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Epidemiological Modelling of Yellow Fever Dynamics

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Abstract

Aims: Yellow fever is a severe and often fatal viral illness caused by the yellow fever virus Despite being largely overlooked, yellow fever continues to silently claim lives in many parts of the world. The study focuses on the epidemiological modelling of yellow fever dynamics between a host (human) and vector (mosquito) populations The human population was divided into five main compartments: Susceptible, Exposed, Infected, Isolated, and Recovered. The vector population was also divided into two compartments: Susceptible and Infected. Nonlinear differential equations describing these compartments were formulated. Stability analysis and numerical simulations were then performed based on the formulated equations.

From the stability analysis, it was observed that the disease-free equilibrium is both locally and globally asymptotically stable. Similarly, the endemic equilibrium was found to be locally and globally asymptotically stable. The simulation also revealed a direct correlation between the transmission rate and disease spread.

Keywords: Stability; yellow fever; equilibrium; disease-free equilibrium; asymptotic.

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1 Introduction

Yellow fever is a severe and often fatal viral illness caused by the yellow fever virus. It is transmitted primarily by Aedes mosquitoes, particularly the Aedes aegypti species, which also serves as the vector for other viruses such as dengue, Chikungunya, and Zika [1], as well as by certain species of Haemagogus mosquitoes. The virus is typically transmitted to humans through the bite of infected mosquitoes, which can occur both during the day and at night. Yellow fever is predominantly found in tropical and subtropical regions of Africa and South America, putting approximately one billion people in forty-seven countries at risk [2]. Despite being largely overlooked, yellow fever continues to silently claim lives in many parts of the world. Yellow fever, a severe and often fatal viral illness, has largely been forgotten by many people, who live their lives without regard for its presence. However, many countries have recently reported cases of yellow fever. In 2016, Angola and the Democratic Republic of Congo (DRC) reported 965 confirmed cases of yellow fever, resulting in approximately 400 fatalities. Additionally, 11 cases were exported to China. From 2021 to 2022, a total of 203 confirmed cases with 40 deaths were recorded across 12 countries, including Cameroon, the Central African Republic, Chad, Côte d'Ivoire, the Democratic Republic of Congo (DRC), Ghana, Kenya, Niger, Nigeria, Sierra Leone, the Republic of the Congo, and Uganda, according to the World Health Organization (WHO). Despite efforts to control the disease, yellow fever remains a persistent threat, causing significant harm and loss of life. The dynamics of infectious disease spread are often analyzed and predicted using mathematical models, which have played a crucial role in developing public health strategies for prevention and control [3]. Several studies have tackled the challenge of yellow fever by formulating mathematical models to understand its spread and devise effective interventions. Mathematical analysis and modeling are essential for studying the dynamics of infectious diseases, providing insights into the origin and evolution of viruses [4]. For instance, [5] formulated a statistical inference model utilizing contemporary likelihood-based methods to evaluate and rebuild key epidemiological mechanisms behind the yellow fever epidemic in Angola. [6] employed the differential transmission approach to resolve the yellow fever dynamics mathematical model that included a secondary host. [7] emphasized the significance of mathematical modeling in making informed decisions and shaping disease management strategies. [8] focused on modeling and stability analysis of yellow fever transmission dynamics, while [9] conducted stability and sensitivity analyses of various yellow fever models. [2] performed optimal control and stability analysis strategies for a yellow fever model considering vertical transmission. Nevertheless, according to [6-9], and [2], academic investigations on yellow fever do not consider the isolation of infected individuals. Infected individuals must be isolated to prevent mosquitoes from transferring the infection, as mosquitoes can serve as vectors for infecting other patients [10]. This study aims to develop and analyze a Susceptible Exposed Infected Isolated Recovered and Susceptible Infected (SEIISR-SI) Host and Vector mathematical model of yellow fever dynamics. This model will consider the isolation of infected individuals. Through mathematical modeling, we seek to enhance our understanding of yellow fever transmission dynamics and identify effective strategies for disease control.

2 Methodology

2.1 Model formulation

The yellow fever model for the study divides the total human population $N_H(t)$ into five classes: susceptible, exposed, infected, isolated, and recovered. Specifically, $S_H(t)$ represents susceptible humans, $E_H(t)$ represents exposed humans, $I_H(t)$ represents infected humans, $I_{SH}(t)$ represents isolated humans, and $R_H(t)$ represents recovered humans. Thus, the total human population is given by $N_H(t) = S_H(t) + E_H(t) + I_H(t) + I_{SH}(t) + R_H(t)$. This model applies to completely unvaccinated geographical areas. The total vector population $N_V(t)$ is divided into two classes: susceptible vectors $S_V(t)$ and infected vectors $I_V(t)$. Therefore, the total vector population is $N_V(t) = S_V(t) + I_V(t)$.

2.1.1 Susceptible human (S_H)

Susceptible human refers to individuals who are capable of or have the highest probability of being infected by a particular disease. The susceptible human population increases only by the birth rate b_H, and decreases by the natural death rate μ_H ,and by being bitten by infected vectors at a rate of $\frac{\beta S_H I_V}{N_V}$. Hence, we have:

$$\frac{dS_V}{dt} = b_V - \mu_V S_V - \frac{\omega S_V I_H}{N_H} \tag{1}$$

2.1.2 Exposed human (E_H)

Exposed human refers to individuals who have the disease but are not showing or experiencing any symptoms and are not capable of transmitting the disease. The exposed population increases as individuals are bitten by infected vectors at a rate of $\frac{\beta S_H I_V}{N_V}$ and decreases as individuals become infected at a rate of γ and by natural death at a rate of μ_H .Thus, the equation for the exposed human population is

$$\frac{dE_H}{dt} = \frac{\beta S_H I_V}{N_V} - \gamma E_H - \mu_H E_H \tag{2}$$

2.1.3 Infected human (I_H)

Infected refers to individuals who have the disease, are showing symptoms, and can transmit the disease. The infected population increases as exposed individuals with weak immunity move to the infected class at a rate of γ . The infected population decreases under the following assumptions:

i. Infected individuals are isolated to avoid mosquito bites, as mosquitoes can transmit the virus to other humans [11], at a rate of δ .
ii. Infected individuals die from the disease at a rate of α_H and from natural causes at a rate of μ_H .

The Infected human equation therefore given by;

$$\frac{dI_H}{dt} = \gamma E_H - I_H\left(\alpha_H + \mu_H + \delta\right) \tag{3}$$

2.1.4 Isolated human (I_{SH})

This refers to individuals being isolated to avoid mosquito bites, as mosquitoes can transmit the virus to other humans [11]. The isolated population increases as infected individuals are isolated at a rate of δ and decreases due to the following reasons:

i. Isolated individuals die from the disease at a rate of α_H
ii. Isolated individuals die from natural causes at a rate of μ_H .
iii. Isolated individuals recover at a rate of ε

Hence, Isolated human equation is;

$$\frac{dI_{SH}}{dt} = \delta I_H - I_{SH}\left(\alpha_H + \mu_H + \varepsilon\right) \tag{4}$$

2.1.5 Recovered human (RH)

This refers to individuals receiving proper medical care and recovering from the disease. The recovered population increases as isolated individuals recover at a rate of ε and μ_H .
Hence, recovered human equation is represented by;

$$\frac{dR_H}{dt} = \varepsilon I_{SH} - \mu_H R_H \tag{5}$$

2.1.6 Susceptible vector (SV)

This refers to vectors that are capable of or have the highest probability of being infected by a particular disease. The susceptible vector population increases only by the birth rate b_V and decreases due to natural death at a rate of μ_V, and decreases by natural death at rate μ_V, and bitten infected human at the rate $\frac{\omega S_V I_H}{N_H}$. Hence, we have;

$$\frac{dS_V}{dt} = b_V - \mu_V S_V - \frac{\omega S_V I_H}{N_H} \tag{6}$$

2.1.7 Infected vector (I_V)

This refers to vectors that can transmit the disease. The infected vector population increases as susceptible vectors bite infected humans at a rate of $\frac{\omega S_V I_H}{N_H}$ and decreases by natural death at the rate μ_V. The equation regarding the infected vector population is given by;

$$\frac{dI_V}{dt} = \frac{\omega S_V I_H}{N_H} - \mu_V I_V \tag{7}$$

2.2 Model assumptions

The following are the model assumptions:

i. Natural death rate is constant in all class.
ii. Host recover from the disease with permanent immunity.
iii. Death and birth occur at equal rates
iv. Migration of both vector and host is ignored
v. The model considers vertical transmission of the infection in the vector and the human population.
vi. The recovered class of the vector is excluded because the vector does not recover from the infection or die from the infection since mosquitoes are only carriers of the virus.
vii. The exposed class of the vector is excluded because the incubation period for the vector is short.

