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THRESHOLD AND STABILITY RESULTS FOR A MALARIA MODEL IN A POPULATION WITH PROTECTIVE INTERVENTION AMONG HIGH-RISK GROUPS

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Abstract. We develop a mathematical model for the dynamics of malaria with a varying population for which new individuals are recruited through immigration and births. In the model, we assume that non-immune travellers move to endemic regions with sprays, smear themselves with jelly that is repellent to mosquitoes on arrival in malarious regions, others take long term antimalarials, and pregnant women and infants receive full treatment doses at intervals even when they are not sick from malaria (commonly referred to as intermittent preventive therapy). We introduce more features that describe the dynamics of the disease for the control strategies that protect the above vulnerable groups. The model analysis is done and equilibrium points are analyzed to establish their local and global stability. The threshold of the disease, the control reproduction number, is established for which the disease can be eliminated.

Key words: Protective intervention, Threshold parameter, Control reproduction number, risk groups.

1 Introduction

Malaria presents a significant public health problem in much of the developing world despite considerable efforts during the last century to eradicate or control it. It remains the most prevalent and devastating parasitic disease in the tropics and causes $350 - 650$ million clinical cases world wide (Snow, 2005) [18], more than 80% of which live in sub-Saharan Africa. Although most control is at the

level of disease management through drug treatment, it can be controlled by preventive health measures.

The successful development of the malaria parasite within the mosquito depends on several critical factors such as ambient temperature and humidity; and whether the mosquito survives long enough for the parasite to complete its life cycle within the host mosquito. The risk of developing severe acquired immunity tends to be location specific. In highly endemic areas, this is limited to young children and immigrants and travellers from non-endemic areas. Another risk group is pregnant women whose natural immunity is depressed during pregnancy.

An important link between the human population and malaria is the spread of malaria due to human migration as it promotes transmission or tends to negate control efforts. There have been ecological changes related to development and human migration. These have disrupted the environment in ways that are favorable to mosquito proliferation that may cause epidemic outbreaks. It is reported (Gratz, 1999)[7] that increased human travel has spread infectious agents, introducing them into areas in which they had been absent. This has been facilitated by high-speed modern transport systems. Population movement (both migration and behavior) affects the pattern of malaria spread.

Despite unprecedented efforts on vaccine research, there is no approved vaccine for malaria and thus vaccination as a control strategy is not yet available and it is unlikely that highly effective vaccine will be available soon. However, some control strategies such as intermittent preventive treatment (IPT) for pregnant women and infants have been widely used. This is due to the fact that drug treatment and immune mechanisms act synergistically to clear infection. The use of mass chemotherapy is an effective strategy for control of malaria which aims to interrupt the transmission. Other malaria control interventions that reduce human-vector contact are widely used in malaria-infected areas. These include use of bed nets impregnated with an insecticide such as permethrin, screened houses that reduce human-vector contact/biting have been widely used as malaria control strategy in malaria-infected areas and have been effective in the control of the disease. However, evolution of chemical resistance in both parasite and mosquitoes has eroded the efficacy of some of these strategies. Thus, there is urgent need to understand the important parameters in the transmission of malaria and design effective intervention strategies for prevention and control of the spread of the disease.

A malaria model with preventive and control measures, temporary immunity, and varying population sizes is studied. Epidemic models for varying population sizes are discussed in Mena-Lorca and Hethcote (1992)[15]. Compartmental models for the spread of malaria with susceptible-exposed-infectious-recovered-susceptible (SEIRS) pattern for humans and a susceptible-exposed-infectious (SEI) pattern for mosquitoes have been proposed in (Ngwa and Shu, 2000)[17] and (Chitnis *et al*, 2006[3]). The Chitnis *et al* (2006) [3] model excludes the direct infectious-to-susceptible recovery in humans that is considered in (Ngwa and Shu, 2000 [17]). In Tumwiine *et al* (2007) [19] a model based on susceptible-infectious-recovered-susceptible (SIRS) pattern for humans and susceptible-infectious (SI) pattern for mosquitoes was considered. It was es-

tablished that recoveries and temporary immunity keep the populations at oscillation patterns and eventually converge to a steady state.

In this paper, we extend the model proposed in Tumwiine *et al*, 2007 [19] to study the dynamics of malaria in which intervention strategies for controlling disease are incorporated to include the protected class in the human population. It is common practice that non-immune travellers move to endemic regions with sprays, smear themselves with jelly that is repellent to mosquitoes on arrival in malarious regions, others take long term antimalarials, and pregnant women and infants receive full treatment doses at intervals even when they are not sick from malaria (commonly referred to as intermittent preventive therapy). Our model also includes infectious-susceptible recovery in the human population due to treatment as immunity is acquired after a period of continuous infections. We find a threshold parameter R_c (control reproduction number) which determines the dynamical behavior of the disease.

This paper is structured as follows. Section 2 presents the model formulation, explanation of the meaning of parameters and variables, and the assumptions they satisfy. In Section 3, the model is analyzed for stability of the steady states. The control reproduction number, R_c, an important threshold parameter is computed. The existence and stability of the equilibrium points are established. In Section 4, we give a brief discussion of results and make conclusions.

2 Formulation of the Model

We consider the total population sizes denoted by $N_V(t)$ and $N_H(t)$ for the female mosquitoes and humans hosts, respectively. These are structured into distinct epidemiological subclasses (compartments) of individuals which are susceptible, protected, infected and temporary immune denoted by $S_H(t), P_H(t)$, $I_H(t)$ and $R_H(t)$, respectively for the human population. The vector population sizes are denoted by $S_V(t)$ and $I_V(t)$ for the susceptible and infected subclasses, respectively. The transfer rates between the subclasses are composed of several epidemiological parameters. The susceptible human bitten by an infectious mosquito may become infected with a finite probability that depends on the abundance of infectious mosquitoes and of human hosts (Killeen *et al.,* 2001 [11]). The susceptible human population is increased by births and/or immigration at a constant rate Λ, with a fraction ρ of the susceptible recruited individuals taken to be under preventive control and enter the protected class. We assume that all the individuals recruited into the community are naive. The protection is assumed to reduce the likelihood of infection by a factor of ϵ. It should be noted that for, $\epsilon = 0$, the protection is effective, while for the case $\epsilon = 1$, then the protection is ineffective.

We also have a proportion $\pi(0 \leq \pi \leq 1)$ of the susceptible individuals under protection from the disease. This protection is non everlasting and wanes with time, and thus, the protected individuals may return to the susceptible class at a constant rate ν_1, when the control measures are relaxed or when the presumptive interventions wane. Infected human individuals return to the susceptible class when they are successfully treated at a constant rate, ν_2, while others

acquire temporary immunity at a constant rate r to join the immune class. Since disease-induced immunity due to malaria is temporary, they leave the recovered/immune state with constant rate γ to return to the susceptible class. There is no vertical transmission of the disease and all the human populations have a constant rate for non-disease related death μ_h. The infected humans have an additional disease-related constant death rate δ. The disease-induced death rate is very small in comparison with the recovery rate.

