



Dynamical Behavior of a Malaria relapse Model with Insecticide Treated Nets (ITNs) as Protection Measure

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Abstract

Malaria is a tropical disease which is mainly spread by *plasmodium falciparum* which has been the principal enemy to the existence of mankind till date. In this paper a version of a malaria model incorporating the use of treated mosquito nets as a disease control strategy is proposed and then transformed into proportions, so as to assess the global impact of ITNs on the prevalence of malaria. Constructing a Lyapunov function using matrix-theoretic approach, a malaria-free equilibrium state is obtained, which is globally asymptotically stable if the control reproduction number, $R_m < 1$. This means that malaria can be controlled or eradicated under such a threshold quantity, R_m . On the other hand, a malaria-persistence equilibrium state exists which is globally stable when $R_m > 1$, using geometric theoretic method with Lozokii measure. Numerical experiments also indicate that prevalence of infection can be driven to zero provided that the proportion of susceptible humans using treated mosquito nets is above a certain threshold value.

Keywords: Malaria; mathematical model; basic reproduction number; Insecticide treated nets; relapse

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1. Introduction

Malaria is one of the oldest and most frequently occurring infectious diseases in humans. It is a parasitic vector borne disease that is endemic in many parts of the world. It is caused by eukaryotic parasites of the genus *Plasmodium*, affecting at least 300 million people globally and causing 1 – 1.5 million malaria related deaths per year. This disease in humans is due to infection

by one of the five species of genus *Plasmodium*, namely; *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale*, *Plasmodium malariae* and *Plasmodium knowlesi*. Malaria is transmitted to vertebrates by female genus *Anopheles* mosquitoes when they feed on blood meal (Balbir et al., 2004; Lawi et al., 2011; Ross, 1911).

Clinical symptoms such as high fever, sweats, chills and headache may develop within 7 days to 4 weeks after an infective mosquito bite. On average the incubation period of *Plasmodium falciparum* is about 12 days in humans and 10 days in mosquitoes. The period can be longer in other strains. The prevention and treatment of the disease have been investigated for hundreds of years, and methods like the use of anti-malarial drugs, fumigation of mosquito breeding reservoirs as well as monitoring of sanitary measures have been employed without yielding maximum results (Ross, 1911). Repeated mass treatment is expensive both with respect to drug purchase, drug resistance and the use of trained personnel (Bradley, 1996). However, the introduction of insecticide treated nets (ITNs), particularly the long lasting insecticidal nets in recent times, is the preferred tool for reducing mosquito bites and alleviating disease burden. ITNs were developed in the 1980s for malaria prevention. They are estimated to be twice as effective as untreated nets and offer greater than 70% protection, see Laith et al. (2011); and Okumu et al. (2013).

A number of mathematical models have been formulated to study the transmission dynamics of the disease, see for example Ngwa and Shu (2000); Tunwiine et al. (2007); Labadin et al. (2009); Agosto et al. (2013); Okumu et al. (2013); Alhassan et al. (2017); Mbogo et al. (2018); Koutou et al. (2018); and Bala and Gimba (2019). None of these models considers the use of ITNs. Tunwiine et al. (2007) proposed a deterministic model describing the dynamics of malaria transmission with temporary immunity for the human host and mosquito vector. They subdivided the human population into three exclusive classes of susceptible, infected and partially immune, while the mosquito vector is divided into two classes of susceptible and infected. Their main aim was to study the role of partial immunity on the dynamics of malaria in the absence of any control measure. They obtained a basic reproduction number, R_0 , that was independent of the rate of loss of immunity, and showed that it is the threshold parameter between extension and persistence of the diseases. The authors employed the Lyapunov theory and the theory of compound matrices and proved that the disease free equilibrium and endemic equilibrium states are globally asymptotically stable when R_0 is less than one and greater than one, respectively. However, their study lacks the numerical support for the established stability results and any control strategy to prevent and contain further spread. They concluded that since temporary immunity wanes over time, and due to reinvasion of the human population by mosquitoes, the use of treatment and a class of treated insecticides bed nets users to limit mosquito bites be considered for future work.

Alhassan et al. (2017) studied the optimal control strategies and cost effectiveness of malaria with respect to treatment, indoor and spray treated bed net. Similarly Bala and Gimba (2019) addresses the global sensitivity analysis of a dual strain malaria model to take account of the impact of bed nets, drug treatment and their efficacies. The model was formulated based on mass action with mosquito biting rate function depending on the proportion of ITNs usage and its efficacy. Their results reiterated that people treated of the disease and using ITNs have 95% success rate with less than 5% treatment failure. As well known, malaria infection is more of

frequency dependent as such this model may not be a true reflection of malaria transmission dynamics. Even though, some of the works (mentioned above) examined the impact of bed nets on malaria transmission, the use of ITNs was not addressed elaborately enough to cater for the concerns raised in Tumwiine et al. (2017) and Osman and Adu (2017).

In this paper, our main objective is to extend the model of Tumwiine et al. (2007) which was without any protection measure by including a separate class of ITNs users and the effect of drugs resistance on the epidemiology of the disease. Other contributions are to explore some of the global stability results and methods and to provide the numerical support on the embedded control effort for malaria. The study is organized as follows: Section 1 introduces the study, and the model equations are formulated in Section 2. The stability analysis of the model is presented in Section 3 while numerical experiments are presented in Section 4. Section 5 discusses the results obtained, and concluding remarks are presented in Section 6.

2. Model Formulation

The present model is a variant of the model by Tumwiine et al. (2007) for the transmission dynamics of malaria in a human host and malaria mosquito vector with temporary immunity. For clarity of presentation we list the state variables and parameters and the basic model equations as follows:

Table 1: The state variables of the basic Model

Variables	Description
$S_H(t)$	The number of susceptible humans at time t
$I_H(t)$	The number of infected humans at time t
$R_H(t)$	The number of partially immune humans at time t
$S_V(t)$	The number of susceptible mosquito vectors at time t
$I_V(t)$	The number of infected mosquito vectors at time t
$N_H(t)$	Total number of human population size at time t
$N_V(t)$	Total mosquito population size at time t

Table 2: The parameters of the basic Model

Parameters	Description
a	The average biting rate on man by a single mosquito
b	The probability that a susceptible human becomes infectious when bitten by an infectious mosquito
c	The probability that a mosquito becomes infectious
γ	The rate of loss of immunity in human hosts
r	The rate at which human hosts acquire immunity
δ	The death rate of infected human hosts due to the disease
ν	The rate of recovery of human hosts from the disease
λ_h	The natural birth rate of humans
λ_v	The natural birth rate of the mosquitoes
μ_h	The natural death rate of humans
μ_v	The natural death rate of the mosquitoes

