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# Mathematical Analysis of an Extended SIRS-SI Malaria Model Incorporating Seasonal Vector Recruitment with Standard Incidence

Alphablondy Achieng[1*], Charles Wachira[2] and Benjamin Kikwai[3]

[1*] alphablondyachieng20@gmail.com

[1,2,3] Department of Mathematics and Statistics, Machakos University

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**Abstract**

In this study an extended deterministic SIRS-SI malaria model incorporating seasonality on vector recruitment with standard incidence and vaccination is developed and analyzed. Positivity and boundedness of model solutions are shown. The vaccine reproductive number $R_v$ is obtained using next generation matrix approach. The Routh Hurwitz criterion is applied to analyze the local stability of Malaria-Free Equilibrium ($MFE$) points which is found to be locally asymptotically stable whenever $R_v < 1$. Gershgorin discs is applied to analyze the local stability of Malaria Persistence Equilibrium ($MPE$) points which is locally asymptotically stable when $R_v > 1$, implying that the disease would persist in the population. The Castillo-Chavez technique is applied to analyze the global stability of $MFE$. The analysis shows that the $MFE$ point is globally asymptotically stable provided that $R_v < 1$. The numerical simulation show that an increase in vaccination rate ($\alpha$) while using a vaccine of higher efficacy ($\epsilon$) leads to reduction of malaria infection. It is shown that during rainy seasons, there is high mosquito recruitment hence there are expectation of malaria infectious periods. Therefore the policy makers should educate on the use of mosquito treated bed nets, clearing of bushes around homestead, and spraying the insecticides during rainy seasons.

**MSC2010 Subject Classification:**
**Keywords:** Seasonality, Malaria Free Infection, Basic Reproduction Number, Vaccine Reproduction Number.

## 1 Introduction

Malaria is a severe and acute fever causing disease caused by parasites known as Plasmodium, the infection is passed to human through bite from an infected female Anopheles Mosquitoes [9]. Malaria disease is still highly prevalent especially in the developing countries, Sub- Saharan Africa, the Caribbean and pacific islands are the most critical endemic regions. There are five species of the Plasmodium ($P$) which infects humans, Plasmodium falciparum, P. vivax, P. ovale, P. malariae, and the zoonotic P. knowelesi. The most virulent pathogen is P. falciparum followed by P. vivax that causes recurrent infections via dormant liver-stage parasites, which leads to hemolysis [12]. The clinical manifestations are fever, headache, nausea, fatigue and in severe cases, anemia, jaundice, cerebral malaria, multi-organ complications and death. Those at the greatest risk of contracting

severe diseases and dying in case of infection are children below the age of five years, expectant mothers, and non-immune humans [20].

Malaria medication is species specific. The most perilous one, P. falciparum is primarily treated using combine therapy with artemisinin ($ACTs$) [17], but infections from other species, the non-falciparum are susceptible to the action of chloroquine or other medications. The prevention strategies measures are; use of insecticides on the nets, indoor spraying, environmental management and vaccination. The first malaria vaccine is RTS,S/AS01 (Mosquirix) which has pilot projects in African countries like Kenya, Ghana and Malawi where the vaccine has partial protection, especially in children under five years old, in existence, conferring the vaccination with partial protection against malaria exposure [22]. Early studies suggest that combining vaccination with vector control and treatment interventions can significantly reduce malaria morbidity and mortality in endemic populations [7].

A deterministic malaria model which incorporates the concept of vaccination and infected immigration into the human population is presented in Duve et al. (2024) [7] in studying the control and prevention of malaria as a mosquito borne disease. Their findings indicated that vaccinated persons considerably limit the number of ineffective classes provided that the inflow of infected immigrants is ongoing and the local transmission of malaria can be reintroduced despite a corresponding decrease. Their study also indicated that the disease burden can be greatly suppressed through the use of the optimal vaccination strategies.

Gatore Sinigirira, Ogana, and Chirove (2025) [10], developed a deterministic model that incorporated environmental variables like temperature, rainfall and vegetation index. In their study, they underscored the role of seasonal and climate-related changes in the population of mosquitoes in the spread of malaria. Their model, by relating the abundance of vectors to environmental variables showed that climatic changes are enough to change the timing and severity of malaria outbreaks and thus, environmental forcing is important in deterministic models even though they did not include optimal control.

Ayalew, Molla, and Woldegbreal (2024) [3], developed a nonlinear deterministic model which paid attention to the malaria dynamics stability analysis. They analyzed disease-free and endemic equilibrium to learn when malaria will survive or disappear. Their findings are used as a theoretical reference point, and they indicate how changes in parameters such as transmission and recovery rates affect the stability and long-term behavior of malaria in a population. A deterministic models with data-driven transmission functions that were determined by temperature and altitude were proposed by [19]. Their methodology enabled spatial heterogeneity of malaria risk indicating that differences in environmental conditions across space can result in large disparities in intensity of transmission, even though they did not include vaccination.

Many populations remain vulnerable to malaria at any given time notwithstanding the number of interventions put in place, the present controls and prevention programs have failed to eradicate the infection [23]. Therefore, there is need to study malaria dynamics under vaccination and seasonal vector recruitment with standard incidence. In this study, an extended $SIRS - SI$ malaria model incorporating seasonality on mosquito recruitment with standard incidence and vaccination is developed

and analyzed.

## 2 Model Formulation and Analysis

In this section, the model is formulated. Positivity and boundedness of model solutions are shown, The equilibrium points of the models are shown. The vaccine reproduction number is computed. The stability analysis for Malaria Persistence Equilibrium ($MPE$) and Malaria Free Equilibrium ($MFE$) are shown.

### 2.1 Model Description

Over the years, mathematical models have been used in the context of malaria to quantify the interactions between processes involving the host, parasite and the vector such as the rates of transmission, waning immunity and the dynamics of the vectors to generate patterns of transmission, and to estimate the effects of interventions such as the use of insecticide-treated nets, therapeutics and vaccination strategies [18]. In this study a deterministic non-linear differential-equations approach is used to develop a malaria $SVIRS - SI$ type model that takes into consideration vaccination, and seasonal vector recruitment. The total human population is taken to be $N_h$ which is subdivided into four compartments namely, $S_h$ the total susceptible humans, $V_h$ vaccinated humans, $I_h$ the number of infected humans, and $R_h$ the number of recovered humans. The susceptible human $S_h$ are recruited at a constant rate $\Lambda_h$. The susceptible human acquires infection through the bite of an infected mosquito at a rate $\beta_1$, the vaccination rate of susceptible human is taken to be $\alpha$ whereas the vaccine efficacy is assumed to be $\epsilon$ and the human immunity waning rate is taken as $\omega$ as vaccine does not guarantee permanent immunity against malaria. The human recovery rate is given as $\gamma$, where the vaccinated individual gets infected at a rate $\beta_2$ where the transmission coefficient of the vaccinees $\beta_2 < \beta_1$ since the vaccinees are assumed to have acquired a vaccine induced immunity.