Mosquito recruitment is via a constant birth rate λ_v. Susceptible mosquitoes become infected by biting infected humans at a rate a, a proportion b of these bites lead to infection and c is the probability that a mosquito becomes infectious. Deaths occurs at a rate μ_v in each of the classes.

Malaria is transmitted horizontally, with the transmission modelled by standard incidence function (Hethcote 1976, 2000 [9, 10]). As in Tumwiine *et al.* (2007) [19], the term abS_HI_V/N_H denotes the rate at which the human hosts S_H get infected by infected mosquitoes I_V and acS_VI_H/N_H refers to the rate at which the susceptible mosquitoes S_V are infected by the infected human hosts I_H. In order to ensure that there is a nonzero flow of individuals into the protected class, we assume that $\pi + \rho\Lambda > 0$.

These assumptions lead to the following system of ordinary differential equations which describe the progress of the disease

$$\begin{aligned}
\frac{dS_H}{dt} &= (1-\rho)\Lambda - \frac{abS_HI_V}{N_H} - \pi S_H + \nu_1 P_H + \nu_2 I_H + \gamma R_H - \mu_h S_H,\\
\frac{dP_H}{dt} &= \pi S_H + \rho\Lambda - \frac{\epsilon abP_HI_V}{N_H} - (\nu_1+\mu_h)P_H,\\
\frac{dI_H}{dt} &= \frac{abS_HI_V}{N_H} + \frac{\epsilon abP_HI_V}{N_H} - (\nu_2 + r + \delta + \mu_h)I_H,\\
\frac{dR_H}{dt} &= rI_H - (\gamma+\mu_h)R_H,\\
\frac{dS_V}{dt} &= \lambda_v N_V - \frac{acS_VI_H}{N_H} - \mu_v S_V,\\
\frac{dI_V}{dt} &= \frac{acS_VI_H}{N_H} - \mu_v I_V.
\end{aligned} \tag{2.1}$$

The total population sizes N_H and N_V can be determined by $S_H + P_H + I_H + R_H = N_H$ and $S_V + I_V = N_V$ or from the differential equations

$$\frac{dN_H}{dt} = \Lambda - \mu_h N_H - \delta I_H, \quad \frac{dN_V}{dt} = (\lambda_v - \mu_v)N_V.$$

It is noted that in the absence of the disease ($\delta = 0$), the total human population size, $N_H \to \Lambda/\mu_h$ as $t \to \infty$. This shows that the feasible region

$$\begin{aligned}
D = \big\{&(S_H, P_H, I_H, R_H, S_V, I_V) :\\
&S_H,\ P_H,\ I_H,\ R_H,\ S_V,\ I_V \geq 0;\ N_H \leq \Lambda/\mu_h;\ N_V > 0\big\},
\end{aligned}$$

is a positively invariant set for the model, and thus, the model is well posed and biologically meaningful.

3 Analysis of the Model

In this Section, we analyze system (2.1) to obtain the equilibrium points of the system and their stability. We consider the equations for the proportions by first scaling the subpopulations for N_H, N_V and using the new variables. Set

$$s_h = \frac{S_H}{N_H},\ p_h = \frac{P_H}{N_H},\ i_h = \frac{I_H}{N_H},\ r_h = \frac{R_H}{N_H},\ s_v = \frac{S_V}{N_V},\ i_v = \frac{I_V}{N_V}$$

in the classes S_H, P_H, I_H, R_H, S_V, I_V, and denote $m = N_V/N_H$. Then we differentiate new variables with respect to time. This is done by differentiating the fractions with respect to time t and simplifying to give the following reduced system of equations

$$\begin{aligned}
\frac{ds_h}{dt} &= (1-\rho)\frac{\Lambda}{N_H} - \left[\frac{\Lambda}{N_H} + \pi - \delta i_h\right] s_h - abms_h i_v + \nu_1 p_h + \nu_2 i_h + \gamma r_h,\\
\frac{dp_h}{dt} &= \pi s_h + \rho\frac{\Lambda}{N_H} - \epsilon abmp_h i_v - \left[\frac{\Lambda}{N_H} + \nu_1 - \delta i_h\right] p_h,\\
\frac{di_h}{dt} &= \epsilon abmp_h i_v + abms_h i_v - \left[\frac{\Lambda}{N_H} + \nu_2 + r + \delta - \delta i_h\right] i_h,\\
\frac{dr_h}{dt} &= ri_h - \left[\frac{\Lambda}{N_H} + \gamma - \delta i_h\right] r_h,\\
\frac{ds_v}{dt} &= \lambda_v(1-s_v) - aci_h s_v,\\
\frac{di_v}{dt} &= acs_v i_h - \lambda_v i_v,
\end{aligned} \tag{3.1}$$

together with total population size N_H satisfying

$$\frac{dN_H}{dt} = \left[\frac{\Lambda}{N_H} - \mu_h - \delta i_h\right] N_H.$$

We observe that the system of proportions involves the total human population size, N_H, in the proportions for the human population.

The system can be reduced to a five-dimensional system by eliminating s_h and s_v, since $s_h = 1 - p_h - r_h - i_h$ and $s_v = 1 - i_v$, respectively in the feasible region (i.e where the model makes biological sense)

$$\begin{aligned}
\Gamma = \{(p_h, r_h, i_h, N_H, i_v) \in \mathbf{R}^5_+ :\ & 0 \le p_h, 0 \le r_h,\ 0 \le i_h,\ p_h + r_h + i_h \le 1,\\
& 0 \le i_v \le 1,\ N_H \le \Lambda/\mu_h\}
\end{aligned}$$

that can be shown to be positively invariant with respect to the system (3.2), where $\mathbf{R}^5_+$ denotes the non-negative cone of $\mathbf{R}^5$ including its lower dimensional faces. We denote the boundary and the interior of Γ by $\partial\Gamma$ and $\overset{\circ}{\Gamma}$, respectively.

