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MODELLING AND STABILITY ANALYSIS OF SVEIRS YELLOW FEVER TWO HOST MODEL

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Abstract. We describe transmission dynamics of yellow fever (YF) within two host populations, and build up a deterministic SVEIRS model with vaccination to the entire new born. The model aims at clarifying contributions of mathematical ideas in studying the impact of YF disease dynamics. We examine existence of equilibrium solutions and give out conditions that are sufficient for existence of realistic equilibria. Threshold of the form $R_0 = \sqrt{R_{hv} + R_{vm}}$ is obtained, where R_{hv} and R_{vm} are reproduction numbers for human-vector and vector-primate compartments respectively. Stability analysis of the equilibria is presented; trace-determinant approach is used in determining local stability of DFE and Metzler matrix for global stability. Lyapunov function is used in establishing conditions for global stability of EE. Our results suggests that eradication of the infection to human population is possible only if $R_{hv} < 1$ and $R_{vm} < 1$. Due to new births and immunity loss to YF after ten years, susceptible class will always be refilled and hence continuous vaccination is essential.

1. Introduction and preliminaries

Yellow fever (YF) is a zoonotic arboviral disease with a long history of outbreaks in human populations [27]. It is a viral hemorrhagic fever caused by yellow fever virus (YFV) and is transmitted through the bite of an infected female yellow fever mosquito, *Aedes aegypti* [25]. YFV is endemic to tropical areas of Africa and South America where it is maintained in sylvatic or jungle cycles between non-human primates and tree-dwelling mosquitoes [31]. Humans and primates are the principle reservoirs for the virus. Increasing migration, accelerating urbanization, and improved travel infrastructure are global trends that increase the risk of YF spreading to parts of the world where the disease had disappeared.

YFV is transmitted from mosquitoes to humans and/or primates by three different epidemiological infectious cycles [4]: sylvatic (or jungle), intermediate (savannah), and urban. Sylvatic yellow fever occurs in tropical rainforests, where primates that are infected by wild mosquitoes can pass the virus to other mosquitoes

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that feed on them. The infected mosquitoes bite humans entering the forest, resulting in sporadic cases of yellow fever. Majority of infections occur in young men working in the forest. In South America, the sylvatic cycle is currently the only way humans can infect each other [4].

Intermediate yellow fever occurs in humid or semi-humid parts of Africa, semi-domestic mosquitoes (that breed in the wild and around households) infect both primates and humans. Increased contact between people and infected mosquitoes leads to transmission. An outbreak can become a more severe epidemic if the infection is carried into an area populated with both domestic mosquitoes and unvaccinated people.

In Africa, savannah is the most common type of outbreak [34]. Large epidemics of urban yellow fever occur when infected people introduce the virus into densely populated areas with a high number of non-immune people and *Aedes* mosquitoes. Infected mosquitoes transmit the virus from person to person.

In humans, yellow fever's incubation period is three to six days. During this time, there are generally no symptoms identifiable to the host [28]. After that time, fever, headache, abdominal pain and vomiting develop which results into shock, bleeding and signs of liver and kidney failure. Liver failure is associated with jaundice hence the name 'yellow fever'.

The annual incidence of YF in South America varied from 46 to 524 cases between 1990-2004, for an average incidence of 168 cases [21], and is endemic in 10 countries. In sub-Saharan Africa, the incidence being in over 30 countries. Thus, it is thought that there is a great degree of under-reporting of this disease. Estimates of the global burden of YF adjusted for underreporting is about 200,000 cases and 30,000 deaths every year in unvaccinated populations [35] and today 90% of the infections occur in African continent [28]. Again, it is noted that up to 50% of severely affected persons without treatment will die from YF.

Also, estimate shows that the overall incidence of YF in Africa has been nearly 5 times higher than in South America [21] although an effective vaccine (yellow fever 17D) has been available since the late 1930s. Again, considering the current level of vaccination coverage, Ferguson *et al.* [9] estimated that the YF burden in Africa for the year 2013 as 130,000 (95% Confidence Interval, CI 51,000-380,000) cases with fever and jaundice or haemorrhage including 78,000 (95%, CI 19,000-180,000) deaths.

Liu *et al.* [18] argued that, mathematical analysis and modelling operation of infectious diseases are critical to the studying of virus spreading dynamics which can state clearly the origination and evolution of viruses. Also, according to Tumwiine *et al.* [29], mathematical modelling can help in figuring out decisions that are of significant importance and increase influence the theory and practice

of disease management and control. They might provide comprehensive examinations that enter into decisions in a way that human reasoning and debate cannot.

Many mathematical models have been developed in the literature to address vector-borne diseases transmission dynamics and control. Most YF models are theoretically based [21, 33, 3, 2, 6, 26] and very few ones [9, 13] use statistical models.

Ferguson *et al.* [9] use generalised linear regression models to fit on to a dataset of the locations of yellow fever outbreaks within the last 25 years to estimate the probability of outbreak across the endemic zone. Johansson *et al.* [13] use statistical model in studying incubation periods of YF virus, Amaku *et al.* [1] use theoretical model in addressing the role of vaccination in reducing YF dynamics and Monath and Cetron [20] conducted a theoretical study to address transmission and prevention of YF in persons traveling to the tropics. All these theoretical and statistical models have considered one host only and are not mathematically based. Thus, a lot need to be done mathematically in addressing the YF dynamics and control.

In this article, we develop and analyse a SVEIRS (Susceptible, Vaccinated, Exposed, Infectious, Recovered, Susceptible) mathematical model of YF between two hosts (primates and human beings) in order to gain an insight of the dynamics and prevention of YF. We consider variable population size into which migration of individuals is allowed but not infectious humans because we assume that most people who are sick will not travel.

2. Model Formulation

We formulate a model for the spread of YF in humans, vector (mosquito) and primates populations with the total population sizes at time t given by $N_H(t)$, $N_V(t)$ and $N_M(t)$ respectively. The populations are further compartmentalized into epidemiological classes as shown in the model flow diagram in Figure 1. The vector and primate compartments of the model do not include the immune class as they never recover from the infection, that is their infective period ends with their death due to their relatively short life cycle.

As indicated in the compartmental diagram Figure 1, the model divides the human population into 5 classes: susceptible, S_H , vaccinated, V_H, exposed, E_H, infectious, I_H and recovered (immune), R_H. People enter susceptible class either through per capita birth at a constant rate b_H or through immigration (Λ) whereby proportion ρ of the immigrants enter to the vaccinated class. We do not include the immigration of infectious humans because we assume that people who are coming from YF endemic zones have YF vaccination certificate. Susceptible individuals may choose to be vaccinated at the rate ε.

We divide the vector (mosquito) population into 3 classes: susceptible, S_V, exposed, E_V, and infectious, I_V. Female YF mosquitoes enter the susceptible class through birth then moves from the susceptible to the exposed class and later to the infectious class. The vector (mosquito) remains infectious for life [7] and leave the population through a per capita density-dependent natural death rate μ_v.

We also divide the primates population which is the source of infection [10] into 3 classes: susceptible, S_M, exposed, E_M, and infectious, I_M. When an infected primate is bitten by a tree-hole breeding mosquito, the mosquito acquires the virus and then the mosquito can pass the virus on to any number of other primates and humans it may bite when it comes across them. When human is bitten by an infected mosquito, the human may acquire the virus. The infected human returns to the city, where an urban mosquito *(Aedes aegypt)* serves as a viral vector spreading infection rapidly by bitting other humans.

In developing the model we assume that new born babies do not have the disease and thus they are directly recruited to the susceptible class, the efficacy of the vaccine is 100% effective for not more than ten years, the disease has no epidemiological effect on the demographic dynamics of the vector (mosquito) and we ignore bites of an infected female mosquito onto an infected human host.

However, we assume that the rate of relapse of vaccinated individual back to susceptibility is the same as that of recovered individuals because individuals who are vaccinated they lose efficacy of the vaccine after ten years and those who recover from the infection after ten years they lose immunity and need to be vaccinated again. Moreover, we do not consider vertical transmission of the infection in the vector population, and migration of primates was ignored, that is to say; mosquitoes that go to the primates habitats are the ones infected by the bites of infected primates and can infect other susceptible primates.