2.1. The Basic Model

Based on their assumptions and the above listed model state variables and parameters, Tumwiine et al. (2007) presented their model equations as follows:

$$\frac{dS_H}{dt} = \lambda_h N_H - \frac{abS_H I_V}{N_H} + v I_H + \gamma R_H - \mu_h S_H, \quad (1)$$

$$\frac{dI_H}{dt} = \frac{abS_H I_V}{N_H} - (v + r + \delta + \mu_h) I_H, \quad (2)$$

$$\frac{dR_H}{dt} = r I_H - (\gamma + \mu_h) R_H, \quad (3)$$

$$\frac{dS_V}{dt} = \lambda_v N_V - \frac{acS_V I_H}{N_H} - \mu_v S_V, \quad (4)$$

$$\frac{dI_V}{dt} = \frac{acS_V I_H}{N_H} - \mu_v I_V, \quad (5)$$

where

$$N_H = S_H + I_H + R_H, \quad (6)$$

$$N_V = S_V + I_V. \quad (7)$$

2.2. Model Incorporating use of ITNs

We now incorporate the use of treated mosquito nets, as a control strategy, into the model by Tunwiine et al. (2007) and also introduce the phenomenon of relapse as done in Hao and Qiu (2014). Relapse is the situation whereby a partially immune human loses immunity and becomes fully infected. Here, we shall denote by σ , the rate of relapse. We only consider the situation where susceptible humans use treated nets, since this is considered as a preventive measure for the susceptible individuals. Infected individuals can be treated using available drugs. Let T_H be the number of susceptible humans using treated nets at time t and n_h be the rate at which susceptible humans use treated nets. Thus the term $n_h S_H$ is subtracted from the right hand side of equation (1), representing the number of susceptible humans using treated nets. This results into an additional equation (9) representing the rate of change of susceptible human population that uses treated nets per unit time. Meanwhile σ which is the relapse rate is removed from (11) and added to (10). The rate, n_0 , at which susceptible people stop using ITNs is removed from (9) and added to (8). The death rates, δ_v , δ_h of mosquitoes and humans due to human activities and infection respectively are also incorporated in (13) and (10). The schematic flow diagram illustrating the dynamics of the system is represented in Figure 1.

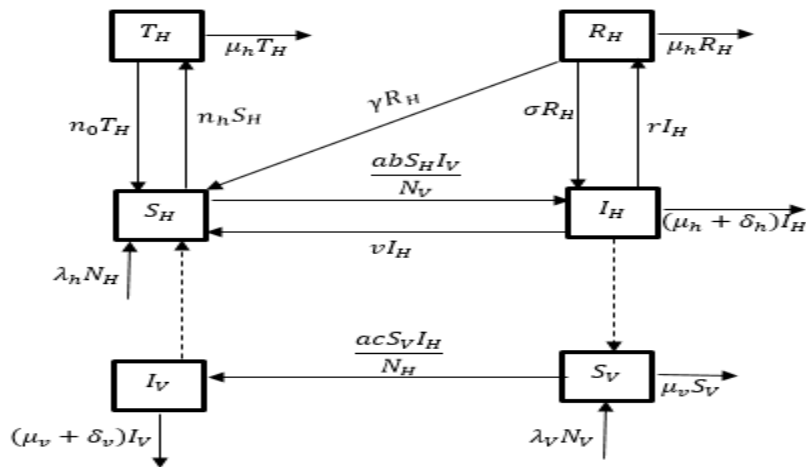


Figure 1. Schematic flow diagram of Malaria transmission dynamics with ITNs and relapse

From the flow-diagram, we obtain the following equations for the dynamics of a malaria relapse model with ITNs as a protective measure

$$\frac{dS_H}{dt} = \lambda_h N_H - \frac{abS_H I_V}{N_V} + vI_H + \gamma R_H - (\mu_h + n_h)S_H + n_0 T_H, \tag{8}$$

$$\frac{dT_H}{dt} = n_h S_H - (n_0 + \mu_h)T_H, \tag{9}$$

$$\frac{dI_H}{dt} = \frac{abS_H I_V}{N_V} - (v + r + \delta_h + \mu_h)I_H + \sigma R_H, \tag{10}$$

$$\frac{dR_H}{dt} = rI_H - (\gamma + \mu_h + \sigma)R_H, \tag{11}$$

$$\frac{dS_V}{dt} = \lambda_v N_V - \frac{acS_V I_H}{N_H} - \mu_v S_V, \tag{12}$$

$$\frac{dI_V}{dt} = \frac{acS_V I_H}{N_H} - (\mu_v + \delta_v)I_V, \tag{13}$$

where

$$N_H = S_H + I_H + R_H + T_H, \tag{14}$$

$$N_v = S_V + I_V. \tag{15}$$

The present model also uses the frequency-dependent mode of transmission as can be seen in the second term in equation (8) and the first term in equation (10). However, the assumption that $m = \frac{N_v}{N_H}$ in the basic model is unnecessary at this point. Adding equations (8) – (11) gives

$$\frac{dN_H}{dt} = (\lambda_h - \mu_h)N_H - \delta_h I_H. \tag{16}$$

Similarly, adding equations (12) and (13) for the vector components gives

$$\frac{dN_V}{dt} = (\lambda_v - \mu_v)N_V - \delta_v I_V. \quad (17)$$

Hence, equations (8) – (13), (16) – (17) constitute the present model.

2.3. Equations in Proportions

In order to define the prevalence of infection, we work with proportions rather than the actual population by scaling each class population by the total population size. As done for example in Kimbir et al. (2006), we let

$$s_h = \frac{S_H}{N_H}, \quad i_h = \frac{I_H}{N_H}, \quad r_h = \frac{R_H}{N_H}, \quad t_h = \frac{T_H}{N_H}, \quad s_v = \frac{S_V}{N_V} \text{ and } i_v = \frac{I_V}{N_V}. \quad (18)$$

Then, the time-derivative of the scaling system (18) is

$$\frac{dx}{dt} = \frac{1}{N} \left(\frac{dX}{dt} - x \frac{dN}{dt} \right), \quad (19)$$

where

$$x = (s_h, t_h, i_h, r_h, s_v, i_v), \quad N = (N_H, N_V) \text{ and } X = (S_H, T_H, I_H, R_H, S_V, I_V).$$

Implementing (19) on human and mosquito components gives

$$\begin{aligned} \frac{ds_h}{dt} &= \lambda_h - (\lambda_h + n_h)s_h - abs_h i_v + vi_h + \gamma r_h + n_0 t_h + \delta_h s_h i_h, \\ \frac{dt_h}{dt} &= n_h s_h + \delta_h i_h t_h - (n_0 + \lambda_h) t_h, \\ \frac{di_h}{dt} &= abs_h i_v - (v + r + \delta_h + \lambda_h) i_h + \sigma r_h + \delta_h i_h^2, \\ \frac{dr_h}{dt} &= r i_h - (\sigma + \gamma + \lambda_h) r_h + \delta_h i_h r_h, \\ \frac{ds_v}{dt} &= \lambda_v (1 - s_v) - ac i_h s_v + \delta_v s_v i_v, \\ \frac{di_v}{dt} &= ac s_v i_h - (\lambda_v + \delta_v) i_v + \delta_v i_v^2. \end{aligned}$$

Since $t_h = 1 - s_h - i_h - r_h$ and $s_v = 1 - i_v$, the governing equations become

$$\left. \begin{aligned} \frac{ds_h}{dt} &= \lambda_h - (\lambda_h + n_h)s_h - abs_h i_v + vi_h + \gamma r_h + n_0(1 - s_h - i_h - r_h) + \delta_h s_h i_h, \\ \frac{di_h}{dt} &= abs_h i_v - (v + r + \delta_h + \lambda_h) i_h + \sigma r_h + \delta_h i_h^2, \\ \frac{dr_h}{dt} &= r i_h - (\sigma + \gamma + \lambda_h) r_h + \delta_h i_h r_h, \\ \frac{di_v}{dt} &= ac i_h (1 - i_v) - (\lambda_v + \delta_v) i_v + \delta_v i_v^2, \end{aligned} \right\} \quad (20)$$

which can be studied in a closed, positively invariant set

$$\Sigma = \{(s_h, i_h, r_h, i_v) \in \mathfrak{R}_+^4 : 0 \leq i_v, 0 \leq i_h, s_h + i_h + r_h \leq 1, i_v \leq 1\},$$

where \mathfrak{R}_+^4 denotes the non-negative cone with its lower dimensional faces.