Thus, we have the following system of equations.

$$\begin{aligned}
\frac{dp_h}{dt} &= \pi(1-p_h-i_h-r_h)+\rho\frac{\Lambda}{N_H}-\epsilon abmp_hi_v-\left[\frac{\Lambda}{N_H}+\nu_1-\delta i_h\right]p_h,\\
\frac{di_h}{dt} &= \epsilon abmp_hi_v+abm(1-p_h-i_h-r_h)i_v-\left[\frac{\Lambda}{N_H}+\nu_2+r+\delta-\delta i_h\right]i_h,\\
\frac{dr_h}{dt} &= ri_h-\left[\frac{\Lambda}{N_H}+\gamma-\delta i_h\right]r_h,\\
\frac{dN_H}{dt} &= \Lambda-\mu_hN_H-\delta i_hN_H, \quad \frac{di_v}{dt}=ac(1-i_v)i_h-\lambda_vi_v.
\end{aligned} \tag{3.2}$$

To compute the steady states of the system (3.2), we set the derivatives with respect to time equal to zero, then the following system of algebraic equations is obtained:

$$\begin{aligned}
&\pi(1-p_h-r_h-i_h)+\rho\frac{\Lambda}{N_H}=\epsilon abmp_hi_v+\left[\frac{\Lambda}{N_H}+\nu_1-\delta i_h\right]p_h,\\
&\epsilon abmp_hi_v+abm(1-p_h-r_h-i_h)i_v=\left[\frac{\Lambda}{N_H}+\nu_2+r+\delta-\delta i_h\right]i_h,\\
&ri_h=\left[\frac{\Lambda}{N_H}+\gamma-\delta i_h\right]r_h,\\
&i_v=\frac{aci_h}{\lambda_v+aci_h}, \quad \frac{\Lambda}{N_H}=\delta i_h+\mu_h.
\end{aligned} \tag{3.3}$$

Then, substituting $\dfrac{\Lambda}{N_H}=\delta i_h+\mu_h$ into the first three expressions of system (3.3) gives a reduced form of expressions in terms of i_h given by the following relations:

$$\begin{aligned}
p_h &= \frac{(\lambda_v+aci_h)\left[\pi+\rho\mu_h+(\rho\delta-\pi(1+\theta))i_h\right]}{[\epsilon a^2bmc+ac(\pi+\mu_h+\nu_1)]i_h+\lambda_v(\pi+\mu_h+\nu_1)}\\
&= \frac{\lambda_v(\pi+\rho\mu_h)+\left[\lambda_v(\rho\delta-\pi(1+\theta))+ac(\pi+\rho\mu_h)\right]i_h+ac[\rho\delta-\pi(1+\theta)]i_h^2}{[\epsilon a^2bmc+ac(\pi+\mu_h+\nu_1)]i_h+\lambda_v(\pi+\mu_h+\nu_1)},
\end{aligned} \tag{3.4}$$

for $\theta=r/(\mu_h+\gamma)$, where i_h can be obtained by substituting for r_h and i_v into the following expression

$$\epsilon abmp_hi_v+abm(1-p_h-i_h-r_h)i_v=\left[\nu_2+r+\mu_h+\delta\right]i_h.$$

This gives the equation

$$\epsilon a^2bmc\,p_hi_h+a^2bmc(1-p_h-i_h-\theta i_h)i_h=(\lambda_v+aci_h)\left[\nu_2+r+\mu_h+\delta\right]i_h. \tag{3.5}$$

From expression (3.5), it is clear that either $i_h=0$, for the disease-free equilibrium point E_0, or, for the endemic equilibrium point E_1, we have

$$p_h=\frac{[\lambda_v(\nu_2+r+\mu_h+\delta)-a^2bmc]+[a^2bmc(1+\theta)+ac(\nu_2+r+\mu_h+\delta)]i_h}{a^2bmc(\epsilon-1)}. \tag{3.6}$$

3.1 Existence and stability of equilibrium points

In order to establish whether we can have an equilibrium point when the disease is absent in the community, we need to verify that if any of the diseased classes of the equilibrium point is zero, then this applies to the rest of the diseased classes. This can easily be deduced from the simplified forms of the algebraic expressions (3.4) at the equilibrium point. We observe that for $i_h = 0$, $i_v = r_h = 0$, $p_h = \pi + \rho\mu_h/\pi + \nu_1 + \mu_h$ and $N_H = \Lambda/\mu_h$. Thus,

$$E_0 = (p_h, i_h, r_h, N_H, i_v) = \left(\frac{\pi + \rho\mu_h}{\pi + \nu_1 + \mu_h}, 0, 0, \frac{\Lambda}{\mu_h}, 0\right)$$

is the disease-free equilibrium point which always exists in Γ.

3.2 Threshold for the disease spread

In this subsection, we analyze system (3.2) to obtain the threshold for the establishment of the disease. This is one of the most important parameters in epidemiology known as the basic reproduction number R_0. It is defined as the number of secondary infections that occur when an infected individual is introduced into a completely susceptible population (Hethcote, 2000 [10]). We define R_c as the control reproduction number of the model. It represents the average number of secondary infections caused by a single infective in a population consisting essentially only of susceptible humans and mosquito vectors with control measures in place. It is a useful quantity in the study of a disease as it sets the threshold both for predicting its outbreak and for evaluating its control strategies. The control reproduction number R_c may be calculated in the same way as the basic reproduction number is calculated using the next generation approach (see Diekmann *et al.*, 1990 [5]; van den Driessche and Watmough, 2002 [20]). First, we need to re-arrange system (3.2) beginning with the infected classes as follows

$$\begin{aligned}
\frac{di_h}{dt} &= \epsilon abm\, p_h i_v + abm\, i_v(1 - p_h - r_h - i_h) - \left[\frac{\Lambda}{N_H} + \nu_2 + r + \delta - \delta i_h\right] i_h,\\
\frac{di_v}{dt} &= ac(1 - i_v)i_h - \lambda_v i_v,\\
\frac{dp_h}{dt} &= \pi(1 - p_h - r_h - i_h) + \rho\frac{\Lambda}{N_H} - \epsilon abm p_h i_v - \left[\frac{\Lambda}{N_H} + \nu_1 - \delta i_h\right] p_h,\\
\frac{dr_h}{dt} &= r i_h - \left[\frac{\Lambda}{N_H} + \gamma - \delta i_h\right] r_h,\\
\frac{dN_H}{dt} &= \left[\frac{\Lambda}{N_H} - \mu_h - \delta i_h\right] N_H,
\end{aligned}$$

and then distinguish the new infections from all other class transitions in the population. The infected class are i_h and i_v in the human population and mosquito, respectively. We thus compute the reproduction number using the next generation operator method (see van den Driessche and Watmough (2002) [20]), where $\mathcal{F}$ is the vector of rates of the appearance of new infections in each

compartment; $\mathcal{V} = \mathcal{V}^+ + \mathcal{V}^-$, where $\mathcal{V}^+$ is the vector of rates of transfer of individuals into the particular compartment; and $\mathcal{V}^-$ is the vector of rates of transfer of individuals out of the particular compartment. Hence, from our model, we have

$$\mathcal{F} = \begin{pmatrix} f_1 \\ ac(1-i_v)i_h \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} (\Lambda/N_H + \nu_2 + r + \delta)i_h - \delta i_h^2 \\ \lambda_v i_v \\ v_3 \\ (\Lambda/N_H + \gamma)r_h - r i_h - \delta i_h r_h \\ (\delta i_h + \mu_h - \Lambda/N_H)N_H \end{pmatrix},$$

where notation $f_1 = \epsilon ab\, m p_h i_v + ab\, m i_v(1 - p_h - i_h - r_h)$, $v_3 = \epsilon ab\, m p_h i_v + (\Lambda/N_H + \nu_1)p_h - \delta i_h p_h - \rho\Lambda/N_H - \pi(1 - p_h - i_h - r_h)$ are used.

