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Qualitative Analysis of a Mathematical Model Applied to Malaria Disease Transmission in Tumaco (Colombia)

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Abstract

In this work we study the malaria disease problem in Tumaco through of the mathematical modelling with the objective of contribute to the understanding of the transmission dynamics. To this end, we formulate a system of ordinary differential equations which tries to describe the malaria disease transmission dynamics for human and mosquito populations considering vectorial transmission, vertical transmission of disease and a force of infection which measure the impact that occurs in the disease transmission rate when an infected human is introduced into the

mosquito population. The results reveal the importance of congenital transmission and death due to disease in the infection outcome.

Mathematics Subject Classification: 34D23, 93D20, 65L05

Keywords: Malaria, vertical transmission, force of infection, differential equation, qualitative analysis

1 Introduction

In Tumaco (Colombia), cases of death due to malaria have been substantially reduced. However, cases of congenital malaria have arisen and in addition the incidence of malaria has been concentrated mainly in specific sectors or communes of the municipality. This suggests that the dynamics of transmission is changing, which generates a new public health alert. In this work we study the malaria disease problem in Tumaco through of the mathematical modelling with the objective of contribute to the understanding of the transmission dynamics. Epidemiological studies address the problem of malaria transmission from different approaches. In particular, mathematical modeling through dynamical systems defined by ordinary differential equations constitutes a tool that contributes to the understanding of the factors that make possible disease spread in a community. Mathematical models help to predict the behavior in the long term of a disease, to evaluate the effectiveness of control programs and to optimize the resources invested for this purpose. Mathematical modeling for malaria began in 1902 with Sir Ronald Ross [13], through his research work he discovered that malaria is transmitted by the bite of the female *Anopheles* mosquito. For this reason he was awarded the Nobel Prize in Medicine. In 1927, Kermack and McKendrick [14] formulate a system of delayed integro-differential equations that models the flow of susceptible, infected and recovered populations in an epidemic process. Using a simplified version of this model, they obtain the so-called *Theorem of threshold*, the cornerstone of Mathematical Epidemiology. In 1957, MacDonald [8] modified the model of Kermack and McKendrick obtaining a two-dimensional model to represent the human population and another to represent the mosquito population. Other research works about malaria transmission are [4, 9, 16].

2 Mathematical model

As in [12], the system of ordinary differential equations proposed below describe the transmission dynamics of malaria disease on human and mosquito populations under the following assumptions: For the human population the

model is of type SEIR (Susceptible-Exposed-Infected-Recovered) and for the mosquito population is of type SI (Susceptible-Infected). $S_H(t)$, $E_H(t)$, $I_H(t)$ and $R_H(t)$ denote the human population for each compartment at time t, while $S_V(t)$ and $I_V(t)$ represent the susceptible and infected mosquito populations at time t, respectively. The total human population is given by $N_H = S_H + E_H + I_H + R_H$ and for mosquitoes its total population is $N_V = S_V + I_V$. The additional hypotheses of SEIR model are specified below: the population of susceptible humans is recruited at a constant rate Λ_H and it decreases by infection due to contact with infected moquitoes by the term $\beta_H S_V$ where $\beta_H = \frac{\beta_{HV}\epsilon\phi I_V}{N_H}$ is the force of infection for humans, being β_{HV} the probability of human infection due to the bite of an infected mosquito, ϵ the per capita mosquito bite rate and ϕ the entomological inoculation rate. The per capita progression rate of exposed humans to infected humans is α which represents the incubation period. The population of susceptible humans increases due to birth of infected humans which is represented by the term $\frac{\lambda}{2}I_H$, where λ represents the per capita vertical transmission rate. On the other hand, population of susceptible humans decreases by death due to the infection represented by the term ρI_H where ρ is mosquito death rate due to infection. We assumed that infected individuals recover to per capita rate δ. The number of humans recovered from the infection becoming susceptible again is represented by the term ωR_H where ω is the per capita rate of loss of immunity. The human population has the same per capita natural death rate in each compartment denoted by μ_H. For the SI model we have the following hypotheses: the population of susceptible mosquitoes is recruited at a constant rate Λ_V and it decreases by infection due to contact with infected humans by the term $\beta_V S_V$ where $\beta_V = \frac{\beta_v I_H}{N_V}$ is the force of infection for mosquitoes being β_{VH} the probability that a mosquito will become infected by biting an infected human. The mosquito population has the same per capita natural death rate in each compartment denoted by μ_V. From above considerations we obtain the following system:

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H + \omega R_H - \beta_H S_H - \mu_H S_H \quad , \quad \frac{dE_H}{dt} = \beta_H S_H - (\alpha + \mu_H)E_H \\
\frac{dI_H}{dt} &= \frac{\lambda}{2}I_H + \alpha E_H - (\delta + \rho + \mu_H)I_H \quad , \quad \frac{dR_H}{dt} = \delta I_H - (\omega + \mu_H)R_H \\
\frac{dS_V}{dt} &= \Lambda_V - \beta_V S_V - \mu_V S_V \quad , \quad \frac{dI_V}{dt} = \beta_V S_V - \mu_V I_V .
\end{aligned} \tag{1}$$

The model (1) will be analyzed in a biologically feasible region for both human and mosquito populations. In this sense, in terms of the following parameters

$$\rho^* = \frac{\lambda/2 - \rho}{\mu_H} \quad and \quad \mathbf{m} = \min\left(\frac{\Lambda_H}{\mu_H}, \frac{\Lambda_H}{\mu_H(1-\rho^*)}\right). \tag{2}$$

We have following result:

Lemma 2.1. *If $\rho^* < 1$, the set Ω defined by*

$$\Omega = \left\{ (S_H, E_H, I_H, R_H, S_V, I_V) \in \mathbb{R}^6_+ : 0 \leq N_H \leq \boldsymbol{m}, \quad 0 \leq N_V \leq \frac{\Lambda_V}{\mu_V} \right\} \quad (3)$$

is positively invariant with respect to the solutions of the system (1).

Proof. Since the vector field defined by the right hand of (1) is continuously differentiable, it follows the existence and uniqueness of solutions of system (1) in Ω. On the other hand, adding the first four equations of (1) we have

$$\dot{N_H} = \Lambda_H - \mu_H N_H + \mu_H \rho^* I_H. \quad (4)$$

From (4) we observe that if $\rho^* \leq 0$, then $\dot{N_H} \leq \Lambda_H - \mu_H N_H$. The solution of above inequality is

$$N_H(t) \leq \frac{\Lambda_H}{\mu_H} + \left(N_H^0 - \frac{\Lambda_H}{\mu_H} \right) e^{-\mu_H t}, \quad (5)$$

where $N_H^0 = S_H^0 + E_H^0 + I_H^0 + R_H^0$. From (5) we verify that $N_H(t) \leq \frac{\Lambda_H}{\mu_H}$ when $N_H^0 \leq \frac{\Lambda_H}{\mu_H}$. Now, substituting $0 < \rho^* < 1$ in (4) we obtain $\dot{N_H} \leq \Lambda_H - \mu_H N_H + \mu_H I_H$. Since $I_H(t) \leq N_H(t)$ from above equation we have

$$\dot{N_H} \leq \Lambda_H + (\rho^* - 1)\mu_H N_H. \quad (6)$$

The solution of (6) is

$$N_H(t) \leq \frac{\Lambda_H}{\mu_H(1-\rho^*)} + \left(N_H^0 - \frac{\Lambda_H}{\mu_H(1-\rho^*)} e^{-\mu_H(1-\rho^*)t} \right). \quad (7)$$

Newly, we observe that $N_H(t) \leq \frac{\Lambda_H}{\mu_H(1-\rho^*)}$ when $N_H^0 \leq \frac{\Lambda_H}{\mu_H(1-\rho^*)}$. En consequence $N_H(t) \leq \mathbf{m}$. Adding the last two equations of (1) and solving the resulting equation we verify that $N_V(t) \leq \Lambda_V/\mu_V$. Finally, it is easy to verify that the vector field determined by the right side of the system (1), points inward to the boundary of Ω, which concludes the proof. □