3. Model Analysis

3.1. Basic reproduction number with relapse.

The unique malaria-free equilibrium, MFE of the model (20) is

$$M_0 = (s_{h0}, i_{h0}, r_{h0}, i_{v0}) = \left(\frac{\lambda_h + n_0}{\lambda_h + n_h + n_0}, 0, 0, 0 \right).$$

Here the standard method of next generation matrix, as outlined in Van den Driessche and Watmough (2002), is used to calculate malaria reproduction number, R_m . In the analysis that follows, we separate the infected state (i_h, r_h, i_v) from the uninfected state (s_h) . Let \mathcal{F} and \mathcal{V} denote the vectors standing for the new and transported cases into the infected states, respectively.

Therefore,

$$\mathcal{F} = \begin{pmatrix} abs_h i_v \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

and

$$\mathcal{V} = \begin{pmatrix} (v + r + \delta_h + \lambda_h)i_h - \sigma r_h - \delta_h i_h^2 \\ (\sigma + \gamma + \lambda_h)r_h - r i_h - \delta_h i_h r_h \\ (\lambda_v + \delta_v)i_v - ac i_h(1 - i_v) - \delta_v i_v^2 \\ (\lambda_h + n_h)s_h + abs_h i_v - \lambda_h - v i_h - \gamma r_h - n_0(1 - s_h - i_h - r_h) - \delta_h s_h i_h \end{pmatrix}.$$

The Jacobian matrices F of \mathcal{F} and V of \mathcal{V} at M_0 are:

$$F = \begin{pmatrix} 0 & 0 & abs_{h0} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad \text{and} \quad V = \begin{pmatrix} H_T & -\sigma & 0 \\ -r & H_R & 0 \\ -ac & 0 & H_V \end{pmatrix}, \text{ respectively,}$$

where

$$H_T = v + r + \delta_h + \lambda_h, H_R = \sigma + \gamma + \lambda_h \text{ and } H_V = \lambda_v + \delta_v.$$

Thus, from Van den Driessche and Watmough (2002), R_m is computed as the spectral radius of the positive matrix FV^{-1} , where

$$FV^{-1} = \begin{pmatrix} \frac{H_R a^2 b c s_{h0}}{H_V \phi} & \frac{\sigma a^2 b c s_{h0}}{H_V \phi} & \frac{a b s_{h0}}{H_V} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

such that

$$R_m = \frac{a^2 b c s_{h0}}{\phi} \frac{H_R}{H_V}, \quad (21)$$

with $\phi = \sigma(v + \delta_h + \lambda_h) + (\gamma + \lambda_h)H_T$.

In the absence of relapse and ITNs, (21) reduces to the basic reproduction number,

$$R_0 = \frac{a^2 b c}{H_V H_T},$$

whereas in Tumwiine et al. (2007),

$$R_0 = m \frac{a^2 b c}{H_V H_T},$$

where $m > 1$.

However, in the presence of relapse without ITNs, (21) reduces to

$$R_{mr} = \frac{a^2 b c H_R}{\phi H_V}.$$

For the scenario where ITNs is introduced with no effect of relapse, then, (21) turns to be

$$R_m = \frac{a^2 b c s_{h0}}{H_V H_T}. \quad (22)$$

By Theorem 2 in Van den Driessche and Watmough (2002) the following result can be accomplished.

Lemma 1.

The malaria-free equilibrium state is locally asymptotically stable provided $R_m < 1$. So, if $R_m < 1$, then, from (22) we get

$$n_h > (\lambda_h + n_0)(R_0 - 1), \quad (23)$$

which is the minimum proportion of human susceptibles that should use ITNs to limit the spread of the disease at the point $R_0 > 1$.

3.2. Global dynamics of the MFE

In this section, we construct a Lyapunov function to study the stability of MFE M_0 globally in Theorem 1. Following Shuai and Van Den Driessche (2013), a matrix-theoretic method is used to guide the construction. Set

$$f(x, y) = (F - V)x - \mathcal{F}(x, y) + \mathcal{V}(x, y),$$

with the disease states expressed as

$$\frac{dx}{dt} = (F - V)x - f(x, y), \quad (24)$$

where F and V remain as above,

$$f(x, y)|_{(\delta_h=\delta_v=0)} = \begin{pmatrix} ab(s_{h0} - s_h)i_v \\ 0 \\ aci_h i_v \end{pmatrix}, \text{ and } y = s_h.$$

Note that the left eigenvector of the nonnegative matrix, $V^{-1}F$, is $\omega^T = \left(\frac{\Phi R_m}{abs_{h0}H_R}, 0, 1\right)$.

Clearly, $f(x, y) \geq 0$ in Σ . If $s_h \leq s_{h0}$ and $f(0, y_0) = 0$. It is also obvious that $F, V^{-1} \geq 0$. By the virtue of Theorem 2.1 in (Shuai and Van Den Driessche (2013)), $L = \omega^T V^{-1}x$ is the Lyapunov function of system (20)

$$L = \frac{R_m}{abs_{h0}H_R} i_h + \frac{\sigma R_m}{abs_{h0}H_R} r_h + \frac{i_v}{H_V}. \quad (25)$$

Theorem 1.

The MFE, M_0 , of system (20)| $\delta_h=\delta_v=0$ is globally asymptotically stable if $R_m \leq 1$ in Σ .

Proof:

Assume (25) is the Lyapunov function of system (20) on Σ with $f(x, y) \geq 0$ and $R_m < 1$. Then, differentiating L along solutions of (20) yields

$$\begin{aligned} \frac{dL}{dt} &= \omega^T V^{-1} \frac{dx}{dt} = \omega^T V^{-1} (F - V)x - \omega^T V^{-1} f(x, y), \\ &= (R_m - 1) \omega^T x - \omega^T V^{-1} f(x, y), \\ &= (R_m - 1) \left(i_v + \frac{\phi R_m}{ab s_{h0} H_R} i_h \right) - \left(R_m (1 + R_m) \left(1 - \frac{s_h}{s_{h0}} \right) + \frac{ac}{H_V} i_h \right) i_v. \end{aligned}$$

If $\frac{dL}{dt} \leq 0$, if $R_m \leq 1$ and $s_h \leq s_{h0}$ and if $R_m = 1$, then

$$\frac{dL}{dt} = 0, \text{ if and only if } i_h = i_v = 0 \text{ and } s_{h0} = s_h.$$

Thus, every solution of the model equations (20) converges to the largest compact invariant set $\{M_0\}$ which is the disease-free state. By LaSalle invariant principle (La Salle, 1976), M_0 is globally asymptotically stable in Σ if $R_m \leq 1$.