Since we have two compartments for the infected classes, then matrices $\mathbf{F}$ and $\mathbf{V}$ obtained from the partial derivatives of $\mathcal{F}$ and $\mathcal{V}$ with respect to the infected classes are given by

$$\mathbf{F} = \begin{pmatrix} -abmi_v & f_{12} \\ ac(1-i_v) & -aci_h \end{pmatrix}, \quad \mathbf{V} = \begin{pmatrix} \Lambda/N_H + \nu_2 + r + \delta - 2\delta i_h & 0 \\ 0 & \lambda_v \end{pmatrix},$$

where $f_{12} = ab\, m\big(\epsilon p_h + (1 - p_h - r_h - i_h)\big)$. Let us evaluate these matrices at the disease-free equilibrium point, E_0, to give

$$\mathbf{F} = \begin{pmatrix} 0 & f_{12}^0 \\ ac & 0 \end{pmatrix}, \quad \mathbf{V} = \begin{pmatrix} \nu_2 + \mu_h + r + \delta & 0 \\ 0 & \lambda_v \end{pmatrix}.$$

where $f_{12}^0 = abm\big(\epsilon(\pi + \rho\mu_h) + \nu_1 + \mu_h(1-\rho)\big)/(\pi + \nu_1 + \mu_h)$. The next generation matrix, $\mathbf{F}\mathbf{V}^{-1}$ has the nonzero eigenvalue corresponding to the spectral radius, $s(\mathbf{F}\mathbf{V}^{-1})$ of the matrix $\mathbf{F}\mathbf{V}^{-1}$. This represents the reproduction number of the disease in the presence of protective measures given by

$$R_c = \sqrt{\frac{ac\,[\epsilon abm(\pi + \rho\mu_h) + abm(\nu_1 + \mu_h(1-\rho))]}{\lambda_v(\pi + \nu_1 + \mu_h)(\nu_2 + \mu_h + r + \delta)}}.$$

The expression for the *control reproduction number*, R_c has a biological meaning that is readily interpreted from terms under the square root. The first term ac/λ_v represents the number of secondary human infections caused by one infected mosquito vector. The second term represents the number of secondary mosquito infections caused by one infected human host. The square root represents the geometric mean R_c for an average individual of both species combined. This control reproduction number serves as an invasion threshold both for predicting outbreaks and evaluating control strategies that would reduce the spread of the disease in the community through the reduction of the control reproduction number and the parameters that enhance the spread of the disease due to the increase in the control reproduction number.

Currently, there is no vaccine against malaria and the main control measures that have been in place include intermittent preventive treatment (IPT) for

pregnant women and infants, use of treated bed nets and curtains, indoor residue spray.

In order to bring a population below the threshold, the number of susceptibles should be reduced by providing them protection from the disease. From the expression for the control reproduction number, we note that the parameters ϵ, ρ and π play important roles in the spread of the disease.

The value of the basic reproduction number can be obtained from the value of the control reproduction number when control measures are ineffective ($\epsilon = 1$) in a sense that both $\pi = 0$ and $\rho = 0$. Thus, the basic reproduction number of the model without control measures is given by

$$R_0 = \sqrt{\frac{a^2bmc}{\lambda_v(\nu_2 + \mu_h + r + \delta)}}.$$

From the two expressions for the reproduction number, we notice that $R_c \leq R_0$ for all $\pi, \rho \geq 0$. This implies that control interventions have a positive impact on reduction of the spread of malaria.

In the next section, we seek to establish whether the unique endemic equilibria exists. This is done by making a more realistic assumption that the protective control measures may not be totally effective, and thus $0 < \epsilon < 1$.

3.3 Existence of endemic equilibrium

In order to determine the existence of the endemic equilibrium in Γ, we assume $i_h \neq 0$ and use the expressions for p_h in system (3.4) and (3.6) in which we make the following substitutions:

$$\begin{aligned}
&a_1 = \lambda_v(\nu_2 + r + \mu_h + \delta) - a^2bmc,\\
&b_1 = a^2bmc(1+\theta) + ac(\nu_2 + r + \mu_h + \delta), \quad c_1 = a^2bmc(\epsilon - 1),\\
&a_2 = \lambda_v(\pi + \rho\mu_h),\ b_2 = \lambda_v[\rho\delta - \pi(1+\theta) + ac(\pi + \rho\mu_h)],\ c_2 = ac[\rho\delta - \pi(1+\theta)],\\
&d_2 = [\epsilon a^2bmc + ac(\pi + \mu_h + \nu_1)], \quad e_2 = \lambda_v(\pi + \mu_h + \nu_1)
\end{aligned}$$

to give respectively,

$$p_h = \frac{a_1 + b_1 i_h}{c_1}, \quad p_h = \frac{a_2 + b_2 i_h + c_2 i_h^2}{d_2 i_h + e_2}.$$

Equating these expressions gives

$$\frac{a_1 + b_1 i_h}{c_1} = \frac{a_2 + b_2 i_h + c_2 i_h^2}{d_2 i_h + e_2}$$

from which we have the following quadratic expression in i_h given by

$$\begin{aligned}
f(i_h) &:= (b_1d_2 - c_1c_2)i_h^2 + (a_1d_2 + b_1e_2 - c_1b_2)i_h + a_1e_2 - c_1a_2\\
&= Ai_h^2 + Bi_h + C = 0. \qquad (3.7)
\end{aligned}$$

For $R_c > 1$, the existence of endemic equilibria is determined by the presence in $(0, 1]$ of positive real solutions of the quadratic expression (3.7).

$$C = \lambda_v[\lambda_v(\nu_2 + r + \mu_h + \delta)(\pi + \mu_h + \nu_1)(1 - R_c^2)] < 0.$$

From the quadratic theorem, it follows that if x_1, x_2 are the roots of equation (3.7), then their product $x_1x_2 = C/A$. Since $C < 0$ and $A > 0$, then $C/A < 0$. Hence, there exists exactly one positive endemic equilibrium for $i_h \in (0, 1]$ whenever $R_c > 1$. This gives the threshold for the endemic persistence.

Therefore, we have proved the existence and uniqueness of the endemic equilibrium E_1 for system (3.2). This result is summarized in the following theorem:

Theorem 1. *If $R_c > 1$, the system (3.2) has a unique endemic equilibrium E_1 in $\overset{\circ}{\Gamma}$.*

3.4 Local stability of disease-free equilibrium E_0

Theorem 2. *The disease-free equilibrium point is locally asymptotically stable if $R_c < 1$ and unstable if $R_c > 1$.*

Proof. The disease-free steady state, E_0 is given by $\left(\frac{\pi + \rho\mu_h}{\pi+\nu_1 + \mu_h}, 0, 0, \frac{\Lambda}{\mu_h}, 0\right)$. The Jacobian matrix of the system (3.2) evaluated at E_0 is given by

$$J_{E_0} = \begin{bmatrix} -(\pi+\mu_h+\nu_1) & -[\pi-\frac{\delta(\pi+\rho\mu_h)}{\pi+\nu_1+\mu_h}] & -\pi & \frac{\mu_h^2}{\Lambda}(p_h-\rho) & -\epsilon abmp_h \\ 0 & -(\nu_2+\mu_h+r+\delta) & 0 & 0 & L \\ 0 & r & -(\mu_h+\gamma) & \frac{\mu_h^2}{\Lambda} & 0 \\ 0 & \frac{-\Lambda\delta}{\mu_h} & 0 & -\mu_h & 0 \\ 0 & ac & 0 & 0 & -\lambda_v \end{bmatrix},$$

where $L = \epsilon abmp_h + abm(1-p_h)$.