2.3 Disease compartmental model

The vertical transmission of yellow fever in Host and vector of the model is shown Fig. 1.

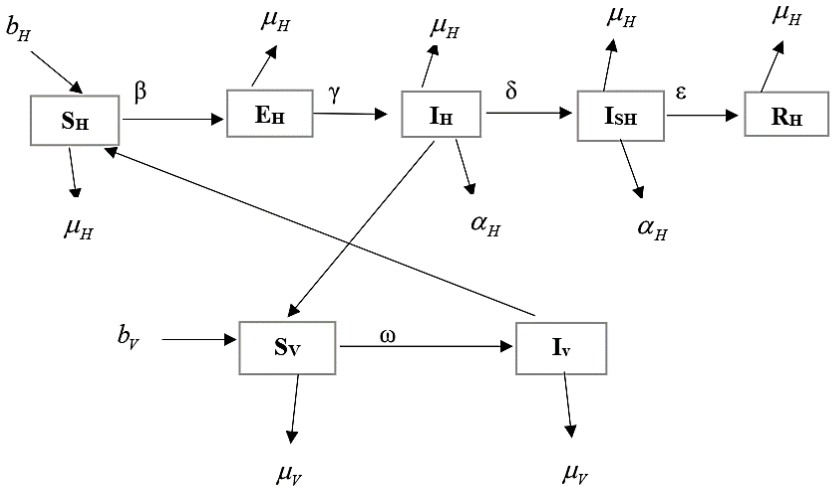

Fig. 1. Compartmental Diagram of the Yellow fever in Host and Vector Population

2.4 Nonlinear equations using SEIISR-SI model (yellow fever)

The differential equations that describe the dynamics of yellow fever in humans and mosquito vectors are formulated in equations (8) and (9).

Host (Human)

$$\left.\begin{aligned}
\frac{dS_H}{dt} &= b_H - \mu_H S_H - \frac{\beta S_H I_V}{N_V} \\
\frac{dE_H}{dt} &= \frac{\beta S_H I_V}{N_V} - E_H\left(\gamma + \mu_H\right) \\
\frac{dI_H}{dt} &= \gamma E_H - I_H\left(\alpha_H + \mu_H + \delta\right) \\
\frac{dI_{SH}}{dt} &= \delta I_H - I_{SH}\left(\alpha_H + \mu_H + \varepsilon\right) \\
\frac{dR_H}{dt} &= \varepsilon I_{SH} - \mu_H R_H
\end{aligned}\right\} \tag{8}$$

Vector (Mosquito)

$$\left.\begin{aligned}
\frac{dS_V}{dt} &= b_V - \mu_V S_V - \frac{\omega S_V I_H}{N_H} \\
\frac{dI_V}{dt} &= \frac{\omega S_V I_H}{N_H} - \mu_V I_V
\end{aligned}\right\} \tag{9}$$

2.5 Basic properties of the model

2.5.1 Invariant region

Theorem 1: All forward solutions in $\mathbb{R}^7_+$ of the system is feasible $\forall t \geq 0$ they enter the invariant region Ω for $\Omega = \Omega_V \times \Omega_H$ where $\Omega_H = \left(S_H, E_H, I_H, I_{SH}, R_H\right) \in \mathbb{R}^5_+ : S_H + E_H + I_H + I_{SH} + R_H \leq N_H$, $\Omega_V = \left(S_V, I_V\right) \in \mathbb{R}^2_+ : S_V + I_V \leq N_V$, then Ω is positively invariant and attracting under the flow described by the system.

Proof:

For Human Population: we need to prove that the solution of the model system is feasible $\forall t \geq 0$ as they enter the invariant region $\Omega_H = \left(S_H, E_H, I_H, I_{SH}, R_H\right) \in \mathbb{R}^5_+$ which is the solution space of the system with nonnegative initial conditions.

Thus, the total human population is given by equation (10);

$$N_H = S_H + E_H + I_H + I_{SH} + R_H . \tag{10}$$

Then,

$$\frac{dN_H}{dt} = \frac{dS_H}{dt} + \frac{dE_H}{dt} + \frac{dI_H}{dt} + \frac{dI_{SH}}{dt} + \frac{dR_H}{dt} \tag{11}$$

$$\frac{dN_H}{dt} = b_H - \mu_H S_H - \frac{\beta S_H I_V}{N_V} + \frac{\beta S_H I_V}{N_V} - E_H(\gamma + \mu_H) + \gamma E_H - I_H(\alpha_H + \mu_H + \delta) + \delta I_H - I_{SH}(\alpha_H + \mu_H + \varepsilon) + \varepsilon I_{SH} - \mu_H R_H \tag{12}$$

$$\frac{dN_H}{dt} = b_H - \mu_H(S_H + E_H + I_H + I_{SH} + R_H) - \alpha_H(I_H + I_{SH}) \tag{13}$$

$$\frac{dN_H}{dt} \le b_H - \mu_H N_H - \alpha_H(I_H + I_{SH}) \tag{14}$$

In many epidemiological models, the invariant region represents the disease-free equilibrium state, where there are no infected individuals. In such a state, it is reasonable to assume that there are no disease-related deaths since the disease is not present in the population.

It implies $\alpha_H = 0$

$$\frac{dN_H}{dt} \le b_H - \mu_H N_H \tag{15}$$

$$\frac{dN_H}{dt} + \mu_H N_H \le b_H s \tag{16}$$

Using First order linear differential equation we have;

$$N_H e^{\int \mu_H dt} \le \int b_H e^{\int \mu_H dt} dt \tag{17}$$

which gives

$$N_H e^{\mu_H t} \le \frac{b_H}{\mu_H} e^{\mu_H t} + C \tag{18}$$

Making N_H the subject it gives;

$$N_H \le \frac{b_H}{\mu_H} + Ce^{-\mu_H t} \tag{19}$$

Using the initial condition $t = 0, N_H(t = 0) = N_H(0)$, then, we get;

$$N_H(0) - \frac{b_H}{\mu_H} \le C \tag{20}$$

Substituting for the constant N_H into (19), results in;

$$N_H \le \frac{b_H}{\mu_H} + \left(N_H(0) - \frac{b_H}{\mu_H}\right) e^{-\mu_H t} \tag{21}$$

Further observation, make known that $N_H(t) \to \frac{b_H}{\mu_H}$ as $t \to \infty$. Therefore, it can be concluded that $N_H(t)$ is bounded as $0 \le N_H(t) \le \frac{b_H}{\mu_H}$. Hence, the feasible region of the human model in the non-negative region define by;

$$\Omega_H = \left\{ \left(S_H, E_H, I_H, I_{SH}, R_H\right) \in \mathbb{R}_+^5 : N_H \le \frac{b_H}{\mu_H} \right\} \tag{22}$$

For Vector Population: we need to prove that the solution of the model system is feasible $\forall t \ge 0$ as they enter the invariant region $\Omega_V = \left(S_V, I_V\right) \in \mathbb{R}_+^2$ be solution space of the system with nonnegative initial conditions. The total human population is;

$$N_V = S_V + I_V \,. \tag{23}$$

Then,

$$\frac{dN_V}{dt} = \frac{dS_V}{dt} + \frac{dI_V}{dt} \tag{24}$$

$$\frac{dN_V}{dt} = b_V - \mu_V S_V - \frac{\omega S_V I_H}{N_H} + \frac{\omega S_V I_H}{N_H} - \mu_V I_V \tag{25}$$

$$\frac{dN_V}{dt} = b_V - \mu_V \left(S_V + I_V\right) \text{ but } N_V = S_V + I_V \tag{26}$$