2.1. **Model Flow Diagram and Description.** Basing on the above assumptions, the model for transmission dynamics of YF in human, vector and primates population is as shown in Figure 1.

When an infectious female *Aedes aegypt* mosquito bites a susceptible human, there is some finite probability that the parasite will be passed on to the human and the person will move to the exposed class. After a certain period of time, people from the exposed class enter the infectious class at a rate δ that is the reciprocal of the duration of the latent period.

After some time, the infectious humans undergo treatment and recover at the rate γ, hence move to the recovered class. The recovered humans have some immunity to the disease and do not get clinically ill, after some years, they lose their immunity and return to the susceptible class at the rate ω. Humans leave the population through natural death rate μ_h, and through a per capita disease-induced death rate α, which is small in this case. However, like any other vector born diseases the YF disease induced death rate is very small in comparison with

TABLE 1. Description of parameters of the model system 4.

Symbol	*Description*
β_1	*transmission probability of vector to human*
β_2	*transmission probability of human to vector*
β_3	*transmission probability of primate to vector*
β_4	*transmission probability of vector to primate*
δ_h	*progression rate from* e_h *to* i_h
δ_v	*progression rate from* e_v *to* i_v
δ_m	*progression rate from* e_m *to* i_m
b_h	*birth rate of human*
b_v	*birth rate of vector*
b_m	*birth rate of primates*
a	*daily bitting rate*
γ	*recovery rate*
α	*death rate due to disease for human*
ω	*rate of relapse of vaccinated and recovered human*
ε	*vaccination rate of susceptible human*
ρ	*proportion of immigrant who are vaccinated*
σ	*arrival rate of immigrant per individual per time*
μ_h	*natural death rate of human*
μ_v	*natural death rate of vector*
μ_m	*natural death rate of primates*

the recovery rate [29].

Applying the assumptions, definition of variables and parameters and description of terms above, the differential equations which describe the dynamics of YF in the human, vector and primates population are formulated as shown below:

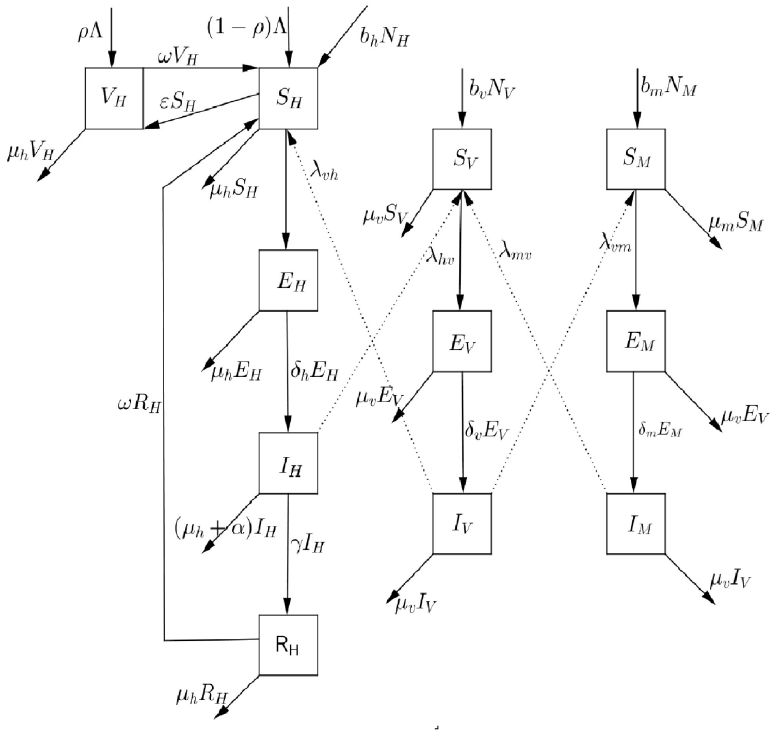

FIGURE 1. *model flow diagram for transmission dynamics of YF.*

Human:

$$\begin{aligned}
\frac{dS_H(t)}{dt} &= b_hN_H + (1-\rho)\Lambda + \omega(V_H + R_H) - \lambda_{vh} - \varepsilon S_H - \mu_hS_H,\\
\frac{dV_H(t)}{dt} &= \rho\Lambda + \varepsilon S_H - \omega V_H - \mu_hV_H,\\
\frac{dE_H(t)}{dt} &= \lambda_{vh} - \delta_hE_H - \mu_hE_H,\\
\frac{dI_H(t)}{dt} &= \delta_hE_H - (\mu_h+\alpha)I_H - \gamma I_H,\\
\frac{dR_H(t)}{dt} &= \gamma I_H - \mu_hR_H - \omega R_H,
\end{aligned} \tag{2.1}$$

Vector:

$$\begin{aligned}
\frac{dS_V(t)}{dt} &= b_vN_V - (\lambda_{hv} + \lambda_{mv}) - \mu_vS_V,\\
\frac{dE_V(t)}{dt} &= (\lambda_{hv} + \lambda_{mv}) - \delta_vE_V - \mu_vE_V,\\
\frac{dI_V(t)}{dt} &= \delta_vE_V - \mu_vI_V,
\end{aligned} \tag{2.2}$$

Primates:

$$\begin{aligned}
\frac{dS_M(t)}{dt} &= b_mN_M - \lambda_{vm} - \mu_mS_M,\\
\frac{dE_M(t)}{dt} &= \lambda_{vm} - \delta_mE_M - \mu_mE_M,\\
\frac{dI_M(t)}{dt} &= \delta_mE_M - \mu_mI_M.
\end{aligned} \tag{2.3}$$

where; $\lambda_{vh} = \dfrac{a\beta_1 S_H I_V}{N_V}$, $\lambda_{hv} = \dfrac{a\beta_2 S_V I_H}{N_H}$, $\lambda_{mv} = \dfrac{a\beta_3 S_V I_M}{N_M}$ and $\lambda_{vm} = \dfrac{a\beta_4 S_M I_V}{N_V}$.

In the model the term $\lambda_{vh} = \dfrac{a\beta_1 S_H I_V}{N_V}$ denotes the rate at which susceptible human hosts S_H get infected from the infected vector I_V (force of infection from vector to human), $\lambda_{hv} = \dfrac{a\beta_2 S_V I_H}{N_H}$ denotes the rate at which susceptible vector S_V get infected from the infected human host I_H (infection force from human host to vector), $\lambda_{mv} = \dfrac{a\beta_3 S_V I_M}{N_M}$ denotes the rate at which the susceptible vector S_V get infected from the infected primate I_M (force of infection from primate to vector) and the term $\lambda_{vm} = \dfrac{a\beta_4 S_M I_V}{N_V}$ denotes the rate at which the susceptible primates S_M get infected from the infected vector I_V. However, it is observed that the infected vector I_V can transmit the infection to both the human hosts and the primates.

The total population sizes $N_H(t)$, $N_V(t)$ and $N_M(t)$ can be determined by:

$$\begin{aligned} N_H(t) &= S_H(t) + V_H(t) + E_H(t) + I_H(t) + R_H(t), \\ N_V(t) &= S_V(t) + E_V(t) + I_V(t), \\ N_M(t) &= S_M(t) + E_M(t) + I_M(t). \end{aligned}$$

Also, adding from the differential equations, of the model system (2.1), (2.2) and (2.3) for the human host population, vector population and primates population, we have;

$$\frac{dN_H(t)}{dt} = \Lambda + (b_h - \mu_h)N_H - \alpha I_H, \tag{2.4}$$

$$\frac{dN_V(t)}{dt} = (b_v - \mu_v)N_V, \tag{2.5}$$

$$\frac{dN_M(t)}{dt} = (b_m - \mu_m)N_M. \tag{2.6}$$

The total population sizes of female mosquitos and primates, N_V and N_M is stationary for $b_v = \mu_v$ and $b_m = \mu_m$, declines for $b_v < \mu_v$ and $b_m < \mu_m$ and grows exponentially for $b_v > \mu_v$ and $b_m > \mu_m$ respectively.