We observe from the first, third and fourth columns of J_{E_0} that this matrix has three distinct negative eigenvalues $-(\pi+\mu_h+\nu_1)$, $-(\mu_h+\gamma)$, $-\mu_h$, and the remaining two eigenvalues are obtained from the following 2×2 block matrix

$$A = \begin{bmatrix} -(\nu_2+\mu_h+r+\delta) & \dfrac{[\epsilon abm(\pi+\rho\mu_h) + abm(\nu_1+\mu_h(1-\rho)]}{(\pi+\nu_1+\mu_h)} \\ ac & -\lambda_v \end{bmatrix}$$

whose trace and determinant are given by

$$TrA = -(\nu_2+\mu_h+r+\delta+\lambda_v) < 0,$$
$$DetA = \lambda_v(\nu_2+\mu_h+r+\delta)(1-R_c^2) > 0 \quad \text{if } R_c < 1,$$

where

$$R_c = \sqrt{\frac{[\epsilon a^2 bmc(\pi+\rho\mu_h) + a^2 bmc(\nu_1+\mu_h(1-\rho))]}{\lambda_v(\pi+\nu_1+\mu_h)(\nu_2+\mu_h+r+\delta)}}.$$

Thus, we have established that the disease-free equilibrium point, E_0 is locally asymptotically stable if $R_c < 1$ and unstable if $R_c > 1$. □

3.4.1 Global stability of the disease-free equilibrium

We establish whether the disease-free equilibrium point is globally asymptotically stable. This is done by proving the following theorem.

Theorem 3. *The equilibrium point E_0 is globally asymptotically stable in Γ for $R_c \leq 1$, and it is unstable for $R_c > 1$. In this case, all the trajectories starting in a sufficiently small neighborhood of E_0 in Γ move away from E_0, except those of the $p_h - N_H$ coordinate plane (the case of no infection), which converge to E_0 in this plane.*

Proof. Consider the following Lyapunov function,

$$L = aci_h + (\nu_2 + \mu_h + r + \delta)i_v.$$

The orbital derivative is given by

$$\begin{aligned}
L' &= aci_h' + (\nu_2 + \mu_h + r + \delta)i_v' = \epsilon a^2 bmcp_h i_v + a^2 bmci_v(1 - p_h - i_h - r_h) \\
&\quad - ac[\nu_2 + \mu_h + r + \delta]i_h + (\nu_2 + \mu_h + r + \delta)[aci_h(1 - i_v) - \lambda_v i_v] \\
&= \epsilon a^2 bmcp_h i_v + a^2 bmci_v(1 - p_h - i_h - r_h) - [\lambda_v + aci_h](\nu_2 + \mu_h + r + \delta)i_v \\
&= \epsilon a^2 bmcp_h i_v + a^2 bmci_v(1 - p_h - i_h - r_h) - [\lambda_v + aci_h](\nu_2 + \mu_h + r + \delta)i_v \\
&\leq \epsilon a^2 bmcp_h i_v + a^2 bmci_v(1 - p_h) - \lambda_v(\nu_2 + \mu_h + r + \delta)i_v \\
&= [\lambda_v(\nu_2 + \mu_h + r + \delta)i_v]\left[\frac{\epsilon a^2 bmcp_h + a^2 bmc(1 - p_h)}{\lambda_v(\nu_2 + \mu_h + r + \delta)} - 1\right] \\
&= [\lambda_v(\nu_2 + \mu_h + r + \delta)i_v]\left[\frac{\epsilon a^2 bmc(\pi + \rho\mu_h) + a^2 bmc[\nu_1 + \mu_h(1 - \rho)]}{\lambda_v(\pi + \nu_1 + \mu_h)(\nu_2 + \mu_h + r + \delta)} - 1\right] \\
&\leq \lambda_v(\nu_2 + \mu_h + r + \delta)i_v[R_c^2 - 1] \leq 0 \quad \text{if} \quad R_c \leq 1.
\end{aligned}$$

We observe that our system has the maximum invariant set for $L' = 0$ if and only if $R_c \leq 1$ holds and $i_h = r_h = i_v = 0$. By Lyapunov-Lasalle's Theorem (see Hale, 1969 [8]), all the trajectories starting in the feasible region where the solutions have biological meaning approach the positively invariant subset of the set where $L' = 0$, which is the set where $i_h = r_h = i_v = 0$. In this set, $p_h'(t) = \pi + \rho\mu_h - (\pi + \mu_h + \nu_1)p_h$, so that as $t \to +\infty$, $p_h \to \frac{\pi + \rho\mu_h}{\pi + \mu_h + \nu_1}$. Hence, the equation $N_H'(t) = (\Lambda/N_H - \mu_h - \delta i_h)N_H$ is asymptotically equivalent to $N_H' = \Lambda - \mu_h N_H$ so that as $t \to +\infty$, $N_H \to \Lambda/\mu_h$. This shows that all solutions in the set where $i_h = r_h = i_v = 0$, go to the disease-free equilibrium E_0. Thus, $R_c \leq 1$ is the necessary and sufficient condition for the disease to be eliminated in the community. If $R_c > 1$, then $L' > 0$ for p_h, i_h, r_h, N_H, i_v sufficiently close to $\left(\frac{\pi + \rho\mu_h}{\pi + \nu_1 + \mu_h}, 0, 0, \Lambda/\mu_h, 0\right)$ in Γ except $i_v = 0$. Thus, E_0 is globally asymptotically stable in Γ if $R_c \leq 1$ and the disease always dies out. □

3.5 Local stability of the endemic equilibrium

The local stability of the equilibrium point, E_1 is determined by investigating the eigenvalues of the linearized system about the equilibrium solution. The