The total mosquito (Vector) population is taken to be $N_v$ which is divided into two compartments namely, $S_v$ the susceptible mosquitoes, $I_v$ the infected mosquitoes. The natural mortality rate for human is denoted by $\mu_h$ while that for mosquitoes is taken to be $\mu_v$, the disease induced human death rate is given as $\delta$ as the susceptible mosquito recruitment rate is assumed to be $\Lambda_v$(t) where seasonality is incorporated in the model through the mosquito recruitment rate $\Lambda_v$(t)

$$\Lambda_v(t) = \Lambda_{v0}\left(1 + a\cos\left(\frac{2\pi}{365}(t-\tau)\right)\right) \tag{1}$$

The amplitude of seasonal variation $a$ is given by $(0 \leq a \leq 1)$, where $\Lambda_{v0}$ is the mosquito recruitment baseline, and $\tau$ is the phase shift that varies, $t$ is the peak timing relative to the start of the year period 365 days (annual cycle). The mosquito biting rate is given as $\phi$ as the transmission rate from infected human to susceptible mosquito is assumed to be $\kappa$. Forces of infection are $\phi\beta_1\frac{I_v}{N_v}$, $\phi\beta_2\frac{I_v}{N_v}$ and $\phi\kappa\frac{I_h}{N_v}$. The standard incidence rates are $\phi\beta_1\frac{S_hI_v}{N_v}$, $\phi\beta_2\frac{V_hI_v}{N_v}$ and $\phi\kappa\frac{S_vI_h}{N_h}$ where $\frac{I_h}{N_h}$ and $\frac{I_v}{N_v}$ are fractions of the infected human and mosquitoes respectively as in [11]. For analytical analysis, the equilibrium analysis is performed using average mosquito recruitment rate $\Lambda_{v0}$ while seasonal vector recruitment is used in numerical simulation. The associated parameters of the model are summarized in table 1.

Table 1: **A descriptive summary of the model parameters, their unit values and sources.**

| Symbol | Description | Baseline Value/Units | Sources |
|---|---|---|---|
| $\Lambda_h$ | Recruitment rate of humans | 0.01547 $day^{-1}$ | [6] |
| $\alpha$ | Vaccination rate of susceptible human | (0, 0.0085,1) varies | [21] |
| $\epsilon$ | Vaccine efficacy | (0, 0.5, 1) (varies) | [2] |
| $\beta_1$ | Transmission rate (mosquito $\rightarrow$ human) | 0.048 $day^{-1}$ | [16] |
| $\beta_2$ | Transmission rate (mosquito $\rightarrow$ vaccinated) | 0.004 $day^{-1}$ | [7] |
| $\phi$ | Mosquito biting rate | 0.33 $day^{-1}$ | [16] |
| $\mu_h$ | Human natural death rate | $4.74\times10^{-5}$ $day^{-1}$ | [16] |
| $\mu_v$ | mosquito natural death rate | 0.1 $day^{-1}$ | [16] |
| $\delta$ | Disease-induced human death rate | 0.001 $day^{-1}$ | [16] |
| $\gamma$ | Recovery rate of infected humans | 0.006 $day^{-1}$ | [7] |
| $\omega$ | Loss of immunity rate | 0.005 $day^{-1}$ | [14] |
| $\Lambda_v(t)$ | Seasonal mosquito recruitment rate | $0.0714(\Lambda_{v0})$varies | [6] |
| $\kappa$ | Transmission rate (Inf. human $\rightarrow$ mosquito) | 0.48 $day^{-1}$ | [16] |

From the description of table 1, the model is represented by the following set of non-linear ordinary differential equations (Odes).

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \beta_1\phi\frac{S_hI_v}{N_v} - (\alpha+\mu_h)S_h + \omega R_h,\\
\frac{dV_h}{dt} &= \alpha S_h - (1-\epsilon)\beta_2\phi\frac{V_hI_v}{N_v} - \mu_h V_h,\\
\frac{dI_h}{dt} &= \beta_1\phi\frac{S_hI_v}{N_v} + (1-\epsilon)\beta_2\phi\frac{V_hI_v}{N_v} - (\mu_h+\gamma+\delta)I_h,\\
\frac{dR_h}{dt} &= \gamma I_h - \mu_h R_h - \omega R_h.\\
\frac{dS_v}{dt} &= \Lambda_v(t) - \phi\kappa\frac{S_vI_h}{N_h} - \mu_v S_v,\\
\frac{dI_v}{dt} &= \phi\kappa\frac{S_vI_h}{N_h} - \mu_v I_v,
\end{aligned} \tag{2}$$

The compartmental diagram of model (2) is given as:

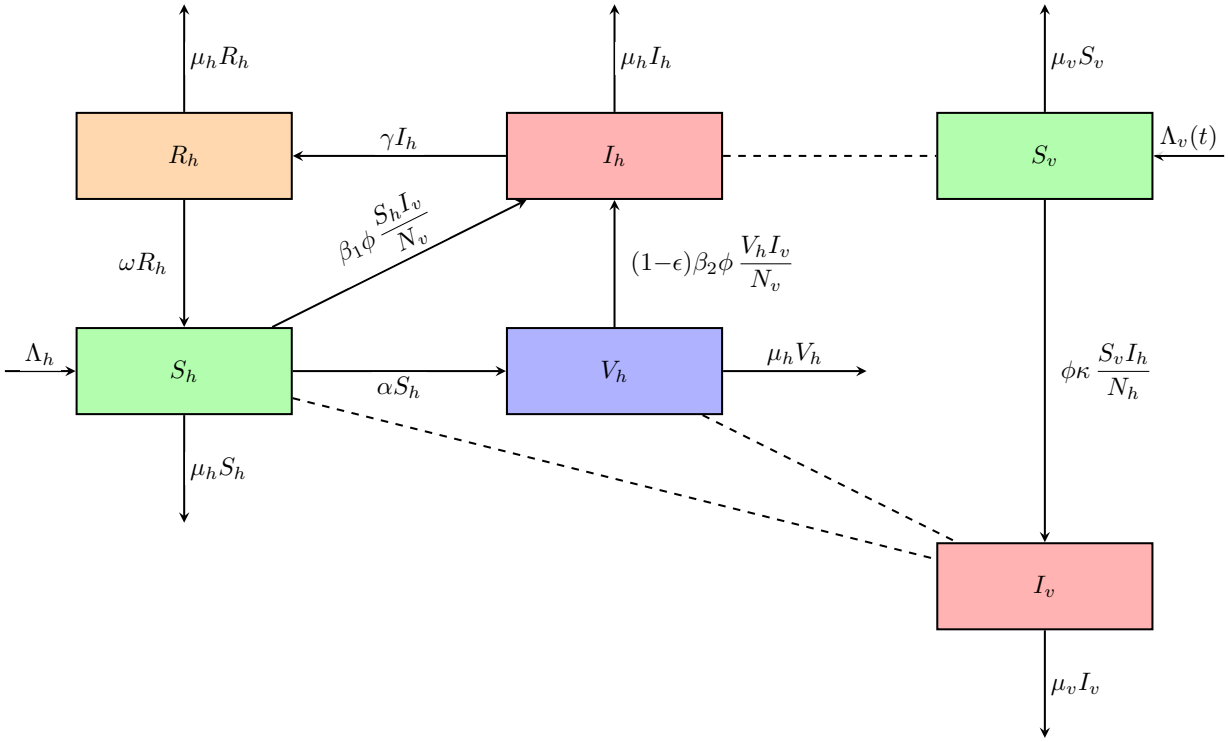



Figure 1: A schematic flow diagram of malaria disease dynamics

## 2.2 The Invariant Region

Since the model describes human and vector population, the solutions remains positive and bounded. The state variables $S_h(t)$, $V_h(t)$, $I_h(t)$, $R_h(t)$, $S_v(t)$ and $I_v(t)$ are always positive for every time $t \geq 0$.

**Proposition 1.** *All solution sets* $\{S_h(t), V_h(t), I_h(t), R_h(t), S_v(t), I_v(t)\}$ *of the model (2) with non–negative initial conditions are positive for all* $t > 0$.

