N_m \rho_m} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\sigma_h \beta_m S_m^{**}}{N_h (\delta + \rho_h + \alpha_1)(\sigma_h + \rho_h)} & \frac{\beta_m S_m^{**}}{N_h (\delta + \rho_h + \alpha_1)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

The characteristic polynomial is,

$$\det(\lambda I - K) = 0$$

and the eigenvalues of K are

$$0, 0, 0, \sqrt{\frac{S_h^{**} S_m^{**} \beta_h \beta_m \sigma_h \sigma_m}{N_h N_m \rho_m (\sigma_h + \rho_h)(\sigma_m + \rho_m)(\delta + \rho_h + \alpha_1)}}, -\sqrt{\frac{S_h^{**} S_m^{**} \beta_h \beta_m \sigma_h \sigma_m}{N_h N_m \rho_m (\sigma_h + \rho_h)(\sigma_m + \rho_m)(\delta + \rho_h + \alpha_1)}}$$

Hence, basic reproduction number of the model is

$$R_0 = \sqrt{\frac{S_h^{**} S_m^{**} \beta_h \beta_m \sigma_h \sigma_m}{N_h N_m \rho_m (\sigma_h + \rho_h)(\sigma_m + \rho_m)(\delta + \rho_h + \alpha_1)}}$$

where all the parameters are positive and biologically meaningful.

After computing the Jacobian analytically,the Center Manifold Theorem is introduced that provides a strong framework to analyze local stability. From here, we find if $R_0 > 1$ and a unique endemic equilibrium exists within a specific region,then EE is locally asymptotically stable. From R_0, the total transmission of both human hosts and mosquitoes overshadows the effects of recovery or hospitalization, hence $R_0 > 1$ and if the dynamic system will remain close to the region of EE.

Since our model satisfies the conditions of the Center Manifold Theorem,hence the EE is locally asymptotically stable.

2.5.3 Global Stability in EE

From the equation of the model,

$$\begin{aligned}
\frac{dE_h}{dt} &= \lambda_h S_h - \sigma_h E_h - \rho_h E_h \\
\frac{dI_h}{dt} &= \sigma_h E_h - \delta I_h - \alpha_1 I_h - \rho_h I_h \\
\frac{dH_h}{dt} &= \delta I_h - \gamma H_h - \alpha_2 H_h - \rho_h H_h \\
\frac{dE_m}{dt} &= \lambda_m S_m - \sigma_m E_m - \rho_m E_m \\
\frac{dI_m}{dt} &= \sigma_m E_m - \rho_m I_m
\end{aligned}$$

Forces of infection:

$$\lambda_h = \frac{\beta_h I_m}{N_m}$$
$$\lambda_m = \frac{\beta_m I_h}{N_h}$$

thus

$$\frac{\beta_h I_m^{**}}{N_m} S_h^{**} = (\sigma_h + \rho_h) E_h^{**}$$
$$\sigma_h E_h^{**} = (\delta + \rho_h + \alpha_1) I_h^{**}$$
$$\delta I_h^{**} = (\gamma + \rho_h + \alpha_2) {H_h}^{*} *$$
$$\frac{\beta_m I_h^{**}}{N_h} S_m^{**} = (\sigma_m + \rho_m) E_m^{**}$$
$$\sigma_m E_m^{**} = \rho_m I_m^{**}$$

here,

$$L = [E_h - E_h^{**} - E_h^{**} \log(\frac{E_h}{E_h^{**}})] + [I_h - I_h^{**} - I_h^{**} \log(\frac{I_h}{I_h^{**}})] + [H_h - H_h^{**} - H_h^{**} \log(\frac{H_h}{H_h^{**}})]$$
$$+ [E_m - E_m^{**} - E_m^{**} \log(\frac{E_m}{E_m^{**}})] + [I_m - I_m^{**} - I_m^{**} \log(\frac{I_m}{I_m^{**}})]$$

$$\dot{L} = (1 - \frac{E_h^{**}}{E_h})\dot{E}_h + (1 - \frac{I_h^{**}}{I_h})\dot{I}_h + (1 - \frac{H_h^{**}}{H_h})\dot{H}_h + (1 - \frac{E_m^{**}}{E_m})\dot{E}_m + (1 - \frac{I_m^{**}}{I_m})\dot{I}_m \qquad (5)$$