3.3. Existence and local stability of malaria-persistence equilibrium state of the model

The usual approach to finding the existence of disease endemic equilibrium state $M_{PES} = (s_{h*}, i_{h*}, r_{h*}, i_{v*})$ is by setting the derivatives of the system (20) to zero as follows:

$$\left. \begin{aligned} 0 &= \lambda_h - (\lambda_h + n_h) s_{h*} - ab s_{h*} i_{v*} + v i_{h*} + \gamma r_{h*} + n_0 (1 - s_{h*} - i_{h*} - r_{h*}) + \delta_h s_{h*} i_{h*}, \\ 0 &= ab s_{h*} i_{v*} - (v + r + \delta_h + \lambda_h) i_{h*} + \sigma r_{h*} + \delta_h i_{h*}^2, \\ 0 &= r i_{h*} - (\sigma + \gamma + \lambda_h) r_{h*} + \delta_h i_{h*} r_{h*}, \\ 0 &= ac i_{h*} (1 - i_{v*}) - (\lambda_v + \delta_v) i_{v*} + \delta_v i_{v*}^2. \end{aligned} \right\} \quad (26)$$

As in Tumwiine et al. (2007), we add up the right hand side of above system to have

$$(\lambda_h + n_0 - \delta_h i_{h*}) (1 - s_{h*} - i_{h*} - r_{h*}) - n_h s_{h*} + ac i_{h*} (1 - i_{v*}) - (\lambda_v + \delta_v) i_{v*} + \delta_v i_{v*}^2 = 0.$$

In view of the last equation in system (26), the above equations reduces to

$$(\lambda_h + n_0 - \delta_h i_{h*}) (1 - s_{h*} - i_{h*} - r_{h*}) = n_h s_{h*}.$$

Since $(s_{h*} + i_{h*} + r_{h*}) < 1$, and from $\lambda_h + n_0 > \delta_h i_{h*}$, we have that $i_{h*} < \left(\frac{\lambda_h + n_0}{\delta_h} \right)$. Therefore, the malaria persistence equilibrium state exists, with i_{h*} lying in the interval

$$\left(0, \min \left\{ 1, \frac{\lambda_h + n_0}{\delta_h} \right\} \right).$$

As in Tumwiine et al. (2007), $\delta_h < \lambda_h + n_0$ has a great impact on the endemic nature of the disease. It indicates that malaria induced death rate should be less than that at which the pool of susceptible humans is refilled by birth and protection failure rate of ITNs.

Now, solving the system (26) using Maple package yields

$$M_{PES} = (s_{h*}, i_{h*}, r_{h*}, i_{v*}), \tag{27}$$

where

$$s_{h*} = \frac{(H_R - \delta_h i_{h*})[q_1 + (q_3 - H_T)i_{h*} + \delta_h i_{h*}^2] + i_{h*}(q_4 + \sigma)r}{(q_2 - \delta_h i_{h*})(H_R - \delta_h i_{h*})},$$

$$r_{h*} = \frac{r i_{h*}}{H_R - \delta_h i_{h*}},$$

$$i_{v*} = \left(\frac{(H_T - \delta_h i_{h*})(H_R - \delta_h i_{h*}) - \sigma r}{\varphi(H_R - \delta_h i_{h*})s_{h*}} \right) i_{h*},$$

and i_{h*} is the root of the polynomial equation

$$p(i_{h*}) = p_7 i_{h*}^7 + p_6 i_{h*}^6 + p_5 i_{h*}^5 + p_4 i_{h*}^4 + p_3 i_{h*}^3 + p_2 i_{h*}^2 + p_1 i_{h*} + p_0 = 0,$$

with

$$p_7 = \delta_h^3(\delta_h \delta_v - \varphi ac),$$

$$p_6 = \delta_h^4 q_2 (\varphi - \delta_h) + 2\delta_h^4 (H_T + H_R) (\varphi - \delta_h \delta_v) + \varphi \delta_h^4 (\varphi - (q_3 + H_V \delta_h)),$$

$$p_5 = \delta_h^3 q_2 q_5 \varphi \left(q_3 - H_T + \frac{3H_R}{q_2} (q_3 - H_T - q_2) - \delta_h s_{ho} \right) + \delta_v \delta_h^4 (q_2^2 + 2(3H_R + 4q_2)) + \delta_h^3 q_5 \varphi r (\sigma + q_4) + \delta_h^3 q_5 \varphi^2 (2(q_3 - H_T) - H_R(2 + 3H_R)) + \varphi H_V \delta_h^4 (H_T + 3H_R + q_2 - q_3),$$

$$p_4 = 4\delta_h^2 q_5 \varphi^2 H_V H_T \left(1 - \frac{q_3}{H_T} - \frac{\delta_h \delta_v}{q_5 \varphi^2 H_T} (H_R^2 + q_2^2) \right) - 12q_2 \delta_h^3 H_R^2 \delta_v + \delta_h^2 q_5 \varphi H_R^3 + \delta_h^3 q_1 q_2 q_5 \varphi + 3H_V H_R \varphi \delta_h^3 (q_3 - (H_T + H_R + q_2)) + \frac{q_5 H_R}{H_V \delta_h} (q_2 - q_3 - H_T) + \frac{q_5 q_2}{H_V \delta_h} (H_T - q_3) + \frac{q_5 q_1}{H_V} + 2q_5 \varphi \delta_h^2 (q_1 \delta_h \varphi - r(\sigma + q_4)(H_R + \varphi) - \varphi H_T q_3) + \delta_h^2 q_5 \varphi^2 (H_T^2 + H_R^2 + q_3^2) + r \varphi \delta_h^2 (\sigma + q_4) (\delta_h H_V - q_2 q_5) + \delta_h^3 q_2 H_V \varphi (q_3 - \delta_h s_{ho} - H_T),$$

$$p_3 = H_R^3 H_V \varphi \delta_h^2 + H_R^2 \delta_v q_2 \delta_h^2 (8H_R + 6q_2) + \delta_v H_R^4 q_2^2 + q_1 q_2 H_V \varphi \delta_h^3 + 2\varphi \delta_h r (\sigma + q_4) (q_2 q_5 H_R + q_5 H_T \varphi + q_5 H_R \varphi - q_3 \varphi q_5 - \delta_h H_R H_V) + \varphi r \delta_h (\sigma + q_4) (H_R^2 q_5 - \delta_h H_V q_2) + 2q_5 \varphi^2 \delta_h (q_3 - H_T) (q_1 \delta_h + H_R^2) + 3q_5 \varphi H_R^2 q_3 \delta_h (q_3 - H_T) + 3\varphi H_R H_V \delta_h^2 q_2 (H_T - q_3) + 3\varphi H_R^2 H_V \delta_h^2 (H_T - q_3 + q_2 - \frac{q_1}{H_V}) - 2q_5 \varphi^2 H_R \delta_h (q_3^2 + H_R^2) + 3\varphi H_R \delta_h^2 q_1 (H_V \delta_h - q_2 q_5) + 4\delta_h q_5 \varphi^2 H_R (H_T q_3 - \delta_h q_1) + q_5 \varphi H_R^3 \delta_h (q_3 - H_T - q_2),$$

$$p_2 = q_5 \varphi^2 r^2 (\sigma^2 + q_4^2) + q_5 \varphi^2 H_R^2 (H_4^2 + q_3^2) + q_5 \varphi^2 q_1^2 \delta_h^2 - q_5 \varphi H_R^2 q_2 r (\sigma + q_4)$$

$$\begin{aligned}
& +q_5\varphi H_R^3 q_2 \left(H_T - q_3 + \delta_h \frac{q_1}{q_2} \right) + \varphi H_R^3 H_v \delta_h (q_3 - H_T - q_2) + 2q_5\varphi^2 r H_R q_4 (q_3 - H_T) \\
& + 3\varphi H_R^2 H_v q_2 \delta_h (q_3 - H_T) + 4q_5\varphi^2 H_R q_1 \delta_h \left(H_T - q_3 - \frac{H_R^2 q_2^2}{q_1 \varphi^2} \delta_v \right) - 2q_5\varphi^2 r q_1 \delta_h (\sigma \\
& + q_4) + 2q_5\varphi^2 H_R^2 (q_1 \delta_h - H_T q_3) + 2q_5\varphi^2 r^2 \sigma q_4 + 2r\varphi H_R H_v \delta_h q_2 (\sigma + q_4) \\
& - 2H_R^4 \delta_h q_2 \delta_v + 2q_5\varphi^2 r \sigma H_R (q_3 - H_T) + r\varphi H_R^2 H_v \delta_h (\sigma + q_4) + 3\varphi H_R H_v q_1 q_2 \left(\frac{H_R}{H_v} q_5 \right. \\
& \left. - \delta_h - \frac{H_R \delta_h}{q_2} \right),
\end{aligned}$$