*Proof.* Considering the first equation in model (2) that is

$$\frac{dS_h}{dt} = \Lambda_h - \frac{\beta_1 \phi S_h I_v}{N_v} - (\alpha + \mu_h)S_h + \omega R_h, \tag{3}$$

where

$$\frac{dS_h}{dt} \geq -\left[\frac{\beta_1 \phi I_v}{N_v} + (\alpha + \mu_h)\right] S_h, \tag{4}$$

Integrating by variable separation we get

$$\int \frac{dS_h}{S_h} \geq -\int \left[\frac{\beta_1 \phi I_v}{N_v} + (\alpha + \mu_h)\right] dt, \tag{5}$$

Thus

$$S_h(t) \geq S_h(0) \exp\left(-\int_0^t [D(\zeta) + (\alpha + \mu_h)] d\zeta\right), \tag{6}$$

where

$$D = \frac{\beta_1 \phi I_v}{N_v}, \tag{7}$$

This implies that

$$S_h(t) \geq 0 \quad \forall t \geq 0, \tag{8}$$

Taking the second last equation that is susceptible mosquito population equation $S_v$, the positivity in relation to the mosquito population that is

$$\frac{dS_v}{dt} = \Lambda_v(t) - \phi\kappa \frac{S_v I_h}{N_h} - \mu_v S_v, \tag{9}$$

where

$$\frac{dS_v}{dt} \geq -\left(\phi\kappa \frac{I_h}{N_h} + \mu_v\right) S_v, \tag{10}$$

Integrating by variable separation we get

$$\int \frac{dS_v}{S_v} \geq -\int \left(\phi\kappa \frac{I_h}{N_h} + \mu_v\right) dt, \tag{11}$$

thus

$$S_v(t) \geq S_v(0) \exp\left[-\int_0^t \left(\phi\kappa \frac{I_h(\zeta)}{N_h(\zeta)} + \mu_v\right) d\zeta\right], \tag{12}$$

This implies that

$$S_v(t) \geq 0 \quad \forall t \geq 0, \tag{13}$$

In a similar way, all other variables, $V_h, I_h, R_h, I_v$ can be shown to be positive in the region $\Omega$. □

**Proposition 2.** *For all time $t \geq 0$, the solutions of model (2) are bounded in the region $\Omega$.*

Model (2) is analyzed on a suitable feasible region.

$$\Omega = \left\{ (S_h, V_h, I_h, R_h, S_v, I_v)(t) \in \mathbb{R}_+^6 \ : \ \begin{array}{l} S_h + V_h + I_h + R_h \leq \dfrac{\Lambda_h}{\mu_h}, \\ S_v(t) + I_v(t) \leq \dfrac{\Lambda_v(t)}{\mu_v} \end{array} \right\} \tag{14}$$

where $(S_h + V_h + I_h + R_h) = N_h$ gives the total human population and $(S_v + I_v) = N_V$ gives the total mosquito population. Using Proposition (2) below, the model solutions are shown to be bounded for all $t \geq 0$ in the region $\Omega$, where $\Omega$=$\Omega_h \times \Omega_v$.

*Proof.* All the solutions of the model (2) are positively invariant in $\Omega$, i.e., all solutions start in $\Omega$ and

remain in the region $\Omega$ for all $t \geq 0$. The rate of change of the human population $N_h(t)$ is given by

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dV_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt} \tag{15}$$

which implies that

$$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h \tag{16}$$

By variation of constant formula, it follows that

$$\limsup_{t\to\infty} N_h(t) \leq \frac{\Lambda_h}{\mu_h}. \tag{17}$$

Thus, $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$. This implies that the solution set $\{S_h(t), V_h(t), I_h(t), R_h(t)\}$ is bounded in the feasible region $\Omega$, that is,

$$\Omega_h = \left\{ (S_h(t), V_h(t), I_h(t), R_h(t)) \;\middle|\; \begin{array}{c} S_h(t) + V_h(t) + I_h(t) + R_h(t) \leq \dfrac{\Lambda_h}{\mu_h}, \\ (S_h, V_h, I_h, R_h)(0) \geq 0 \end{array} \right\}. \tag{18}$$

A similar procedure for the mosquito population $N_v$ shows $N_v(t) \leq \frac{\Lambda_v(t)}{\mu_v}$ since the seasonal recruitment $\Lambda_v(t)$ is bounded by its periodic maximum, the solution set is contained within region $\Omega$ for all $t \geq 0$. Hence all solutions of the model (2) are bounded in the region $\Omega$. □

Clearly from propositions (1) and (2), all solutions of the model (2) are positive and bounded in the invariant region $\Omega$. Thus, model(2) is mathematically and epidemiologically well posed in a biological feasible region $\Omega$.

### 2.3 Malaria Free Equilibrium Points $(MFE)$

The steady-state solution of the malaria population is the Malaria-Free Equilibrium $(MFE)$, it is a state in which there is no malaria disease in the population, it is achieved when all the malaria parasites are eliminated.

**Proposition 3.** *There exist a $MFE$ of model (2) given by*

$$E^0 = (S_h^0, V_h^0, I_h^0, R_h^0, S_v^0, I_v^0) = \left( \frac{\Lambda_h}{\alpha + \mu_h},\ \frac{\alpha\Lambda_h}{\mu_h(\alpha + \mu_h)},\ 0,\ 0,\ \frac{\Lambda_v}{\mu_v},\ 0 \right). \tag{19}$$

*Proof.* There is no malaria infection in the population at malaria free equilibrium $(MFE)$. Therefore

having $I_h = R_h = I_v = 0$, the model (2) is given as follows:

$$\begin{aligned}
\Lambda_h - \beta_1\phi\frac{S_hI_v}{N_v} - (\alpha+\mu_h)S_h + \omega R_h &= 0\\
\alpha S_h - (1-\epsilon)\beta_2\phi\frac{V_hI_v}{N_v} - \mu_h V_h &= 0\\
\beta_1\phi\frac{S_hI_v}{N_v} + (1-\epsilon)\beta_2\phi\frac{V_hI_v}{N_v} - (\mu_h+\gamma+\delta)I_h &= 0\\
\gamma I_h - \mu_h R_h - \omega R_h &= 0\\
\Lambda_v(t) - \phi\kappa\frac{S_vI_h}{N_h} - \mu_v S_v &= 0\\
\phi\kappa\frac{S_vI_h}{N_h} - \mu_v I_v &= 0
\end{aligned} \tag{20}$$

Solving model (2), $S_h^0 = \frac{\Lambda_h}{\alpha+\mu_h}$, $V_h^0 = \frac{\alpha\Lambda_h}{\mu_h(\alpha+\mu_h)}$, $S_v^0 = \frac{\Lambda_v}{\mu_v}$. Therefore model(2) is having a malaria free equilibrium given by

$$E^0 = \left(\frac{\Lambda_h}{\alpha+\mu_h},\ \frac{\alpha\Lambda_h}{\mu_h(\alpha+\mu_h)},\ 0,\ 0,\ \frac{\Lambda_v}{\mu_v},\ 0\right). \tag{21}$$