Now,

$$(1 - \frac{E_h^{**}}{E_h})\dot{E}_h = -(\sigma_h + \rho_h)\frac{(E_h - E_h^{**})^2}{E_h}$$
$$(1 - \frac{I_h^{**}}{I_h})\dot{I}_h = -(\delta + \rho_h + \alpha_1)\frac{(I_h - I_h^{**})^2}{I_h}$$
$$(1 - \frac{H_h^{**}}{H_h})\dot{H}_h = -(\gamma + \rho_h + \alpha_2)\frac{(H_h - H_h^{**})^2}{H_h}$$
$$(1 - \frac{E_m^{**}}{E_m})\dot{E}_m = -(\sigma_m + \rho_m)\frac{(E_m - E_m^{**})^2}{E_m}$$
$$(1 - \frac{I_m^{**}}{I_m})\dot{I}_m = -\rho_m\frac{(I_m - I_m^{**})^2}{I_m}$$

Now ,from equation (5),

$$\dot{L} = -\frac{(\sigma_h+\rho_h)(E_h-E_h^{**})^2}{E_h} - \frac{(\delta+\rho_h+\alpha_1)(I_h-I_h^{**})^2}{I_h} - \frac{(\gamma+\rho_h+\alpha_2)(H_h-H_h^{**})^2}{H_h} - \frac{(\sigma_m+\rho_m)(E_m-E_m^{**})^2}{E_m} - \frac{\rho_m(I_m-I_m^{**})^2}{I_m}$$

here,

$$-\frac{(\sigma_h+\rho_h)(E_h-E_h^{**})^2}{E_h} < 0$$
$$-\frac{(\delta+\rho_h+\alpha_1)(I_h-I_h^{**})^2}{I_h} < 0$$
$$-\frac{(\gamma+\rho_h+\alpha_2)(H_h-H_h^{**})^2}{H_h} < 0$$
$$-\frac{(\sigma_m+\rho_m)(E_m-E_m^{**})^2}{E_m} < 0$$
$$-\frac{\rho_m(I_m-I_m^{**})^2}{I_m} < 0$$

Therefore, we have $\bar{L} \leq 0$ for $R_0 > 1$. Thus, by Lyapunav function L and the Lasalle's invariance principle, every solution to the equations in the model 1 approaches as $t \to \infty$ for $R_0 > 1$.

Hence, the EE is globally asymptotically stable.

3. Numerical Simulation

In this section, numerical and analytical outcomes, the relations between classes and parameters and how they change over time have been discussed using MATLAB. Furthermore, the demonstration of the basic reproduction number of the graph and how it effects the infected classes for different values are analyzed thoroughly. The section examines the changes in population of infected humans and infected mosquitoes for $R_0 < 1$ and $R_o > 1$. Below, a table is presented having certain values of parameters for simulation.

Parameters	Values	References
μ_h	0.0001	[4, 19]
μ_m	0.1	[14]
ρ_h	0.0001	[19, 23]
ρ_m	0.1	[36, 28]
σ_h	0.083	[25, 7]
σ_m	0.083	[33]
δ	0.08	[6, 37]
γ	0.17	[14]
α_1	0.0008	[8]
α_2	0.0002	[8]
β_h	0.5	[34]
β_m	0.03	[8]

Table 2: Values of parameters of the model 1 for simulation.