It implies

$$\frac{dN_V}{dt} \le b_V - \mu_V N_V \tag{27}$$

Which can be rewrite as

$$\frac{dN_V}{dt} + \mu_V N_V \le b_V \tag{28}$$

Using First order linear differential equation we have;

$$N_V e^{\int \mu_V dt} \le \int b_V e^{\int \mu_V dt} dt \tag{29}$$

which gives

$$N_V e^{\mu_V t} \le \frac{b_V}{\mu_V} e^{\mu_V t} + C \tag{30}$$

Making NV the subject in equation (30);

$$N_V \le \frac{b_V}{\mu_V} + Ce^{-\mu_V t} \tag{31}$$

Using the initial condition $t = 0, N_V(t=0) = N_V(0)$, then, we get;

$$N_V(0) - \frac{b_V}{\mu_V} \leq C \tag{32}$$

Substituting for the constant C into (4.24), we had

$$N_V \leq \frac{b_V}{\mu_V} + \left(N_V(0) - \frac{b_V}{\mu_V} \right) e^{-\mu_V t} \tag{33}$$

Further observation, make known that $N_V(t) \to \frac{b_V}{\mu_V}$ as $t \to \infty$. Therefore, concluded that $N_V(t)$ is bounded as $0 \leq N_V(t) \leq \frac{b_V}{\mu_V}$. Therefore, the feasible region of the vector model in the non-negative region is define as

$$\Omega_V = \left\{ (S_V, I_V) \in \mathbb{R}_+^2 : N_V \leq \frac{b_V}{\mu_V} \right\} \tag{34}$$

2.6 Positivity of solution

In epidemiological models, ensuring the positivity of solutions is crucial because the state variables and parameters of the model must be nonnegative $\forall t \geq 0$.

Theorem 2: The solution of both human and vector model systems with the initial conditions $(S_H(0), E_H(0), I_H(0), I_{SH}(0), R_H(0), S_V(0), I_V(0)) \geq 0$ is positive in $\mathbb{R}_+^7$ for all $t \geq 0$.

For Susceptible Human (S_H)

$$\frac{dS_H}{dt} = b_H - \mu_H S_H - \frac{\beta S_H I_V}{N_V} \tag{35}$$

Further simplification we have;

$$\frac{dS_H}{dt} = b_H - S_H \left(\mu_H + \frac{\beta I_V}{N_V} \right) \tag{36}$$

Considering the S_H terms, we have;

$$\frac{dS_H}{dt} \geq -S_H \left(\mu_H + \frac{\beta I_V}{N_V} \right) \tag{37}$$

Using separable method, we have;

$$\int \frac{dS_H}{S_H} \geq -\int_0^t \left(\mu_H + \frac{\beta I_V}{N_V} \right) dt \tag{38}$$

Which gives

$$\ln(S_H) \geq -\left(\mu_H + \frac{\beta I_V}{N_V} \right) t \tag{39}$$

Which further gives

$$S_H \geq e^{-\left(\mu_H + \frac{\beta I_V}{N_V}\right)t} > 0\,, \tag{40}$$

$$\text{Since } e^{-\left(\mu_H + \frac{\beta I_V}{N_V}\right)t} > 0 \tag{41}$$

From the equation (2), we have

$$\frac{dE_H}{dt} = \frac{\beta S_H I_V}{N_V} - E_H\left(\gamma + \mu_H\right) \tag{42}$$

Thus

$$\frac{dE_H}{dt} \geq -E_H\left(\gamma + \mu_H\right) \tag{43}$$

Using separable method, we have

$$\int \frac{dE_H}{E_H} \geq -\int_0^t (\gamma + \mu_H)dt \tag{44}$$

Which gives

$$\ln\left(E_H\right) \geq -\left(\gamma + \mu_H\right)t \tag{45}$$

Which further gives

$$E_H \geq e^{-(\gamma+\mu_H)t} > 0 \tag{46}$$

$$\text{Since } e^{-(\gamma+\mu_H)t} > 0 \tag{47}$$

From equation (3), we have

$$\frac{dI_H}{dt} = \gamma E_H - I_H\left(\alpha_H + \mu_H + \delta\right) \tag{48}$$

Thus

$$\frac{dI_H}{dt} \geq -I_H(\alpha_H + \mu_H + \delta) \tag{49}$$

Using separable method, we have

$$\int \frac{dI_H}{I_H} \geq -\int_0^t (\alpha_H + \mu_H + \delta)dt \tag{50}$$

Which gives

$$\ln\left(I_H\right) \geq -(\alpha_H + \mu_H + \delta)t \tag{51}$$

Which further gives

$$I_H \geq e^{-(\alpha_H+\mu_H+\delta)t} > 0 \tag{52}$$

$$\text{Since } e^{-(\alpha_H+\mu_H+\delta)t} > 0 \tag{53}$$

From equation (4), we have

$$\frac{dI_{SH}}{dt} = \delta I_H - I_{SH}\left(\alpha_H + \mu_H + \varepsilon\right) \tag{54}$$

Thus

$$\frac{dI_{SH}}{dt} \geq -I_{SH}(\alpha_H + \mu_H + \varepsilon) \tag{55}$$

Using separable method, we have

$$\int \frac{dI_{SH}}{I_{SH}} \geq -\int_0^t (\alpha_H + \mu_H + \varepsilon)dt \tag{56}$$

Which gives

$$\ln\left(I_{SH}\right) \geq -(\alpha_H + \mu_H + \varepsilon)t \tag{57}$$

Which further gives

$$I_{SH} \geq e^{-(\alpha_H+\mu_H+\varepsilon)t} > 0 \tag{58}$$

Since

$$e^{-(\alpha_H+\mu_H+\varepsilon)t} > 0\,. \tag{59}$$

From the equation (5),

$$\frac{dR_H}{dt} = \varepsilon I_{SH} - \mu_H R_H \tag{60}$$

Thus

$$\frac{dR_H}{dt} \geq -\mu_H R_H \tag{61}$$

Using separable method,

$$\int \frac{dR_H}{R_H} \geq -\int_0^t \mu_H dt \tag{62}$$

Which gives

$$\ln\left(R_H\right) \geq -\mu_H t \tag{63}$$

Which further gives

$$R_H \geq e^{-\mu_H t} > 0\,, \tag{64}$$

$$\text{Since } e^{-\mu_H t} > 0 \tag{65}$$

For the vector population:

From the equation (6), we have

$$\frac{dS_V}{dt} = b_V - \mu_V S_V - \frac{\omega S_V I_H}{N_H} \tag{66}$$

Further simplification we have;

$$\frac{dS_V}{dt} = b_V - S_V\left(\mu_V + \frac{\omega I_H}{N_H}\right) \tag{67}$$

Thus

$$\frac{dS_V}{dt} \geq -S_V\left(\mu_V + \frac{\omega I_H}{N_H}\right) \tag{68}$$

Using separable method, we have

$$\int \frac{dS_V}{S_V} \geq -\int_0^t \left(\mu_V + \frac{\omega I_H}{N_H}\right) dt \tag{69}$$

Which gives

$$\ln\left(S_V\right) \geq -\left(\mu_V + \frac{\omega I_H}{N_H}\right) t \tag{70}$$

Which further gives

$$S_V \geq e^{-\left(\mu_V + \frac{\omega I_H}{N_H}\right) t} > 0 \tag{71}$$

Since

$$e^{-\left(\mu_V + \frac{\omega I_H}{N_H}\right) t} > 0 \tag{72}$$

From the second equation, we have

$$\frac{dI_V}{dt} = \frac{\omega S_V I_H}{N_H} - \mu_V I_V \tag{73}$$

Thus

$$\frac{dI_V}{dt} \geq -\mu_V I_V \tag{74}$$

Using separable method, we have

$$\int \frac{dI_V}{I_V} \geq -\int_0^t \mu_V dt \tag{75}$$

Which gives

$$\ln\left(I_V\right) \geq -\mu_V t \tag{76}$$

Which further gives

$$I_V \geq e^{-\mu_V t} > 0\,, \tag{77}$$

$$\text{Since } e^{-\mu_V t} > 0 \tag{78}$$

Since all the seven systems are greater than zero, it implies positives theorem is proved and tested.

