

Demographic impact and controllability of malaria in an SIS model with proportional fatality

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Abstract: The aim of this paper is to study the demographic impact of malaria on the host population and investigate the possibility to eliminate it with a strategy based on prevention (e.g., the use of bed-nets). In this paper, a deterministic model for the transmission of malaria (with variable human and mosquito population sizes) has been studied. The model equilibria have been found. Their local and global stability have been analyzed. An exact formula for the critical case fatality required to stop the growth of the host has been found. The conditions to eliminate malaria with a strategy based on the use of bed nets have been found.

Keywords: Malaria model, equilibria, local and global stability, case fatality, and elimination.

1 Introduction

Malaria is a life-threatening vector-borne disease caused by parasites which are transmitted to people through the bites of infected mosquitoes. Recently, it has been estimated that there were about 219 million cases of malaria in 2010 (with an uncertainty range of 154 million to 289 million) and an estimated 660000 deaths (with an uncertainty range of 490000 to 836000) [31].

The mathematical modeling of malaria has a long history since the discovery of malaria mode of transmission. This history goes back to the work of the pioneers (Ross [26] and Macdonald [17]) in this field. Ross [26] set the first model that describes the transmission dynamics of malaria. Then, Macdonald [17] considered major extensions for Ross's model, where he improved the model to a two dimensional model with one variable representing humans and one variable representing mosquitoes. An important extension of the model was proposed by Dietz et al [12] who added the inclusion of immunity. Thereafter, other extensive studies for malaria models have been carried out (see [3, 19, 21, 22, 29] and the references therein). Other studies concerning the effect of climatic changes and weather on malaria dynamics are shown in [13, 23, 24]. Some good reviews on the mathematical modeling and analysis of malaria are in [2, 4, 10, 15, 18, 20]. However, almost all these studies concentrate on the epidemiology of malaria infection, while this study gathers the epidemiology and demography of malaria infection on the host population.

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We consider an SIS model for malaria, where the human as well as the mosquito populations are assumed to have variable (in time) size. The human population is assumed to be exponentially growing with a growth rate ρ . Since the infectious period of malaria is relatively short (in days), we assumed that individuals dying as a result of malaria infection die after passing the infectious period, an assumption that we believe to be more realistic than assuming differential mortality approach as considered in the literature (see for example [8, 22, 32]). Therefore, we consider the proportional fatality approach [28]. We further investigate the impact of malaria on the demography of the host.

The paper is organized as follows. In section 2, the model is formulated and the corresponding model in subpopulation proportions is introduced. In section 3, the reduced model is considered and thoroughly analyzed, where the endemic equilibria have been found. Both the local and global stability of equilibria are evaluated in section 4. The effect of case fatality on the demography of the host is shown in section 5. In section 6, conditions to eliminate malaria with a strategy based on prevention (e.g., the use of bed nets) are found. Finally, summary and conclusion on the results is given in section 7.

2 Formulation of the model

The model supposes a homogeneous mixing of the human and vector (mosquito) populations so that each mosquito bite has equal chance of transmitting the virus to susceptible humans in the population. The total host (human) population at time t , whose size is denoted by $N_h(t)$, is subdivided into two sub-populations: susceptible humans $S_h(t)$ and infectious humans $I_h(t)$, so that $N_h(t) = S_h(t) + I_h(t)$. Similarly, the total vector (mosquitoes) population at time t , whose size is denoted by $N_v(t)$, is split into two sub-population: susceptible mosquitoes $S_v(t)$ and infectious mosquitoes $I_v(t)$, so that $N_v(t) = S_v(t) + I_v(t)$.

The susceptible human population is generated via recruitment of humans by births at rate β_h . This population is decreased due to natural deaths at rate μ_h or due to an infection by effective contacts with infectious vectors at rate $\lambda_h(t)$ (the force of infection), given by $\lambda_h = C_{vh} \frac{I_v}{N_v}$. Thus, the rate of change in the susceptible human population is given by

$$\frac{dS_h}{dt} = \beta_h N_h - (\lambda_h + \mu_h) S_h + (1 - c) \gamma_h I_h.$$

Infected humans either die with natural death rate μ_h , die due to the infection with rate $c\gamma_h$ or recover with temporal immunity to be susceptible again with rate $(1 - c)\gamma_h$. Thus,

$$\begin{aligned} \frac{dI_h}{dt} &= \lambda_h S_h - \mu_h I_h - c\gamma_h I_h - (1 - c)\gamma_h I_h \\ &= \lambda_h S_h - (\mu_h + \gamma_h) I_h. \end{aligned}$$

Physically, the parameter c represents the proportion of removed individuals (from the I_h state) who die as a result of malaria in the human population.

The susceptible vector population is generated by births at rate β_v . This susceptible population is reduced by infection following effective contact rate $\lambda_v(t) = C_{hv} \frac{I_h}{N_h}$ and natural deaths at rate μ_v . Infected vectors die with rate μ_v . Thus,

$$\begin{aligned}\frac{dS_v}{dt} &= \beta_v N_v - (\lambda_v + \mu_v) S_v, \\ \frac{dI_v}{dt} &= \lambda_v S_v - \mu_v I_v.\end{aligned}$$

Since mosquitoes bite both susceptible and infected humans, it is assumed that the average number of mosquito bites received by humans depends on the total sizes of the population of mosquitoes and humans in the community. It is assumed further that each susceptible mosquito bites an infected human at an average biting rate b_s , and the human hosts are always sufficient in abundance so that it is reasonable to assume that the biting rate b_s is constant. let $C_{hv} = P_{hv} b_s$, be the rate at which mosquitoes acquire infection from infected humans, where P_{hv} is the transmission probability from an infected human to a susceptible mosquito and b_s is the number of human bites one mosquito has per unit time [8] .

Similarly, let $C_{vh} = P_{vh} b_i$, be the rate at which humans acquire infection from infected mosquitoes, where P_{vh} is the transmission probability from an infected mosquito to a susceptible human and b_i is the number of mosquito bites one human received per unit time [8]. A full description for the model variable states and parameters is given in Table 1 and 2, respectively.

Table 1: The state variables for the original model (1) and for the model with scaled population sizes (2).

| State variable | Description |
|----------------|--|
| S_h | Number of susceptible humans at time t . |
| I_h | Number of infectious humans at time t . |
| N_h | Total human population size at time t . |
| S_v | Number of susceptible mosquitoes at time t . |
| I_v | Number of infectious mosquitoes at time t . |
| N_v | Total mosquito population size at time t . |
| s_h | Proportion of susceptible humans at time t . |
| i_h | Proportion of infectious humans at time t . |
| s_v | Proportion of susceptible mosquitoes at time t . |
| i_v | Proportion of infectious mosquitoes at time t . |

The mathematical representation of the model is

$$\begin{aligned}
\frac{dS_h}{dt} &= \beta_h N_h - (\lambda_h + \mu_h) S_h + (1 - c) \gamma_h I_h, \\
\frac{dI_h}{dt} &= \lambda_h S_h - (\mu_h + \gamma_h) I_h, \\
\frac{dS_v}{dt} &= \beta_v N_v - (\lambda_v + \mu_v) S_v, \\
\frac{dI_v}{dt} &= \lambda_v S_v - \mu_v I_v.
\end{aligned} \tag{1}$$

All parameters are strictly positive with the exception of the parameter c , representing the proportional fatality, which is assumed to be nonnegative. The mosquito birth rate β_v is assumed to be bigger than the mosquito death rate μ_v to ensure that there is a stable positive mosquito population.