Jacobian matrix for the system is given by

$$J_{E_1} = \begin{bmatrix} -J_{11} & -J_{12} & -J_{13} & J_{14} & -J_{15} \\ -J_{21} & -J_{22} & -J_{23} & J_{24} & J_{25} \\ 0 & J_{32} & -J_{33} & J_{34} & 0 \\ 0 & -J_{42} & 0 & -J_{44} & 0 \\ 0 & J_{52} & 0 & 0 & -J_{55} \end{bmatrix}$$

where

$$J_{11} = \pi + \epsilon abmi_v + \mu_h + \nu_1,\ J_{12} = (\pi - \delta p_h),\ J_{13} = \pi,$$
$$J_{14} = \frac{(\mu_h + \delta i_h)^2(p_h - \rho)}{\Lambda},\ \ J_{15} = \epsilon abmp_h$$
$$J_{21} = abmi_v(1-\epsilon),\ J_{22} = abmi_v + \nu_2 + \mu_h + r + \delta - \delta i_h,$$
$$J_{23} = abmi_v,\ J_{24} = \frac{(\mu_h + \delta i_h)^2 i_h}{\Lambda},\ J_{25} = \epsilon abmp_h + abm(1 - p_h - i_h - r_h),$$
$$J_{32} = (r - \delta r_h),\ J_{33} = \mu_h + \gamma,\ J_{34} = \frac{(\mu_h + \delta i_h)^2 r_h}{\Lambda},\ J_{42} = \frac{\delta\Lambda}{(\mu_h + \delta i_h)},$$
$$J_{44} = \mu_h + \delta i_h,\ J_{52} = ac(1 - i_v),\ J_{55} = (\lambda_v + aci_h).$$

The characteristic equation for the equilibrium point, E_1 is given by

$$\det(J_{E_1} - \lambda I_5) = 0,$$

where I_5 is the 5×5 identity matrix. In order to determine the stability of E_1, we use the Routh-Hurwitz stability criteria on the characteristic equation of a fifth-degree polynomial given by

$$p_5(\lambda) = \lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5, \tag{3.8}$$

whose coefficients a_i in terms of the elements of J_{E_1} are

$$a_1 = J_{11} + J_{22} + J_{33} + J_{44} + J_{55}$$
$$a_2 = J_{11}J_{22} + (J_{11} + J_{22})(J_{33}+J_{44}+J_{55}) + J_{33}J_{44} + (J_{33} + J_{44})J_{55} + J_{23}J_{32} + J_{24}J_{42} - (J_{12}J_{21} + J_{25}J_{52}),$$
$$a_3 = J_{32}(J_{11}J_{23} - J_{13}J_{21} + J_{23}(J_{44}+J_{55}) + (J_{11}J_{22} - J_{12}J_{21})(J_{33}+J_{44}+J_{55}) + [J_{33}J_{44} + J_{55}(J_{33} + J_{44})][J_{11} + J_{22}] + (J_{33}J_{44}J_{55}) + J_{42}(J_{11}J_{24} - J_{21}J_{14}) + (J_{24}J_{42})(J_{33} + J_{55}) - J_{23}J_{34}J_{42} + J_{52}[J_{21}J_{15} - J_{11}J_{25} - J_{25}(J_{33} + J_{44})],$$
$$a_4 = J_{32}[(J_{44} + J_{55})(J_{11}J_{23} - J_{13}J_{21}) + J_{23}J_{44}J_{55}] + J_{33}J_{44}J_{55}(J_{11} + J_{22}) + J_{42}[(J_{11}J_{24} - J_{21}J_{14})(J_{33} + J_{55}) + J_{24}J_{33}J_{55}] + J_{34}J_{42}[J_{13}J_{21} - J_{11}J_{23} - J_{23}J_{55}] + J_{52}[(J_{33} + J_{44})(J_{21}J_{15} - J_{11}J_{25}) - J_{25}J_{33}J_{44}] - J_{12}J_{21}[(J_{33} + J_{44})J_{55} + J_{33}J_{44}],$$
$$a_5 = J_{32}J_{44}J_{55}(J_{11}J_{23} - J_{13}J_{21}) + J_{33}J_{44}J_{55}(J_{11}J_{22} - J_{12}J_{21}) + J_{52}J_{33}J_{44}(J_{21}J_{15} - J_{11}J_{25}) + J_{42}J_{33}J_{55}(J_{11}J_{24} - J_{21}J_{14}) + J_{34}J_{42}J_{55}(J_{13}J_{21} - J_{11}J_{23}).$$

The criteria that provides necessary and sufficient conditions for the endemic equilibrium to be asymptotically stable is the Routh-Hurwitz's theorem (see Lancaster, 1969 pp.272 [12]) which states that all the eigenvalues of J_{E_1} have negative real parts if and only if the Hurwitz determinants H_i are all positive. From the polynomial (3.8), the endemic equilibrium point is locally asymptotically stable provided

$$H_1 = a_1 > 0, \quad H_2 = a_1a_2 - a_3 > 0,$$
$$H_3 = a_1a_2a_3 + a_1a_5 - a_1^2a_4 - a_3^2 > 0,$$
$$H_4 = (a_3a_4 - a_2a_5)(a_1a_2 - a_3) - (a_1a_4 - a_5)^2 > 0, \quad H_5 = a_5H_4 > 0.$$

3.5.1 Global stability of the endemic equilibrium

In this section, we show that the disease persists when $R_c > 1$. This occurs if the infected fractions of the populations persist above a certain positive level for sufficiently large time. From the system (3.2), we observe that the dynamics of the disease depends on the total human population N_H. From the equation

$$\frac{dN_H}{dt} = \Lambda - \mu_h N_H - \delta i_h N_H$$

we get the solution in an explicit form

$$N_H(t) = \frac{\Lambda}{\mu_h + \delta i_h} + N_H(0)e^{-(\mu_h+\delta i_h)t}.$$

Since $N_H \to \Lambda/\mu_h + \delta i_h$, as $t \to +\infty$, then, substituting for N_H into the first three equations of the system (3.2) gives the following four-dimensional asymptotically autonomous differential system (3.9).

$$\begin{aligned}
\frac{dp_h}{dt} &= \pi(1 - p_h - i_h - r_h) + \rho(\mu_h + \delta i_h) - \epsilon abmp_h i_v - (\mu_h + \nu_1)p_h,\\
\frac{di_h}{dt} &= \epsilon abmp_h i_v + abm(1 - p_h - i_h - r_h)i_v - (\nu_2 + \mu_h + r + \delta)i_h, \qquad (3.9)\\
\frac{dr_h}{dt} &= ri_h - (\mu_h + \gamma)r_h,\\
\frac{di_v}{dt} &= ac(1 - i_v)i_h - \lambda_v i_v.
\end{aligned}$$

Let $\Omega = \{(p_h, i_h, r_h, i_v) \in {\mathbf{R}_+}^4 : p_h, i_h, r_h, i_v \geq 0; p_h + i_h + r_h \leq 1; 0 \leq i_v \leq 1\}$. We now study the global dynamics in $\overset{\circ}{\Omega}$, the interior of Ω, when $R_c > 1$. The global dynamics of the system (3.9) is said to be uniformly persistent (see Butler and Waltman, 1986 [2]) if there exists a positive constant $0 < \epsilon_0 < 1$ (independent of the choice of the solution) such that any solution $(p_h(t), i_h(t), r_h(t), i_v(t))$ of (3.9) with $(p_h(0), i_h(0), r_h(0), i_v(0)) \in \overset{\circ}{\Omega}$ satisfies

$$min\{\lim\inf_{t\to\infty} p_h(t), \lim\inf_{t\to\infty} i_h(t), \lim\inf_{t\to\infty} r_h(t), \lim\inf_{t\to\infty} i_v(t)\} \geq \epsilon_0. \quad (3.10)$$