2.7 The disease-free equilibrium (DFE)

Disease-free equilibrium points are steady-state solutions where no disease exists in either the human host or mosquito vector populations. To understand the dynamical behavior of all the compartmental equations in the system, we set the right-hand side of all the equations in the system to zero as follows:

$$\left.\begin{aligned}
&b_H - \mu_H S_H - \frac{\beta S_H I_V}{N_V} = 0\\
&\frac{\beta S_H I_V}{N_V} - E_H\left(\gamma + \mu_H\right) = 0\\
&\gamma E_H - I_H\left(\alpha_H + \mu_H + \delta\right) = 0\\
&\delta I_H - I_{SH}\left(\alpha_H + \mu_H + \varepsilon\right) = 0\\
&\varepsilon I_{SH} - \mu_H R_H = 0\\
&b_V - \mu_V S_V - \frac{\omega S_V I_H}{N_H} = 0\\
&\frac{\omega S_V I_H}{N_H} - \mu_V I_V = 0
\end{aligned}\right\} \tag{79}$$

Hence the result for the disease-free equilibrium is given as;

$$\left[S_H = \frac{b_H}{\mu_H}, E_H = 0, I_H = 0, I_{SH} = 0, R_H = 0, S_V = \frac{b_V}{\mu_V}, I_V = 0\right]$$

2.8 Determination of the basic reproductive number ($\mathcal{R}_0$)

The basic reproductive number $\Re_0$ is the estimated number of subsequent infections caused by an index case in a fully susceptible community (Driessche and Watmough, 2008). To calculate the basic reproductive number, the next generation method is applied. The basic reproduction number is computed as $\Re_0 = \rho(FV^{-1})$,where ρ is the spectral radius, also known as the dominant eigenvalue of FV^{-1}. In determining the basic reproductive number, the disease compartments considered are exposed host $\left(E_H\right)$, infected host $\left(I_H\right)$, isolated host $\left(I_{SH}\right)$,and infected vector $\left(I_V\right)$. The disease compartmental equations are as follows:

$$\left.\begin{aligned}
\frac{dE_H}{dt} &= \frac{\beta S_H I_V}{N_V} - E_H(\gamma + \mu_H) \\
\frac{dI_H}{dt} &= \gamma E_H - I_H(\alpha_H + \mu_H + \delta) \\
\frac{dI_{SH}}{dt} &= \delta I_H - I_{SH}(\alpha_H + \mu_H + \varepsilon) \\
\frac{dI_V}{dt} &= \frac{\omega S_V I_H}{N_H} - \mu_V I_V
\end{aligned}\right\} \tag{80}$$

From the above equations, next generation approach gives the transmission(Ƒ) and transition (ν) as;

$$f = \begin{bmatrix} \frac{\beta S_H I_V}{N_V} \\ 0 \\ 0 \\ \frac{\omega S_V I_H}{N_H} \end{bmatrix} \tag{81}$$

And

$$v = \begin{bmatrix} -E_H(\gamma + \mu_H) \\ \gamma E_H - I_H(\mu_H + \alpha_H + \delta) \\ \delta I_H - I_{SH}(\mu_H + \alpha_H + \varepsilon) \\ -\mu_V I_V \end{bmatrix} \tag{82}$$

The Jacobian matrix of the transmission (F) and transition (V) states of the model evaluated at the disease-free equilibrium are as follows:

$$F = \partial f\,|_{E_H, I_H, I_{SH}, I_V} = \begin{bmatrix} 0 & 0 & 0 & \frac{\beta b_H}{N_V \mu_H} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\omega b_V}{N_H \mu_V} & 0 & 0 \end{bmatrix} \tag{83}$$

$$V = \partial v\,|_{E_H, I_H, I_{SH}, I_V} = \begin{bmatrix} -(\gamma + \mu_H) & 0 & 0 & 0 \\ \gamma & -(\alpha_H + \mu_H + \delta) & 0 & 0 \\ 0 & \delta & -(\alpha_H + \mu_H + \varepsilon) & 0 \\ 0 & 0 & 0 & -\mu_V \end{bmatrix} \tag{84}$$

The inverse of V is obtained as;

$$V^{-1} = \begin{bmatrix} -\frac{1}{(\gamma + \mu_H)} & 0 & 0 & 0 \\ -\frac{\gamma}{(\gamma + \mu_H)(\alpha_H + \mu_H + \delta)} & -\frac{1}{(\alpha_H + \mu_H + \delta)} & 0 & 0 \\ -\frac{\gamma\delta}{(\gamma + \mu_H)(\alpha_H + \mu_H + \delta)(\alpha_H + \mu_H + \varepsilon)} & -\frac{\delta}{(\alpha_H + \mu_H + \delta)(\alpha_H + \mu_H + \varepsilon)} & -\frac{1}{(\alpha_H + \mu_H + \varepsilon)} & 0 \\ 0 & 0 & 0 & -\frac{1}{\mu_V} \end{bmatrix} \tag{85}$$

$$FV^{-1}=\begin{bmatrix} 0 & 0 & 0 & -\dfrac{\beta b_H}{\mu_V \mu_H N_V} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ -\dfrac{\gamma\omega b_V}{N_H\mu_V(\gamma+\mu_H)(\alpha_H+\mu_H+\delta)} & -\dfrac{\omega b_V}{N_H\mu_V(\alpha_H+\mu_H+\delta)} & 0 & 0 \end{bmatrix} \tag{86}$$

Therefore, eigenvalues for FV^{-1} are; $\lambda = 0,0,-\dfrac{\sqrt{N_H N_V \mu_V \mu_H b_V b_H \beta\gamma abd}}{N_H N_V \mu_V \mu_H abd}$, $\dfrac{\sqrt{N_H N_V \mu_V \mu_H b_V b_H \beta\gamma abd}}{N_H N_V \mu_V \mu_H abd}$.

Where $a=(\gamma+\mu_H), b=(\alpha_H+\mu_H+\delta), c=(\alpha_H+\mu_H+\varepsilon), d=\mu_V$

The dominant eigenvalue is $\dfrac{\sqrt{N_H N_V \mu_V \mu_H b_V b_H \beta\gamma abd}}{N_H N_V \mu_V \mu_H abd}$

Therefore, the basic reproductive number for the model is given by $\Re_0 = \dfrac{\sqrt{N_H N_V \mu_V \mu_H b_V b_H \beta\gamma abd}}{N_H N_V \mu_V \mu_H abd}$

Which can further be simplified as

$$\Re_0 = \sqrt{\frac{b_V b_H \beta\gamma\omega}{N_H N_V \mu_V^2 \mu_H (\gamma+\mu_H)(\alpha_H+\mu_H+\delta)}} \tag{87}$$

2.9 The endemic equilibrium (EE)

The endemic equilibrium (EE) is given by the system below:

$$\left.\begin{aligned} S_H^* &= \frac{b_H(\beta b_V+\mu_H\mu_V N_V\Re_0^2)}{\mu_H\eta_2\Re_0^2}, E_H^* = \frac{\eta_1(\Re_0^2-1)}{\eta_2\gamma\omega} \\ I_H^* &= \frac{\mu_V^2\mu_H N_H N_V(\Re_0^2-1)}{\omega\eta_2}, I_{SH}^* = \frac{\mu_V^2\mu_H N_H N_V\delta(\Re_0^2-1)}{\omega(\alpha_H+\mu_H+\varepsilon)\eta_2} \\ R_H^* &= \frac{\mu_V^2 N_H N_V\varepsilon\delta(\Re_0^2-1)}{\omega(\alpha_H+\mu_H+\varepsilon)\eta_2}, S_V^* = \frac{b_V N_V\eta_2}{\mu_V N_H\left(\beta b_V+\mu_V\mu_H N_V\Re_0^2\right)} \\ I_V^* &= \frac{\mu_H N_V\eta_3(\Re_0^2-1)}{\beta\left(\eta_3+\gamma\omega b_H\right)} \end{aligned}\right\} \tag{88}$$

Where $\eta_1=\mu_V^2\mu_H N_V N_H(\alpha_H+\delta+\mu_H)$, $\eta_2=(\mu_H\mu_V N_V+\beta b_V)$, and $\eta_3=\mu_V N_H(\gamma+\mu_H)(\alpha_H+\delta+\mu_H)$

2.10 Local stability of equilibrium solutions

The stability analysis for the system of DFE and EE are performed in this section

2.10.1 Local stability of disease-free equilibrium

Theorem 3: The disease-free equilibrium of the system equation is locally asymptotically stable if and only if $\Re_0 < 1$ [2,12].