□

### 2.4 The Reproductive Number

The basic reproduction number denoted by $R_0$, is defined as the average number of secondary infections produced by a single individual introduced into a fully susceptible population during an individual entire infectious period [1, 13]. The vaccine reproduction number denoted by $R_v$ of model (2) is computed using the next generation matrix approach [15]. The basic reproduction number is given by the spectral radius of the matrix $FV^{-1}$, that is, $\mathcal{R}_0 = \rho(FV^{-1})$, where $F$ and $V$ are the next-generation matrices [15]. The operator $FV^{-1}$, known as the next-generation matrix, is constructed from the matrices of partial derivatives of $F_i$ and $V_i$, where: $F_i$ represents the rate of appearance of new infections in the $i$-th compartment, $V_i = V_i^- - V_i^+$ represents the rate of transfer (transition rate) into and out of the disease compartment $i$, with respect to the infected compartments (for example, $I_h$ and $I_v$) evaluated at $MFE$. The infected compartments from model (2) are given as

$$\frac{dI_h}{dt} = \beta_1\phi\frac{S_hI_v}{N_v} + (1-\epsilon)\beta_2\phi\frac{V_hI_v}{N_v} - (\mu_h+\gamma+\delta)I_h, \tag{22}$$

$$\frac{dI_v}{dt} = \phi\kappa\frac{S_vI_h}{N_h} - \mu_v I_v. \tag{23}$$

The matrices $F$ and $V$ are given by

$$F = \begin{bmatrix} \frac{\phi I_v}{N_v}\left[\beta_1 S_h + (1-\epsilon)\beta_2 V_h\right] \\ \frac{\phi\kappa S_v I_h}{N_h} \end{bmatrix}, \quad V = \begin{bmatrix} (\mu_h+\gamma+\delta)I_h \\ \mu_v I_v \end{bmatrix}. \tag{24}$$

$$F = \left(\frac{\partial F_i(E^0)}{\partial x_j}\right), \qquad V = \left(\frac{\partial V_i(E^0)}{\partial x_j}\right). \tag{25}$$

where the transition matrices $F$ and $V$ evaluated at $E^0$ are given by

$$F = \begin{pmatrix} 0 & \frac{\phi S_v^0}{(N_v^0)^2}\left[\beta_1 S_h^0 + (1-\epsilon)\beta_2 V_h^0\right] \\ \frac{\phi\kappa S_v^0}{(N_h^0)^2}\left[S_h^0 + V_h^0\right] & 0 \end{pmatrix} \tag{26}$$

When the variables $N_v^0$,$S_v^0$,$S_h^0$,$V_h^0$,$N_h^0$, where $N_h^0$, is the total human population at $MFE$ given as $N_h^0 = S_h^0 + V_h^0 = \frac{\Lambda_h}{\mu_h}$, are inserted equation (26), then it becomes

$$F = \begin{pmatrix} 0 & \frac{\phi\mu_v\Lambda_h}{\Lambda_v}\left[\frac{\beta_1\mu_h + (1-\epsilon)\alpha\beta_2}{\mu_h(\alpha+\mu_h)}\right] \\ \frac{\phi\kappa\Lambda_v\mu_h}{\mu_v\Lambda_h} & 0 \end{pmatrix} \tag{27}$$

$$\mathbf{V} = \begin{pmatrix} \mu_h + \gamma + \delta & 0 \\ 0 & \mu_v \end{pmatrix} \tag{28}$$

Matrix $V$ is invertible and

$$\mathbf{V}^{-1} = \begin{pmatrix} \frac{1}{\mu_h + \gamma + \delta} & 0 \\ 0 & \frac{1}{\mu_v} \end{pmatrix} \tag{29}$$

Thus the matrix $FV^{-1}$ is

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\phi\Lambda_h}{\Lambda_v}\left[\frac{\beta_1\mu_h + (1-\epsilon)\alpha\beta_2}{\mu_h(\alpha+\mu_h)}\right] \\ \frac{\phi\kappa\Lambda_v\mu_h}{\mu_v\Lambda_h(\mu_h+\gamma+\delta)} & 0 \end{pmatrix} \tag{30}$$

Upon simplification it yield $R_v = \rho(FV^{-1}) = \sqrt{ab}$

$$\sqrt{\frac{\phi^2\kappa\left(\beta_1\mu_h + (1-\epsilon)\beta_2\alpha\right)}{\mu_v(\alpha+\mu_h)\ (\mu_h+\gamma+\delta)}} \tag{31}$$

Since model (2) involves vaccination as an intervention, its associated reproduction number is called the vaccine reproduction number denoted by $R_v$. This is the threshold quantity that can predict the spread of the disease in a given population in the presence of vaccination. The vaccine reproduction number computed by using the next generation matrix approach above is given by,

$$R_v = \sqrt{\frac{\phi^2\kappa\left(\beta_1\mu_h + (1-\epsilon)\beta_2\alpha\right)}{\mu_v(\alpha+\mu_h)\ (\mu_h+\gamma+\delta)}}. \tag{32}$$

$$R_v = R_0 \times \sqrt{\frac{\beta_1\mu_h + (1-\epsilon)\beta_2\alpha}{\beta_1(\alpha+\mu_h)}}. \tag{33}$$

If the vaccination rate of susceptible humans $\alpha$=0, vaccine efficacy $\epsilon$=0, then $R_v$=$R_0$, which is the basic reproduction number given by $R_0 = \sqrt{\frac{\phi^2\kappa\beta_1}{\mu_v(\mu_h+\gamma+\delta)}}$.

The vaccine reproduction number, $R_v$, is the measure of the severity of an epidemic in the presence of vaccination and one of the most important parameters since it determines whether or not malaria will invade a population. Epidemiologically, if $R_v < 1$, then by definition, the infection does not spread in the population. On the other hand, if $R_v > 1$, then the infection spreads in the population and may result into an epidemic.

### 2.5 Local Stability of Malaria Free Equilibrium

The malaria-free equilibrium ($MFE$) is the state at which there is no infection in a certain population(absence of infection) [6].

**Proposition 4.** *The malaria-free equilibrium $E^0$ is locally asymptotically stable whenever $R_v < 1$ and unstable whenever $R_v > 1$*

*Proof.* The Jacobian matrix of model (2), is given by

$$J = \begin{pmatrix} -\frac{\beta_1\phi I_v}{N_v} - (\alpha+\mu_h) & 0 & 0 & \omega & \frac{\beta_1\phi S_h I_v}{N_v^2} & -\frac{\beta_1\phi S_h S_v}{N_v^2} \\ \alpha & -\frac{(1-\epsilon)\beta_2\phi I_v}{N_v} - \mu_h & 0 & 0 & \frac{(1-\epsilon)\beta_2\phi V_h I_v}{N_v^2} & -\frac{(1-\epsilon)\beta_2\phi V_h S_v}{N_v^2} \\ \frac{\beta_1\phi I_v}{N_v} & \frac{(1-\epsilon)\beta_2\phi I_v}{N_v} & -(\mu_h+\gamma+\delta) & 0 & -(\frac{\beta_1\phi S_h I_v+(1-\epsilon)\beta_2\phi V_h I_v}{N_v^2}) & \frac{\beta_1\phi S_h S_v+(1-\epsilon)\beta_2\phi V_h S_v}{N_v^2} \\ 0 & 0 & \gamma & -(\mu_h+\omega) & 0 & 0 \\ \frac{\phi\kappa S_v I_h}{N_h^2} & \frac{\phi\kappa S_v I_h}{N_h^2} & -\frac{\phi\kappa S_v(S_h+V_h+R_h)}{N_h^2} & \frac{\phi\kappa S_v I_h}{N_h^2} & -\frac{\phi\kappa I_h}{N_h} - \mu_v & 0 \\ -\frac{\phi\kappa S_v I_h}{N_h^2} & -\frac{\phi\kappa S_v I_h}{N_h^2} & \frac{\phi\kappa S_v(S_h+V_h+R_h)}{N_h^2} & -\frac{\phi\kappa S_v I_h}{N_h^2} & \frac{\phi\kappa I_h}{N_h} & -\mu_v \end{pmatrix} \tag{34}$$