This implies that the disease becomes endemic if the infectious population persists above a certain positive level. It has already been established that the

disease-free equilibrium point is stable when $R_c \leq 1$, and the disease always dies out. The condition for uniform persistence (see Freedman *et al.*, 1994 [6]) is equivalent to the condition, when the disease-free equilibrium point is unstable and this occurs if and only if $R_c > 1$. Thus, when the disease-free equilibrium is unstable and the condition $R_c > 1$ holds, then system (3.9) is uniformly persistent in Ω. Thus, the boundedness of Ω and condition (3.10) imply that there exists a compact set $K \subset \overset{\circ}{\Omega}$ for system (3.9) such that $x(t) = (p_h(t), i_h(t), r_h(t), i_v(t)) \in K$ for $t > T$ and $x(0) = (p_h(0), i_h(0), r_h(0), i_v(0)) \in \overset{\circ}{\Omega}$, where K is referred to as an absorbing set.

In order to study the global stability of the unique endemic equilibrium, we apply the novel approach of convergence of trajectories within an invariant manifold in $\mathbf{R^n}$. The detailed exposition of these techniques may be found in (Li and Muldowney, 1996 [13]) and (Muldowney, 1995 [16]). The aim of this section is to prove the following theorem

Theorem 4. *When $R_0 > 1$, the endemic equilibrium E_1 of system (3.2) is globally asymptotically stable in $\overset{\circ}{\Gamma}$.*

Some of the following results found in (Li and Muldowney, 2000 [14]) are used to prove Theorem 4 that each positive semiorbit in $\overset{\circ}{\Gamma}$ converges to an equilibrium. We denote the vector field defined by the system (3.9) and $x(t) = (p_h, i_h, r_h, i_v)$ by $f(x)$. Then system (3.9) can be written as

$$x' = f(x), \quad z' = \frac{\partial f}{\partial x}^{[3]}(x)z,$$

where $z = (z_1, z_2, z_3, z_4) \in \mathbf{R}^4 \cong \mathbf{R}^{\begin{pmatrix} 4 \\ 3 \end{pmatrix}}$. The Jacobian matrix of system (3.9) at the point (p_h, i_h, r_h, i_v) is given by

$$J_E = \begin{bmatrix} j_{11} & -\pi + \rho\delta & -\pi & -\epsilon abmp_h \\ -abmi_v(1-\epsilon) & -abmi_v & -abmi_v & j_{24} \\ 0 & r & -(\mu_h + \gamma) & 0 \\ 0 & ac(1-i_v) & 0 & -(\lambda_v + aci_h) \end{bmatrix}, \tag{3.11}$$

where $j_{11} = -(\pi + \epsilon abm\, i_v + \mu_h + \nu_1)$, $j_{24} = \epsilon abmp_h + abm(1 - p_h - i_h - r_h)$. Using the Appendix, the third additive compound matrix $\frac{\partial f}{\partial x}^{[3]}$ for the system (3.9), is given by

$$\frac{\partial f}{\partial x}^{[3]} = -(\pi + \epsilon abmi_v + \mu_h + \nu_1 + abmi_v + \mu_h + \gamma + \lambda_v + aci_h)\mathbf{I} + \Phi,$$

where Φ is the following matrix

$$\Phi = \begin{bmatrix} \lambda_v + aci_h & 0 & -j_{24} & -\epsilon abmp_h \\ 0 & \mu_h + \gamma & -abmi_v & \pi \\ -ac(1-i_v) & r & abmi_v & -(\pi - \rho\delta) \\ 0 & 0 & -abmi_v(1-\epsilon) & -j_{11} \end{bmatrix}.$$

Let us denote $V(x,z) = max\{a_1|z_1| + |z_2|, \frac{i_h}{r_h}(|z_3| + a_2|z_4|)\}$. We have the following differential inequalities from (3.11)

$$\begin{aligned}
D_+a_1|z_1(t)| &\le -(\pi + \epsilon abmi_v + \mu_h + \nu_1 + abmi_v + \mu_h + \gamma)a_1|z_1(t)| \\
&\quad - (\epsilon abmp_h + abm(1 - p_h - i_h - r_h))a_1|z_3(t)| - \epsilon abmp_h a_1|z_4(t)| \\
&\le -(\pi + \epsilon abmi_v + \mu_h + \nu_1 + abmi_v + \mu_h + \gamma)a_1|z_1(t)| \\
&\quad - (\epsilon abmp_h + abm(1 - p_h - i_h - r_h))a_1|z_3(t)| - \epsilon abmp_h a_1|z_4(t)| \\
&\le -(\pi + \epsilon abmi_v + \mu_h + \nu_1 + abmi_v + \mu_h + \gamma)a_1|z_1(t)| \\
&\quad - \frac{r_h}{i_h}\left(\epsilon abmp_h + abm(1 - p_h - i_h - r_h)\right) a_1 \frac{i_h}{r_h}|z_3(t)| \\
&\quad - \frac{\epsilon abmp_h a_1 r_h}{a_2 i_h}\frac{i_h}{r_h}a_2|z_4(t)|, \qquad (3.12)
\end{aligned}$$

$$\begin{aligned}
D_+|z_2(t)| &\le -(\pi + \epsilon abmi_v + \mu_h + \nu_1 + abmi_v + \lambda_v + aci_h)|z_2(t)| \\
&\quad - abmi_v|z_3(t)| + \pi|z_4(t)| \le -(\pi + \epsilon abmi_v + \mu_h + \nu_1 + abmi_v \\
&\quad + \lambda_v + aci_h)|z_2(t)| - abmi_v|z_3(t)| + \frac{\pi r_h}{a_2 i_h}\frac{i_h}{r_h}a_2|z_4(t)|, \qquad (3.13)
\end{aligned}$$

$$\begin{aligned}
D_+|z_3(t)| &\le -ac(1 - i_v)|z_1(t)| + r|z_2(t)| \\
&\quad - (\pi + \epsilon abmi_v + \mu_h + \nu_1 + \mu_h + \gamma + \lambda_v + aci_h)|z_3(t)| - (\pi - \rho\delta)|z_4(t)| \\
&\le -ac(1 - i_v)i_h a_1|z_1(t)| + r|z_2(t)| - (\pi + \epsilon abm\, i_v + \mu_h \\
&\quad + \nu_1 + \mu_h + \gamma + \lambda_v + aci_h)|z_3(t)| - \pi a_2|z_4(t)|, \qquad (3.14)
\end{aligned}$$

$$\begin{aligned}
D_+a_2|z_4(t)| &\le -abmi_v(1 - \epsilon)a_2|z_3(t)| - (abmi_v + \mu_h + \nu_1 + \lambda_v + aci_h) \\
&\quad \times a_2|z_4(t)| \le -abmi_v|z_3(t)| - (abmi_v + \mu_h + \nu_1 + \lambda_v + aci_h)a_2|z_4(t)|, \qquad (3.15)
\end{aligned}$$

where D_+ denotes the right-hand derivative with respect to time t. Set

$$w_1(t) = a_1|z_1(t)| + |z_2(t)|, \quad w_2(t) = \frac{i_h(t)}{r_h(t)}\left(|z_3(t)| + a_2|z_4(t)|\right).$$