3 Qualitative analysis of equilibrium solutions

In this section, we determine the basic reproductive number, the steady states and their stability as well as the bifurcation behavior of system (1). The disease free equilibrium (DFE) of the malaria model (1) exists and is given by

$$E_0 = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, \frac{\Lambda_V}{\mu_V}, 0 \right). \quad (8)$$

The basic reproduction number was calculated by using the next generation matrix [15] and it is given by

$$\mathcal{R}_0 = \sqrt{\beta_h \beta_v \frac{\alpha}{\alpha + \mu_H} \frac{1}{\mu_H(1-\rho^*) + \delta} \frac{1}{\mu_V}}. \tag{9}$$

Now, we will proof the local asymptotic stability of DFE. To this end, following the same idea presented in [10, 11] we verify that the eigenvalues of Jacobian matrix of the system (1) evaluated in E_0 are $\xi_1 = -\mu_H$, $\xi_2 = -\mu_V$, $\xi_3 = -(\omega + \mu_H)$ and the solutions of the following cubic equation

$$\xi^3 + a_1\xi^2 + a_2\xi + a_3 = 0, \tag{10}$$

where

$$\begin{aligned} a_1 &= \mu_V + \rho + \delta + 2\mu_H - \lambda/2 + \alpha \\ a_2 &= (\alpha + \mu_H)(\mu_H + \mu_V + \rho + \delta - \lambda/2) + \mu_V(\mu_H(1-\rho^*) + \delta) \\ a_3 &= (\mu_H(1-\rho^*) + \delta)(\mu_H + \alpha)\mu_V - \alpha\beta_h\beta_v. \end{aligned} \tag{11}$$

From Routh Hurwitz criterion [5, 7, 6], we establish that the roots of cubic equation (10) to have a real part if the following determinants

$$\begin{aligned} \Delta_1 &= 1 \\ \Delta_2 &= (\mu_V + \rho + \delta + 2\mu_H - \lambda/2 + \alpha)\left[(\mu_H + \alpha)\mu_V + (\mu_H(1-\rho^*) + \delta)(\mu_H + \mu_V + \alpha)\right] \\ &\quad + \alpha\beta_h\beta_v - (\mu_H(1-\rho^*) + \delta)(\mu_H + \alpha)\mu_V \\ \Delta_3 &= \left[(\mu_H(1-\rho^*) + \delta)(\mu_H + \alpha)\mu_V - \alpha\beta_h\beta_v\right]\Delta_2 \end{aligned}$$

are positive. Note that $\Delta_1 > 0$ and, if

$$(\mu_H(1-\rho^*) + \delta)(\mu_H + \alpha)\mu_V - \alpha\beta_h\beta_v > 0. \tag{12}$$

then $\Delta_2 > 0$ and $\Delta_3 > 0$. From inequality (12) we obtain $\mathcal{R}_0^2 < 1$, the following proposition summarizes the previous result.

Proposition 3.1. *If $\mathcal{R}_0 < 1$, then disease free equilibrium E_0 defined in (8) is local and asymptotically stable in Ω. If $\mathcal{R}_0 > 1$ then E_0 is unstable.*

In next proposition we prove the global stability of E_0 under the condition $\rho^* \leq 0$.

Proposition 3.2. *If $\rho^* \leq 0$ and $\mathcal{R}_0 < 1$, then E_0 is global and asymptotically stable in Ω.*

Proof. Let $(S_H(t), E_H(t), I_H(t), R_H(t), S_V(t), I_V(t))$ a positive solution of (1) for which $N_V(t) = S_V(t) + I_V(t)$, then by Lemma 2.1 we have $N_V(t) \leq \Lambda_V/\mu_V$. Since $I_H(t) < N_H(t)$ and $\rho^* \leq 0$ we have $\dot{N}_H(t) > \Lambda_H + \mu_H(1-\rho^*)N_H$ which implies $N_H(t) > \Lambda_H/\mu_H(1-\rho^*)$. Let us consider the following functions

$$V^*(S_H, E_H, I_H, R_H, S_V, I_V) = I_V + \frac{\mu_V}{\beta_h} E_H + \frac{\mu_V(\alpha+\mu_H)}{\alpha\beta_h} I_H,$$

and

$$V(\bar{S_H}, \bar{E_H}, \bar{I_H}, \bar{R_H}, \bar{S_V}, \bar{I_V}) = V^*\left(S_H - \frac{\Lambda_H}{\mu_H}, E_H, I_H, R_H, S_V - \frac{\Lambda_V}{\mu_V}, I_V\right). \tag{13}$$