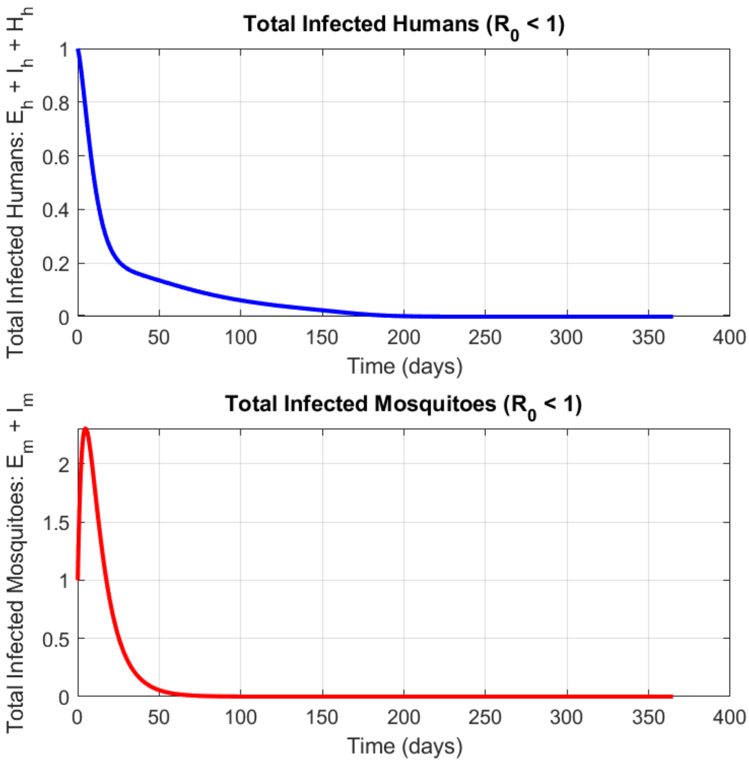

Figure 2: time vs infected population for $R_0 < 1$

Figure (2) shows graphs of time vs infected population for both human and mosquito population when $R_0 < 1$.This means less than one new individuals, on average, gets infected by every infected person. At the beginning,the decreasing blue curve represent that the disease dies out quickly causing no outbreak which is an indication of DFE. The red curve gives a brief rise which mountains at first but declines immediately to zero subsequently. This represents the transmission rate of human to mosquito is low restricting both human and mosquito population from sustaining the parasite and seizing the disease transmission.

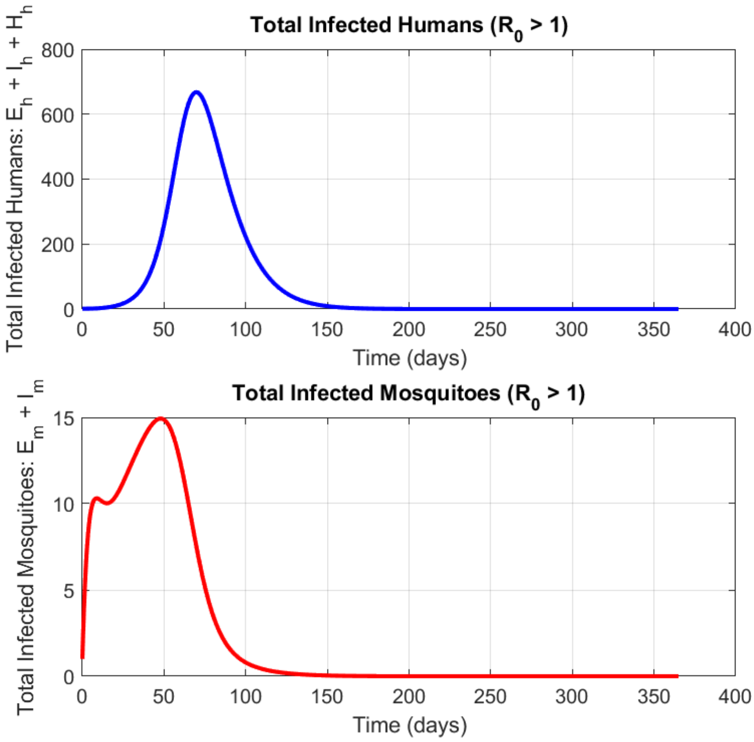

Figure 3: time vs infected population for $R_0 > 1$

Figure (3) displays graphs of time vs infected population for both human and mosquito population when $R_0 > 1$.This means more than one new individuals on average gets infected by every infected person. High mosquito infection rate is crucial to get $R_0 >$ 1.The blue graph consists of infected humans(i.e. $E_h + I_h + H_h$) which has a sharp peak around day 75.It decreases gradually to near zero after 150 days. Since$R_0 > 1$,the disease spreads rapidly indicating endemic behavior. It's initial rapid growth dictates an outbreak and the infection successfully spreads among human population. Since, either people become immune to the disease or die with time, the infection dies out. The red curve represents the total infected mosquito population (i.e. $E_m + I_m$).It jumps up rapidly and pinnacles around day 40 and declines later on like the human infection curve. The initial peak compared to human illustrates that mosquitoes get infected sooner which drives initial humans to get infected. Further, the decline of the graph asserts that mosquito infection drops as infected humans either recover or die.