Then from the equations (3.12) and (3.13) we have

$$\begin{aligned}
D_+|w_1(t)| &\le -(\pi + \epsilon abmi_v + \mu_h + \nu_1 + abmi_v + \mu_h + \gamma)w_1(t) \qquad (3.16) \\
&\quad - \frac{r_h}{i_h}\big(\epsilon abmp_h + abm(1 - p_h - i_h - r_h))a_1 - \frac{\epsilon abmp_h a_1 r_h}{i_h} + \frac{\pi r_h}{a_2 i_h}\big)w_2(t).
\end{aligned}$$

From equations (3.14) and (3.15) we derive

$$\begin{aligned}
D_+|w_2(t)| &= \left(\frac{i_h'}{i_h} - \frac{r_h'}{r_h}\right)w_2(t) + \frac{i_h}{r_h}D_+|(z_3(t)| + a_2|z_4(t)|) \\
&\le -\frac{ac(1 - i_v)i_h}{r_h}a_1|z_1(t)| + \frac{r i_h}{r_h}|z_2(t)| \\
&\quad + \left(\frac{i_h'}{i_h} - \frac{r_h'}{r_h} - (\pi + abmi_v + \mu_h + \nu_1 + \lambda_v + aci_h)\right)w_2(t)
\end{aligned}$$

$$\leq \left(-\frac{ac(1-i_v)i_h}{r_h}+\frac{ri_h}{r_h}\right)w_1(t)$$
$$+\left(\frac{i_h'}{i_h}-\frac{r_h'}{r_h}-(\pi+abmi_v+\mu_h+\nu_1+\lambda_v+aci_h)\right)w_2(t)$$
$$\leq \frac{ri_h}{r_h}w_1(t)+\left(\frac{i_h'}{i_h}-\frac{r_h'}{r_h}-(\pi+abmi_v+\mu_h+\nu_1+\lambda_v+aci_h)\right)w_2(t). \qquad (3.17)$$

Using the differential inequalities (3.16) and (3.17) we can show that

$$D_+V(t)\leq -\phi(t)V(t),$$

where $-\phi(t)=max\{h_1(t),h_2(t)\}$, and

$$h_1(t)=-(\pi+\epsilon abmi_v+\mu_h+\nu_1+abmi_v+\mu_h+\gamma)$$
$$-\frac{i_h}{r_h}\Big(\epsilon abmp_h+abm(1-p_h-i_h-r_h))-\frac{\epsilon abmp_ha_1r_h}{i_h}+\frac{\pi r_h}{a_2i_h}\Big)$$
$$\leq -(\pi+\epsilon abmi_v+\mu_h+\nu_1+abmi_v+\mu_h+\gamma)$$
$$-\frac{i_h}{r_h}\Big(\epsilon abmp_h+abm(1-p_h-i_h-r_h))-\frac{\epsilon abmp_ha_1r_h}{i_h}+\frac{\pi r_h}{a_2i_h}\Big),$$
$$h_2(t)=\left(\frac{ri_h}{r_h}+\frac{i_h'}{i_h}-\frac{r_h'}{r_h}-abmi_v-\mu_h-\nu_1-\lambda_v-aci_h\right)$$
$$\leq \left(\frac{i_h'}{i_h}+\frac{i_v'}{i_v}-\frac{aci_h}{i_v}\right).$$

Rewriting the equations of system (3.9) gives the following relations:

$$(\epsilon abmp_h+abm(1-p_h-i_h-r_h))\frac{i_v}{i_h}=\frac{i_h'}{i_h}+\nu_2+\mu_h+r+\delta,$$
$$\frac{ri_h}{r_h}=\frac{r_h'}{r_h}+\mu_h+\gamma,\quad \lambda_v+aci_h=\frac{aci_h}{i_v}-\frac{i_v'}{i_v},$$

so that we have

$$\max\{h_1(t),h_2(t)\}\leq\left(\frac{i_h'}{i_h}+\frac{i_v'}{i_v}-\frac{aci_h}{i_v}\right),$$
$$\int_0^t\max\{h_1(\tau),h_2(\tau)\}\,d\tau\leq[\log i_h(\tau)+\log i_v(\tau)]_0^t-\int_0^t\frac{aci_h(\tau)}{i_v(\tau)}\,d\tau$$
$$=2\log\epsilon_0-ac\epsilon_0t.$$

This implies that the linear system (3.9) is asymptotically stable and therefore the periodic solution is asymptotically orbitally stable.

4 Discussion

In this paper, a model that monitors the dynamics of malaria with interventions to control the spread of the disease has been proposed to understand its

effect on the disease transmission. The model considers a varying total human population that incorporates recruitment of new individuals in the susceptible class through either birth or immigration. Our model incorporates features that are effective to control the transmission of malaria. The model analysis revealed that the system has both a disease-free equilibrium and a unique endemic equilibrium. The prominent parameter in our model, the basic reproduction number, R_c, as a modified control intervention measure was computed. This was used to determine the stability of the disease-free steady state as well as the existence of the endemic equilibria. It was proved that under the condition that $R_c \leq 1$ the disease-free equilibrium E_0 is globally asymptotically stable, and no other equilibria exist. This means that after some period of time the disease will die out. After $R_c > 1$, the steady state E_0 loses its stability, and an endemic equilibrium point, E_1 appears. The control reproduction number is an important tool for effective disease management. The thresholds for control reproduction number and the basic production number in the absence of control strategies are compared. If $R_c \leq 1$, the disease can not persist in a community, hence R_c is a useful indication of the effort required to eliminate an infection.

It is also noted that $R_c \leq R_0$ which implies that increasing preventive and control measures in newborns and other people at high risk of malaria has a great effect on reduction of R_c. Thus, malaria can be eradicated out of the community by deployment of a combination of strategies such as effective mass drug administration and vector control that are of significance in its fight.

Appendix. Third Additive Compound Matrix

The third additive compound matrix $A^{[3]}$ for a 4×4 matrix $A = (a_{ij})$ is

$$A^{[3]} = \begin{bmatrix} a_{11}+a_{22}+a_{33} & a_{34} & -a_{24} & a_{14} \\ a_{43} & a_{11}+a_{22}+a_{44} & a_{23} & -a_{13} \\ -a_{42} & a_{32} & a_{11}+a_{33}+a_{44} & a_{12} \\ a_{41} & -a_{31} & a_{21} & a_{22}+a_{33}+a_{44} \end{bmatrix},$$

$$A^{[3]} = a_{11}+a_{22}+a_{33}$$

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