Table 2: Description of the model parameters and their dimensions.

| Parameter | Description and dimension |
|------------|--|
| β_h | Per capita birth rate of humans. Time^{-1} |
| β_v | Per capita birth rate of mosquitoes. Time^{-1} |
| μ_h | Per capita natural death rate of humans (i.e., deaths due to causes other than malaria). Time^{-1} |
| μ_v | Per capita death rate of mosquitoes. Time^{-1} |
| b_s | The number of human bites one mosquito has per unit time. Time^{-1} |
| b_i | The number of mosquito bites one human has per unit time. Time^{-1} |
| P_{vh} | Transmission probability from an infectious human to a susceptible mosquito. Dimensionless |
| P_{hv} | Transmission probability from an infectious mosquito to a susceptible human. Dimensionless |
| C_{hv} | Rate at which mosquitoes acquire infection from infectious humans. Time^{-1} |
| C_{vh} | Rate at which humans acquire infection from infectious mosquitoes. Time^{-1} |
| γ_h | Removal rate from the I_h state with causes other than natural death. Time^{-1} |
| c | Proportion of removed individuals (from the I_h state) dying due to the infection in the human population. Dimensionless |

Reformulation of the model

Let us now reformulate model (1) to express it in terms of proportions rather than numbers. Assume that $s_h = \frac{S_h}{N_h}$, $i_h = \frac{I_h}{N_h}$, $s_v = \frac{I_v}{N_v}$, $i_v = \frac{I_v}{N_v}$ where $s_h + i_h = 1$ and

$s_v + i_v = 1$. Thus, the model (1) reads, see appendix A:

$$\begin{aligned}\frac{ds_h}{dt} &= \beta_h - (\lambda_h + \beta_h - c\gamma_h i_h)s_h + (1-c)\gamma_h i_h, \\ \frac{di_h}{dt} &= \lambda_h s_h - (\beta_h + \gamma_h)i_h + c\gamma_h i_h^2, \\ \frac{ds_v}{dt} &= \beta_v - (\lambda_v + \beta_v)s_v, \\ \frac{di_v}{dt} &= \lambda_v s_v - \beta_v i_v.\end{aligned}\tag{2}$$

For this system the closed set $\mathcal{D} = \{(s_h, i_h, s_v, i_v) \in \mathbb{R}^4 : 0 \leq s_h, i_h, s_v, i_v \leq 1, s_h + i_h = 1, s_v + i_v = 1\}$ is positively invariant, and the model (2) is mathematically and epidemiologically well-posed, in the sense that solutions starting in \mathcal{D} remain in it all the time.

3 Reduced model

Since $s_h + i_h = 1$ and $s_v + i_v = 1$, then model (2) can be reduced to the following model with two equations

$$\begin{aligned}\frac{di_h}{dt} &= \lambda_h(1 - i_h) - (\beta_h + \gamma_h)i_h + c\gamma_h i_h^2, \\ \frac{di_v}{dt} &= \lambda_v(1 - i_v) - \beta_v i_v,\end{aligned}\tag{3}$$

where $\lambda_h = C_{vh}i_v$ and $\lambda_v = C_{hv}i_h$.

3.1 Existence and stability of the infection-free equilibrium and the basic reproduction number

To find the equilibria we put the derivatives in the left hand side in model (3) equal zero. The infection-free equilibrium (IFE) is obtained by putting $i_h = i_v = 0$ and solving the resulting system with respect to i_h and i_v . Therefore, the IFE is $E_0 = (i_h^0, i_v^0)^t = (0, 0)^t$, where t denotes vector transpose.

To find the basic reproduction number we use the next generation operator method [30] to find the matrices \mathbf{F} (for the new infection terms) and \mathbf{V} (for the transition terms) as, see appendix B

$$\mathbf{F} = \begin{pmatrix} 0 & C_{vh} \\ C_{hv} & 0 \end{pmatrix} \quad \text{and} \quad \mathbf{V} = \begin{pmatrix} \beta_h + \gamma_h & 0 \\ 0 & \beta_v \end{pmatrix}$$

It follows then that the basic reproduction number is given by

$$R_0 = \rho(\mathbf{FV}^{-1}) = \sqrt{\frac{C_{hv}C_{vh}}{\beta_v(\beta_h + \gamma_h)}}\tag{4}$$

where ρ is the spectral radius (dominant eigenvalue in magnitude) of the matrix \mathbf{FV}^{-1} .

To establish the local stability of the infection-free equilibrium we consider its corresponding jacobian matrix

$$J_{E_0} = \begin{pmatrix} -(\beta_h + \gamma_h) & C_{vh} \\ C_{hv} & -\beta_v \end{pmatrix}. \quad (5)$$

It is clear that the trace of J_{E_0} is less than zero, while its determinant is positive if and only if the basic reproduction number $R_0 < 1$. Therefore, we show the following proposition:

Proposition 1. *The infection-free equilibrium E_0 is locally asymptotically stable (LAS) if and only if $R_0 < 1$.*

The proof of this proposition is done using the next generation matrix method [30] and is deferred to appendix C.

3.2 Endemic equilibria

The characteristic epidemiologic equation:

The endemic equilibria are obtained by solving the following nonlinear algebraic system of two equations in the variables i_v and i_h

$$\begin{aligned} 0 &= \lambda_h(1 - i_h) - (\beta_h + \gamma_h)i_h + c\gamma_h i_h^2, \\ 0 &= \lambda_v(1 - i_v) - \beta_v i_v. \end{aligned} \quad (6)$$

We further assume that $i_h \neq i_v \neq 0$. Therefore, we get

$$i_v = \frac{i_h(\beta_h + \gamma_h - c\gamma_h i_h)}{C_{vh}(1 - i_h)}, \quad (7)$$

$$i_h = \frac{\beta_v i_v}{C_{hv}(1 - i_v)}. \quad (8)$$

On reducing i_h between the last two equations, we get an equation in the proportion of endemic infected vector i_v as

$$G(i_v) =: Ai_v^2 + Bi_v + C = 0 \quad (9)$$

where

$$\begin{aligned} A &= C_{hv}C_{vh}(C_{hv} + \beta_v), \\ B &= C_{hv}\beta_v(\beta_h + \gamma_h) + c\gamma_h\beta_v^2 - C_{hv}C_{vh}(2C_{hv} + \beta_v), \\ C &= C_{hv}[C_{hv}C_{vh} - \beta_v(\beta_h + \gamma_h)]. \end{aligned}$$

Equation (9) is the characteristic epidemiologic equation whose solutions have one to one correspondence with the endemic equilibria of system (3). A feasible

solution of this equation satisfies the inequality $0 < i_v < 1$. Moreover, the left hand side of (9) is a polynomial of the second degree and henceforth it can have up to two feasible (nonnegative) roots, depending on the values of the model parameters. However, to study the possibility for the existence of positive equilibria, we analyze the bifurcation near the infection-free equilibrium.