Evaluating (34) at $MFE$, $E^0 = \left(\frac{\Lambda_h}{\alpha+\mu_h},\ \frac{\alpha\Lambda_h}{\mu_h(\alpha+\mu_h)},\ 0,\ 0,\ \frac{\Lambda_v}{\mu_v},\ 0\right)$, we obtain $J_{(E^0)}$,

$$J_{(E^0)} = \begin{bmatrix} -(\alpha+\mu_h) & 0 & 0 & \omega & 0 & -\frac{\beta_1\phi\mu_v\Lambda_h}{(\alpha+\mu_h)\Lambda_v} \\ \alpha & -\mu_h & 0 & 0 & 0 & -\frac{(1-\epsilon)\beta_2\phi\alpha\mu_v\Lambda_h}{\mu_h(\alpha+\mu_h)\Lambda_v} \\ 0 & 0 & -(\mu_h+\gamma+\delta) & 0 & 0 & \frac{\beta_1\phi\mu_v\Lambda_h}{(\alpha+\mu_h)\Lambda_v} + \frac{(1-\epsilon)\beta_2\phi\alpha\Lambda_h\mu_v}{\mu_h(\alpha+\mu_h)\Lambda_v} \\ 0 & 0 & \gamma & -(\mu_h+\omega) & 0 & 0 \\ 0 & 0 & -\frac{\phi\kappa\Lambda_v\mu_h}{\Lambda_h\mu_v} & 0 & -\mu_v & 0 \\ 0 & 0 & \frac{\phi\kappa\Lambda_v\mu_h}{\Lambda_h\mu_v} & 0 & 0 & -\mu_v \end{bmatrix} \tag{35}$$

Clearly the four eigenvalues are negative as shown, -$(\alpha+\mu_h)$, -$\mu_h$, -$(\mu_h+\omega)$,-$\mu_v$, the remaining

eigenvalues can be obtained from,

$$B = \begin{bmatrix} -(\mu_h + \gamma + \delta) & \frac{\beta_1 \phi \mu_v \Lambda_h}{(\alpha + \mu_h)\Lambda_v} + \frac{(1-\epsilon)\beta_2 \phi \alpha \Lambda_h \mu_v}{\mu_h(\alpha + \mu_h)\Lambda_v} \\ \frac{\phi \kappa \Lambda_v \mu_h}{\Lambda_h \mu_v} & -\mu_v \end{bmatrix} \tag{36}$$

where B is a 2×2 reduced matrix of $J_{(E^0)}$. Routh-Hurwitz Criterion is applied to compute the eigenvalues of matrix B. A negative trace and positive determinant of matrix B, which will ensure that the eigenvalues of matrix B are of negative real part. The trace: $tr(B) = -(\mu_h + \gamma + \delta) - \mu_v < 0$, and the determinant of matrix B given as: $\det(B) = \mu_v(\mu_h + \gamma + \delta)(1 - R_v^2)$. Since the $tr(B) < 0$ and the determinant $\det(B) = \mu_v(\mu_h + \gamma + \delta)(1 - R_v^2)$ is positive when $R_v < 1$. Therefore, the $MFE$ is locally asymptotically stable whenever $R_v < 1$ and unstable when $R_v > 1$. □

Proposition (4) implies that for a small perturbation of the $MFE$, the solutions of model (2) will eventually converge to the $MFE$ whenever $R_v < 1$. Epidemiologically, this implies that if a few infectious individuals are introduced into a fully susceptible population, malaria will die out whenever $R_v < 1$, otherwise it will spread.

### 2.6 Global Stability of Malaria Free Equilibrium

The Castillo-Chavez theorem in [5] is applied to compute the global stability of the malaria-free equilibrium. Rewriting the model (2) in the form

$$\frac{dX}{dt} = H(X, Z), \qquad \frac{dZ}{dt} = G(X, Z), \qquad G(X, 0) = 0, \tag{37}$$

where $X \in \mathbb{R}^4$ represents uninfected compartments X=$(S_h, V_h, R_h, S_v)$ and $Z \in \mathbb{R}^2$, represents infected compartments $Z = (I_h, I_v)$. At the malaria-free equilibrium $(MFE)$,

$$E^0 = (X^*, Z^*) = (X^*, 0), \tag{38}$$

where

$$X^* = \left( \frac{\Lambda_h}{\alpha + \mu_h}, \frac{\alpha \Lambda_h}{\mu_h(\alpha + \mu_h)}, 0, \frac{\Lambda_v}{\mu_v} \right). \tag{39}$$

For global stability, the conditions in (40) has to be achieved:

$$\begin{aligned} \frac{dX}{dt} &= H(X, 0), \quad X^* \text{ is globally asymptotically stable,} \\ G(X, Z) &= BZ - \hat{G}(X, Z), \qquad \hat{G}(X, Z) \geq 0. \end{aligned} \tag{40}$$

where $B$=$D_Z G(X^*, 0)$ is a $M$-matrix (the off-diagonal elements of $B$ are non-negative). Conditions (40) must be satisfied to guarantee a globally asymptotic stability of the fixed point, otherwise it is unstable.

**Proposition 5.** *The fixed point $E^0$=$(X^*, 0)$ is proved to be globally asymptotically stable equilibrium point of the model whenever $R_v < 1$ and the conditions in(40)are satisfied, otherwise it is unstable*

*Proof.* From the model (2), we have

$$H(X,0) = \begin{bmatrix} \Lambda_h - (\alpha+\mu_h)S_h + \omega R_h \\ \alpha S_h - \mu_h V_h \\ -(\mu_h+\omega)R_h \\ \Lambda_v - \mu_v S_v \end{bmatrix}, \quad \text{and} \quad G(X,Z) = BZ - \hat{G}(X,Z) \tag{41}$$

where

$$B = \begin{bmatrix} -(\mu_h+\gamma+\delta) & \frac{\beta_1\phi S_h^*}{N_v^*} + \frac{(1-\epsilon)\beta_2\phi V_h^*}{N_v^*} \\ \frac{\phi\kappa S_v^*}{N_h^*} & -\mu_v \end{bmatrix}, \quad \hat{G}(X,0) = \begin{pmatrix} \hat{G}_1(X,Z) \\ \hat{G}_2(X,Z) \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \end{pmatrix} \tag{42}$$

Evaluating the Jacobian of the infected subsystem at the DFE where $N_h^0 = S_h^0 + V_h^0 = \frac{\Lambda_h}{\mu_h}$, gives

$$B = \begin{bmatrix} -(\mu_h+\gamma+\delta) & \frac{\beta_1\phi\mu_v\Lambda_h}{(\alpha+\mu_h)\Lambda_v} + \frac{(1-\epsilon)\beta_2\phi\alpha\Lambda_h\mu_v}{\mu_h(\alpha+\mu_h)\Lambda_v} \\ \frac{\phi\kappa\Lambda_v\mu_h}{\Lambda_h\mu_v} & -\mu_v \end{bmatrix} \tag{43}$$

$$\hat{G}(X,0) = \begin{pmatrix} \hat{G}_1(X,Z) \\ \hat{G}_2(X,Z) \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \end{pmatrix} \tag{44}$$

Since $\hat{G}(X,Z) \geq 0$ as in (44) and the conditions in (40) are satisfied, it follows that the malaria-free equilibrium $E^0$ is globally asymptotically stable (GAS) whenever $R_v < 1$. □

Proposition (5) implies that given a large perturbation of the $MFE$, the solutions of the model will eventually converge to the $MFE$ whenever $R_v < 1$. Epidemiologically, this means that if a large number of infectious individuals are introduced into a fully susceptible population, malaria will die out whenever $R_v < 1$; otherwise, it will spread.