We are going to proof that the function V defined on (13) is a Lyapunov function. In fact, V is definite positive well $V(E_0) = V^*(0) = 0$ and $V > 0$ for all $(\bar{S_H}, \bar{E_H}, \bar{I_H}, \bar{R_H}, \bar{S_V}, \bar{I_V}) \neq E_0 \in \Omega$. Furthermore, the orbital derivative of V is given by:

$$\begin{aligned} \dot{V} &= \left[\frac{\mu_V(\alpha+\mu_H)(\mu_H(1-\rho^*)+\delta)}{\alpha\beta_h}\right]\left[\frac{\beta_h\beta_v\alpha}{\mu_V(\alpha+\mu_H)(\mu_H(1-\rho^*)} - 1\right] \\ &< \left[\frac{\mu_V(\alpha+\mu_H)(\mu_H(1-\rho^*)+\delta)}{\alpha\beta_h}\right]\left(\mathcal{R}_0^2 - 1\right). \end{aligned}$$

Therefore $\dot{V} \leq 0$ if only if $\mathcal{R}_0 < 1$, and by Lyapunov Theorem E_0 is asymptotically stable when $\mathcal{R}_0 < 1$. From LaSalle principle, E_0 is a global attractor, that is E_0 is global and asymptotically stable when $\mathcal{R}_0 < 1$. □

Now, we will determine the existence of endemic equilibrium solutions of the system (1) which are the solutions of the following algebraic system:

$$\begin{aligned} \Lambda_H + \omega R_H - \beta_H S_H - \mu_H S_H = 0 \qquad & \beta_H S_H - (\alpha+\mu_H)E_H = 0 \\ \lambda/2 I_H + \alpha E_H - (\delta+\rho+\mu_H)I_H = 0 \qquad & \delta I_H - (\omega+\mu_H)R_H = 0 \\ \Lambda_V - \beta_V S_V - \mu_V S_V = 0 \qquad & \beta_V S_V - \mu_V I_V = 0. \end{aligned} \tag{14}$$

Adding the two last equations of (14) we obtain $N_V = \frac{\Lambda_V}{\mu_V}$.Substituting $\beta_V = \frac{\beta_v I_H}{N_V}$ and N_V defined in (5) in the sixth equation of (14) we obtain

$$\beta_{VH}\epsilon\phi I_H S_V \frac{\mu_V}{\Lambda_V} - \mu_V I_V = 0.$$

From above equation we obtain

$$I_H = \frac{\mu_V}{\beta_v}\frac{\Lambda_V}{\mu_V}\frac{I_V}{S_V}. \tag{15}$$

Substituting (15) in third and fourth equations of (14) we obtain

$$E_H = \frac{\beta_h}{\alpha+\mu_H}\frac{1}{(\mathcal{R}_0)^2}\frac{\Lambda_V}{\mu_V}\frac{I_V}{S_V} \quad , \quad R_H = \frac{\delta}{\omega+\mu_H}\frac{\mu_V}{\beta_v}\frac{\Lambda_V}{\mu_V}\frac{I_V}{S_V}. \tag{16}$$