2.2. **Dimensionless Transformation.** We transform our model equations into normalized quantities such that the total population for the normalized model is equal to 1. This can be done by scaling the population of each class by the total species population. We make the following transformation:

$s_h = \frac{S_H}{N_H}$, $v_h = \frac{V_H}{N_H}$, $e_h = \frac{E_H}{N_H}$, $i_h = \frac{I_H}{N_H}$, $r_h = \frac{R_H}{N_H}$, $s_v = \frac{S_V}{N_V}$, $e_v = \frac{E_V}{N_V}$,

$i_v = \frac{I_V}{N_V}$, $s_m = \frac{S_M}{N_M}$, $e_m = \frac{E_M}{N_M}$ and $i_m = \dfrac{I_M}{N_M}$.

in the classes S_H, V_H, E_H, I_H, R_H, S_V, E_V, I_V, S_M, E_M and I_M of the

populations respectively. Also, we define $\sigma = \frac{\Lambda}{N_H}$ as the arrival rate of immigrants per individual per unit time.

Differentiating the scaling equations and solving for the derivatives of the scaled variables, system (2.1), (2.2), (2.3) are combined to becomes a single normalised model as:

$$\begin{aligned}
\frac{ds_h}{dt} &= b_h + \sigma(1-\rho) + \omega v_h + \omega r_h - a\beta_1 s_h i_v - s_h(\varepsilon + b_h + \sigma) + \alpha s_h i_h,\\
\frac{dv_h}{dt} &= \rho\sigma + \varepsilon s_h - v_h(\omega + b_h + \sigma) + \alpha v_h i_h,\\
\frac{de_h}{dt} &= a\beta_1 s_h i_v - e_h(\delta_h + b_h + \sigma) + \alpha e_h i_h,\\
\frac{di_h}{dt} &= \delta_h e_h - i_h(\gamma + \alpha + b_h + \sigma) + \alpha i_h^2,\\
\frac{dr_h}{dt} &= \gamma i_h - r_h(\omega + b_h + \sigma) + \alpha r_h i_h\\
\frac{ds_v}{dt} &= b_v - (a\beta_2 s_v i_h + a\beta_3 s_v i_m) - s_v b_v,\\
\frac{de_v}{dt} &= a\beta_2 s_v i_h + a\beta_3 s_v i_m - e_v(\delta_v + b_v),\\
\frac{di_v}{dt} &= \delta_v e_v - i_v b_v,\\
\frac{ds_m}{dt} &= b_m - a\beta_4 s_m i_v - s_m b_m,\\
\frac{de_m}{dt} &= a\beta_4 s_m i_v - e_m(\delta_m + b_m),\\
\frac{di_m}{dt} &= \delta_m e_m - i_m b_m.
\end{aligned} \tag{2.7}$$

However, it is easy to show that $\frac{dn_h}{dt} = 0$, $\frac{dn_v}{dt} = 0$ and $\frac{dn_m}{dt} = 0$ for the humans, vector and primates population respectively. Where solutions are restricted to the hyperplanes $s_h + v_h + e_h + i_h + r_h = 1$, $s_v + e_v + i_v = 1$ and $s_m + e_m + i_m = 1$.

The YF model system (2.7) monitors human, mosquito (vector) and primates populations, we assume that all state variables and parameters of the model are non-negative $\forall t \geq 0$. Thus, the model will be analysed in a suitable feasible region where it makes biological sense. This region will be obtained as follows:

Lemma 2.1. *Solutions of the normalised model system (2.7) are contained in the region* $\Phi = \Phi_H \cup \Phi_V \cup \Phi_M \subset \Gamma_+^5 \times \Gamma_+^3 \times \Gamma_+^3$.

Proof. We split the model system into three parts; namely the human component (n_h), vector (mosquito) component (n_v) and the primates component (n_m), given respectively by, $n_h = s_h + v_h + e_h + i_h + r_h$, $n_v = s_v + e_v + i_v$ and $n_m = s_m + e_m + i_m$. Such that

$$\Phi_H = \{(s_h, v_h, e_h, i_h, r_h) \in \Gamma_+^5 : 0 < s_h + v_h + e_h + i_h + r_h \leq 1\}$$
$$\Phi_V = \{(s_v, e_v, i_v) \in \Gamma_+^3 : 0 < s_v + e_v + i_v \leq 1\}$$

$$\Phi_M = \{(s_m, e_m, i_m) \in \Gamma_+^3 : 0 < s_m + e_m + i_m \leq 1\}$$

Thus,

$$\Phi = \Phi_H \cup \Phi_V \cup \Phi_M \subset \Gamma_+^5 \times \Gamma_+^3 \times \Gamma_+^3. \tag{2.8}$$

that can be shown to be positively invariant with respect to the model system (2.7). From this lemma, we conclude that it is sufficient to consider the dynamics of the model system (2.7) in Φ. In this region, the model can be considered as being epidemiologically and mathematically well-posed [11]. □

3. Model Analysis

We now investigate the existence of disease-free equilibrium (E_0), basic reproduction number R_0, and disease endemic equilibrium EE. E_0 is obtained by setting the derivatives with respect to time of the model system (2.7), equal to zero. On computations, the following E_0 is obtained:

$$E_0 = \left(\frac{b_h + (1-\rho)\sigma + \omega}{\omega + \varepsilon + b_h + \sigma}, \frac{\rho\sigma + \varepsilon}{\omega + \varepsilon + b_h + \sigma}, 0, 0, 0, 1, 0, 0, 1, 0, 0\right) \tag{3.1}$$

3.1. The Basic Reproduction Number, R_0.

One of the most important concerns in the analysis of epidemiological models is the determination of the asymptotic behaviour of their solutions which is usually based on the stability of the associated equilibria [19]. These models typically consist of a disease-free equilibrium and at least one endemic equilibrium. The local stability of the disease-free equilibrium is determined based on a threshold parameter, known as the basic reproduction number, R_0.

An easy way to theoretically compute R_0 is to follow the approach described by Van den Driessche and Watmough [32]. According to this method, R_0 for our model system (2.7) is given by;

$$R_0 = \sqrt{\frac{a^2\beta_1\beta_2\delta_h\delta_v s_h^\circ}{(\delta_h + b_h + \sigma)(\gamma + \alpha + b_h + \sigma)b_v(\delta_v + b_v)} + \frac{a^2\beta_3\beta_4\delta_v\delta_m}{b_m(\delta_m + b_m)b_v(\delta_v + b_v)}} \tag{3.2}$$

where $s_h^\circ = \dfrac{b_h + (1-\rho)\sigma + \omega}{\omega + \varepsilon + b_h + \sigma}$ from the first component of E_0 in (3.1). [We refer the reader to Kung'aro *et al.* [16] for procedure of computing R_0 and its explanations].

In our model we have two hosts and one vector, and it is indicated in the model flow diagram (Figure 1) that the vector can transmit infection to both the human host and the primates. Thus, for easy understanding, we can represent the reproduction number as, $R_0 = \sqrt{R_{hv} + R_{vm}}$ or $R_0^2 = R_{hv} + R_{vm}$, such that

$$R_{hv} = \frac{(b_h + \sigma(1-\rho) + \omega)a^2\beta_1\beta_2\delta_h\delta_v}{(\omega + \varepsilon + b_h + \sigma)(\delta_h + b_h + \sigma)(\gamma + \alpha + b_h + \sigma)b_v(\delta_v + b_v)} \tag{3.3}$$

which is the reproduction number of human-vector compartments and represents the infection from vector to human and human to vector. And,

$$R_{mv} = \frac{a^2\beta_3\beta_4\delta_v\delta_m}{b_m(\delta_m + b_m)b_v(\delta_v + b_v)} \tag{3.4}$$

as the reproduction number of (monkey) primate-vector compartments. It represents the infection from primate to vector and vector to primate.

3.2. **Stability Analysis of E_0.** We determine condition under which the equilibrium points are asymptotically stable or unstable. Asymptotic stability implies that a solution starts close to the equilibrium remains close to the equilibrium and approaches the equilibrium over time $0 \leq t < \infty$, while instability of the equilibrium implies that there are solutions starting arbitrary close to the equilibrium but they do not approach it over indefinite time. However, if nearby initial conditions of a fixed point remain close to that point over a positive time then the fixed point is said to be locally stable and if it remains close over indefinite time it is said to be globally stable.