## 3 Malaria Persistence Equilibrium (MPE)

The model state of (2) where the malaria disease persists is known as the malaria persistence equilibrium ($MPE$). This is where the malaria disease is still present in the population understudy. The ($MPE$) of the model (2) is given by setting $(S_h^*, V_h^*, I_h^*, R_h^*, S_v^*, I_v^*) \neq 0$, thus $E^* = (S_h^*, V_h^*, I_h^*, R_h^*, S_v^*, I_v^*) \in \mathbb{R}_+^6$

**Proposition 6.** *There exist malaria persistence equilibrium* ($MPE$) *state of the model(2) if* $(I_h^*, I_v^*) > 0$ *whenever* $R_v > 1$.

*Proof.* At equilibrium the last equation of model (2) becomes:

$$\frac{\phi\kappa S_v I_h^*}{N_h} - \mu_v I_v^* = 0$$

which can be expressed as:

$$I_v^* = \frac{\phi\kappa S_v I_h^*}{\mu_v N_h} \tag{45}$$

Substituting equation(45)into the third equation of model(2)and solving for $I_h$ yields:

$$\frac{dI_h^*}{dt} = \frac{\beta_1\phi S_h\phi\kappa S_v I_h^*}{\mu_v N_h N_v} + \frac{(1-\epsilon)\beta_2\phi V_h\phi\kappa S_v I_h^*}{\mu_v N_h N_v} - (\mu_h+\gamma+\delta)I_h^* \tag{46}$$

Thus,

$$\begin{aligned}\frac{dI_h^*}{dt} &= \left[\frac{\phi^2\kappa\beta_1 S_h S_v}{\mu_v N_h N_v} + \frac{(1-\epsilon)\beta_2\phi^2\kappa V_h S_v}{\mu_v N_h N_v} - (\mu_h+\gamma+\delta)\right] I_h^* \\ &= \left[\phi^2\kappa\frac{(\beta_1 S_h S_v + (1-\epsilon)\beta_2 V_h S_v)}{\mu_v N_h N_v} - (\mu_h+\gamma+\delta)\right] I_h^* \\ &= (\mu_h+\gamma+\delta)\left[\frac{\phi^2\kappa(\beta_1 S_h S_v + (1-\epsilon)\beta_2 V_h S_v)}{\mu_v(N_h N_v)(\mu_h+\gamma+\delta)} - 1\right] I_h^* \end{aligned} \tag{47}$$

Substituting $R_v$ that is equation (32) into (47) yields:

$$\frac{dI_h^*}{dt} = (\mu_h+\gamma+\delta)(R_v^2-1)I_h^* \tag{48}$$

Integrating by separation of variables, (48) becomes:

$$\int \frac{dI_h^*}{I_h^*} = \int (\mu_h+\gamma+\delta)(R_v^2-1)dt$$

$$\ln I_h^* = (\mu_h+\gamma+\delta)(R_v^2-1)t + \ln I_h^*(0)$$

hence

$$I_h^*(t) = I_h^*(0)e^{(\mu_h+\gamma+\delta)(R_v^2-1)t} \tag{49}$$

$$\lim_{t\to\infty} I_h^*(t) > 0$$

Therefore $I_h^* > 0$ when $R_v > 1$ More still at equilibrium, the third equation of the model (2) becomes:

$$I_h^* = \left[\frac{\frac{\beta_1\phi S_h}{N_v} + (1-\epsilon)\beta_2\phi\frac{V_h}{N_v}}{(\mu+\gamma+\delta)}\right] I_v^*$$

which can be expressed again as

$$\frac{dI_v^*}{dt} = \left[\frac{\phi\kappa S_v}{N_h}\left[\frac{\beta_1\phi\frac{S_h}{N_v} + (1-\epsilon)\beta_2\phi\frac{V_h}{N_v}}{(\mu+\gamma+\delta)}\right] - \mu_v\right] I_v^*,$$

Thus

$$\left[\frac{\phi^2\kappa\beta_1 S_v S_h + (1-\epsilon)\phi^2\beta_2\kappa S_v V_h}{N_h N_v(\mu+\gamma+\delta)} - \mu_v\right] I_v^*,$$

then

$$\mu_v\left[\frac{\phi^2\kappa\left[\beta_1 S_v S_h + (1-\epsilon)\beta_2 S_v V_h\right]}{N_h N_v\mu_v(\mu+\gamma+\delta)} - 1\right] I_v^*$$

substituting $R_v$ one obtains

$$\frac{dI_v^*}{dt} = \mu_v(R_v^2 - 1)I_v^* \tag{50}$$

Integrating by separation of variables equation (50) becomes,

$$\int \frac{dI_v^*}{I_v^*} = \int \mu_v(R_v^2 - 1)dt$$

$$\ln I_v^* = \mu_v(R_v^2 - 1)t + \ln I_v^*(0)$$

$$I_v^* = I_v^*(0)e^{\mu_v(R_v^2-1)t} \tag{51}$$

Therefore $I_v^* > 0$ when $R_v > 1$ That means there exists malaria persistence equilibrium $(MPE)$ whenever $R_v > 1$ and this therefore completes the proof. □

### 3.1 Local Stability of Malaria Persistence Equilibrium

**Proposition 7.** *The malaria persistence equilibrium $(MPE)$ of the model (2) is locally asymptotically stable whenever $R_v > 1$ and unstable whenever $R_v < 1$.*

*Proof.* Consider the jacobian matrix (34) of the model (2) evaluated at $MPE$ given by $J^*$ where $J^* = J(E^*)$

$$J(E^*) = \begin{pmatrix} -\frac{\beta_1\phi I_v^*}{N_v^*} - (\alpha + \mu_h) & 0 & 0 & \omega & \frac{\beta_1\phi S_h^* I_v^*}{(N_v^*)^2} & -\frac{\beta_1\phi S_h^* S_v^*}{(N_v^*)^2} \\ \alpha & -\frac{(1-\epsilon)\beta_2\phi I_v^*}{N_v^*} - \mu_h & 0 & 0 & \frac{(1-\epsilon)\beta_2\phi V_h^* I_v^*}{(N_v^*)^2} & -\frac{(1-\epsilon)\beta_2\phi V_h^* S_v^*}{(N_v^*)^2} \\ \frac{\beta_1\phi I_v^*}{N_v^*} & \frac{(1-\epsilon)\beta_2\phi I_v^*}{N_v^*} & -(\mu_h + \gamma + \delta) & 0 & -(\frac{\beta_1\phi S_h^* I_v^* + (1-\epsilon)\beta_2\phi V_h^* I_v^*}{(N_v^*)^2}) & \frac{\beta_1\phi S_h^* S_v^* + (1-\epsilon)\beta_2\phi V_h^* S_v^*}{(N_v^*)^2} \\ 0 & 0 & \gamma & -(\mu_h + \omega) & 0 & 0 \\ \frac{\phi\kappa S_v^* I_h^*}{(N_h^*)^2} & \frac{\phi\kappa S_v^* I_h^*}{(N_h^*)^2} & -\frac{\phi\kappa S_v^*(S_h^* + V_h^* + R_h^*)}{(N_h^*)^2} & \frac{\phi\kappa S_v^* I_h^*}{(N_h^*)^2} & -\frac{\phi\kappa I_h^*}{N_h^*} - \mu_v & 0 \\ -\frac{\phi\kappa S_v^* I_h^*}{(N_h^*)^2} & -\frac{\phi\kappa S_v^* I_h^*}{(N_h^*)^2} & \frac{\phi\kappa S_v^*(S_h^* + V_h^* + R_h^*)}{(N_h^*)^2} & -\frac{\phi\kappa S_v^* I_h^*}{(N_h^*)^2} & \frac{\phi\kappa I_h^*}{N_h^*} & -\mu_v \end{pmatrix} \tag{52}$$

Since the diagonal elements of the matrix $J(E^*)$ are negative and eigenvalues of any square matrix $A$ are the same as $A^T$, an argument using Gershgorin discs [4] and [8] shows that $J(E^*)$ is stable if it is diagonally dominant in rows and columns. Applying the Gershgorin discs theorem in proposition below as in [4],

**Proposition 8.** *Let $A = (a_{ij})$ be a square complex matrix. Then every eigenvalue of A lies in one of the Gershgorin circles*

$D_i = \{z \in \mathbb{C} : |z - a_{ii}| \leq R_i\} \quad \vee \quad D_j = \{z \in \mathbb{C} : |z - a_{jj}| \leq R_j\}$, *where,*

$R_i = \sum_{\substack{j=1 \\ j\neq i}}^{n} |a_{ij}| \quad R_j = \sum_{\substack{i=1 \\ i\neq j}}^{n} |a_{ij}|$. *The union of the n Gershgorin disks is called the Gershgorin set,*

$D = \bigcup_{i=1}^{n} D_i$ .