The Jacobian matrix, J of the system evaluated at the DFE, represented below as

$$J(DFE)=\begin{bmatrix} -\mu_H & 0 & 0 & 0 & 0 & 0 & -\frac{\beta b_H}{N_V \mu_H} \\ 0 & -(\mu_H+\gamma) & 0 & 0 & 0 & 0 & \frac{\beta b_H}{N_V \mu_H} \\ 0 & \gamma & -(\mu_H+\alpha_H+\delta) & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta & -(\mu_H+\alpha_H+\varepsilon) & 0 & 0 & 0 \\ 0 & 0 & 0 & \varepsilon & -\mu_H & 0 & 0 \\ 0 & 0 & -\frac{\omega b_V}{N_H \mu_V} & 0 & 0 & -\mu_V & 0 \\ 0 & 0 & \frac{\omega b_V}{N_H \mu_V} & 0 & 0 & 0 & -\mu_V \end{bmatrix} \quad (89)$$

To prove the local stability of the DFE, Geshgorin's Theorem [9]. Thus, the following are obtained;

$$\left.\begin{array}{l} -\frac{\beta b_H}{\mu_H N_V} < \mu_H \\ \frac{\beta b_H}{\mu_H N_V} < (\mu_H+\gamma) \\ \gamma < (\mu_H+\alpha_H+\delta) \\ \delta < (\mu_H+\alpha_H+\varepsilon) \\ \varepsilon < \mu_H \\ -\frac{\omega b_V}{N_H \mu_V} < \mu_V \\ \frac{\omega b_V}{N_H \mu_V} < \mu_V \end{array}\right\} \quad (90)$$

By multiplying the square of second equation by the square of the third and then by square of the seventh gives;

$$\left(\frac{\beta b_H}{\mu_H N_V}\right)^2 (\gamma)^2 \left(\frac{\omega b_V}{\mu_V N_H}\right)^2 < (\mu_H+\gamma)^2 (\mu_H+\alpha_H+\delta)^2 (\mu_V)^2 \quad (91)$$

Further simplification gives;

$$\frac{(\beta b_H)^2}{(\mu_H N_V)^2}(\gamma)^2 \frac{(\omega b_V)^2}{(\mu_V N_H)^2} < (\mu_H+\gamma)^2 (\mu_H+\alpha_H+\delta)^2 (\mu_V)^2 \quad (92)$$

Dividing through the simplified equation by the right-hand side yields;

$$\frac{(\beta b_H)^2 (\gamma)^2 (\omega b_V)^2}{(\mu_H N_V)^2 (\mu_V N_H)^2 (\mu_H+\gamma)^2 (\mu_H+\alpha_H+\delta)^2 (\mu_V)^2} < 1 \quad (93)$$

Further simplification gives;

$$\left(\frac{\beta b_H \omega b_V \gamma}{\mu_V{}^2 \mu_H N_H N_V (\mu_H+\gamma)(\mu_H+\alpha_H+\delta)}\right)^2 < 1 \quad (94)$$

$$\text{But } \Re_0{}^2 = \left(\frac{\beta b_H \omega b_V \gamma}{\mu_V{}^2 \mu_H N_H N_V \left(\mu_H + \gamma\right)\left(\mu_H + \alpha_H + \delta\right)} \right) \tag{95}$$

It implies

$$(\Re_0{}^2)^2 < 1 \tag{96}$$

Which can further be simplified as;

$$\Re_0^4 < 1 \tag{97}$$

Hence $\Re_0 < 1$.

This proves that the disease-free equilibrium of the system is locally asymptotically stable.

2.10.2 Local stability of endemic equilibrium

In likewise manner as above, the Jacobian matrix, J of the system evaluated at the endemic equilibrium (EE). This is done by differentiating each of the system with respect to each compartment and then substituting endemic equilibrium of each compartment. The Jacobian matrix evaluated at the endemic equilibrium is represented below;

$$J(EE) = \begin{bmatrix} -\left(\mu_H + \frac{\mu_H \eta_3 (\Re_0^2 - 1)}{(\eta_3 + \gamma\omega b_H)}\right) & 0 & 0 & 0 & 0 & 0 & -\frac{\beta b_H (\beta b_V + \mu_H \mu_V N_V \Re_0^2)}{N_V \mu_H \eta_2 \Re_0^2} \\ \frac{\mu_H \eta_3 (\Re_0^2 - 1)}{(\eta_3 + \gamma\omega b_H)} & -(\mu_H + \gamma) & 0 & 0 & 0 & 0 & \frac{\beta b_H (\beta b_V + \mu_H \mu_V N_V \Re_0^2)}{N_V \mu_H \eta_2 \Re_0^2} \\ 0 & \gamma & -(\alpha_H + \mu_H + \delta) & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta & -(\alpha_H + \mu_H + \varepsilon) & 0 & 0 & 0 \\ 0 & 0 & 0 & \varepsilon & -\mu_H & 0 & 0 \\ 0 & 0 & -\frac{b_V N_V \omega \eta_2}{\mu_V N_H^2 (\beta b_V + N_V \mu_V \mu_H \Re_0^2)} & 0 & 0 & -\left(\mu_V + \frac{\mu_V^2 \mu_H N_V (\Re_0^2 - 1)}{\eta_2}\right) & 0 \\ 0 & 0 & \frac{b_V N_V \omega \eta_2}{\mu_V N_H^2 (\beta b_V + N_V \mu_V \mu_H \Re_0^2)} & 0 & 0 & \frac{\mu_V^2 \mu_H N_V (\Re_0^2 - 1)}{\eta_2} & -\mu_V \end{bmatrix} \tag{98}$$

Applying the Geshgorin's Corollary to the above matrix gives;

$$\left.\begin{aligned} &\left(\mu_H + \frac{\mu_H \eta_3 (\Re_0^2 - 1)}{(\eta_3 + \gamma\omega b_H)}\right) > -\frac{\beta b_H (\beta b_V + \mu_H \mu_V N_V \Re_0^2)}{N_V \mu_H \eta_2 \Re_0^2} \\ &(\mu_H + \gamma) > \frac{\mu_H \eta_3 (\Re_0^2 - 1)}{(\eta_3 + \gamma\omega b_H)} + \frac{\beta b_H (\beta b_V + \mu_H \mu_V N_V \Re_0^2)}{N_V \mu_H \eta_2 \Re_0^2} \\ &(\alpha_H + \mu_H + \delta) > \gamma \\ &(\alpha_H + \mu_H + \varepsilon) > \delta \\ &\mu_H > \varepsilon \\ &\left(\mu_V + \frac{\mu_V^2 \mu_H N_V (\Re_0^2 - 1)}{\eta_2}\right) > -\frac{b_V \omega \eta_2 N_V}{N_H^2 \mu_V \left(\beta b_V + N_V \mu_V \mu_H \Re_0^2\right)} \\ &\mu_V > \frac{b_V N_V \omega \eta_2}{N_H^2 \mu_V \left(\beta b_V + N_V \mu_V \mu_H \Re_0^2\right)} + \frac{\mu_V^2 \mu_H N_V (\Re_0^2 - 1)}{\eta_2} \end{aligned}\right\} \tag{99}$$

From the above equations, first, second, fifth and sixth equation gives us; $\Re_0^2 - 1$. $\Re_0^2$ is positive on itself. Due to that, For the inequalities above to hold $\Re_0^2 - 1 > 0$, which implies that $\Re_0^2 > 1$. Hence $\Re_0 > 1$.