$$\begin{aligned}
p_1 = & q_2 H_v \varphi H_R^2 (3q_1 \delta_h - r(\sigma + q_4)) + \delta_v H_R^4 q_2^2 + q_2 H_v H_T \varphi H_R^3 \left(1 - \frac{q_3}{H_T} - \frac{q_5 q_1}{H_v H_T} + \frac{\delta_h}{H_T} S_{ho} \right) \\
& + 2\delta_h^2 q_5 \varphi^2 H_R q_1 (H_R (q_3 - H_T) + r(\sigma + q_4) - q_1 \delta_h),
\end{aligned}$$

$$p_0 = q_1 q_2 H_R H_v ac \left(r\sigma + H_R H_T \left(\frac{\phi}{H_R} R_m - 1 \right) \right),$$

and

$$\varphi = ab, q_1 = \lambda_h + n_0, q_2 = \lambda_h + n_h + n_0, q_3 = v - n_0, q_4 = \gamma - n_0, q_5 = ac.$$

3.4. Local stability of M_{PES}

The Jacobian matrix of the system (20) at the disease state is

$$J_f = \begin{pmatrix} -(\zeta_1 - \delta_h i_h) & q_2 + \delta_h s_h & q_3 & -\varphi s_h \\ \varphi i_v & -(H_T - 2\delta_h i_h) & \sigma & \varphi s_h \\ 0 & r + \delta_h r_h & -(H_R - 2\delta_h i_h) & 0 \\ 0 & ac(1 - i_v) & 0 & -(\zeta_2 - 2\delta_v i_v) \end{pmatrix}, \quad (28)$$

where $\zeta_1 = q_1 + \varphi i_v$ and $\zeta_2 = H_v + ac i_h$, with the partition matrix taking the form

$$J_f = \begin{pmatrix} J_{11} & J_{12} \\ J_{21} & J_{22} \end{pmatrix},$$

where

$$J_{11} = \begin{pmatrix} -(\zeta_1 - \delta_h i_h) & q_2 + \delta_h s_h \\ \varphi i_v & -(H_T - 2\delta_h i_h) \end{pmatrix}, \quad J_{12} = \begin{pmatrix} q_3 & -\varphi s_h \\ \sigma & \varphi s_h \end{pmatrix},$$

$$J_{21} = \begin{pmatrix} 0 & r + \delta_h r_h \\ 0 & ac(1 - i_v) \end{pmatrix}, \quad J_{22} = \begin{pmatrix} -(H_R - 2\delta_h i_h) & 0 \\ 0 & -(\zeta_2 - 2\delta_v i_v) \end{pmatrix}.$$

The M_{PES} is locally asymptotically stable if J_{11} and J_{22} have negative real parts. The eigenvalues of J_{22} are $-(H_R - 2\delta_h i_h)$ and $-(\zeta_2 - 2\delta_v i_v)$, and that of J_{11} are:

$$\lambda_i = \frac{-(m_1+m_2) \pm \sqrt{(m_1-m_2)^2 + 4n_1n_2}}{2}, i = 3, 4,$$

with $m_1 = (\varsigma_1 - \delta_h i_h), m_2 = (H_T - 2\delta_h i_h), n_1 = q_2 + \delta_h s_h$ and $n_2 = \varphi i_v$.

Therefore, since all the roots are real and negative, the persistent endemic equilibrium is locally asymptotically stable for $R_m > 1$.

3.5. Global dynamics of M_{PES}

In this section, we adopt the result on geometric theoretic approach to prove the global stability of M_{PES} . As given in (Ozaire et al., 2016), the Lozinskii measure for an $n \times n$ matrix B is defined as $\bar{\mu}(B) = \inf\{\zeta: D_+ \|z\| \leq \zeta \|z\| \text{ for all solutions of } \dot{z} = Bz\}$, where D_+ is the right hand derivative. The unique endemic equilibrium is globally asymptotically stable if there exist a norm on \mathbb{R}^6 satisfying the condition $\bar{\mu}(B) < 0$ provided that $R_m > 1$ for all $x \in \Sigma$.

Theorem 2.

The unique M_{PES} of the model (20) is globally asymptotically stable if and only if $R_m > 1$.

Proof:

The second additive compound matrix, see Buonomo and Lacitignola (2010); Ozaire et al. (2016) is given by

$$J_f^{[2]} = \begin{pmatrix} \tau_{11} & \sigma & \varphi s_h & -q_3 & \varphi s_h & 0 \\ r + \delta_h r_h & \tau_{22} & 0 & q_2 + \delta_h s_h & 0 & \varphi s_h \\ ac(1 - i_v) & 0 & \tau_{33} & 0 & q_2 + \delta_h s_{h*} & q_3 \\ 0 & \varphi i_v & 0 & \tau_{44} & 0 & -\varphi s_h \\ 0 & 0 & \varphi i_v & 0 & \tau_{55} & \sigma \\ 0 & 0 & 0 & -ac(1 - i_v) & r + \delta_h r_h & \tau_{66} \end{pmatrix},$$

having

$$\begin{aligned} \tau_{11} &= -(\varsigma_1 + H_T - 3\delta_h i_h), \tau_{22} = -(\varsigma_1 + H_R - 3\delta_h i_h), \\ \tau_{33} &= -(\varsigma_1 + \varsigma_2 - \delta_h i_h - 2\delta_v i_v), \tau_{44} = -(H_T + H_R - 4\delta_h i_h), \\ \tau_{55} &= -(\varsigma_2 + H_T - 2\delta_h i_h - 2\delta_v i_v), \tau_{66} = -(\varsigma_2 + H_R - 2\delta_h i_h - 2\delta_v i_v). \end{aligned}$$

Let $p = \text{diag} \left\{ 1, \frac{i_h}{i_v}, \frac{i_h}{i_v}, \frac{i_h}{i_v}, \frac{i_h}{i_v}, \frac{i_h}{i_v} \right\}$, then, we have

$$p_f = \text{diag} \left\{ 0, \frac{i_h'}{i_v} - \frac{i_h i_v'}{i_v^2}, \frac{i_h'}{i_v} - \frac{i_h i_v'}{i_v^2}, \frac{i_h'}{i_v} - \frac{i_h i_v'}{i_v^2}, \frac{i_h'}{i_v} - \frac{i_h i_v'}{i_v^2}, \frac{i_h'}{i_v} - \frac{i_h i_v'}{i_v^2} \right\},$$

and

$$B = p_f p^{-1} + p]_f^{[2]} p^{-1} = \begin{pmatrix} B_{11} & \sigma \frac{i_v}{i_h} & \varphi S_h \frac{i_v}{i_h} & -q_3 \frac{i_v}{i_h} & \varphi S_h \frac{i_v}{i_h} & 0 \\ (r + \delta_h r_h) \frac{i_h}{i_v} & B_{22} & 0 & q_2 + \delta_h S_h & 0 & \varphi S_h \\ ac(1 - i_v) \frac{i_h}{i_v} & 0 & B_{33} & 0 & q_2 + \delta_h S_h & q_3 \\ 0 & \varphi i_v & 0 & B_{44} & 0 & -\varphi S_h \\ 0 & 0 & \varphi i_v & 0 & B_{55} & \sigma \\ 0 & 0 & 0 & -ac(1 - i_v) & r + \delta_h r_h & B_{66} \end{pmatrix},$$

where $B_{11} = \tau_{11}$, $B_{jj} = \frac{i_h'}{i_h} - \frac{i_v'}{i_v} + \tau_{jj}$, $j = 2, 3, \dots, 6$.