Adding the two first equation of (14) we obtain

$$S_H = \frac{\Lambda_H + \omega R_H - (\alpha+\mu_H)E_H}{\mu_H}. \tag{17}$$

Substituting R_H and E_H defined in (16) in (17) we obtain

$$S_H = \frac{\Lambda_H}{\mu_H} - \frac{\beta_h}{\mu_H}(1-\theta)\frac{1}{(\mathcal{R}_0)^2}\frac{\Lambda_V}{\mu_V}\frac{I_V}{S_V}, \tag{18}$$

where

$$\theta = \frac{\omega}{\omega+\mu_H}\frac{\alpha}{\alpha+\mu_H}\frac{\delta}{\mu_H(1-\rho^*)+\delta}. \tag{19}$$

Substituting S_H, I_H, R_H and E_H defined in (18), (15) and (16) respectively, in the total population we obtain

$$N_H = \frac{\Lambda_H}{\mu_H} + \frac{\rho^*}{\rho_1}\frac{\Lambda_V}{\mu_V}\frac{I_V}{S_V}, \tag{20}$$

where

$$\rho_1 = \frac{\beta_v}{\mu_V}. \tag{21}$$

Substituting $\beta_H = \frac{\beta_{HV}\epsilon\phi I_V}{N_H}$ in the second equation of (14) we obtain

$$\frac{\beta_{HV}\epsilon\phi I_V}{N_H}S_H - (\alpha+\mu_H)E_H = 0. \tag{22}$$

Now, replacing (16), (18) and (20) in the equation (22) we obtain

$$-\left(\frac{\beta_h}{\mu_H}(1-\theta)\frac{1}{(\mathcal{R}_0)^2} + \frac{1}{S_V}\frac{1}{(\mathcal{R}_0)^2}\frac{\Lambda_V}{\mu_V}\frac{\rho^*}{\rho_1}\right)\frac{I_V}{S_V} + \left(\frac{\mu_V}{\Lambda_V} - \frac{1}{(\mathcal{R}_0)^2}\frac{1}{S_V}\right)\frac{\Lambda_H}{\mu_H} = 0. \tag{23}$$

From the sum of the last two equilibrium equations of (14) we

$$\frac{I_V}{S_V} = \frac{\Lambda_V}{\mu_V}\frac{1}{S_V} - 1. \tag{24}$$

substituting (24) in (23) we obtain

$$-\left(\frac{\beta_h}{\mu_H}(1-\theta) + \frac{1}{S_V}\frac{\Lambda_V}{\mu_V}\frac{\rho^*}{\rho_1}\right)\left(\frac{\Lambda_V}{\mu_V}\frac{1}{S_V} - 1\right) + \left(\frac{\mu_V}{\Lambda_V}(\mathcal{R}_0)^2 - \frac{1}{S_V}\right)\frac{\Lambda_H}{\mu_H} = 0. \tag{25}$$

Multiplying (25) by $(S_V)^2$ we obtain the following quadratic equation

$$a(S_V)^2 + bS_V + c = 0, \tag{26}$$

where

$$\begin{aligned} a &= \frac{\beta_h}{\mu_H}(1-\theta) + \frac{\Lambda_H}{\mu_H}\frac{\mu_V}{\Lambda_V}(\mathcal{R}_0)^2 \\ b &= -\left[\frac{\beta_h}{\mu_H}(1-\theta)\frac{\Lambda_V}{\mu_V} + \frac{\Lambda_H}{\mu_H}\right] + \frac{\Lambda_V}{\mu_V}\frac{\rho^*}{\rho_1} \\ c &= -\frac{\rho^*}{\rho_1}\left(\frac{\Lambda_V}{\mu_V}\right)^2, \end{aligned} \tag{27}$$

The solutions of the quadratic equation (26) are given by

$$S_V^1 = \frac{-b + |b|\sqrt{1-\eta}}{2a} \quad , \quad S_V^2 = \frac{-b - |b|\sqrt{1-\eta}}{2a}, \tag{28}$$

where $\eta = \dfrac{4ac}{b^2}$. Note that $a > 0$ and the signs of b and c depend on the sign of ρ^*. In consequence, from (28) we obtain The following results.