Therefore, this proves the endemic equilibrium to be locally asymptotically stable.

2.11 Global stability of equilibrium solutions

2.11.1 Global stability of DFE

Using the approach by [13], the Lyapunov function is defined as:

$$W(t) = (S_H - S_H^{**} \ln S_H) + E_H + I_H + I_{SH} + R_H + (S_V - S_V^{**} \ln S_V) + I_V \tag{100}$$

With the assumption that $\mu_H = \frac{\omega S_V^{**}}{N_H}$ and $\mu_V = \frac{\beta S_H^{**}}{N_V}$, where S_H^{**} and S_V^{**} are respectively the equilibrium points of the susceptible host and susceptible vector populations.

Differentiating the above Lyapunov function with respect to time gives;

$$\dot{W}(t) = \dot{S}_H \left(1 - \frac{S_H^{**}}{S_H}\right) + \dot{E}_H + \dot{I}_H + \dot{I}_{SH} + \dot{R}_H + \dot{S}_V \left(1 - \frac{S_V^{**}}{S_V}\right) + \dot{I}_V \tag{101}$$

By substituting system of equations (14.1) of the model yields;

$$\begin{aligned}\dot{W}(t) = &\left(b_H - \mu_H S_H - \frac{\beta S_H I_V}{N_V}\right)\left(1 - \frac{S_H^{**}}{S_H}\right) + \left(\frac{\beta S_H I_V}{N_V} - \mu_H E_H - \gamma E_H\right) + \\ &\left(\gamma E_H - \mu_H I_H - \alpha_H I_H - \delta I_H\right) + \left(\delta I_H - \mu_H I_{SH} - \alpha_H I_{SH} - \varepsilon I_{SH}\right) + \left(\varepsilon I_{SH} - \mu_H R_H\right) \\ &+ \left(b_V - \mu_V S_V - \frac{\omega S_V I_H}{N_H}\right)\left(1 - \frac{S_V^{**}}{S_V}\right) + \left(\frac{\omega S_V I_H}{N_H} - \mu_V I_V\right)\end{aligned} \tag{102}$$

Further simplification gives;

$$\begin{aligned}\dot{W}(t) = &\, b_H \left(1 - \frac{S_H^{**}}{S_H}\right) - \mu_H S_H \left(1 - \frac{S_H^{**}}{S_H}\right) + \left(\frac{\beta S_H^{**}}{N_V} - \mu_V\right) I_V - \mu_H E_H - \alpha_H I_H \\ &- \mu_H I_{SH} - \alpha_H I_{SH} - \mu_H R_H + b_V \left(1 - \frac{S_V^{**}}{S_V}\right) - \mu_V S_V \left(1 - \frac{S_V^{**}}{S_V}\right) + \left(\frac{\omega S_V^{**}}{N_H} - \mu_H\right) I_H\end{aligned} \tag{103}$$

Substituting the assumptions $\mu_H = \frac{\omega S_V^{**}}{N_H}$ and $\mu_V = \frac{\beta S_H^{**}}{N_V}$,the simplified equation may be rewritten as;

$$\begin{aligned}\dot{W}(t) = &\, b_H \left(1 - \frac{S_H^{**}}{S_H}\right) - \mu_H S_H^{**} \left(1 - \frac{S_H^{**}}{S_H}\right) - \mu_H E_H - \alpha_H I_H - \left(\mu_H + \alpha_H\right) I_{SH} \\ &- \mu_H R_H + b_V \left(1 - \frac{S_V^{**}}{S_V}\right) - \mu_V S_V^{**} \left(1 - \frac{S_V^{**}}{S_V}\right)\end{aligned} \tag{104}$$

At the DFE, $S_H^{**} = \frac{b_H}{\mu_H}$ and $S_V^{**} = \frac{b_V}{\mu_V}$, substitution into rewritten equation gives;

$$\begin{aligned}\dot{W}(t) = &\, b_H \left(1 - \frac{S_H^{**}}{S_H}\right) + b_H \left(1 - \frac{S_H}{S_H^{**}}\right) - \mu_H E_H - \alpha_H I_H - \left(\mu_H + \alpha_H\right) I_{SH} \\ &- \mu_H R_H + b_V \left(1 - \frac{S_V^{**}}{S_V}\right) + b_V \left(1 - \frac{S_V}{S_V^{**}}\right)\end{aligned} \tag{105}$$

Further simplification gives;

$$\dot{W}(t) = -b_H\left(\frac{(S_H^{**} - S_H)^2}{S_H S_H^{**}}\right) - b_V\left(\frac{(S_V^{**} - S_V)^2}{S_V S_V^{**}}\right) - \mu_H E_H - \alpha_H I_H - (\mu_H + \alpha_H) I_{SH} - \mu_H R_H \tag{106}$$

It implies that $\dot{W}(t) < 0$, therefore this proves that the disease-free equilibrium (DFE) of the system is globally asymptotically stable.

2.11.2 Global stability of EE

Theorem 4.3: Endemic equilibrium of the model is globally asymptotically stable in whenever $\Re_0 > 1$.
Using the approach by [14], the Lyapunov function is defined as:

$$\begin{aligned} V(t) &= \left(S_H - S_H^* \ln S_H\right) + \left(E_H - E_H^* \ln E_H\right) + \left(I_H - I_H^* \ln I_H\right) + \left(I_{SH} - I_{SH}^* \ln I_{SH}\right) \\ &+ \left(R_H - R_H^* \ln R_H\right) + \left(S_V - S_V^* \ln S_V\right) + \left(I_V - I_V^* \ln I_V\right) \end{aligned} \tag{107}$$

Differentiating the above Lyapunov function with respect to time gives;

$$\begin{aligned} \dot{V}(t) &= \left(1 - \frac{S_H^*}{S_H}\right)\dot{S}_H + \left(1 - \frac{E_H^*}{E_H}\right)\dot{E}_H + \left(1 - \frac{I_H^*}{I_H}\right)\dot{I}_H + \left(1 - \frac{I_{SH}^*}{I_{SH}}\right)\dot{I}_{SH} \\ &+ \left(1 - \frac{R_H^*}{R_H}\right)\dot{R}_H + \left(1 - \frac{S_V^*}{S_V}\right)S_V + \left(1 - \frac{I_V^*}{I_V}\right)\dot{I}_V \end{aligned} \tag{108}$$

By substituting system of equation (4.1) of the model yields;

$$\begin{aligned} \dot{V}(t) &= \left(1 - \frac{S_H^*}{S_H}\right)\left(b_H - \mu_H S_H - \frac{\beta S_H I_V}{N_V}\right) + \left(1 - \frac{E_H^*}{E_H}\right)\left(\frac{\beta S_H I_V}{N_V} - \mu_H E_H - \gamma E_H\right) \\ &+ \left(1 - \frac{I_H^*}{I_H}\right)\left(\gamma E_H - I_H(\mu_H + \alpha_H + \delta)\right) + \left(1 - \frac{I_{SH}^*}{I_{SH}}\right)\left(\delta I_H - I_{SH}(\mu_H + \alpha_H + \varepsilon)\right) \\ &+ \left(1 - \frac{R_H^*}{R_H}\right)\left(\varepsilon I_{SH} - \mu_H R_H\right) + \left(1 - \frac{S_V^*}{S_V}\right)\left(b_V - \mu_V S_V - \frac{\omega S_V I_H}{N_H}\right) + \left(1 - \frac{I_V^*}{I_V}\right)\left(\frac{\omega S_V I_H}{N_H} - \mu_V I_V\right) \end{aligned} \tag{109}$$