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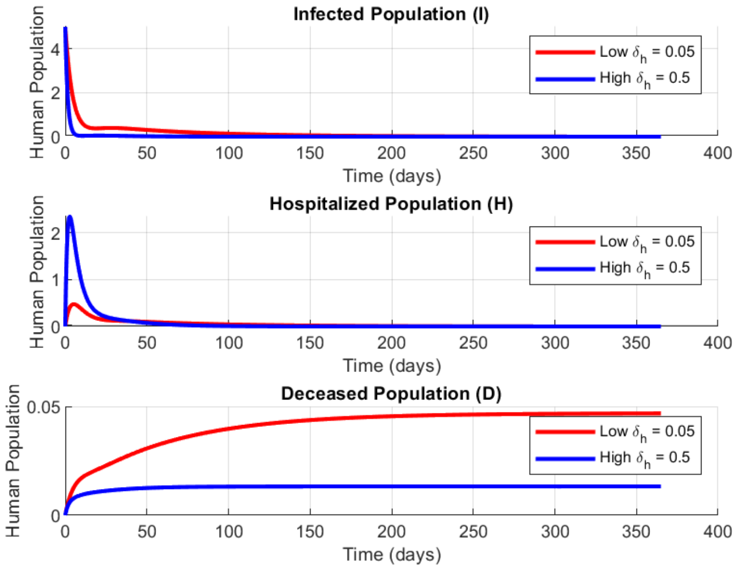

Figure 4: time vs population graph for low and high hospitalization rate

Figure (4) describes how the low hospitalization rate and high hospitalization rate contribute to the changes of infected, hospitalized and deceased population. In first figure, the curve skyrockets following by a sudden decline. Higher hospitalization rate gives a faster infection reduction comparing to the lower hospitalization rate because of the isolation of infected ones and rapid reduction of the disease. For the second graph, before it's declination, hospitalized population rise and the blue line has a higher initial peak than the red one. With time,the apex lowers down to zero since after hospitalization, individuals either die or recover. Third figure exhibits a rising death count which stabilizes with time. Low hospitalization rate has a particularly higher death count than the earlier one. So,it is clear that low hospitalization rate increases the total death count whereas later one reduces the death count as this has a speedy access to health care.

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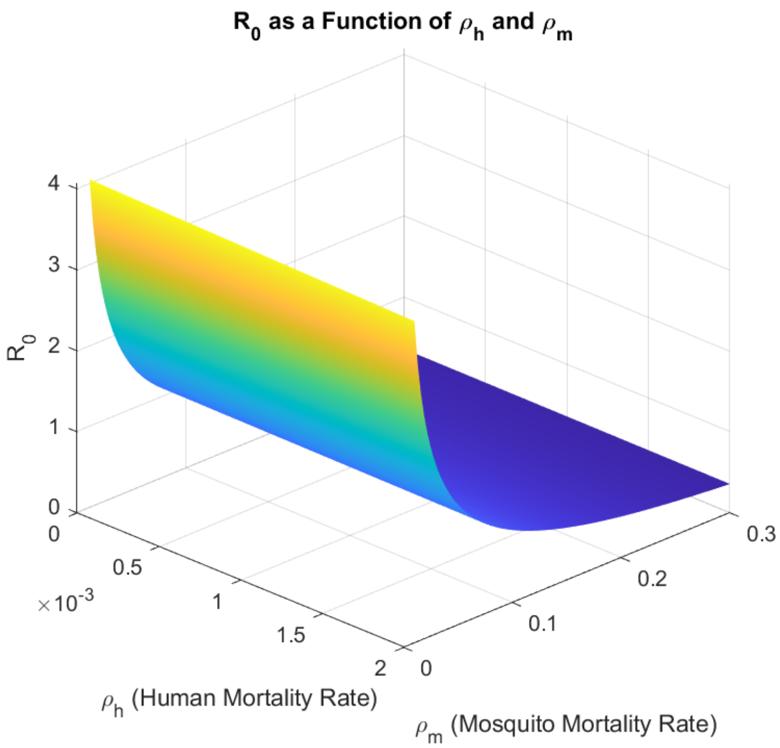

Figure 5: A 3D graph of human and mosquito mortality rate.