Proposition 3.3. *The system (1) always has a trivial disease free equilibrium E_0 defined on (8). Besides,*

1. *If $0 \leq \rho^* < 1$, then there is a unique endemic equilibrium E_1.*
2. *If $\rho^* < 0$, the following options are presented:*
   (a) *If $0 < \eta < 1$, then there are two endemic equilibria E_1 and E_2.*
   (b) *If $\eta = 1$, then there is a unique endemic equilibrium E_1.*
   (c) *If $\eta > 1$, there are not endemic equilibria.*

3.1 Forward bifurcation analysis

Using the thoery of Center Manifold we carry out a bifurcation analysis of system (1) at $\mathcal{R}_0 = 1$ [1]. Since $N_V(t)$ converges to Λ_V/μ_V when $t \to \infty$, then $S_V(t)$ converges to $\frac{\Lambda_V}{\mu_V} - I_V(t)$. Above implies that the system (1) approaches asymptotically the following system:

$$\begin{aligned} \frac{dS_H}{dt} &= \Lambda_H + \omega R_H - \frac{\beta_h}{N_H} I_V S_H - \mu_H S_H \quad , \quad \frac{dE_H}{dt} = \frac{\beta_h}{N_H} I_V S_H - (\alpha + \mu_H) E_H \\ \frac{dI_H}{dt} &= \frac{\lambda}{2} I_H + \alpha E_H - (\delta + \rho + \mu_H) I_H \quad , \quad \frac{dR_H}{dt} = \delta I_H - (\omega + \mu_H) R_H \\ \frac{dI_V}{dt} &= \frac{\beta_v}{N_V}\frac{\Lambda_V}{\mu_V} I_H - \left[\frac{\beta_v}{N_H} I_H + \mu_V\right] I_V. \end{aligned} \tag{29}$$

Now, we consider the transmission rate β_h as a bifurcation parameter so that $\mathcal{R}_0 = 1$ if only if

$$\beta_h = \beta^* := \frac{\mu_V(\alpha+\mu_H)(\mu_H(1-\rho^*)+\delta)}{\beta_v \alpha}. \tag{30}$$

The Jacobian matrix of (29) evaluated in E_0 is given by

$$J(E_0) = \begin{pmatrix} -\mu_H & 0 & 0 & \omega & -\beta^* \\ 0 & -(\alpha+\mu_H) & 0 & 0 & \beta^* \\ 0 & \alpha & -(\rho+\delta+\mu_H) & 0 & 0 \\ 0 & 0 & \delta & -(\omega+\mu_H) & 0 \\ 0 & 0 & \beta_v & 0 & -\mu_V. \end{pmatrix}. \tag{31}$$

The eigenvalues of the matrix $J(E_0)$ are $\xi_1 = 0$, $\xi_2 = -\mu_H$, $\xi_3 = -(\mu_H + \omega)$ and the roots of the following quadratic equation

$$\xi^2 + n\xi + m \;\; = \;\; 0, \tag{32}$$

where

$$\begin{aligned} n &= (\alpha_H + \mu_H) - (\mu_H(1-\rho^*)+\delta) + \mu_V \\ m &= \mu_V(\alpha_H+\mu_H) - (\alpha_H+\mu_H)(\mu_H(1-\rho^*)+\delta) - \mu_V(\mu_H(1-\rho^*)+\delta). \end{aligned} \tag{33}$$

Note that n and m given on (33) are positive. Then, the solutions of the quadratic equation (32) are given by

$$\xi_{1,2} = \frac{-n \pm \sqrt{n^2-4m}}{2}. \tag{34}$$

Observe that the real part of $\xi_{1,2}$ always is negative. In consequence, matrix (31) has an eigenvalue $\xi_1 = 0$ and four eigenvalues with negative real part. We are now in position to apply the center manifold approach. We start calculating right and left eigenvectors of $J(E_0)$ associated with the eigenvalue $\xi = 0$ denoted by $W = (w_1, w_2, w_3, w_4, w_5)$ and $V = (v_1, v_2, v_3, v_4, v_5)$, respectively. After some computations we obtain