Bifurcation points:

The scalar equation (9) could be seen as a bifurcation equation. We keep the parameters $C_{vh}, \beta_v, \beta_h, \gamma_h$ and c fixed and express the equation in terms of C_{hv} and i_v . Since the infection-free equilibrium exists for all parameter values, we put $i_v = 0$ in equation (9) to get $C_{hv} = 0$ or $C_{hv} = C_{hv}^0$ where

$$C_{hv}^0 = \beta_v(\beta_h + \gamma_h)/C_{vh}. \tag{10}$$

Hence, there are two bifurcation points, $(0, 0)$ and $(C_{hv}^0, 0)$. We shall compute the direction of bifurcation at each of them. It is shown in appendix D that the bifurcation is forward at the bifurcation point $(0, 0)$.

Center manifold analysis near the infection-free equilibrium:

Since the infection-free equilibrium exists for all parameter values, we put $i_v = 0$ in equation (9) to get $C_{hv} = 0$ or $C_{hv} = C_{hv}^0$ where

$$C_{hv}^0 = \beta_v(\beta_h + \gamma_h)/C_{vh}. \tag{11}$$

Hence, $(C_{hv}^0, 0)$ is a bifurcation point at which we compute the direction of bifurcation. We use the center manifold theory [30, 6, 7] to analyze the model near the infection-free equilibrium. To this end, we consider the jacobian matrix J_{E_0} evaluated at the infection-free equilibrium with $C_{hv} = C_{hv}^0$ (denoted by J^*) which becomes

$$J^* = \begin{pmatrix} -(\beta_h + \gamma_h) & C_{vh} \\ \beta_v(\beta_h + \gamma_h)/C_{vh} & -\beta_v \end{pmatrix}.$$

It is easy to check that the matrix J^* has a simple zero eigenvalue with another one having negative real part. Therefore, the center manifold theory [30, 6, 7] can be used to analyze the model. The method is based on evaluating a left eigenvector of J^* (corresponding to the zero eigenvalue), given by $V = (\beta_v, C_{vh})$ and a right eigenvector of it which is given by $W = (C_{vh}, (\beta_h + \gamma_h))^T$. Then we compute the two quantities a and b which are given by

$$\begin{aligned}
a &= \sum_{i,j,k=1}^2 v_i w_j w_k \frac{\partial^2 f_i}{\partial x_j \partial x_k}(x_0, C_{hv}^0) \\
&= -2C_{vh}[C_{vh}\beta_v(\beta_h + (1-c)\gamma_h) + \beta_v(\beta_h + \gamma_h)^2] < 0.
\end{aligned} \tag{12}$$

$$\begin{aligned}
b &= \sum_{i,j=1}^2 v_i w_j \frac{\partial_i^f}{\partial x_i \partial C_{hv}^0}(x_0, C_{hv}^0) \\
&= C_{vh}^2 > 0.
\end{aligned} \tag{13}$$

Therefore, according to theorem 4.1 of [7], the direction of bifurcation at the bifurcation point $(C_{hv}^0, 0)$ is forward and we show the following proposition.

Proposition 2. *The model we consider has two bifurcation points $(0, 0)$ and $(C_{hv}^0, 0)$ at which the bifurcation direction is forward (i.e., at these points the bifurcation is supercritical).*

Existence of endemic equilibria:

It can be checked that

$$G(0) = C = C_{hv}[C_{hv}C_{vh} - \beta_v(\beta_h + \gamma_h)] = C_{hv}\beta_v(\beta_h + \gamma_h)(R_0 - 1) \tag{14}$$

and

$$G(1) = A + B + C = c\gamma_h\beta_v^2 > 0. \tag{15}$$

Hence, there are two cases:

Case 1: $R_0 < 1$. In this case $G(0) < 0$ which implies that there is a unique positive solution for (9). This solution is given by

$$i_v^+ = \frac{-B + \sqrt{D}}{2A} \tag{16}$$

where

$$D = B^2 - 4AC = \beta_v^2 \{[(\beta_h + \gamma_h - C_{vh}) + c\gamma_h\beta_v]^2 + 4C_{vh}(\beta_h + (1-c)\gamma_h)C_{hv}^2\} > 0. \tag{17}$$

Thus, for model (3), there is a unique endemic equilibrium $E^+ = (i_h^+, i_v^+)^t$ where

$$i_h^+ = \frac{\beta_v i_v^+}{C_{hv}(1 - i_v^+)}. \tag{18}$$

Case 2: $R_0 > 1$. In this case, $G(0) > 0, G(1) > 0$ and $D > 0$. Also, it is easy to check that $G'(0) < 0$. Therefore, equation (9) has two positive solutions i_v^+ (given by (16)) and i_v^- , given by

$$i_v^- = \frac{-B - \sqrt{D}}{2A}. \tag{19}$$

Hence, model (3) has two equilibria $E^+ = (i_h^+, i_v^+)^t$ and $E^- = (i_h^-, i_v^-)^t$, where i_h^- has a similar form like (18).

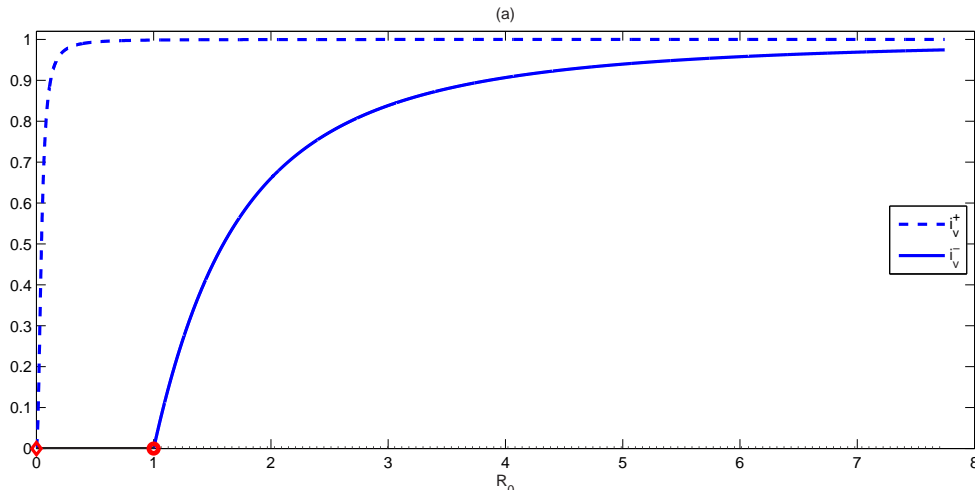


Figure 1: Bifurcation diagram showing the endemic equilibrium values for the proportion of infectious mosquito population for the parameters in Table 3. The figure shows that there are two bifurcation points, at $R_0 = 0$ and at $R_0 = 1$, at which the bifurcation is supercritical. In addition to the infection-free equilibrium E_0 , an unstable endemic equilibrium E^+ (represented by the dashed curve, i_v^+) exists for all values of R_0 . For $R_0 < 1$, E_0 is globally stable, while for $R_0 > 1$, E_0 loses its stability and another endemic equilibrium E^- (represented by the solid curve, i_v^-) exists and is globally stable.