Further simplification gives;

$$\begin{aligned} \dot{V}(t) &= b_H\left(1 - \frac{S_H^*}{S_H}\right) - \mu_H S_H\left(1 - \frac{S_H^*}{S_H}\right) + \frac{\beta S_H I_V}{N_V}\left(\frac{S_H^*}{S_H} - \frac{E_H^*}{E_H}\right) - \mu_H E_H\left(1 - \frac{E_H^*}{E_H}\right) \\ &+ \gamma E_H\left(\frac{E_H^*}{E_H} - \frac{I_H^*}{I_H}\right) - I_H(\mu_H + \alpha_H)\left(1 - \frac{I_H^*}{I_H}\right) + \delta I_H\left(\frac{I_H^*}{I_H} - \frac{I_{SH}^*}{I_{SH}}\right) \\ &- I_{SH}(\mu_H + \alpha_H)\left(1 - \frac{I_{SH}^*}{I_{SH}}\right) + \varepsilon I_{SH}\left(1 - \frac{I_{SH}^*}{I_{SH}}\right) - \mu_H R_H\left(1 - \frac{R_H^*}{R_H}\right) + \\ &b_V\left(1 - \frac{S_V^*}{S_V}\right) - \mu_V S_V\left(1 - \frac{S_V^*}{S_V}\right) + \frac{\omega S_V I_H}{N_H}\left(\frac{S_V^*}{S_V} - \frac{I_V^*}{I_V}\right) - \mu_V I_V\left(1 - \frac{I_V^*}{I_V}\right) \end{aligned} \tag{110}$$

Which can be rewritten as;

$$\dot{V}(t) = -b_H\left(\frac{S_H^*}{S_H}-1\right)-\mu_H\left(\left(S_H-S_H^*\right)+\left(E_H-E_H^*\right)\right)-\frac{\beta S_H I_V}{N_V}\left(\frac{E_H^*}{E_H}-\frac{S_H^*}{S_H}\right)-\gamma E_H\left(\frac{I_H^*}{I_H}-\frac{E_H^*}{E_H}\right)$$
$$-(\mu_H+\alpha_H)\left(\left(I_H-I_H^*\right)+\left(I_{SH}-I_{SH}^*\right)\right)-\delta I_H\left(\frac{I_{SH}^*}{I_{SH}}-\frac{I_H^*}{I_H}\right)-\varepsilon I_{SH}\left(\frac{R_H^*}{R_H}-\frac{I_{SH}^*}{I_{SH}}\right) \quad (111)$$
$$-\mu_H R_H\left(1-\frac{R_H^*}{R_H}\right)-b_V\left(\frac{S_V^*}{S_V}-1\right)-\mu_V S_V\left(1-\frac{S_V^*}{S_V}\right)-\frac{\omega S_V I_H}{N_H}\left(\frac{I_V^*}{I_V}-\frac{S_V^*}{S_V}\right)-\mu_V I_V\left(1-\frac{I_V^*}{I_V}\right)$$
$$\Rightarrow \dot{V}(t) \le 0$$

Thus $\dot{V}(t) \le 0$ for $\Re_0 > 1$, therefore this proves that the endemic equilibrium (EE) of the system is globally asymptotically stable according to LaSalle's Invariance Principle [15].

2.12 Numerical simulations

In this section, the numerical simulations of yellow fever model are presented values which were taken from various sources and others estimated. These parameter values are indicated in Table 1. All simulations in this section were performed using MATLAB Software.

Table 1. Parameters, Values, Interpretation, and their sources

Parameter	Interpretation	Value	Source
SH (0)	Susceptible Human	90,000	Estimated
EH (0)	Exposed Human	30,000	Estimated
IH (0)	Infected Human	10,000	Estimated
ISH (0)	Isolation Human	200	Estimated
RH (0)	Recovered Human	0	Estimated
SV (0)	Susceptible Vector	40,000	Estimated
IV (0)	Infected Vector	5,000	Estimated
NH	Total population of Human	130,200	Estimated
bH	Birth (Recruitment) Human	4.9×10-5	[16]
μH	Natural death rate of Human	3×10-5 6×10-5	[12]
αH	Disease induced death rate of Humans	0.0001 0.0004	[12]
β	Transmission probability from IV to SH	0.167-0.3	[12]
ε	Rate of recovery	0.25 – 0.33	[12]
NV	Total population of Vector	450,000	Estimated
μV	Natural death rate of vector	0.0287 – 0.25	[12]
bV	Birth (Recruitment) Vector	0.05 – 0.1	[12]
γ	Progression rate of exposed human	0.01 – 0.04	[12]
ω	Transmission probability from IH to SV	0.15 – 1.0	[12]
δ	Rate of Isolation	0.04	Estimated

The graph of the transmission dynamics of the various compartments considered in the study is shown in Fig. 2. As observed from Fig. 2, there is a steady decline in the number of susceptible humans, corresponding to an increase in the exposed, infective, isolated, and recovered human populations during the initial stage of the disease. Over time, the susceptible, exposed, infective, isolated, and recovered human populations become asymptotic to the horizontal axis. Similarly, the susceptible vector population shows a sharp decline at the onset of the disease and, after some time, follows a characteristic curve. The infectious vector population exhibits a similar pattern to that of the infective human population.

Fig. 3 shows the dynamics of the susceptible human population. Initially, there is no disease infection, and no individuals have immunity to yellow fever, so the susceptible human population equals the total human population. At the onset of infection, the susceptible human population decreases gradually and becomes asymptotic over time as the disease spreads through the population. This occurs because, as the disease enters the population, individuals progress from the susceptible class to the infectious class through the exposed class,

causing the susceptible population to decrease and become asymptotic to the horizontal axis over time. Since the disease induces permanent immunity in individuals, those who recover do not return to the susceptible class.