Figure (5) represents a three dimensional graph which shows how the basic reproduction number gets affected by human mortality and mosquito mortality. Human mortality rate ρ_h, mosquito mortality ρ_m and R_0 are placed in x, y, and z axes respectively. The curve shows if ρ_h increases then R_0 drops and there is a downward slope along x-axis .As human mortality rate increases, infected population die quicker lowering the time needed to infect the mosquitoes which ultimately lowers the disease transmission i.e. $R_0 < 1$. The similar result can be seen for the mosquito mortality rate (ρ_m) too.

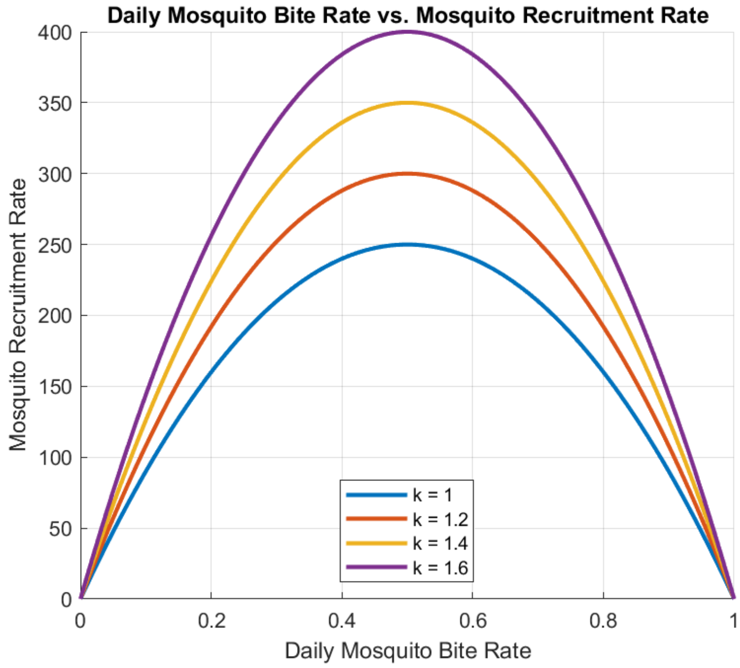

Figure 6: Mosquito bite rate vs Mosquito recruitment rate.

Figure (6) compares a daily mosquito bite rate with mosquito recruitment rate. Here,k is the breeding capacity modifier that indicates the suitable environment for recruitment. Each of the curve represents the increasing behavior of mosquito bite rate with respect to mosquito recruitment rate starting from zero and as bite rate rises mosquitoes breed more multiplying the number. The curves mountain when bite rates reach mid point and starts declining later. The breeding environment becomes more suitable as k increases making the curves peak higher than the rest.

A table for the people of Bangladesh who have fallen victim to malaria from year 2014-2023 is shown below[16, 1, 30, 38, 18, 21]:

Year	**Infected** (I_h)	**Deceased** (D_h)	**Recovered** ($R_h = I_h - D_h$)
2014	57,480	45	57,435
2017	5,133	13	5,120
2018	1,919	7	1,912
2020	7,294	9	7,285
2021	6,130	9	6,121
2022	18,195	14	18,181
2023	16,567	6	16,561

Table 3: Malaria Statistics in Bangladesh (2014–2023)