We summarize our result in the following proposition.

Proposition 3. *In addition to the infection-free equilibrium E_0 , the reduced model (3) has an endemic equilibrium if $R_0 < 1$ and two endemic equilibria if $R_0 > 1$.*

4 Stability analysis

4.1 Local stability analysis of the endemic equilibria

To study the stability of the endemic equilibria of model (3) we make a small perturbation $(x(t), y(t))^t$ around a general equilibrium $(i_h, i_v)^t$ of that model. It is easy to get the linearized system of model (3) in the matrix form as

$$\frac{d}{dt} \begin{pmatrix} x \\ y \end{pmatrix} = \begin{pmatrix} -(C_{vh}i_v + \beta_h + \gamma_h - 2c\gamma_h i_h) & (1 - i_h)C_{vh} \\ (1 - i_v)C_{hv} & -(\beta_v + C_{hv}i_h) \end{pmatrix} \begin{pmatrix} x \\ y \end{pmatrix}. \quad (20)$$

We denote the coefficient matrix by J . Hence the characteristic equation of J is

$$\lambda^2 - tr(J)\lambda + det(J) = 0, \quad (21)$$

where, using (6)

$$tr(J) = -\{(\beta_v + C_{hv}i_h) + (C_{vh}i_v + \beta_h + \gamma_h - 2c\gamma_h i_h)\}, \quad (22)$$

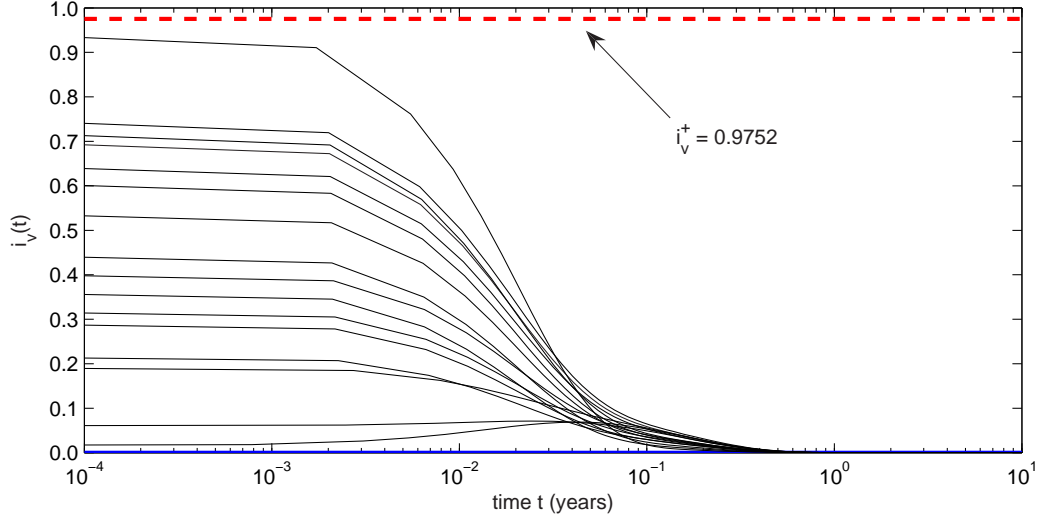


Figure 2: Time-dependent solutions in the region $R_0 < 1$. An unstable endemic equilibrium, E^+ , coexists with the infection-free equilibrium which is the global attractor. Simulations are made with parameter values in Table 3 and for $C_{hv} = 0.06$ per day which means that $R_0 = 0.25$.

and

$$\begin{aligned}
\det(J) &= \frac{\beta_v}{C_{hv}(1-i_v)^2} \{C_{hv}(\beta_h + \gamma_h + C_{vh}i_v)(1-i_v) - 2c\gamma_h\beta_v i_v\} - C_{vh}(C_{hv}(1-i_v) - \beta_v i_v) \\
&= \frac{1}{C_{hv}(1-i_v)^2} \{C_{hv}[\beta_v(\beta_h + \gamma_h) - C_{hv}C_{vh}] \\
&\quad + [C_{hv}C_{vh}(2\beta_v + 3C_{hv}) - C_{hv}\beta_v(\beta_h + \gamma_h) - 2c\gamma_h\beta_v^2]i_v \\
&\quad + C_{hv}C_{vh}(\beta_v + C_{hv})i_v^2(i_v - 3)\} \\
&= \frac{1}{C_{hv}(1-i_v)^2} \{2C + Bi_v - 2Ci_v - Bi_v^2\} \\
&= \frac{-i_v(1-i_v)(2Ai_v + B)}{C_{hv}(1-i_v)^2} \\
&= \frac{-i_v(1-i_v)(\mp\sqrt{B^2 - 4AC})}{C_{hv}(1-i_v)^2} \\
&= \begin{cases} < 0 & \text{if } i_v = i_v^+, \\ > 0 & \text{if } i_v = i_v^-. \end{cases}
\end{aligned}$$

It is shown in appendix E that $tr(J) < 0$. Therefore, we get the following proposition.

Proposition 4. *The endemic equilibrium $E^+ = (i_h^+, i_v^+)^t$ is unstable whenever it exists, while the other one $E^- = (i_h^-, i_v^-)^t$ is locally asymptotically stable whenever it exists (i.e., for $R_0 > 1$).*