3.2.1. *Local Stability Analysis.* To establish local stability of this equilibrium, the Jacobian of the model system (2.7) is computed and evaluated at E_0. The local stability of E_0 is then determined based on the trace-determinant approach of this Jacobian. The equilibrium E_0 is locally stable if trace of the Jacobian matrix is less than zero and determinant of the same matrix is greater than zero.

Thus, by letting the functions $(f_1, f_2, ..., f_{11})$, to represent the right hand sides of the model equations (2.7), at steady state the Jacobian of (2.7) is given by:

$$J_i = \frac{\partial f_i}{\partial x_j} \tag{3.5}$$

where,
f_i , $i = 1, 2, ..., 11$. and $x_j (j = 1, 2, ..., 11)$ represent s_h, v_h, e_h, i_h, r_h, s_v, e_v, i_v, s_m, e_m, i_m, respectively. The following matrix is obtained;

$$J_{E_0} = \begin{bmatrix} -b_1 & \omega & 0 & \alpha s_h^\circ & \omega & 0 & 0 & -\eta & 0 & 0 & 0 \\ \varepsilon & -b_2 & 0 & \alpha v_h^o & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -b_3 & 0 & 0 & 0 & 0 & \eta & 0 & 0 & 0 \\ 0 & 0 & \delta_h & -b_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -b_5 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -b_v & 0 & 0 & 0 & 0 & -a\beta_3 \\ 0 & 0 & 0 & 0 & 0 & 0 & -b_7 & 0 & 0 & 0 & a\beta_3 \\ 0 & 0 & 0 & 0 & 0 & 0 & \delta_v & -b_v & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -a\beta_4 & -b_m & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a\beta_4 & 0 & -b_{10} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \delta_m & -b_m \end{bmatrix} \tag{3.6}$$

note that, $b_1 = (\varepsilon + b_h + \sigma)$, $b_2 = (\omega + b_h + \sigma)$, $b_3 = (\delta_h + b_h + \sigma)$, $\eta = a\beta_1 s_h^\circ$
$b_4 = (\alpha + \gamma + b_h + \sigma)$, $b_5 = (\omega + b_h + \sigma)$, $b_7 = (\delta_v + b_v)$, $b_{10} = (\delta_m + b_m)$.

As seen from (3.6) trace of $J_{E_0} < 0$. Also, by reducing the dimensions of matrix

J_{E_0} and little computation, we obtain sub-matrix

$$M = (b_v b_m) \begin{bmatrix} -b_1 & \omega & 0 & \alpha s_h^\circ & \omega & 0 & -\eta & 0 & 0 \\ \varepsilon & -b_2 & 0 & \alpha v_h^o & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -b_3 & 0 & 0 & 0 & \eta & 0 & 0 \\ 0 & 0 & \delta_h & -b_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -b_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -b_7 & 0 & a\beta_3 \\ 0 & 0 & 0 & 0 & 0 & \delta_v & -b_v & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a\beta_4 & -b_{10} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \delta_m & -b_m \end{bmatrix} \tag{3.7}$$

determinant of M is given by

$$det\ M = B[b_v(\delta_v + b_v)b_m(\delta_m + b_m) - a^2\beta_3\beta_4\delta_v\delta_m)] \tag{3.8}$$

where $B = b_v b_m(b_1 b_2 - \varepsilon\omega)(b_5 b_4 b_3)$. Further simplification leads to

$$det\ M = B\left[1 - R_{mv}\right] b_v(\delta_v + b_v)b_m(\delta_m + b_m) \tag{3.9}$$

For $det\ M > 0$, we should have $R_{mv} < 1$ which leads to the following theorem.

Theorem 3.1. *The disease-free equilibrium point E_0 of model system (2.7) is locally asymptotically stable if $R_{mv} < 1$ and unstable if $R_{mv} > 1$.*

The epidemiological implication of Theorem (3.1) is that the YF infection can be eradicated from the community (when $R_{mv} < 1$) if the initial sizes of the sub-population of the model system (2.7) are in basin of attraction of the DFE, E_0. The threshold quantity, R_0, represents the average number of secondary infections that one infected individual (or infected vector) can generate if introduced into a completely-susceptible population.

3.2.2. *Global Stability Analysis.* We address the issue of global asymptotic stability of the disease-free equilibrium. We give a sufficient condition for the global asymptotic stability for the DFE of YF. For some systems we can show that the global asymtotic stability (GAS) of the DFE is equivalent to $R_0 \leq 1$. Thus we can state the following theorem

Theorem 3.2. *The DFE, E_0 of the YF model system given by (2.7) is globally asymptotically stable (GAS) if $R_0 \leq 1$.*

Proof. To prove the theorem, we will use the equations of the normalised model system (2.7) and approach by Kamgang and Sallet and Dumont *et al.* [14, 7], it is possible to rewrite (2.7) in the following manner;

$$\begin{cases} \dfrac{dX_s}{dt} = A_1(x)(X_s - X_{DFE,s}) + A_3(x)X_i \\ \dfrac{dX_i}{dt} = A_2(x)X_i \end{cases} \tag{3.10}$$

where X_s is the vector representing the state of different compartments of non-transmitting individuals (e.g. susceptible, vaccinated, immune) and the vector X_i represents the state of compartments of different transmitting individuals (e.g. exposed, infected). Here, we have

$$X_s = (s_h, v_h, r_h, s_v, s_m)^T, X_i = (e_h, i_h, e_v, i_v, e_m, i_m)^T, \tag{3.11}$$

and

$$X_{DFE,s} = (n_h, 0, s_v, s_m). \tag{3.12}$$

with

$$A_1(x) = \begin{bmatrix} -(\varepsilon + b_h + \sigma) & \omega & \omega & 0 & 0 \\ \varepsilon & -(\omega + b_h + \sigma) & 0 & 0 & 0 \\ 0 & 0 & -(\omega + b_h + \sigma) & 0 & 0 \\ 0 & 0 & 0 & -b_v & 0 \\ 0 & 0 & 0 & 0 & -b_m \end{bmatrix} \tag{3.13}$$

$$A_3(x) = \begin{bmatrix} 0 & \alpha s_h & 0 & -a\beta_1 s_h & 0 & 0 \\ 0 & \alpha v_h & 0 & 0 & 0 & 0 \\ 0 & \gamma & 0 & 0 & 0 & 0 \\ 0 & -a\beta_2 s_v & 0 & 0 & 0 & a\beta_3 s_v \\ 0 & 0 & 0 & -a\beta_4 s_m & 0 & 0 \end{bmatrix} \tag{3.14}$$

and

$$A_2(x) = \begin{bmatrix} -(\delta_h + b_h + \sigma) & 0 & 0 & a\beta_1 s_h & 0 & 0 \\ \delta_h & -(\alpha + \gamma + b_h + \sigma) & 0 & 0 & 0 & 0 \\ 0 & a\beta_2 s_v & -(\delta_v + b_v) & 0 & 0 & a\beta_3 s_v \\ 0 & 0 & \delta_v & -bv & 0 & 0 \\ 0 & 0 & 0 & a\beta_4 s_m & -(\delta_m + b_m) & 0 \\ 0 & 0 & 0 & 0 & \delta_m & -b_m \end{bmatrix} \tag{3.15}$$

A direct computation shows that the eigenvalues of $A_1(x)$ are real and negative. Thus, the system

$$\frac{dX_s}{dt} = A_1(x)(X_s - X_{DFE,s}) + A_3(x)X_i, \tag{3.16}$$

is globally asymptotically stable at X_{DFE}. □

To check the stability of system $A_2(x)$, we are going to employ the idea of Metzler matrix and use the *lemma* by Kamgang and Sallet and Dumont *et al.* [14] and [7]. A Metzler matrix A is a matrix such that $A(i, j) > 0$, for any indices $i \neq j$ [5, 12]. These matrices are also called quasi-positive matrices or matrices whose off diagonal elements are non-negative.

Lemma 3.3. *Let Z be a square Metzler matrix written in block form*

$$Z = \begin{bmatrix} P & Q \\ R & S \end{bmatrix}$$

with P and S are square matrices. Z is Metzler stable if and only if the matrices P and $S - RP^{-1}Q$ are Metzler stable.