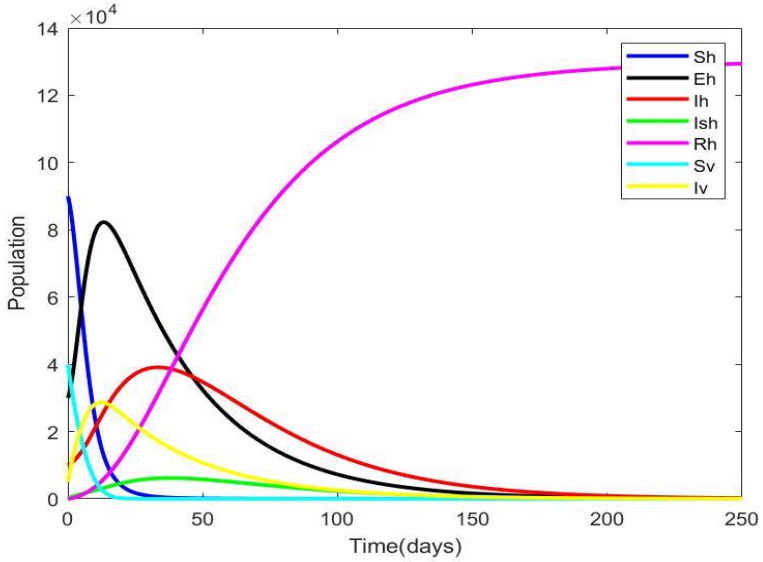

Fig. 2. Simulation of the human and vector (Mosquito) population

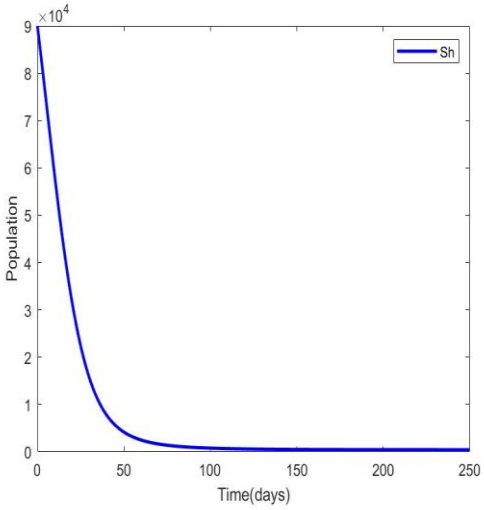

Fig. 3. Simulation of susceptible human

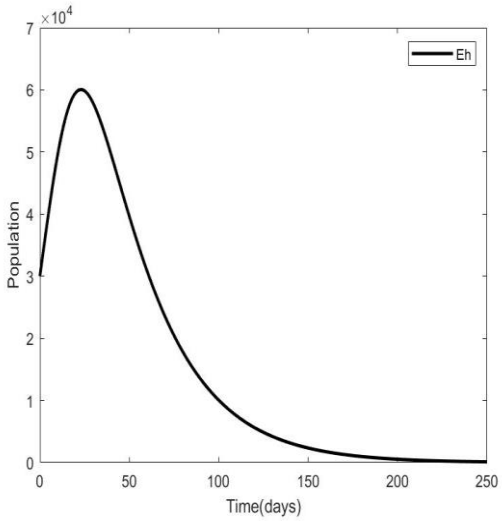

Fig. 4. Simulation of exposed human

Observations from Fig. 4 indicate that as infectives are introduced into the susceptible class, they become exposed to the disease. This results in a continuous increase in the exposed human class until it reaches its maximum, then decreases and becomes asymptotic to the horizontal axis over time. After a few days, members of the exposed class progress to the infectious human class. In Fig. 5, it is observed that initially, few individuals get infected with the disease, but as time progresses, the infection multiplies and the population becomes increasingly at risk. The infection rises to its peak and then begins to decline, eventually becoming asymptotic to the horizontal axis. The graph also demonstrates that the transmission rate significantly impacts the spread of the disease through the population. A higher transmission rate correlates with a higher rate of infection. Fig. 6 shows that initially, few infected individuals are isolated, but as time progresses, more infected individuals are isolated. This causes the isolation class to increase to its maximum and then begin to decrease, eventually becoming asymptotic to the horizontal axis.

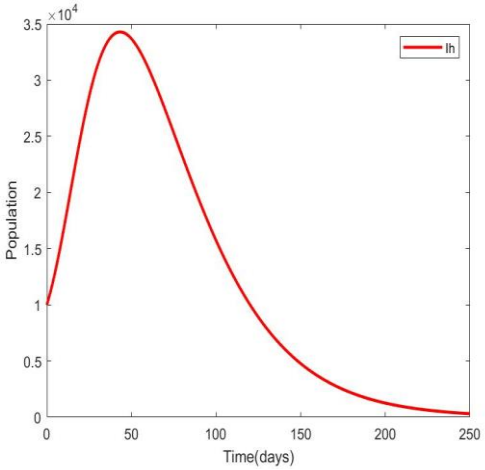

Fig. 5. Simulation of Infected Human

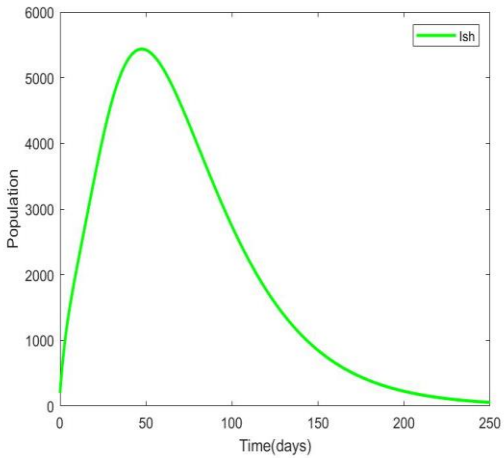

Fig. 6. Simulation of Isolated Human

Fig. 7 is the simulation of the recovered human class which shows gradual rise from 0 to around 130,000 human population size. It then continues to remain constant due to the moderate human recovery rate and very low disease-induced death rate. Recovered individuals gain permanent immunity to the disease and hence individuals do not become susceptible again.

Fig. 8 the susceptible vector population reveals a similar dynamic of the disease with respect to susceptible host population. It shows a gradual decline of susceptible vector with time. As number of susceptible vectors become infected to the disease, the susceptible vector population begin to reduce as they migrate to the infected class and becomes asymptotic to the horizontal as time goes on.

From Fig. 9 observing the graph of infected vector, the number of infectious increases as the vector encounter infected human. It continues to rise to a maximum point after which it begins to decline as some of the infected vectors die naturally and there is no disease-induced death as vectors do not die from the yellow fever virus infection. The number of infected vectors will continue to decrease until they all become extinct leaving only susceptible vectors in the system and becomes asymptotic to the horizontal as time goes on. The graph also demonstrates that the transmission rate has significant impact on the spread of the disease through the population. If the transmission rate is observed to be high then the rate of infection of the disease will also be high.

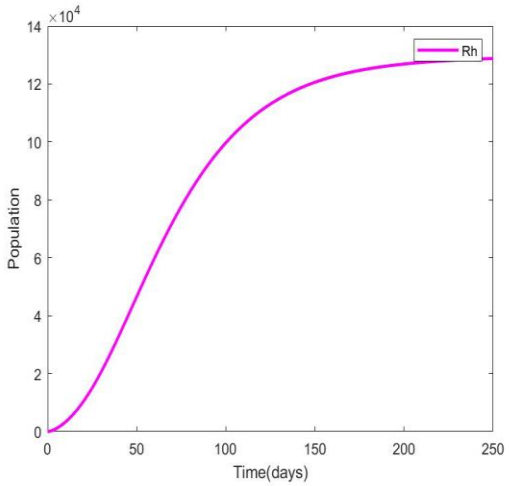

Fig. 7. Simulation of Recovered Human

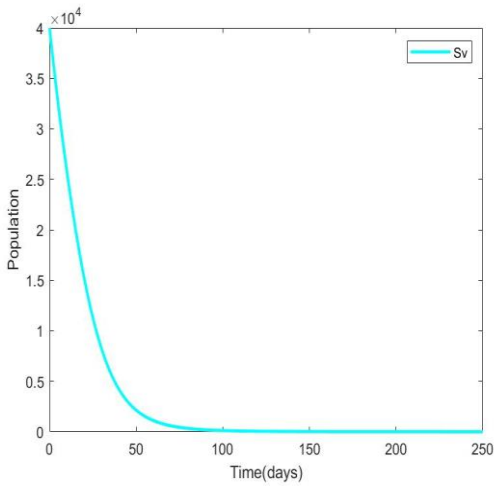

Fig. 8. Simulation of Susceptible Vector

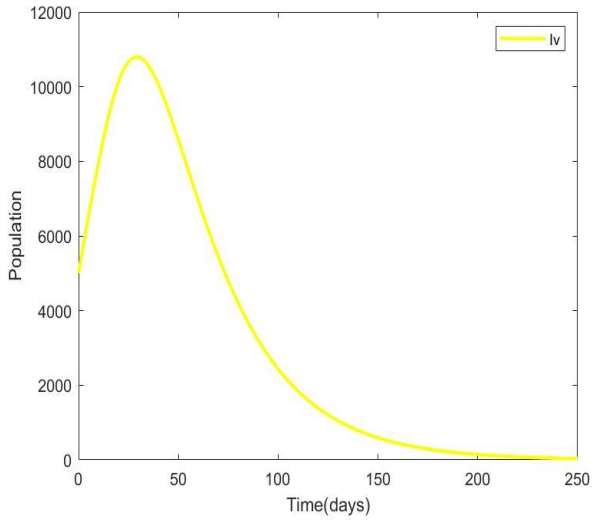

Fig. 9. Simulation of Infected Vector

3 Conclusion

The study utilized an SEIISR-SI epidemic model to analyze the stability of Yellow Fever transmission dynamics. Key components of the research included the determination of the basic reproduction number, comprehensive stability analysis, and numerical simulations. The analytical solution revealed that the basic reproduction number (R0) was less than the critical threshold of one, indicating that the infection would eventually die out under these conditions. The analysis demonstrated that the disease-free equilibrium is both locally and globally asymptotically stable, suggesting that the disease will be eradicated if no new infections are introduced. Similarly, the endemic equilibrium was found to be locally and globally asymptotically stable, meaning that if the disease persists, it will reach a steady state without leading to further outbreaks. An increase in the transmission rate led to a higher number of exposed and infectious individuals, highlighting a direct correlation between the transmission rate and disease spread. An increase in the number of infected vectors resulted in a decline in the susceptible vector population, underscoring the importance of vector control measures in managing the disease dynamics. This thorough analysis offers valuable insights into Yellow Fever transmission and provides a basis for effective control strategies derived from the model's findings.

Disclaimer (Artificial Intelligence)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of manuscripts.

Competing Interests

Authors have declared that no competing interests exist.

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