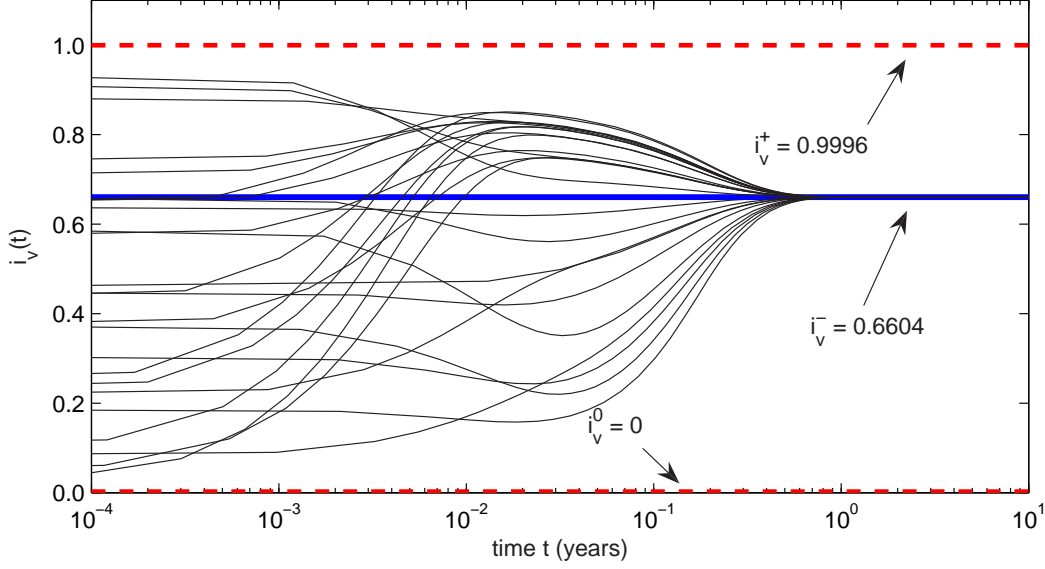


Figure 3: Time-dependent solutions in the region $R_0 > 1$. Two positive equilibria exist. The infection-free equilibrium loses its stability, while the equilibrium infection level corresponding to the equilibrium solution E^- is the global attractor. The other equilibrium solution E^+ is unstable. Simulations are made with parameter values in Table 3 and for $C_{hv} = 0.48$ per day which means that $R_0 = 2$.

4.2 Global stability

To study the global stability, we consider the reduced model (3) and define the set $\Omega := \{(i_h, i_v) : 0 < i_h, i_v < 1\}$. On putting $i_h = 0$ in (3.1) we get $di_h/dt = C_{vh}i_v > 0$ for all $i_v > 0$ and putting $i_v = 0$ in (3.2) we get $di_v/dt = C_{hv}i_h > 0$ for all $i_h > 0$. Therefore, the first quadrant and consequently the set Ω are positively invariant regions for the model, for any solution of (3) starting in the interior of Ω . Hence the ω -limit set of trajectory must be contained in Ω . Now, assume that

$$\begin{aligned} X(i_h, i_v) &= C_{vh}i_v(1 - i_h) - (\beta_h + \gamma_h)i_h + c\gamma_h i_h^2, \\ Y(i_h, i_v) &= C_{hv}i_h(1 - i_v) - \beta_v i_v, \end{aligned}$$

and consider the Dulac's function

$$\mathcal{D} = \frac{1}{\beta_v i_h i_v [(1 - ci_h)\gamma_h + \beta_h]} \quad (23)$$

where both i_h and i_v are positive. Hence

$$\frac{\partial(\mathcal{D}X)}{\partial i_h} + \frac{\partial(\mathcal{D}Y)}{\partial i_v} = -\mathcal{D}^2 \{C_{vh}\beta_v[(1 - i_h)^2 c\gamma_h + (1 - c)\gamma_h + \beta_h] + \beta_v C_{hv}[(1 - ci_h)\gamma_h + \beta_h]\} < 0.$$

Therefore, using the Dulac's criterion [25], the reduced model (3) has no limit cycle in the first quadrant. We show the following proposition.

Proposition 5. *The reduced model (3) has no periodic solutions.*

Hence, based on the Poincare-Bendixon theorem and propositions 4 and 5, the local stability of the equilibria E_0 and E^- implies their global stability. Thus, we show the following proposition.

Proposition 6. *For model (3), the following hold:*

1. *If $R_0 < 1$, then an unstable endemic equilibrium E^+ exists in addition to the infection-free equilibrium which is globally stable, see figure 2.*
2. *If $R_0 > 1$, then two endemic equilibria (E^- and E^+) exist in addition to the infection-free equilibrium. Both the infection free equilibrium E_0 and the endemic equilibrium with higher infection level E^+ are unstable, while the endemic equilibrium with lower infection level E^- is globally asymptotically stable, see figure 3.*

Table 3: The parameter values for which we do simulations for the model. The unit of time is days.

| Parameter | Value | Reference |
|-------------------|----------------------|-----------|
| β_h | 1.1×10^{-4} | [9] |
| β_v | 0.13 | [9] |
| μ_v | 0.033 | [9] |
| C_{vh} | 0.022×0.5 | [9] |
| $c\gamma_h$ | 9.0×10^{-5} | [9] |
| $(1 - c)\gamma_h$ | 0.02 | [1] |

A bifurcation diagram, with parameter values in Table 3, has been created, figure 1. The figure shows that there are two bifurcation points; one at $R_0 = 0$ and the other at $R_0 = 1$. It further shows that at both points, the bifurcation is supercritical. The equilibrium solution represented by the curve bifurcating at $R_0 = 0$ is always unstable while that represented by the curve bifurcating at $R_0 = 1$ is stable.

Figure 2 shows the proportion of the infectious mosquito population for many randomly generated initial conditions, for parameter values in table 3 and with $C_{hv} = 0.06$ per day which corresponds to a $R_0 = 0.25 < 1$. It shows that an unstable endemic equilibrium ($i_v = i_v^+ = 0.9752$) co-exists with the infection-free equilibrium ($i_v = 0$) that attracts all solutions (i.e., it is globally stable).

Simulations for the same parameter values except for C_{hv} which has been increased to $C_{hv} = 0.48$ per day (i.e., $R_0 = 2$) are shown in figure 3. The figure shows that solutions are attracted by an endemic positive equilibrium ($i_v = i_v^- = 0.6604$) that is globally stable and lie in between an unstable endemic equilibrium ($i_v = i_v^+ = 0.9996$) and the infection-free equilibrium ($i_v = i_v^0 = 0$) which loses its stability at $R_0 = 1$.

5 Effects on demography

To study the effects of malaria infection on the human population demography, we assume that ρ is the growth/decay rate of the human population. Since, system (1) is homogeneous, then the stationary solution of the human model (the first two equations in model (1)) is not a stationary (fixed) point, but an exponential "persistent" solution, i.e., a solution of the form $(S_h, I_h)e^{\rho t}$. Thus, on applying the theory of finite-dimensional homogeneous systems ([14], [5], [27]), we get

$$\rho I_h = C_{vh}i_v S_h - (\mu_h + \gamma_h)I_h, \quad (24)$$

$$\rho N_h = (\beta_h - \mu_h)N_h - c\gamma_h I_h \quad (25)$$

which implies

$$(\rho + \mu_h + \gamma_h)i_h = C_{vh}i_v(1 - i_h), \quad (26)$$

$$i_h = \frac{\beta_h - (\rho + \mu_h)}{c\gamma_h}. \quad (27)$$

Now, we rewrite (8) to get

$$i_v = \frac{C_{hv}i_h}{\beta_v + C_{hv}i_h}. \quad (28)$$

On omitting i_v and i_h among equations (26), (27) and (28) we get an equation in the exponent of growth ρ

$$[(\rho + \mu_h) + \gamma_h][c\gamma_h\beta_v + C_{hv}(\beta_h - (\rho + \mu_h))] = C_{hv}C_{vh}[c\gamma_h - (\beta_h - (\rho + \mu_h))]. \quad (29)$$