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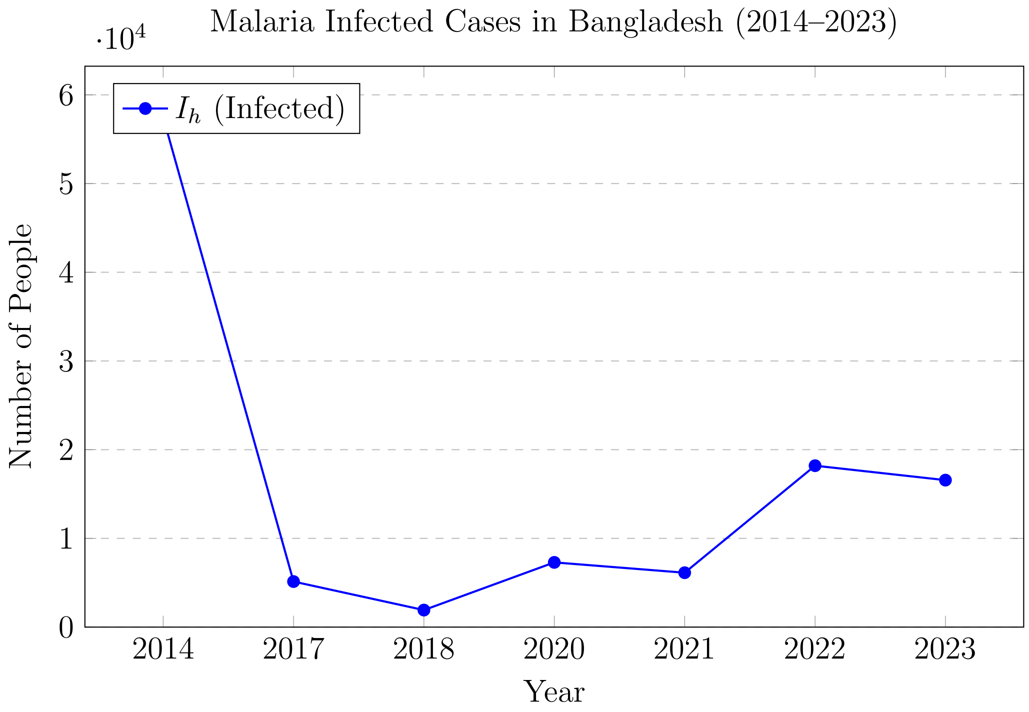