Let $z = (z_1, z_2, z_3, z_4, z_5, z_6)^T$ be the solution of the linear system $\dot{z}(t) = Bz$, where

$$\dot{z}_1 = B_{11}z_1 + \sigma \frac{i_v}{i_h} z_2 + \varphi S_h \frac{i_v}{i_h} z_3 - q_3 \frac{i_v}{i_h} z_4 + \varphi S_h \frac{i_v}{i_h} z_5,$$

$$\dot{z}_2 = r \frac{i_h}{i_v} z_1 + B_{22}z_2 + (q_2 + \delta_h S_h)z_4 + \varphi S_h z_6,$$

$$\dot{z}_3 = ac(1 - i_v) \frac{i_h}{i_v} z_1 + B_{33}z_3 + (q_2 + \delta_h S_h)z_5 + q_3 z_6,$$

$$\dot{z}_4 = \varphi i_v z_2 + B_{44}z_4 - \varphi S_h z_6,$$

$$\dot{z}_5 = \varphi i_v z_3 + B_{55}z_5 + \sigma z_6,$$

$$\dot{z}_6 = -ac(1 - i_v)z_4 + rz_5 + B_{66}z_6.$$

Thus, taking into account that;

$$\frac{i_h'}{i_h} = \varphi S_h \frac{i_v}{i_h} - (v + r + \lambda_h + \delta_h) + \sigma \frac{r_h}{i_h} + \delta_h i_h,$$

$$\frac{i_v'}{i_v} = ac(1 - i_v) \frac{i_h}{i_v} - (\lambda_v + \delta_v) + \delta_v i_v,$$

we have

$$B_{22} = \lambda_v - (q_1 + H_T + H_R) - \varphi \left(1 - \frac{S_h}{i_h}\right) i_v + \sigma \frac{r_h}{i_h} + 4\delta_h i_h + \left(\delta_v - ac \frac{i_h}{i_v}\right) (1 - i_v),$$

$$B_{33} = -(q_1 + H_T) + \left\{ \delta_v - \varphi \left(1 - \frac{S_h}{i_h}\right) \right\} i_v + \sigma \frac{r_h}{i_h} + i_h \left(2\delta_h - \frac{ac}{i_v}\right),$$

$$B_{44} = \lambda_v - (2H_T + H_R) + \varphi \frac{S_h}{i_h} i_v + \sigma \frac{r_h}{i_h} + 5\delta_h i_h + \left(\delta_v - ac \frac{i_h}{i_v}\right) (1 - i_v),$$

$$B_{55} = -2H_T + \varphi \frac{S_h}{i_h} i_v + \sigma \frac{r_h}{i_h} + \delta_v i_v + \left(3\delta_h - \frac{ac}{i_v}\right) i_h,$$

$$B_{66} = -(H_T + H_R) + \varphi \frac{S_h}{i_h} i_v + \sigma \frac{r_h}{i_h} + \delta_v i_v + \left(2\delta_h - \frac{ac}{i_v}\right) i_h.$$

Following a similar fashion as in Buonomo and Lacitignola (2010), we concentrate on the norm in \mathbb{R}^6 ;

$$|z| = \max(G_1, G_2), \tag{29}$$

where $z \in \mathbb{R}^6$, with components $z_j = 1, 2, \dots, 6$, and

$$G_1(z_1, z_2, z_3) = \max \begin{cases} \max\{|z_1|, |z_2| + |z_3|\}, & \text{if } \text{sign}(z_1) = \text{sign}(z_2) = \text{sign}(z_3), \\ \max\{|z_2|, |z_1| + |z_3|\}, & \text{if } \text{sign}(z_1) = \text{sign}(z_2) = -\text{sign}(z_3), \\ \max\{|z_1|, |z_2|, |z_3|\}, & \text{if } \text{sign}(z_1) = -\text{sign}(z_2) = \text{sign}(z_3), \\ \max\{|z_1| + |z_2|, |z_2| + |z_3|\}, & \text{if } -\text{sign}(z_1) = \text{sign}(z_2) = \text{sign}(z_3), \end{cases}$$

$$G_2(z_4, z_5, z_6) = \max \begin{cases} \max\{|z_4| + |z_5| + |z_6|\}, & \text{if } \text{sign}(z_4) = \text{sign}(z_5) = \text{sign}(z_6), \\ \max\{|z_4| + |z_5|, |z_4| + |z_6|\}, & \text{if } \text{sign}(z_4) = \text{sign}(z_5) = -\text{sign}(z_6), \\ \max\{|z_5|, |z_4| + |z_6|\}, & \text{if } \text{sign}(z_4) = -\text{sign}(z_5) = \text{sign}(z_6), \\ \max\{|z_4| + |z_6|, |z_5| + |z_6|\}, & \text{if } -\text{sign}(z_4) = \text{sign}(z_5) = \text{sign}(z_6), \end{cases}$$

subject to the following

$$|z_2| < G_1, |z_3| < G_1, |z_2 + z_3| < G_1,$$

and

$$|z_i|, |z_i + z_j|, |z_4 + z_5 + z_6| \leq G_2(z); i = 4, 5, 6, i \neq j.$$

Case 1:

$G_1 > G_2, z_1, z_2, z_3 > 0$ and $|z_1| > |z_2| + |z_3|$. Then,

$$\|z\| = |z_1|, \tag{30}$$

so that

$$D_+ \|z\| = \dot{z}_1 = -(\zeta_1 + H_T - 3\delta_h i_{h*})z_1 + \sigma \frac{i_v}{i_h} z_2 - q_3 \frac{i_v}{i_h} z_4 + \varphi s_h \frac{i_v}{i_h} (z_3 + z_5)$$

$$\leq -(\zeta_1 + H_T - 3\delta_h i_{h*})|z_1| + \sigma \frac{i_v}{i_h} |z_2| - q_3 \frac{i_v}{i_h} |z_4| + \varphi s_h \frac{i_v}{i_h} |z_3 + z_5|.$$

Using $|z_3 + z_5| < G_2 < |z_1|, |z_4| < G_2 < |z_1|, |z_2| < G_2 < |z_1|$ and (30), it follows that

$$D_+ \|z\| \leq -\left(\zeta_1 + H_T - 3\delta_h i_h - \sigma \frac{i_v}{i_h} + q_3 \frac{i_v}{i_h} - \varphi s_h \frac{i_v}{i_h}\right) \|z\|$$

$$\leq -\eta_1 \|z\|,$$

where

$$\eta_1 = \varsigma_1 + H_T - 3\delta_h i_h - \sigma \frac{i_v}{i_h} + q_3 \frac{i_v}{i_h} - \varphi s_h \frac{i_v}{i_h}.$$

Case 2:

$G_1 > G_2, z_1, z_2, z_3 > 0$ and $|z_1| < |z_2| + |z_3|$. Then,

$$\|z\| = |z_2| + |z_3|. \quad (31)$$

Thus,

$$\begin{aligned} D_+ \|z\| &= \dot{z}_2 + \dot{z}_3 \\ &= (r + ac(1 - i_v)) \frac{i_h}{i_v} z_1 + (q_2 + \delta_h s_h)(z_4 + z_5) + (q_3 + \varphi s_h) z_6 \\ &\quad + \left\{ \lambda_v - (q_1 + H_T + H_R) - \varphi \left(1 - \frac{s_h}{i_h}\right) i_v + \sigma \frac{r_h}{i_h} + 4\delta_h i_h + \left(\delta_v - ac \frac{i_h}{i_v}\right) (1 - i_v) \right\} z_2 \\ &\quad + \left[-(q_1 + H_T) + \left\{ \delta_v - \varphi \left(1 - \frac{s_h}{i_h}\right) \right\} i_v + \sigma \frac{r_h}{i_h} + i_h \left(2\delta_h - \frac{ac}{i_v}\right) \right] z_3, \\ &\leq (r + ac(1 - i_v)) \frac{i_h}{i_v} |z_1| + (q_2 + \delta_h s_h) |z_4 + z_5| + (q_3 + \varphi s_h) |z_6| \\ &\quad + \left\{ \lambda_v - (q_1 + H_T + H_R) - \varphi \left(1 - \frac{s_h}{i_h}\right) i_v + \sigma \frac{r_h}{i_h} + 4\delta_h i_h + \left(\delta_v - ac \frac{i_h}{i_v}\right) (1 - i_v) \right\} |z_2| \\ &\quad + \left[-(q_1 + H_T) + \left\{ \delta_v - \varphi \left(1 - \frac{s_h}{i_h}\right) \right\} i_v + \sigma \frac{r_h}{i_h} + i_h \left(2\delta_h - \frac{ac}{i_v}\right) \right] |z_3|. \end{aligned}$$

Again using,

$$|z_4 + z_5| < G_2 < |z_2| + |z_3|, |z_3| < G_2 < |z_2| + |z_3|, |z_6| < G_2 < |z_2| + |z_3|, |z_1| < G_2 < |z_2| + |z_3|,$$

and (31), we get

$$\begin{aligned} D_+ \|z\| &\leq \left(\lambda_v + q_2 + \delta_h s_h + q_3 + \varphi s_h + (r + ac(1 - i_v)) \frac{i_h}{i_v} + 2\sigma \frac{r_h}{i_h} + 4\delta_h i_h - (q_1 + H_T + H_R) \right. \\ &\quad \left. - \varphi \left(1 - \frac{s_h}{i_h}\right) i_v + \left(\delta_v - ac \frac{i_h}{i_v}\right) (1 - i_v) - (q_1 + H_T) + \left\{ \delta_v - \varphi \left(1 - \frac{s_h}{i_h}\right) \right\} i_v + i_h \left(2\delta_h - \frac{ac}{i_v}\right) \right) \|z\|, \\ &\leq -\eta_2 \|z\|, \end{aligned}$$

where

$$\eta_2 = 2(q_1 + H_T) + H_R + \varphi \left(1 - \frac{s_h}{i_h}\right) i_v + \left(-\delta_v + ac \frac{i_h}{i_v}\right) (1 - i_v) + \left\{ -\delta_v + \varphi \left(1 - \frac{s_h}{i_h}\right) \right\} i_v$$

$$+i_h \left(-2\delta_h + \frac{ac}{i_v} \right) - \left(\lambda_v + q_2 + \delta_h s_h + q_3 + \varphi s_h + (r + ac(1 - i_v)) \frac{i_h}{i_v} + 2\sigma \frac{i_h}{i_h} + 4\delta_h i_h \right).$$

Case 3:

$G_1 > G_2, z_1 < 0, z_2, z_3 > 0$ and $|z_1| > |z_2|$. Then,

$$\|z\| = |z_1| + |z_3|, \quad (32)$$

so that

$$\begin{aligned} D_+ \|z\| &= -z_1 + z_3, \\ &= - \left(B_{11} - ac(1 - i_v) \frac{i_h}{i_v} \right) z_1 - \sigma \frac{i_v}{i_h} z_2 - \left(\varphi s_h \frac{i_v}{i_h} - B_{33} \right) z_3 + q_3 \frac{i_v}{i_h} z_4 \\ &\quad - \left(\left(\varphi \frac{i_v}{i_h} - \delta_h \right) s_h - q_2 \right) z_5 + q_3 z_6, \\ &\leq - \left(B_{11} - ac(1 - i_v) \frac{i_h}{i_v} \right) |z_1| - \sigma \frac{i_v}{i_h} |z_2| - \left(\varphi s_h \frac{i_v}{i_h} - B_{33} \right) |z_3| + q_3 \frac{i_v}{i_h} |z_4| \\ &\quad - \left(\left(\varphi \frac{i_v}{i_h} - \delta_h \right) s_h - q_2 \right) |z_5| + q_3 |z_6|. \end{aligned}$$

Using

$$|z_1| < G_2 < |z_1| + |z_3|, |z_3| < G_2 < |z_1| + |z_3|, |z_4| < G_2 < |z_1| + |z_3|, |z_5| < G_2 < |z_1| + |z_3|, |z_6| < G_2 < |z_1| + |z_3|, -\sigma \frac{i_v}{i_h} |z_2| < \sigma \frac{i_v}{i_h} |z_2|,$$

and taking (32) into account gives

$$D_+ \|z\| \leq -\eta_3 \|z\|,$$

where

$$\eta_3 = B_{11} - ac(1 - i_v) \frac{i_h}{i_v} + \sigma \frac{i_v}{i_h} + \varphi s_h \frac{i_v}{i_h} - B_{33} - q_3 \frac{i_v}{i_h} + \left(\varphi \frac{i_v}{i_h} - \delta_h \right) s_h - q_2 - q_3.$$

In a similar fashion, other cases can be achieved, and after some tedious manipulation we obtain $\eta_4, \eta_5, \dots, \eta_{16}$. Assume $\eta = \min\{\eta_1, \eta_2, \dots, \eta_{16}\}$ and $\eta > 0$ under the condition that $R_m > 1$, we have the Lozinkii measure $\bar{\mu}(K) < 0$. The result of Ozaire et al. (2016) draws us to the conclusion that the unique endemic equilibrium is globally asymptotically stable, and that ends the proof.

4. Numerical Experiments

This section illustrates numerically the analytical results obtained above based on the parameter values in Table 3. The simulation results are displayed in the figures below:

Table 3: Parameters of the Model

Parameter	Value.	Ref.
a	0.35 day^{-1}	Assumed
b	0.52 day^{-1}	Assumed
c	0.8333 day^{-1}	Huai and Qiu (2014)
γ	$\frac{1}{730} \text{ day}^{-1}$	Agusto et al. (2012)
r	0.0001 day^{-1}	Assumed
n_h	(0, 1)	Assumed
v	0.0035 day^{-1}	Osman and Adu (2016)
λ_h	$0.00007666 \text{ day}^{-1}$	Assumed
λ_v	0.7 day^{-1}	Osman and Adu (2016)
δ_h	0.068 day^{-1}	Osman and Adu (2016)
δ_v	0.01 day^{-1}	Assumed
n_0	0.01 day^{-1}	Assumed
σ	0.0001 day^{-1}	Huai and Qiu (2014)

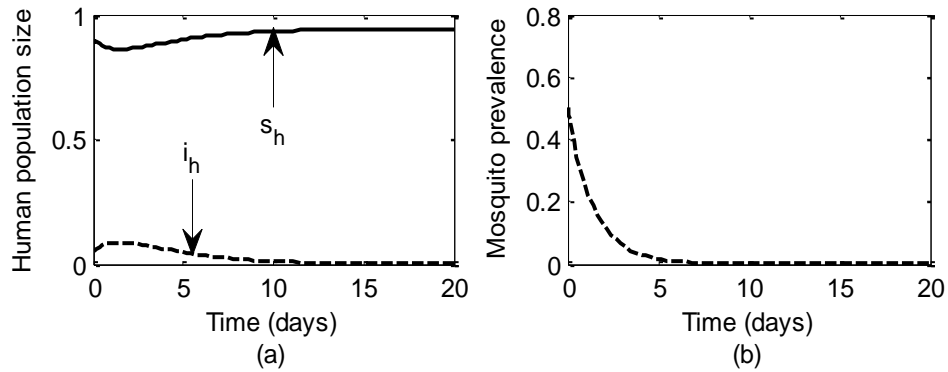


Figure 2. $R_m = 0.02238674921 < 1$, the malaria-free equilibrium, M_0 , is globally stable using parameter values in Table 2 with $n_h = 5 \times 10^{-4}$, $v = 0.25$ and $c = 0.08333$