The first hypothesis $J(E^*)_{ii} < 0$ for $i = 1, \ldots, 6$ of the Jacobian (52) is satisfied. The second hypothesis $R_i < |J(E^*)_{ii}|$ for $i = 1, \ldots, 6$ is verified if and only if

$$D_1 = \left\{ z : \left| z + \frac{\beta_1 \phi I_v^*}{N_v^*} + \alpha + \mu_h \right| \leq R_1 \right\},$$

$$D_2 = \left\{ z : \left| z + \frac{(1-\epsilon)\beta_2 \phi I_v^*}{N_v^*} + \mu_h \right| \leq R_2 \right\},$$

$$D_3 = \{ z : |z + \mu_h + \gamma + \delta| \leq R_3 \},$$

$$D_4 = \{ z : |z + \mu_h + \omega| \leq R_4 \},$$

$$D_5 = \left\{ z : \left| z + \frac{\phi \kappa I_h^*}{N_h^*} + \mu_v \right| \leq R_5 \right\},$$

$$D_6 = \{ z : |z + \mu_v| \leq R_6 \}$$

In consequence, $J(E^*)$ is locally asymptotically stable when all of the above inequalities are satisfied as in [4]. Taking the theorem and proofing by columns as in [8], we have that;

setting $\rho = \max\{g_1, g_2, g_3, g_4, g_5, g_6\}$

where

$$g_1 = -\frac{\beta_1 \phi I_v^*}{N_v^*} - (\alpha + \mu_h) + \alpha + \frac{\beta_1 \phi I_v^*}{N_v^*} + 0 + \frac{\phi \kappa S_v^* I_h^*}{(N_h^*)^2} - \frac{\phi \kappa S_v^* I_h^*}{(N_h^*)^2} = -\mu_h.$$

$$g_2 = 0 - \frac{(1-\epsilon)\beta_2 \phi I_v^*}{N_v^*} - \mu_h + \frac{(1-\epsilon)\beta_2 \phi I_v^*}{N_v^*} + 0 + \frac{\phi \kappa S_v^* I_h^*}{(N_h^*)^2} - \frac{\phi \kappa S_v^* I_h^*}{(N_h^*)^2} = -\mu_h.$$

$$g_3 = 0 + 0 - (\mu_h + \gamma + \delta) + \gamma - \frac{\phi \kappa S_v^* (S_h^* + V_h^* + R_h^*)}{(N_h^*)^2} + \frac{\phi \kappa S_v^* (S_h^* + V_h^* + R_h^*)}{(N_h^*)^2} = -(\mu_h + \delta).$$

$$g_4 = \omega + 0 + 0 - (\mu_h + \omega) + \frac{\phi \kappa S_v^* I_h^*}{(N_h^*)^2} - \frac{\phi \kappa S_v^* I_h^*}{(N_h^*)^2} = -\mu_h.$$

$$g_5 = \frac{\beta_1 \phi S_h^* I_v^*}{(N_v^*)^2} + \frac{(1-\epsilon)\beta_2 \phi V_h^* I_v^*}{(N_v^*)^2} - \frac{\beta_1 \phi S_h^* I_v^* + (1-\epsilon)\beta_2 \phi V_h^* I_v^*}{(N_v^*)^2} + 0 - \frac{\phi \kappa I_h^*}{N_h^*} - \mu_v + \frac{\phi \kappa I_h^*}{N_h^*} = -\mu_v.$$

$$g_6 = -\frac{\beta_1 \phi S_h^* S_v^*}{(N_v^*)^2} - \frac{(1-\epsilon)\beta_2 \phi V_h^* S_v^*}{(N_v^*)^2} + \frac{\beta_1 \phi S_h^* S_v^* + (1-\epsilon)\beta_2 \phi V_h^* S_v^*}{(N_v^*)^2} + 0 + 0 - \mu_v = -\mu_v.$$

gives,

$$\rho = \max\{-\mu_h,\ -(\mu_h + \delta),\ -\mu_v\} < 0$$

This implies diagonal dominance as claimed and hence the proof since each eigenvalue lies in at least one of the discs, giving a sufficient condition for stability. Therefore the Malaria Persistence Equilibrium ($MPE$) point is locally asymptotically stable provided that $R_v > 1$, otherwise it is unstable. □

Proposition(7) implies that a small perturbation of the $MPE$, the solution of the model (2) will always converge to the $MPE$ whenever $R_v > 1$. Epidemiologically it implies that if a few infectious individuals are introduced in a fully susceptible population and there are new secondary infections

produced whenever $R_v > 1$ then malaria would persist in the population.

## 4 Numerical Simulation and Discussion

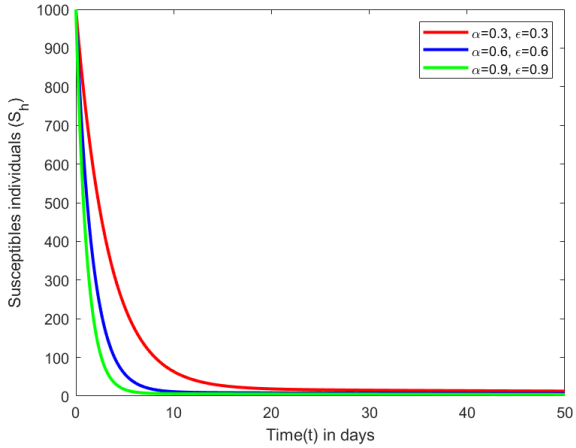
For the purpose of illustrating the behavior of the solutions of the model (2) graphically, simulation analysis of the model with varying parameter values is performed in this section. This helps in deducing control strategies against malaria disease. Currently malaria disease is controlled through control strategies that include strategies such as clearing of Some bushes close to homesteads, insecticide spraying and insecticide treated nets for sleeping, vaccination and treatment [18, 7]. For the purpose of the simulation, the initial population are taken to be $S_h(0)$=1000, $V_h(0)$=700, $I_h(0)$=500, $R_h(0)$=300, $S_v(0)$=1500, $I_v(0)$=1000. The mosquito population is taken to be higher than the human population so as to allow the spread of the malaria infection [6].