I.e.,

$$C_{hv}(\rho + \mu_h)^2 + [(C_{vh} + \gamma_h - \beta_h)C_{hv} - c\gamma_h\beta_v](\rho + \mu_h) + C_{hv}C_{vh}(c\gamma_h - \beta_h) - \gamma_h(\beta_h C_{hv} + c\gamma_h\beta_v) = 0. \quad (30)$$

It should be noted that the relations (27) and (28) imply

$$i_v = \frac{C_{hv}(\beta_h - (\rho + \mu_h))}{c\gamma_h\beta_v + C_{hv}(\beta_h - (\rho + \mu_h))} \quad (31)$$

or equivalently,

$$\rho = \rho_0 - \frac{c\gamma_h\beta_v}{C_{hv}(1 - i_v)} \quad (32)$$

where $\rho_0 = \beta_h - \mu_h$ is the malthusian growth rate (i.e., the growth rate in the absence of infection) for the human population. Thus, there is a one-to-one correspondence between solutions of (9) and those of (30). On the other hand, the case fatality

([11], [16], [28]) is

$$\begin{aligned}
f &= \frac{\text{Total number of human deaths}}{\text{Total number of human infections}} \\
&= \frac{c\gamma_h \int_0^\infty I_h(t)dt}{\int_0^\infty C_{vh}i_v(t)S_h(t)dt} \\
&= \frac{c\gamma_h \int_0^\infty I_h(t)dt}{(\rho + \mu_h + \gamma_h) \int_0^\infty I_h(t)dt} \\
&= \frac{c\gamma_h}{\rho + \mu_h + \gamma_h}. \tag{33}
\end{aligned}$$

Now, we investigate whether the exponent of growth ρ can be driven to zero and what the case fatality required to do so is. To this end, we put $\rho = 0$ and $c = c^*$ in (29) to get

$$\begin{aligned}
c^* &= \frac{C_{hv}(\beta_h - \mu_h)(\gamma_h + \mu_h - C_{vh})}{\gamma_h[C_{hv}C_{vh} - \beta_v(\mu_h + \gamma_h)]} \\
&= \frac{\mu_h}{\gamma_h} \cdot \frac{R_0^2(R_D - 1)}{(R_0^2 - 1)} \left(\frac{C_{hv}^0}{\beta_v} - 1 \right). \tag{34}
\end{aligned}$$

where $R_D = \beta_h/\mu_h$ is the demographic reproduction number for the human population. Thus, the critical case fatality f^* [28] required to drive the human population growth rate to zero is

$$\begin{aligned}
f^* &= \frac{c^*\gamma_h}{\mu_h + \gamma_h} \\
&= c^* \left(1 - \frac{D_{Ih}}{L_0} \right) \\
&= c^*(1 - P_{Ih}) \tag{35}
\end{aligned}$$

where

- $D_{Ih} = 1/(\mu_h + \gamma_h)$ is the average time spent in the I_h state for a human,
- $L_0 = 1/\mu_h$ is the expected time of life at birth in the absence of infection for a human,
- $P_{Ih} = D_{Ih}/L_0$ is the proportion of life spent in the I_h state for a human.

It is clear that the existence of the critical case fatality f^* depends on the existence of a feasible value of the proportion c . I.e., the critical proportion c^* must satisfy $c^* \in [0, 1]$. We summarize our result in the following proposition.

Proposition 7. *If the model parameters satisfy that $c^* \in [0, 1]$, then the critical case fatality f^* required to stop the growth of the human population is given by (35) and any slightly higher case fatality could drive the population to extinction.*

6 Elimination of the infection

Since the infection-free equilibrium is the global attractor if $R_0 < 1$, then eliminating the infection could be implemented if R_0 is reduced to slightly below one. One way to do so is to use nets as a barrier to prevent mosquitoes biting. In the infection transmission process, the bite is important when it occurs between an infected human and a susceptible mosquito or vice versa (i.e., when it occurs between an infected mosquito and a susceptible human). Now, using nets could be applied in three different scenarios: by susceptible humans only, by infected humans only or by both (susceptible and infected humans). We shall study the three different scenarios.

Scenario #1 : In this case, we assume that a proportion p of susceptible human individuals would use nets to protect themselves from the biting of mosquitoes. Thus, a proportion $(1 - p)$ of susceptible human individuals are subject to get bitten by infected mosquitoes. Hence, the total number of susceptible humans available for contacts with infected mosquitoes is reduced to $(1 - p)S_h$. In other words, the effect of applying this strategy on model (1) appears in the term representing new infections in humans, where the term $\lambda_h S_h$ in the first two equations of model (1) modifies to $(1 - p)\lambda_h S_h = (1 - p)C_{vh}i_v S_h$. The mathematical analysis of the model does not get affected, except we replace every C_{vh} with $(1 - p)C_{vh}$. Thus, the basic reproduction number for the modified model is simply $\sqrt{(1 - p)R_0}$ and eliminating the infection could be achieved if a proportion p of the susceptible humans use nets such that

$$p > 1 - \frac{1}{R_0^2}. \quad (36)$$

Scenario #2 : In this case, we assume that a proportion q of infected human individuals would use nets to prevent susceptible mosquitoes from infecting itself while taking its meals from the infected humans. This means that a proportion q of infected humans are excluded from contacts with mosquitoes and, therefore, the probability that a susceptible mosquito gets its meal from an infected human individual will be reduced from I_h/N_h to $(1 - q)I_h/N_h$, which in turn means that the term $\lambda_v S_v$ representing new infections in the vector population in model (1) is modified to $(1 - q)\lambda_v S_v$. The effect of this modification can be considered if we replace every C_{hv} with $(1 - q)C_{hv}$ in the analysis of the original model. This implies that the basic reproduction number for the modified model becomes $\sqrt{(1 - q)R_0}$. Hence, to eliminate the infection, a proportion q of the infected humans could use nets such that

$$q > 1 - \frac{1}{R_0^2}. \quad (37)$$

Scenario #3 : In this case we assume that the above two scenarios are combined together. I.e., a proportion p of susceptible humans and a proportion q of

infected humans would use the nets. As, in the above two scenarios we replace C_{vh} with $(1-p)C_{vh}$ and C_{hv} with $(1-q)C_{hv}$. Thus, the basic reproduction number for the new model reads $\sqrt{(1-p)(1-q)}R_0$. Hence, eliminating the infection requires

$$\sqrt{(1-p)(1-q)} < \frac{1}{R_0}. \quad (38)$$

We notice that if $p = q := \hat{p}$, then a proportion

$$\hat{p} > 1 - \frac{1}{R_0} \quad (39)$$

of the total human population is required to use nets to ensure an effective elimination of the infection. We end this section by the following proposition.