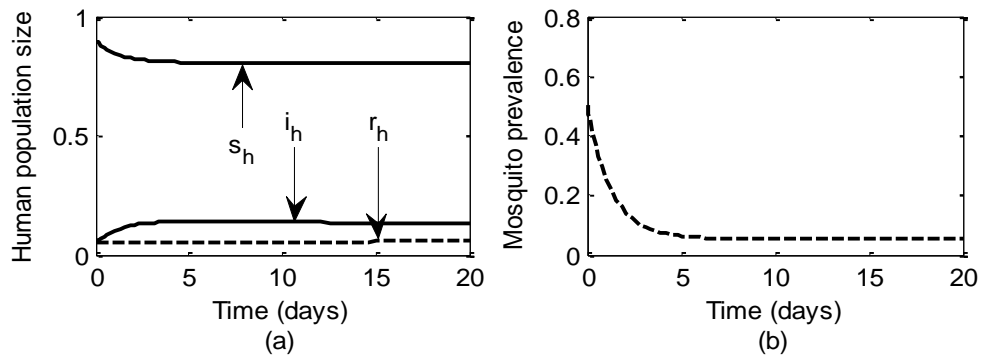


Figure 3. $R_m = 1.000090220 > 1$, the malaria-persist equilibrium state, M_{PES} , is uniformly persist using values in Table 2 with $n_h = 4.339700053 \times 10^{-4}$

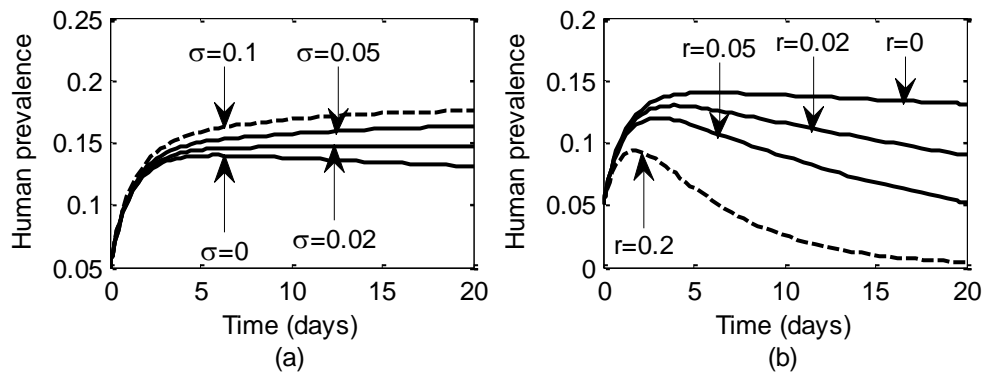


Figure 4. The impact of relapse (a) and recovery (b) rates on the human prevalence using values in Table 3.

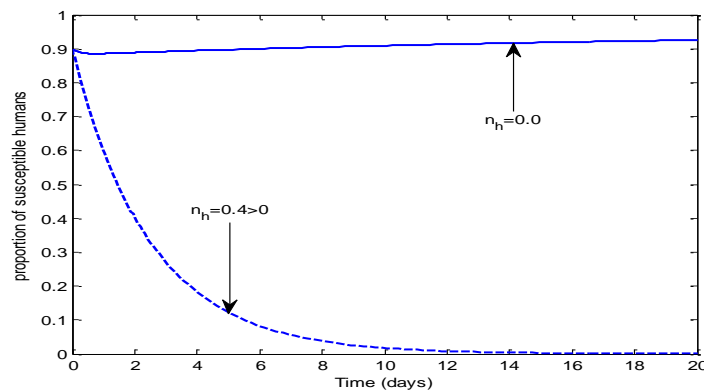


Figure 5. The impact of ITNs on the susceptible humans. Parameter values are as given on Table 3

5. Discussion

The present model is a variant of that of Tumwiine et al. (2007) for the dynamics of malaria in a human host and mosquito vector. Their aim was to study the effort of partial immunity on the dynamics of malaria in the absence of any control measure. In the present study the use of treated mosquito nets is incorporated as a control measure and is consistent with Tumwiine et al. (2007) recommendation. From stability analysis, we derive two steady states, namely, malaria-free equilibrium and malaria persistence equilibrium state. Using next generation operator, we computed effective reproduction number with(out) relapse, from which we calculated the minimum number of humans to use the treated mosquito nets. By constructing a Lyapunov function from matrix-theoretic method, MFE is shown to be globally stable subject to the condition $R_m \leq 1$. The proof of globally stable M_{PES} was done by adopting a geometric-matrix approach taking into account the Lozinkii measure with respect to the constraint that $R_m > 1$.

The Figures 2 and 3 derived from numerical experiments affirm that if the number of people sleeping under ITNs supersedes the threshold value $n_h = 4.29780993 \times 10^{-4}$, with moderate acquired immunity, the malaria epidemic could be driven to extinction. However, if the minimum number of susceptible individuals using treated nets are less than or equal to the above stated threshold value, then the mosquito invades the population and malaria will continue to persist as seen in Figure 3. Furthermore, impact of drug resistance actually compounds the malaria burden by increasing human prevalence and thus making the disease elimination difficult as demonstrated in Figure 4a. This result agrees with the outcome of the work by Hao and Quio (2014) which says that the basic reproduction number increases with respect to the relapse rate. On the contrary, human prevalence declines with increase in the rates of recovery (see Figure 4b). In the same vein, the proper use of ITNs decreases the susceptibility of human hosts from being infected as Figure 5 clearly illustrates. This is consistent with the outcome from Bala and Gimba (2019).

The earlier study (Tumwiine et al., 2007) concluded that due to re-infection the disease persists. Our results therefore suggest importantly that the use of treated mosquito nets should be maintained and be encouraged for every household until the mosquito vectors are possibly eliminated from the human population. Antimalarial drugs could be improved upon so as to avoid the consequences of relapse and patients be guided on new malaria treatment regimens to boost their immunity.

6. Conclusion

In this study, we present a version of the model by Tunwinne et al. (2007) for the dynamics of malaria in a human host and mosquito vector with temporary immunity. As recommended in their study we incorporate the use of treated mosquito nets in their model as a control strategy. Furthermore, we apply frequency dependent transmission mode in both the human and mosquito populations. The model equations were transformed into proportions to make more biological sense in terms of infected classes as they define prevalence of malaria. The control reproduction number with relapse R_m is calculated using the next generation operator. A malaria –free equilibrium state of the model is found to be globally asymptotically stable for $R_m \leq 1$, which means that the disease is controllable provided that $R_m < 1$. This condition also gives a threshold value for the proportion of susceptible humans to use treated nets for eradication of infection in a situation where the effect of drug resistance is negligible. Numerical experiments carried out using the transformed equations show that prevalence of malaria can be driven to zero provided that the proportion of susceptible humans using treated mosquito nets is above a certain threshold value. However, the presence of relapse makes malaria elimination difficult. Therefore, future work can be centered on awareness creation on the importance of ITNs and the best practices of treatment regimens with respect to drug resistance.

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