Proof. Comparing our matrix $A_2(x)$ and a square Metzler matrix Z given, we have matrices P, Q, R and S defined as;

$$P = \begin{bmatrix} -(\delta_h + b_h + \sigma) & 0 & 0 \\ \delta_h & -(\alpha + \gamma + b_h + \sigma) & 0 \\ 0 & a\beta_2 & -(\delta_v + b_v) \end{bmatrix}$$

$$Q = \begin{bmatrix} a\beta_1 s_h^\circ & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & a\beta_2 \end{bmatrix} \quad R = \begin{bmatrix} 0 & 0 & \delta_v \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad S = \begin{bmatrix} -bv & 0 & 0 \\ a\beta_4 & -(\delta_m + b_m) & 0 \\ 0 & \delta_m & -b_m \end{bmatrix}$$

Clearly, P is a stable Metzler matrix.

Then, after some computations we obtain

$$S - RP^{-1}Q = \begin{bmatrix} -b_v + \frac{\delta_v \delta_h a^2 \beta_1 \beta_2 s_h^\circ}{(\delta_h + b_h + \sigma)(\alpha + \gamma + b_h + \sigma)(\delta_v + b_v)} & 0 & \frac{\delta_v a \beta_2}{\delta_v + b_v} \\ a\beta_4 & -(\delta_m + b_m) & 0 \\ 0 & \delta_m & -b_m \end{bmatrix}$$

Thus, $(S - RP^{-1}Q)$ is Metzler stable iff

$$-b_v + \frac{\delta_v \delta_h a^2 \beta_1 \beta_2 s_h^\circ}{(\delta_h + b_h + \sigma)(\alpha + \gamma + b_h + \sigma)(\delta_v + b_v)} < 0$$

that is,

$$\frac{\delta_v \delta_h a^2 \beta_1 \beta_2 s_h^\circ}{(\delta_h + b_h + \sigma)(\alpha + \gamma + b_h + \sigma)(\delta_v + b_v)} < b_v$$

and so,

$$\frac{\delta_v \delta_h a^2 \beta_1 \beta_2 s_h^\circ}{(\delta_h + b_h + \sigma)(\alpha + \gamma + b_h + \sigma) b_v (\delta_v + b_v)} < 1 \tag{3.17}$$
$$\implies R_{hv} < 1$$

Thus, the DFE, E_0 is globally asymptotically stable if $R_{hv} < 1$ and unstable if $R_{hv} > 1$. □

In here we find that if the number of human new infections is greater than one, then the disease will persist. Alternatively, if the number of human new infections is less than one, then the disease will die out. Essentially, we need to find ways of making sure that the number of newly human infecteds does not exceed one.

3.3. **Endemic Equilibrium Point (EE).** In the presence of infection in the population, the model system (2.7) has an equilibrium point called disease endemic equilibrium point denoted by P^*. P^* as the endemic equilibrium denotes the fraction of the population that is infected at an infinite time in the future and is given by
$P^* = (s_h^*, v_h^*, e_h^*, i_h^*, r_h^*, s_v^*, e_v^*, i_v^*, s_m^*, e_m^*, i_m^*)$.

Thus using model system (2.7), we can express each variable in terms of the other in endemic equilibrium as follows;

$$s_h^* = \frac{b_h + (1-\rho)\sigma + \omega(v_h^* + r_h^*)}{a\beta_1 i_v^* + (\varepsilon + b_h + \sigma) - \alpha i_h^*}, \qquad v_h^* = \frac{\rho\sigma + \varepsilon s_h^*}{(\omega + b_h + \sigma) - \alpha i_h^*}$$
$$e_h^* = \frac{a\beta_1 s_h^* i_v^*}{(\delta_h + b_h + \sigma) - \alpha i_h^*}, \qquad i_h^* = \frac{\delta_h e_h^*}{(\alpha + \gamma + b_h + \sigma) - \alpha i_h^*},$$
$$r_h^* = \frac{\gamma i_h^*}{(\omega + b_h + \sigma) - \alpha i_h^*}, \qquad s_v^* = \frac{b_v}{(a\beta_2 i_h^* + a\beta_3 i_m^*) + b_v},$$
$$e_v^* = \frac{(a\beta_2 s_v^* i_h^* + a\beta_3 s_v^* i_m^*)}{(\delta_v + b_v)}, \qquad i_v^* = \frac{\delta_v e_v^*}{b_v},$$
$$s_m^* = \frac{b_m}{(a\beta_4 i_v^* + b_m)}, \qquad e_m^* = \frac{(a\beta_4 s_m^* i_v^*)}{(\delta_m + b_m)}, \qquad i_m^* = \frac{\delta_m e_m^*}{b_m}.$$

3.3.1. *Stability Analysis of EE.* Since the DFE is locally asymptotically stable in Φ, this suggests the local stability of the EE for the reverse condition [32]. Hence, we only need to investigate the global stability of the EE. We explore the global stability of the EE through construction of suitable Lyapunov function using the approach by [23, 15, 8, 24, 30]. In this approach we construct Lyapunov function basing on the following form;

$$L = \sum a_i \left(x_i - x_i^* \; ln \; x_i\right) \tag{3.18}$$

where a_i is the constant selected properly, that is $a_i > 0$, x_i is the population of the i^{th} compartment and x_i^* is the equilibrium point, and for this case, it is an endemic equilibrium. The approach has been found to be useful for compartmental epidemic models regardless number of compartments [15]. Thus, consider the Lyapunov function

$$\begin{aligned} L = a_1 \left(s_h - s_h^* \; ln \; s_h\right) + a_2 \left(v_h - v_h^* \; ln \; v_h\right) + a_3 \left(e_h - e_h^* \; ln \; e_h\right) \\ + a_4 \left(i_h - i_h^* \; ln \; i_h\right) + a_5 \left(r_h - r_h^* \; ln \; r_h\right) + a_6 \left(s_v - e_v^* \; ln \; e_v\right) \\ + a_7 \left(e_v - e_v^* \; ln \; e_v\right) + a_8 \left(i_h - i_h^* \; ln \; i_h\right) + a_9 \left(s_m - s_m^* \; ln \; s_m\right) \\ + a_{10} \left(e_m - e_m^* \; ln \; e_m\right) + a_{11} \left(i_m - i_m^* \; ln \; i_m\right) \end{aligned} \tag{3.19}$$

where $a_i > 0$ for $i = 1, 2, ...11$.
Analyzing in three different compartments for human, vector and primate respectively we have;

$$\begin{aligned} L_h(s_h, v_h, e_h, i_h, r_h) = a_1 \left(s_h - s_h^* \; ln \; s_h\right) + a_2 \left(v_h - v_h^* \; ln \; v_h\right) \\ + a_3 \left(e_h - e_h^* \; ln \; e_h\right) + a_4 \left(i_h - i_h^* \; ln \; i_h\right) \\ + a_5 \left(r_h - r_h^* \; ln \; r_h\right) \end{aligned} \tag{3.20}$$

Differentiating (3.20) with respect to time we get

$$\begin{aligned}
\frac{dL_h}{dt} &= a_1\left[1-\frac{s_h^*}{s_h}\right]\frac{ds_h}{dt} + a_2\left[1-\frac{v_h^*}{v_h}\right]\frac{dv_h}{dt} + a_3\left[1-\frac{e_h^*}{e_h}\right]\frac{de_h}{dt} \\
&\quad + a_4\left[1-\frac{i_h^*}{i_h}\right]\frac{di_h}{dt} + a_5\left[1-\frac{r_h^*}{r_h}\right]\frac{dr_h}{dt} \\
&= a_1\left(1-\frac{s_h^*}{s_h}\right)\left[b_h+\sigma(1-\rho)+\omega v_h+\omega r_h - a\beta_1 s_h i_v - s_h(\varepsilon+b_h+\sigma)+\alpha s_h i_h\right] \\
&\quad + a_2\left(1-\frac{v_h^*}{v_h}\right)\left[\rho\sigma+\varepsilon s_h - v_h(\omega+b_h+\sigma)+\alpha v_h i_h\right] \\
&\quad + a_3\left(1-\frac{e_h^*}{e_h}\right)\left[a\beta_1 s_h i_v - e_h(\delta_h+b_h+\sigma)+\alpha e_h i_h\right] \\
&\quad + a_4\left(1-\frac{i_h^*}{i_h}\right)\left[\delta_h e_h - i_h(\gamma+\alpha+b_h+\sigma)+\alpha i_h^2\right] \\
&\quad + a_5\left(1-\frac{r_h^*}{r_h}\right)\left[\gamma i_h - r_h(\omega+b_h+\sigma)+\alpha r_h i_h\right]
\end{aligned}$$