Proposition 8. *Malaria infection could be eliminated from the population if*

1. *a proportion $p > 1 - 1/R_0^2$ of the susceptible human population uses nets to protect themselves from the biting of infected mosquitoes, or*
2. *a proportion $q > 1 - 1/R_0^2$ of the infected human population uses nets to prevent susceptible mosquitoes from getting infected while taking its meals from the infected humans, or*
3. *a proportion $\hat{p} > 1 - 1/R_0$ of the total human population uses nets to prevent the incidence of new infections.*

7 Summary and conclusion

As malaria is one of the most serious health problems facing the world today, several studies have been made in attempt to understand its dynamics and predict what may happen and then suggest strategies to eliminate it [3, 8, 10, 12, 15, 21, 22].

In this paper, a deterministic model (SIS for humans and SI for mosquitoes) has been studied. Both the human and mosquito populations are of varying size. Though its simplicity, the model shows interesting dynamics. Its analysis shows that in addition to the infection free equilibrium (denoted by E_0) two endemic equilibria exist. One of them (denoted by E^+ and has high level of endemic infection) exists forever, while the other (denoted by E^- and has low level of endemic infection) exists only if $R_0 > 1$. The analysis shows further that E_0 is globally stable if $R_0 < 1$ and is unstable if $R_0 > 1$, while E^+ is unstable but E^- is globally stable whenever they exist. Thus any control strategy aiming to eliminate malaria should consider the inequality $R_0 < 1$. In other words, reducing R_0 to slightly below one is not only a necessary but a sufficient condition to eliminate malaria.

Since malaria is preventable (i.e., preventing the recruitment of new infections), our model has been modified to study the possibility to eliminate malaria using nets. The analysis shows that there are three scenarios to eliminate malaria. The first is

that a proportion $p > 1 - 1/R_0^2$ of the susceptible human population use nets to protect themselves from the bites of infected mosquitoes. This scenario is applicable if the total number of infected humans is much bigger than that of susceptible human population. The second is that a proportion $q > 1 - 1/R_0^2$ of the infected humans should use nets to prevent the transmission of infection to susceptible mosquitoes while taking its meals. This is applicable if the number of infected humans is much less than that of susceptible human population. Finally, if the proportion of infected humans is close to 50% then a proportion $\hat{p} > 1 - 1/R_0$ of the total human population should use bed nets as barriers to prevent the transmission of malaria to new cases.

On the other hand, the impact of malaria fatality on the demography of the host has been investigated. It has been shown that malaria case fatality could stop the growth of the host if the model parameters are such that the critical proportional fatality $c^* \in [0, 1]$.

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Appendices:

Appendix A

Since $N_h = S_h + I_h$ and $N_v = S_v + I_v$, then

$$\begin{aligned}\frac{dN_h}{dt} &= \frac{dS_h}{dt} + \frac{dI_h}{dt} = (\beta_h - \mu_h)N_h - c\gamma_h I_h, \\ \frac{dN_v}{dt} &= \frac{dS_v}{dt} + \frac{dI_v}{dt} = (\beta_v - \mu_v)N_v.\end{aligned}$$

Hence,

$$\begin{aligned}\frac{1}{N_h} \frac{dN_h}{dt} &= (\beta_h - \mu_h) - c\gamma_h i_h, \\ \frac{1}{N_v} \frac{dN_v}{dt} &= \beta_v - \mu_v.\end{aligned}$$

Since $s_h = \frac{S_h}{N_h}$, then

$$\begin{aligned}
\frac{ds_h}{dt} &= \frac{1}{N_h^2} \left(N_h \frac{dS_h}{dt} - S_h \frac{dN_h}{dt} \right) \\
&= \frac{1}{N_h} \frac{dS_h}{dt} - s_h \cdot \frac{1}{N_h} \cdot \frac{dN_h}{dt} \\
&= \frac{1}{N_h} (\beta_h N_h - (\lambda_h + \mu_h) S_h + (1-c)\gamma_h I_h) - s_h (\beta_h - \mu_h - c\gamma_h i_h) \\
&= \beta_h - (\lambda_h + \mu_h) s_h + (1-c)\gamma_h i_h - s_h (\beta_h - \mu_h - c\gamma_h i_h) \\
&= \beta_h - (\lambda_h + \beta_h - c\gamma_h i_h) s_h + (1-c)\gamma_h i_h.
\end{aligned}$$

Similarly, $i_h = \frac{I_h}{N_h}$ implies that

$$\begin{aligned}
\frac{di_h}{dt} &= \frac{1}{N_h} \frac{dI_h}{dt} - i_h \cdot \frac{1}{N_h} \cdot \frac{dN_h}{dt} \\
&= \frac{1}{N_h} (\lambda_h S_h - (\mu_h + \gamma_h) I_h) - i_h (\beta_h - \mu_h - c\gamma_h i_h) \\
&= \lambda_h s_h - (\mu_h + \gamma_h) i_h - i_h (\beta_h - \mu_h - c\gamma_h i_h) \\
&= \lambda_h s_h - (\beta_h + \gamma_h) i_h + c\gamma_h i_h^2.
\end{aligned}$$

Also,

$$\begin{aligned}
\frac{ds_v}{dt} &= \frac{1}{N_v} \frac{dS_v}{dt} - s_v \cdot \frac{1}{N_v} \cdot \frac{dN_v}{dt} = \beta_v - (\lambda_v + \beta_v) s_v, \\
\frac{di_v}{dt} &= \frac{1}{N_v} \frac{dI_v}{dt} - i_v \cdot \frac{1}{N_v} \cdot \frac{dN_v}{dt} = \lambda_v s_v - \beta_v i_v.
\end{aligned}$$

Appendix B:

To obtain the martinis F and V we rearrange the model (2) so that the equations representing infections come first. Therefore,

$$\begin{aligned}
\frac{di_h}{dt} &= C_{vh} i_v s_h - (\beta_h + \gamma_h) i_h + c\gamma_h i_h^2, \\
\frac{di_v}{dt} &= C_{hv} i_h s_v - \beta_v i_v, \\
\frac{ds_h}{dt} &= \beta_h - (C_{vh} i_v + \beta_h - c\gamma_h i_h) s_h + (1-c)\gamma_h i_h, \\
\frac{ds_v}{dt} &= \beta_v - (C_{hv} i_h + \beta_v) s_v.
\end{aligned}$$