Figure 2 show the effect of varying vaccination rate $\alpha$ and vaccine efficacy $\epsilon$ on the susceptible human $S_h$ and vaccinated human $V_h$ that is (2a) and (2b) respectively. It is observed that the susceptible humans $S_h$ figure (2a), reduces at a higher rate as vaccine efficacy ($\epsilon$) and vaccination rate ($\alpha$) increases, Sharp decrease can be seen when vaccination rate ($\alpha$)=90% and vaccine efficacy ($\epsilon$)=90%. In figure (2b), the vaccinated individuals ($V_h$) increases gradually as the susceptible humans ($S_h$) are being moved to the vaccinated class as a result of increased vaccination rate ($\alpha$) and vaccine efficacy ($\epsilon$), at around 90% vaccine efficacy ($\epsilon$) and vaccination rate ($\alpha$) a sharp increment in the vaccinated population is experienced. For the eradication of malaria infection, the control strategies should focus and target vaccine efficacy ($\epsilon$) and the vaccination rate ($\alpha$) for elimination of malaria disease.

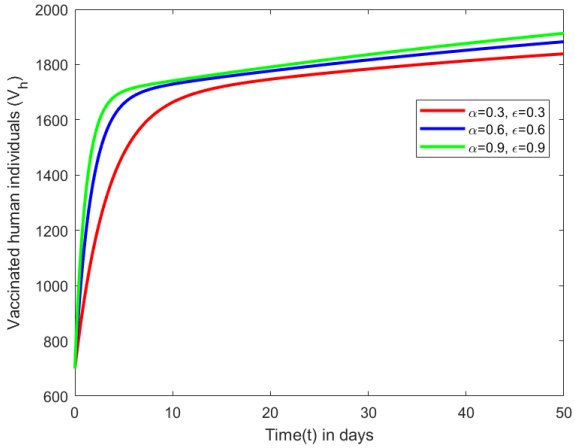
Figure (3a) show the effect of varying vaccination rate ($\alpha$) and vaccine efficacy ($\epsilon$) on the infected human ($I_h$), with vaccination the infected humans ($I_h$) reduces sharply as the rate of transmission is low since most of the population are vaccinated, hence control strategies should focus on increased vaccination rate ($\alpha$) using vaccine of higher efficacy ($\epsilon$), as can be seen in figure (3a), with vaccination rate and vaccine efficacy at around 90%, the infected population reduces sharply hence high effective malaria control strategy.

Figure (3b) shows the recruitment of mosquito population seasonally during rainy seasons and dry seasons. Mosquitoes are recruited in different swarms, therefore the population depends on the seasons. It can be clearly seen from figure (3b) that, during rainy seasons there are high population of mosquito compared to dry seasons, in January and February dry season when the temperatures are high, the mosquito population is reduced as their breeding places are few. From March to June wet season a lot of stagnant water are experienced hence a spike and sharp increment on mosquito population, in July and September there is low recruitment since it is dry season, in October to December a high spike is seen again hence high biting rate leading to high infection transmission. However areas of irrigation may not be seasonal as mosquitoes are prone to these areas, hence malaria is endemic throughout the year. It is clear that for malaria to be eradicated there should be an interplay between vaccination, treatment and personal protection such as clearing bushes, use of insecticides treated nets, and mosquito repellents. This shows that for eradication of malaria disease the effective control measures should target reduction the mosquito population that is susceptible and infected vectors by

destroying their breeding places. Therefore, reducing the interaction with humans and the spread of the infection would be reduced.

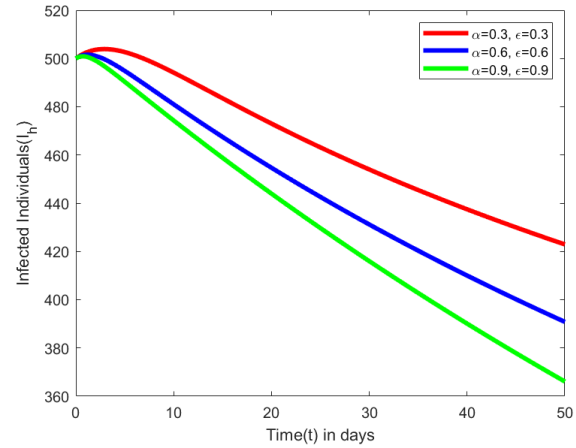



(a) Effect of varying vaccination rate ($\alpha$) and vaccine efficacy ($\epsilon$) on susceptible human ($S_h$) and

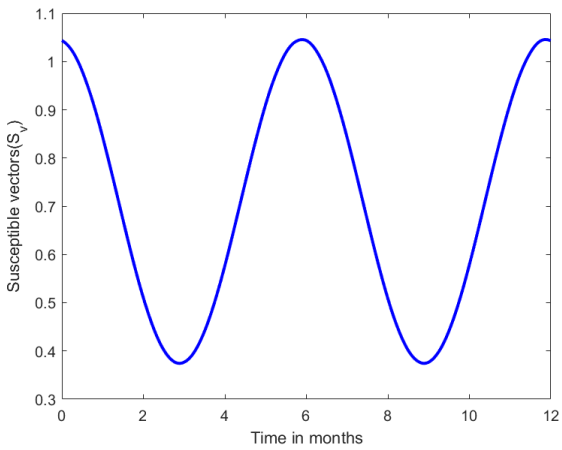



(b) Effect of varying vaccination rate ($\alpha$) and vaccine efficacy ($\epsilon$) on Vaccinated human ($V_h$)

Figure 2: **Simulation of effect of varying vaccination rate $\alpha$ and vaccine efficacy $\epsilon$ on the susceptible human ($S_h$) and vaccinated human ($V_h$)**




(a) **Effect of varying vaccination rate ($\alpha$) and vaccine efficacy ($\epsilon$) on Infected human ($I_h$)**




(b) **Seasonal recruitment of mosquitoes**

Figure 3: **Effect of varying vaccination rate ($\alpha$) and vaccine efficacy ($\epsilon$) on Infected human ($I_h$) and seasonal recruitment of mosquitoes**

## 5 Conclusion

In this study an extended deterministic SIRS-SI malaria model incorporating seasonality on vector recruitment with standard incidence and vaccination is presented and analyzed. Positivity and boundedness of the model solution is shown. The vaccine reproduction number $R_v$ is computed using the next generation matrix approach. Stability analysis shows that the $MFE$ is locally asymptotically stable when $R_v < 1$, this implies that for a small perturbation of the $MFE$, the solutions of model (2) will eventually converge to the $MFE$ whenever $R_v < 1$. Epidemiologically, this implies that if a few infectious individuals are introduced into a fully susceptible population, malaria will die out whenever $R_v < 1$, otherwise it will spread. The $MPE$ is locally asymptotically stable when $R_v > 1$, implies that a small perturbation of the $MPE$, the solution of the model (2) will always converge to the $MPE$ whenever $R_v > 1$. Epidemiologically it implies that if a few infectious individuals are introduced in a fully susceptible population and there are new secondary infections produced whenever $R_v > 1$ then malaria would persist in the population. From numerical simulation on the effect of the vaccination rate ($\alpha$), vaccine efficacy ($\epsilon$) control strategies such as increasing the vaccination rate ($\alpha$) while using a vaccine of higher efficacy ($\epsilon$) can lead to eradication of malaria disease, this reduction in the infected individuals is clearly seen in the figures (2, and 3a,) when $\epsilon$=90% and $\alpha$=90% there is a sharp decrease in the total number of the infected. It is shown that when rainfall is high, recruitment of mosquitoes is also high hence high malaria infectious periods, the policy makers should thus campaign for the use of mosquito treated bed nets during rainy seasons. Other control measures identified in other studies that can be implemented to eradicate malaria include using insecticide treated mosquito bed nets, clearing of bushes near the homesteads, spraying insecticide hence elimination of malaria disease in the population [18, 7]. This model assumes homogeneous mixing, ignores spatial heterogeneity, and uses periodic mosquito recruitment without incorporating climatic data directly. Future studies may incorporate, fractional order differential equations, stochastic models and their optimal control as discussed in [7].

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