At endemic equilibrium point (P^*), we have

$$\begin{aligned}
\frac{dL_h}{dt} &= a_1\left(1-\frac{s_h^*}{s_h}\right)\left[(a\beta_1 i_v^*+\varepsilon+b_h+\sigma)s_h^* - \omega(v_h^*+r_h^*) - \alpha s_h^* i_h^*\right. \\
&\quad \left. + \omega(v_h+r_h) - a\beta_1 s_h i_v - s_h(\varepsilon+b_h+\sigma)+\alpha s_h i_h\right] \\
&\quad + a_2\left(1-\frac{v_h^*}{v_h}\right)\left[v_h^*(\omega+b_h+\sigma) - \varepsilon s_h^* - \alpha v_h^* i_h^*\right. \\
&\quad \left. + \varepsilon s_h - v_h(\omega+b_h+\sigma)+\alpha v_h i_h\right] \\
&\quad + a_3\left(1-\frac{e_h^*}{e_h}\right)\left[a\beta_1 s_h i_v - e_h\left(\frac{a\beta_1 s_h^* i_v^* + \alpha e_h^* i_h^*}{e_h^*}\right)+\alpha e_h i_h\right] \\
&\quad + a_4\left(1-\frac{i_h^*}{i_h}\right)\left[\delta_h e_h - i_h\left(\frac{\delta_h e_h^* + \alpha i_h^{*2}}{i_h^*}\right)+\alpha i_h^2\right] \\
&\quad + a_5\left(1-\frac{r_h^*}{r_h}\right)\left[\gamma i_h - r_h\left(\frac{\gamma i_h^* + \alpha r_h^* i_h^*}{r_h^*}\right)+\alpha r_h i_h\right]
\end{aligned}$$

Further simplification yields

$$\begin{aligned}\frac{dL_h}{dt} = &-a_1\Big(\varepsilon + b_h + \sigma\Big)s_h\Big(1-\frac{s_h^*}{s_h}\Big)^2 - a_1\Big(1-\frac{s_h^*}{s_h}\Big)\Big[a\beta_1 s_h i_v\Big(1-\frac{i_v^* s_h^*}{i_v s_h}\Big)\\ &- \alpha s_h i_h\Big(1-\frac{i_h^* s_h^*}{i_h s_h}\Big) - \omega v_h\Big(1-\frac{v_h^*}{v_h}\Big) - \omega r_h\Big(1-\frac{r_h^*}{r_h}\Big)\Big]\\ &- a_2\Big(\omega + b_h + \sigma\Big)v_h\Big(1-\frac{v_h^*}{v_h}\Big)^2 - a_2\Big(1-\frac{v_h^*}{v_h}\Big)\Big[\varepsilon s_h^*\Big(1-\frac{s_h}{s_h^*}\Big)\\ &+ \alpha v_h^* i_h^*\Big(1-\frac{v_h i_h}{v_h^* i_h^*}\Big)\Big]\\ &\frac{-a_3}{e_h^*}\Big(1-\frac{e_h^*}{e_h}\Big)\Big[a\beta_1 s_h^* e_h i_v^*\Big(1-\frac{s_h i_v e_h^*}{s_h^* i_v^* e_h}\Big) - \alpha e_h i_h e_h^*\Big(1-\frac{i_h^*}{i_h}\Big)\Big]\\ &\frac{-a_4}{i_h^*}\Big(1-\frac{i_h^*}{i_h}\Big)\Big[\delta_h e_h^* i_h\Big(1-\frac{e_h i_h^*}{e_h^* i_h}\Big) - \alpha i_h^* i_h^2\Big(1-\frac{i_h^*}{i_h}\Big)\Big]\\ &\frac{-a_5}{r_h^*}\Big(1-\frac{r_h^*}{r_h}\Big)\Big[r_h\gamma i_h^*\Big(1-\frac{i_h r_h^*}{i_h^* r_h}\Big) - \alpha r_h i_h r_h^*\Big(1-\frac{i_h^*}{i_h}\Big)\Big]\end{aligned}$$

which can also be written as

$$\begin{aligned}\frac{dL_h}{dt} = &-a_1\Big(\varepsilon + b_h + \sigma\Big)s_h\Big(1-\frac{s_h^*}{s_h}\Big)^2 - a_2\Big(\omega + b_h + \sigma\Big)v_h\Big(1-\frac{v_h^*}{v_h}\Big)^2\\ &+ F_h(s_h, v_h, e_h, i_h, r_h)\end{aligned} \tag{3.21}$$

where

$$\begin{aligned}F_h = &-a_1\Big(1-\frac{s_h^*}{s_h}\Big)\Big[a\beta_1 s_h i_v\Big(1-\frac{i_v^* s_h^*}{i_v s_h}\Big) - \alpha s_h i_h\Big(1-\frac{i_h^* s_h^*}{i_h s_h}\Big)\\ &- \omega v_h\Big(1-\frac{v_h^*}{v_h}\Big) - \omega r_h\Big(1-\frac{r_h^*}{r_h}\Big)\Big]\\ &- a_2\Big(1-\frac{v_h^*}{v_h}\Big)\Big[\varepsilon s_h^*\Big(1-\frac{s_h}{s_h^*}\Big) + \alpha v_h^* i_h^*\Big(1-\frac{v_h i_h}{v_h^* i_h^*}\Big)\Big]\\ &\frac{-a_3}{e_h^*}\Big(1-\frac{e_h^*}{e_h}\Big)\Big[a\beta_1 s_h^* e_h i_v^*\Big(1-\frac{s_h i_v e_h^*}{s_h^* i_v^* e_h}\Big) - \alpha e_h i_h e_h^*\Big(1-\frac{i_h^*}{i_h}\Big)\Big]\\ &\frac{-a_4}{i_h^*}\Big(1-\frac{i_h^*}{i_h}\Big)\Big[\delta_h e_h^* i_h\Big(1-\frac{e_h i_h^*}{e_h^* i_h}\Big) - \alpha i_h^* i_h^2\Big(1-\frac{i_h^*}{i_h}\Big)\Big]\\ &\frac{-a_5}{r_h^*}\Big(1-\frac{r_h^*}{r_h}\Big)\Big[r_h\gamma i_h^*\Big(1-\frac{i_h r_h^*}{i_h^* r_h}\Big) - \alpha r_h i_h r_h^*\Big(1-\frac{i_h^*}{i_h}\Big)\Big]\end{aligned} \tag{3.22}$$

With the same procedure, we can show the analysis of vector and primate populations respectively as

$$L_v(s_v, e_v, i_v) = a_6\ (s_v - s_v^*\ ln\ s_v) + a_7\ (e_v - e_v^*\ ln\ e_v) + a_8\ (i_v - i_v^*\ ln\ i_v) \quad (3.23)$$

into which time derivative of L_v yields;

$$\begin{aligned}
\frac{dL_v}{dt} &= a_6\left[1 - \frac{s_v^*}{s_v}\right]\frac{ds_v}{dt} + a_7\left[1 - \frac{e_v^*}{e_v}\right]\frac{de_v}{dt} + a_8\left[1 - \frac{i_v^*}{i_v}\right]\frac{di_v}{dt} \\
&= a_6\left(1 - \frac{s_v^*}{s_v}\right)\left[b_v - (a\beta_2 s_v i_h + a\beta_3 s_v i_m) - s_v b_v\right] \\
&\quad + a_7\left(1 - \frac{e_v^*}{e_v}\right)\left[a\beta_2 s_v i_h + a\beta_3 s_v i_m - e_v(\delta_v + b_v)\right] \\
&\quad + a_8\left(1 - \frac{i_v^*}{i_v}\right)\left[\delta_v e_v - i_v b_v\right]
\end{aligned}$$