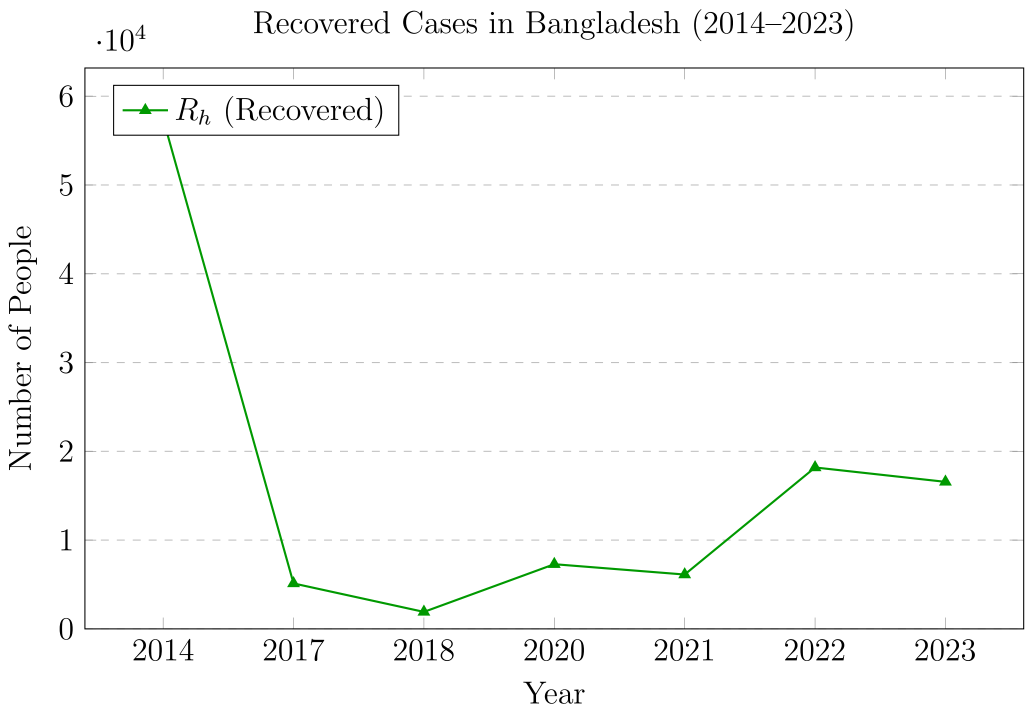

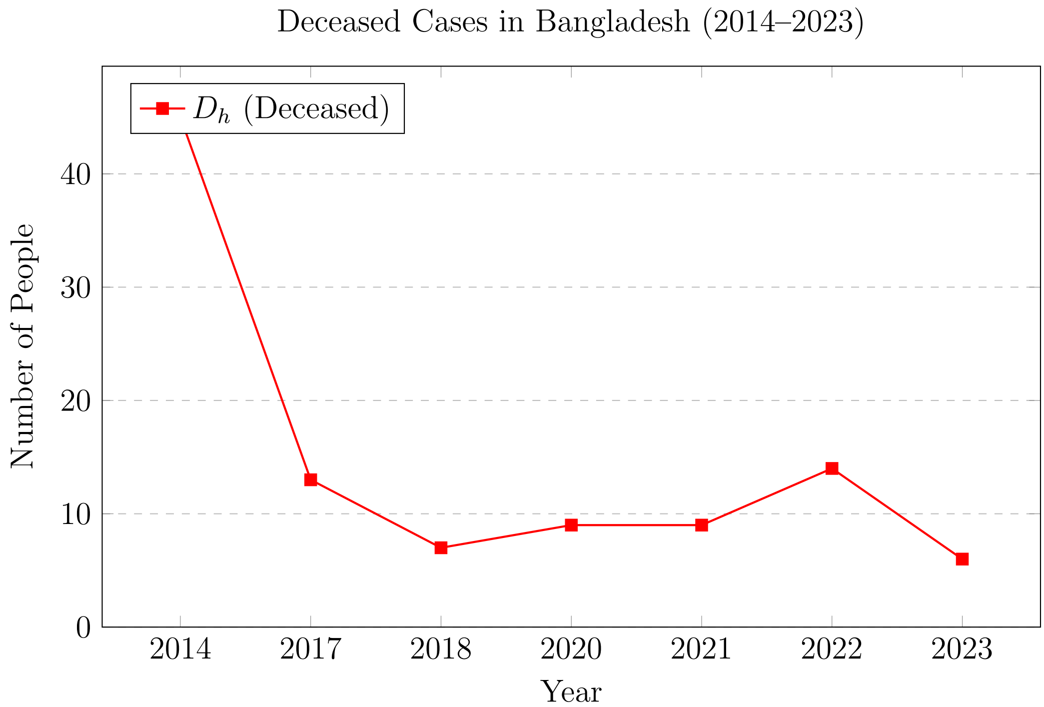

Figure-1,figure-2 and figure-3 show the flow of malaria cases in Bangladesh for the selected years 2014,2017,2018,2020,2021,2022 and 2023 for infected, recovered and deceased population. In figure-1,it is clear that infected cases were at peak in 2014 with more than 57000 infected individuals. Then the graph declined in the subsequent years because of many malaria control efforts. Recovered(Rh) graph, which is figure-2 follows the infected graph almost similarly since the deaths are minimal. It indicates that majority of people survived the disease in the mentioned time period. In figure-3,it is clear that the deceased curve stays low throughout all these years with lower deaths every year hinting the improvement of treatment and early detection. In conclusion,the figures represent a significant eradication of malaria over time , especially between 2014 and 2018 but there is a little resurgence in 2022-2023 because of the transmission in endemic hill areas.

4. Conclusion

In this project, a malaria transmission model is formulated and analyzed meticulously getting couple of findings as follows:

Mathematical findings

1. The disease free equilibrium (DFE), $\bar{E}_0$ of the model is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.Moreover, DFE model is globally asymptotically stable if $R_0 < 1$.

2. The Endemic Equilibrium (EE) exists when the basic reproduction number, R_0 is greater than one .Again, EE is globally asymptotically stable if $R_0 > 1$.

Epidemiological findings

1. Controlling and lowering the probability of transmission can reduce disease spread. Regular monitoring of mosquito population is important to ensure the continued effectiveness of vector control measures.

2. During disease outbreak, isolation and controlling the contact rate may help as well. Using mosquito repellents to eliminate mosquito breeding sites and insecticide-treated bed nets (ITNs) to prevent mosquitoes from biting can control the transmission.

3. Pregnant women in malaria endemic areas should receive IPTp with antimalarial drugs even if they do not have symptoms. Rapid diagnostic tests and microscopy are elementary steps to quickly identify malaria cases and further treatment must be ensured to prevent and cure transmission.

4. Educating communities about malaria transmission,prevention methods and the importance of seeking prompt treatment is crucial for success.

CRediT Authorship Contribution Statement

S.M. Saydur Rahman : Conceptualization, Writing - review & editing, Supervision. **Ferdousi Begum :** Conceptualization, Writing - original draft, Visualization - figures & graphs, Writing - review & editing. **Sayma Mostafa :** Conceptualization, Writing - original draft, Visualization - figures & graphs, Writing - review & editing.

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