The above system can be written as $\dot{x}_i = f_i(x) = \mathcal{F}_i(x) - \mathcal{V}_i(x)$, where $i = 1, 2, 3, 4$; $x_1 = i_h, x_2 = i_v, x_3 = s_h, x_4 = s_v$; $\mathcal{F}_i(x)$ is the rate of appearance of new infections in compartment i , and $\mathcal{V}_i(x) = \mathcal{V}_i^-(x) - \mathcal{V}_i^+(x)$ with $\mathcal{V}_i^+(x)$ representing the rate of transfer of individuals into compartment i by all other means and $\mathcal{V}_i^-(x)$ representing

the rate of transfer of individuals out of compartment. It has the infection-free equilibrium $x_0 = (i_h^0, i_v^0, s_h^0, s_v^0)' = (0, 0, 1, 1)'$. Thus,

$$\mathcal{F} = \begin{pmatrix} C_{vh}i_v s_h \\ C_{hv}i_h s_v \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} (\beta_h + \gamma_h)i_h - c\gamma_h i_h^2 \\ \beta_v i_v \\ -\beta_h + (C_{vh}i_v + \beta_h - c\gamma_h i_h)s_h - (1-c)\gamma_h i_h \\ -\beta_v + (C_{hv}i_h + \beta_v)s_v \end{pmatrix}$$

Hence,

$$F = \begin{pmatrix} \frac{\partial \mathcal{F}_1}{\partial i_h} & \frac{\partial \mathcal{F}_1}{\partial i_v} \\ \frac{\partial \mathcal{F}_2}{\partial i_h} & \frac{\partial \mathcal{F}_2}{\partial i_v} \end{pmatrix}_{x=x_0} = \begin{pmatrix} 0 & C_{vh} \\ C_{hv} & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} \frac{\partial \mathcal{V}_1}{\partial i_h} & \frac{\partial \mathcal{V}_1}{\partial i_v} \\ \frac{\partial \mathcal{V}_2}{\partial i_h} & \frac{\partial \mathcal{V}_2}{\partial i_v} \end{pmatrix}_{x=x_0} = \begin{pmatrix} \beta_h + \gamma_h & 0 \\ 0 & \beta_v \end{pmatrix}.$$

Appendix C:

Proof of proposition 1.

Let

$$J_1 = F - V = \begin{pmatrix} -(\beta_h + \gamma_h) & C_{vh} \\ C_{hv} & -\beta_v \end{pmatrix}.$$

Following the approach shown in [30], the stability of the infection-free equilibrium is determined by the sign of the quantity $s(j_1)$ representing the maximum real part of all the eigenvalues of the matrix J_1 (i.e., it is the spectral abscissa of J_1). Thus the proof summarizes in showing that

1. $s(J_1) < 0 \iff R_0 = \rho(FV^{-1}) < 1$,
2. $s(J_1) = 0 \iff R_0 = \rho(FV^{-1}) = 1$,
3. $s(J_1) > 0 \iff R_0 = \rho(FV^{-1}) > 1$.

It is easy to evaluate that

$$s(J_1) = \frac{1}{2} \left(-[\beta_h + \gamma_h + \beta_v] + \sqrt{[\beta_h + \gamma_h + \beta_v]^2 - 4\beta_v(\beta_h + \gamma_h)(1 - R_0^2)} \right).$$

Now, if $R_0 < 1$, then the value of the expression under the square root is less than $[\beta_h + \gamma_h + \beta_v]^2$ which implies that $s(J_1) < 0$ and vice versa. Also, if $R_0 > 1$ then the expression under the square root will have a value that is bigger than $[\beta_h + \gamma_h + \beta_v]^2$ which induces that $s(J_1) > 0$. It is easy to check that a value of $R_0 = 1$ makes $s(J_1) = 0$. Thus, the infection-free equilibrium is locally stable if and only if $s(J_1) < 0 \iff R_0 = \rho(FV^{-1}) < 1$, while it is unstable if and only if $s(J_1) > 0 \iff R_0 = \rho(FV^{-1}) > 1$.

Appendix D:

Direction of bifurcation at $(0, 0)$

Using the implicit function theorem, the direction of bifurcation at a point depends on the sign of the expression

$$\frac{di_v}{dC_{hv}} = -\frac{G_{C_{hv}}}{G_{i_v}}$$

at this point. It is easy to check that

$$\begin{aligned} G_{i_v}|_{(0,0)} &= c\gamma_h\beta_v^2, \\ G_{C_{hv}}|_{(0,0)} &= -\beta_v(\beta_h + \gamma_h). \end{aligned}$$

Hence

$$\frac{di_v}{dC_{hv}}|_{(0,0)} = \frac{\beta_h + \gamma_h}{c\gamma_h\beta_v} > 0$$

Thus, the bifurcation is forward at the point $(0, 0)$.

Appendix E:

We have

$$tr(J) = -\{(\beta_v + C_{hv}i_h) + (C_{vh}i_v + \beta_h + \gamma_h - 2c\gamma_h i_h)\}.$$

However, from the second equation of (6) we have

$$\beta_v + C_{hv}i_h = C_{hv}\frac{i_h}{i_v} = \frac{\beta_v}{1 - i_v}.$$

Also, from the first equation of (6) we have

$$c\gamma_h i_h = \beta_h + \gamma_h - C_{vh}\frac{i_v(1 - i_h)}{i_h}.$$

This implies that

$$\begin{aligned} C_{vh}i_v + \beta_h + \gamma_h - 2c\gamma_h i_h &= -\beta_h - \gamma_h - C_{vh}i_v + 2C_{vh}\frac{i_v}{i_h} \\ &= -\beta_h - \gamma_h - C_{vh}i_v + 2\frac{C_{vh}C_{hv}(1 - i_v)}{\beta_v}. \end{aligned}$$

Hence,

$$tr(J) = -\frac{1}{\beta_v(1 - i_v)}H(i_v),$$

where

$$H(i_v) = \beta_v^2 + 2C_{vh}C_{hv}(1 - i_v)^2 - \beta_v(1 - i_v)\{C_{vh}i_v + \beta_h + \gamma_h\} \quad (40)$$

$$= C_{vh}(\beta_v + 2C_{hv})i_v^2 + [\beta_v(\beta_h + \gamma_h) - 4C_{vh}C_{hv} - \beta_v C_{vh}]i_v + [\beta_v^2 + 2C_{vh}C_{hv} - \beta_v(\beta_h + \gamma_h)]. \quad (41)$$

Now, we use (9) in (41) to reduce the degree by one. Therefore, we get

$$H(i_v) = \frac{(H_1 - i_v H_2)}{A}, \quad (42)$$

where

$$H_1 = \beta_v C_{vh} C_{hv} \{\beta_v^2 + C_{hv}(C_{vh} + \beta_v + \beta_h + \gamma_h)\} > 0,$$

$$H_2 = \beta_v C_{vh} \{(\beta_h + \gamma_h + C_{vh})C_{hv}^2 + c\gamma_h \beta_v (\beta_v + 2C_{hv})\} > 0.$$

It is clear that H is linear in i_v . Moreover, $H(0) = H_1/A > 0$ and $H'(0) = -H_2/A < 0$. On the other hand, since i_v^- tends to 1 as C_{hv} tends to ∞ , then $H(1) = \beta_v^2$, this could be obtained also if we put $i_v = 1$ in (40). Thus $H(i_v^-) > 0$. Hence, $tr(J) < 0$ for $i_v = i_v^-$.

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