At P^*, we have

$$\begin{aligned}
\frac{dL_v}{dt} &= a_6\left(1 - \frac{s_v^*}{s_v}\right)\left[a\beta_2 s_v^* i_h^* + a\beta_3 s_v^* i_m^* + s_v^* b_v - a\beta_2 s_v i_h - a\beta_3 s_v i_m - s_v b_v\right] \\
&\quad + a_7\left(1 - \frac{e_v^*}{e_v}\right)\left[a\beta_2 s_v i_h + a\beta_3 s_v i_m - e_v\left(\frac{a\beta_2 s_v^* i_h^* + a\beta_3 s_v^* i_m^*}{e_v^*}\right)\right] \\
&\quad + a_8\left(1 - \frac{i_v^*}{i_v}\right)\left[\delta_v e_v - i_v\left(\frac{\delta_v e_v^*}{i_v^*}\right)\right]
\end{aligned}$$

Further simplification yields

$$\begin{aligned}
\frac{dL_v}{dt} &= -a_6\left(1 - \frac{s_v^*}{s_v}\right)^2 b_v s_v - a_6\left(1 - \frac{s_v^*}{s_v}\right)\left[a\beta_2 s_v i_h\left(1 - \frac{s_v^* i_h^*}{s_v i_h}\right)\right. \\
&\quad \left. + a\beta_3 s_v i_m\left(1 - \frac{s_v^* i_m^*}{s_v i_m}\right)\right] - \frac{a_7}{e_v^*}\left(1 - \frac{e_v^*}{e_v}\right)\left[a\beta_2 s_v^* i_h^* e_v\right. \\
&\quad \left.\left(1 - \frac{s_v i_h e_v^*}{s_v^* i_h^* e_v}\right) + a\beta_3 e_v s_v^* i_m^*\left(1 - \frac{e_v^* s_v i_m}{e_v s_v^* i_m^*}\right)\right] \\
&\quad - \frac{a_8}{i_v^*}\left(1 - \frac{i_v^*}{i_v}\right)\left[i_v \delta_v e_v^*\left(1 - \frac{e_v i_v^*}{e_v^* i_v}\right)\right]
\end{aligned}$$

which can also be written as

$$\frac{dL_v}{dt} = -a_6\left(1 - \frac{s_v^*}{s_v}\right)^2 b_v s_v + F_v(s_v, e_v, i_v). \quad (3.24)$$

where

$$F_v = -a_6\left(1-\frac{s_v^*}{s_v}\right)\left[a\beta_2 s_v i_h\left(1-\frac{s_v^* i_h^*}{s_v i_h}\right)+a\beta_3 s_v i_m\left(1-\frac{s_v^* i_m^*}{s_v i_m}\right)\right] - \frac{a_7}{e_v^*}\left(1-\frac{e_v^*}{e_v}\right)\left[a\beta_2 s_v^* i_h^* e_v\left(1-\frac{s_v i_h e_v^*}{s_v^* i_h^* e_v}\right)+a\beta_3 e_v s_v^* i_m^* \left(1-\frac{e_v^* s_v i_m}{e_v s_v^* i_m^*}\right)\right]-\frac{a_8}{i_v^*}\left(1-\frac{i_v^*}{i_v}\right)\left[i_v\delta_v e_v^*\left(1-\frac{e_v i_v^*}{e_v^* i_v}\right)\right] \tag{3.25}$$

Similarly,

$$L_m(s_m,e_m,i_m) = a_9(s_m - s_m^*\ ln\ s_m)+a_{10}(e_m-e_m^*\ ln\ e_m)+a_{11}(i_m-i_m^*\ ln\ i_m) \tag{3.26}$$

which gives

$$\begin{aligned}\frac{dL_m}{dt} &= a_9\left[1-\frac{s_m^*}{s_m}\right]\frac{ds_m}{dt}+a_{10}\left[1-\frac{e_m^*}{e_m}\right]\frac{de_m}{dt}+a_{11}\left[1-\frac{i_m^*}{i_m}\right]\frac{di_m}{dt}\\ &= a_9\left(1-\frac{s_m^*}{s_m}\right)\left[b_m-a\beta_4 s_m i_v - s_m b_m\right]\\ &\quad + a_{10}\left(1-\frac{e_m^*}{e_m}\right)\left[a\beta_4 s_m i_v - e_m(\delta_m+b_m)\right]\\ &\quad + a_{11}\left(1-\frac{i_m^*}{i_m}\right)\left[\delta_m e_m - i_m b_m\right]\end{aligned}$$

At P^*, we also have

$$\begin{aligned}\frac{dL_m}{dt} &= a_9\left(1-\frac{s_m^*}{s_m}\right)\left[a\beta_4 s_m^* i_v^* + s_m^* b_m - a\beta_4 s_m i_v - s_m b_m\right]\\ &\quad + a_{10}\left(1-\frac{e_m^*}{e_m}\right)\left[a\beta_4 s_m i_v - e_m\left(\frac{a\beta_4 s_m^* i_v^*}{e_m^*}\right)\right]\\ &\quad + a_{11}\left(1-\frac{i_m^*}{i_m}\right)\left[\delta_m e_m - i_m\left(\frac{\delta_m e_m^*}{i_m^*}\right)\right]\end{aligned}$$

Again, further simplification yields

$$\begin{aligned}\frac{dL_m}{dt} &= -a_9\left(1-\frac{s_m^*}{s_m}\right)^2 b_m s_m - a_9\left(1-\frac{s_m^*}{s_m}\right)\left[a\beta_4 s_m i_v\left(1-\frac{s_m^* i_v^*}{s_m i_v}\right)\right]\\ &\quad - \frac{a_{10}}{e_m^*}\left(1-\frac{e_m^*}{e_m}\right)\left[a\beta_4 s_m^* i_v^* e_m\left(1-\frac{s_m i_v e_m^*}{s_m^* i_v^* e_m}\right)\right]\\ &\quad - \frac{a_{11}}{i_m^*}\left(1-\frac{i_m^*}{i_m}\right)\left[i_m\delta_m e_m^*\left(1-\frac{e_m i_m^*}{e_m^* i_m}\right)\right]\end{aligned}$$

which can also be written as

$$\frac{dL_m}{dt} = -a_9\left(1-\frac{s_m^*}{s_m}\right)^2 b_m s_m + F_m(s_m, e_m, i_m). \tag{3.27}$$

where

$$\begin{aligned} F_m = &-a_9\left(1-\frac{s_m^*}{s_m}\right)\left[a\beta_4 s_m i_v\left(1-\frac{s_m^* i_v^*}{s_m i_v}\right)\right] \\ &-\frac{a_{10}}{e_m^*}\left(1-\frac{e_m^*}{e_m}\right)\left[a\beta_4 s_m^* i_v^* e_m\left(1-\frac{s_m i_v e_m^*}{s_m^* i_v^* e_m}\right)\right] \\ &-\frac{a_{11}}{i_m^*}\left(1-\frac{i_m^*}{i_m}\right)\left[i_m\delta_m e_m^*\left(1-\frac{e_m i_m^*}{e_m^* i_m}\right)\right] \end{aligned} \tag{3.28}$$

Thus, considering equations (3.21), (3.24) and (3.27) we can have

$$\begin{aligned} \frac{dL}{dt} &= \frac{dL_h}{dt} + \frac{dL_v}{dt} + \frac{dL_m}{dt} \\ &= -a_1\Big(\varepsilon + b_h + \sigma\Big)s_h\left(1-\frac{s_h^*}{s_h}\right)^2 - a_2\Big(\omega + b_h + \sigma\Big)v_h\left(1-\frac{v_h^*}{v_h}\right)^2 \\ &\quad + F_h\big(s_h, v_h, e_h, i_h, r_h\big) \\ &\quad - a_6\left(1-\frac{s_v^*}{s_v}\right)^2 b_v s_v + F_v(s_v, e_v, i_v) \\ &\quad - a_9\left(1-\frac{s_m^*}{s_m}\right)^2 b_m s_m + F_m(s_m, e_m, i_m) \end{aligned} \tag{3.29}$$

which can be written as

$$\begin{aligned} \frac{dL}{dt} = &-a_1\Big(\varepsilon + b_h + \sigma\Big)s_h\left(1-\frac{s_h^*}{s_h}\right)^2 - a_2\Big(\omega + b_h + \sigma\Big)v_h\left(1-\frac{v_h^*}{v_h}\right)^2 \\ &- a_6\left(1-\frac{s_v^*}{s_v}\right)^2 b_v s_v - a_9\left(1-\frac{s_m^*}{s_m}\right)^2 b_m s_m \\ &+ F(s_h, v_h, e_h, i_h, r_h, s_v, e_v, i_v, s_m, e_m, i_m), \end{aligned} \tag{3.30}$$

where

$$
\begin{aligned}
F = &-a_1\left(1-\frac{s_h^*}{s_h}\right)\left[a\beta_1 s_h i_v\left(1-\frac{i_v^* s_h^*}{i_v s_h}\right)-\alpha s_h i_h\left(1-\frac{i_h^* s_h^*}{i_h s_h}\right)\right.\\
&\left.-\omega v_h\left(1-\frac{v_h^*}{v_h}\right)-\omega r_h\left(1-\frac{r_h^*}{r_h}\right)\right]\\
&-a_2\left(1-\frac{v_h^*}{v_h}\right)\left[\varepsilon s_h^*\left(1-\frac{s_h}{s_h^*}\right)+\alpha v_h^* i_h^*\left(1-\frac{v_h i_h}{v_h^* i_h^*}\right)\right]\\
&\frac{-a_3}{e_h^*}\left(1-\frac{e_h^*}{e_h}\right)\left[a\beta_1 s_h^* e_h i_v^*\left(1-\frac{s_h i_v e_h^*}{s_h^* i_v^* e_h}\right)-\alpha e_h i_h e_h^*\left(1-\frac{i_h^*}{i_h}\right)\right]\\
&\frac{-a_4}{i_h^*}\left(1-\frac{i_h^*}{i_h}\right)\left[\delta_h e_h^* i_h\left(1-\frac{e_h i_h^*}{e_h^* i_h}\right)-\alpha i_h^* i_h^2\left(1-\frac{i_h^*}{i_h}\right)\right]\\
&\frac{-a_5}{r_h^*}\left(1-\frac{r_h^*}{r_h}\right)\left[r_h\gamma i_h^*\left(1-\frac{i_h r_h^*}{i_h^* r_h}\right)-\alpha r_h i_h r_h^*\left(1-\frac{i_h^*}{i_h}\right)\right]\\
&-a_6\left(1-\frac{s_v^*}{s_v}\right)\left[a\beta_2 s_v i_h\left(1-\frac{s_v^* i_h^*}{s_v i_h}\right)+a\beta_3 s_v i_m\left(1-\frac{s_v^* i_m^*}{s_v i_m}\right)\right]\\
&-\frac{a_7}{e_v^*}\left(1-\frac{e_v^*}{e_v}\right)\left[a\beta_2 s_v^* i_h^* e_v\left(1-\frac{s_v i_h e_v^*}{s_v^* i_h^* e_v}\right)+a\beta_3 e_v s_v^* i_m^*\right.\\
&\left.\left(1-\frac{e_v^* s_v i_m}{e_v s_v^* i_m^*}\right)\right]-\frac{a_8}{i_v^*}\left(1-\frac{i_v^*}{i_v}\right)\left[i_v\delta_v e_v^*\left(1-\frac{e_v i_v^*}{e_v^* i_v}\right)\right]\\
&-a_9\left(1-\frac{s_m^*}{s_m}\right)\left[a\beta_4 s_m i_v\left(1-\frac{s_m^* i_v^*}{s_m i_v}\right)\right]\\
&-\frac{a_{10}}{e_m^*}\left(1-\frac{e_m^*}{e_m}\right)\left[a\beta_4 s_m^* i_v^* e_m\left(1-\frac{s_m i_v e_m^*}{s_m^* i_v^* e_m}\right)\right]\\
&-\frac{a_{11}}{i_m^*}\left(1-\frac{i_m^*}{i_m}\right)\left[i_m\delta_m e_m^*\left(1-\frac{e_m i_m^*}{e_m^* i_m}\right)\right]
\end{aligned}
\tag{3.31}
$$

As it is seen in equations (3.30) and (3.31), F is non-positive by following the approach of Mukandavire *et al.* and McCluskey [22, 23].

Thus, $F \leq 0$ for $s_h, v_h, e_h, i_h, r_h, s_v, e_v, i_v, s_m, e_m, i_m > 0$.

Hence $\dfrac{dL}{dt} \leq 0$ for all $s_h, v_h, e_h, i_h, r_h, s_v, e_v, i_v, s_m, e_m, i_m > 0$ and is zero when

$s_h = s_h^*,\ v_h = v_h^*,\ e_h = e_h^*,\ i_h = i_h^*,\ r_h = r_h^*,\ s_v = s_v^*,\ e_v = e_v^*,\ i_v = i_v^*$
$s_m = s_m^*,\ e_m = e_m^*,\ i_m = i_m^*.$

Therefore, the largest compact invariant set in Φ such that $\dfrac{dL}{dt} = 0$ is the singleton P^* which is the endemic equilibrium point.

LaSalles invariant principle [17] guarantees that P^* is globally asymptotically

stable (GAS) in $\overset{\circ}{\Phi}$, (the interior of Φ). Thus, we have established the following result;

Theorem 3.4. *If $R_0 > 1$, then, the model system (2.7) has a unique EE point which is globally asymptotically stable (GAS) in $\overset{\circ}{\Phi}$.*

4. Discussions and Conclusion

In this paper, we have formulated and analysed a mathematical model with standard incidence that tracks the dynamics of human-vector-primate in YF infection. The model is of type SVEIRS and incorporates immigration of human individuals by which proportions of the immigrants are vaccinated and others are susceptible. All new borne are considered susceptible. The model was then reformulated in terms of proportions of the classes of the respective populations. We also established a region where the model is epidemiologically feasible and mathematically well-posed. The model was analysed for the existence and stabilities of disease-free and endemic equilibrium points.

We investigate the stability of DFE point E_0 using the threshold parameter, R_0, which had the form $R_0^2 = R_{hv} + R_{vm}$, where R_{hv} represents the reproduction number of human host-vector compartment and R_{vm} represents the reproduction number of primate-vector compartment. Local stability of the E_0 was established using trace-determinant approach (Jacobian technique) of the model system (2.7), while Metzler matrix approach was used to carry out global stability analysis of E_0. It was found that the disease-free equilibrium point is locally asymptotically stable if $R_{vm} < 1$ and unstable if $R_{vm} > 1$. Also, it was established that the disease-free equilibrium point is globally stable if $R_{hv} < 1$ and unstable otherwise. Since the disease threshold parameter had the form $R_0 = \sqrt{R_{hv} + R_{vm}}$, and from the local and global stabilities of the disease-free point, we can generally conclude that the disease-free equilibrium point is stable locally and globally if and only if $R_0 < 1$ so that the disease always dies out. For $R_0 > 1$, the disease-free equilibrium point is unstable while the endemic equilibrium emerges as a unique equilibrium point, thus re-invasion is always possible and the disease never dies out.

We further analyse the stability of EE point using a suitable Lyapunov function. Results show that a unique endemic equilibrium point exists and is globally stable when $R_0 > 1$. The results of this study indicate that the Lyapunov functions of the form $L(x_1, x_2, ...x_n) = \sum a_i \ (x_i - x_i^* \ ln \ x_i)$ can be useful especially for compartmental human-vector and human-vector-primate models with many number of compartments.

This analysis enables us to gain valuable insights and it introduces an important step in theoretical analysis of the disease. The vector (mosquitoes) being at the middle part of human host and primates and the transmitters of the disease to both hosts need to be controlled in order to make the human host free from

the infection. Thus, interventional strategies are suggested important to reduce the span of the lives of parasites and vectors.

Moreover, numerous additions in the model will be required in order to provide further insights in assessing the impact, dynamics and transmissions of YFV like temperature variations and vertical transmission of the vector. Vaccination could remain as the single most important measure for preventing yellow fever. Again, control of the yellow fever mosquito (*Aedes aegypti*) is of a major importance, especially because the same mosquito can also transmit dengue fever and chikungunya disease.

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