$$W = \begin{bmatrix} \frac{(\alpha+\mu_H)(\mu_H(1-\rho^*)+\delta)(\theta-1)}{\mu_H \alpha} \\ \frac{(\mu_H(1-\rho^*)+\delta)}{\alpha} \\ 1 \\ \frac{\delta}{\omega+\mu_H} \\ \frac{\beta_v}{\mu_V} \end{bmatrix} \frac{\mu_V}{\beta_v} \tag{35}$$

and

$$V = \begin{bmatrix} 0 \\ \frac{\alpha}{(\mu_H(1-\rho^*)+\delta)(\alpha+\mu_H)} \\ \frac{1}{(\mu_H(1-\rho^*)+\delta))} \\ 0 \\ \frac{1}{\beta_v} \end{bmatrix} \frac{(\mu_H(1-\rho^*)+\delta))(\alpha+\mu_H)\beta_v}{(\mu_H(1-\rho^*)+\delta)(\mu_V+\alpha+\mu_H)+\mu_V(\alpha+\mu_H)}. \tag{36}$$

Parameter	High transm. zone	Low transm. zone	Range	Reference
α	0.10	0.1050	$0.007 - 0.20$	[3, 2]
μ_V	0.0039	0.0025	$0.0010 - 0.12$	[3, 2]
μ_H	0.00102	0.00125	$0.000001 - 0.002$	[3, 2]
ρ	0.0090	0.009	$0 - 0.00041$	[3, 2]
δ	0.0029	0.0029	$0.0014 - 0.017$	[3, 2]
β_{HV}	0.20	0.45	$0.010 - 0.69$	[3, 2]
β_{VH}	0.20	0.15	$0.072 - 0.69$	[3, 2]
ϵ	0.3	0.29	$0.25 - 0.87$	[3, 2]
ϕ	0.27	0.10	$0.13 - 0.77$	[3, 2]
λ	0.0091	0.0002	$0.000000012 - 0.00012$	[3, 2]
Λ_V	460	200	$100 - 1000$	[3, 2]
Λ_H	91	100	10-200	[3, 2]
ω	0.01	0.01	$0.000055 - 0.01$	[3, 2]

Table 1: Parameter values of the system (1) for high and low transmission zones.

Newly, after some computations we verify that a and b defined in page 13 of [1], are given by

$$\begin{aligned} a &= -\frac{\mu_V \beta_h \mu_H[(\mu_H(1-\rho^*)+\delta)+\alpha+\delta]}{\Lambda_H[(\mu_H(1-\rho^*)+\delta)(\alpha+\mu_H+\mu_V)+\mu_V(\alpha+\mu_H)]} \\ &- \frac{\mu_V^2(\mu_H(1-\rho^*)+\delta)(\alpha+\mu_H)}{\Lambda_V[(\mu_H(1-\rho^*)+\delta)(\alpha+\mu_H+\mu_V)+\mu_V(\alpha+\mu_H)]}, \end{aligned} \tag{37}$$

and

$$b = \frac{\beta_v(\alpha+\mu_H)(\mu_H(1-\rho^*)+\delta)}{(\mu_H(1-\rho^*)+\delta)(\mu_V+\alpha+\mu_H)+\mu_V(\alpha+\mu_H)}.$$

Since $b > 0$ and $a < 0$ when $\rho^* < 1$, then the SEIR malaria model exhibits a forward bifurcation.

4 Numerical solutions

In Tumaco (municipality of Colombia) there was an accelerated growth in the incidence of malaria between 2000 and 2001. In this sense, National System of Vigilance for Public Health (SIVIGILA) carried out a census to evaluate the level of incidence. We used data from Chitnis et al [2] and data reported by SIVIGILA to estimate two sets of parameter which are shown in the Table 1: the first consists of the values for low transmission zone which refer to urban areas of Tumaco, where $\mathcal{R}_0 = 0.7144$ and the second consists of the values for high transmission zone which refer to rural areas of Tumaco, where $\mathcal{R}_0 = 6.2638$. In the Figures 1 and 2 it shown the numerical simulations of (1) made with data of Table 1 for low and high transmission zones.

5 Discussion

In this paper we formulated a mathematical model for malaria disease transmission in Tumaco (Colombia). Our main objective was to explore the effect of vertical

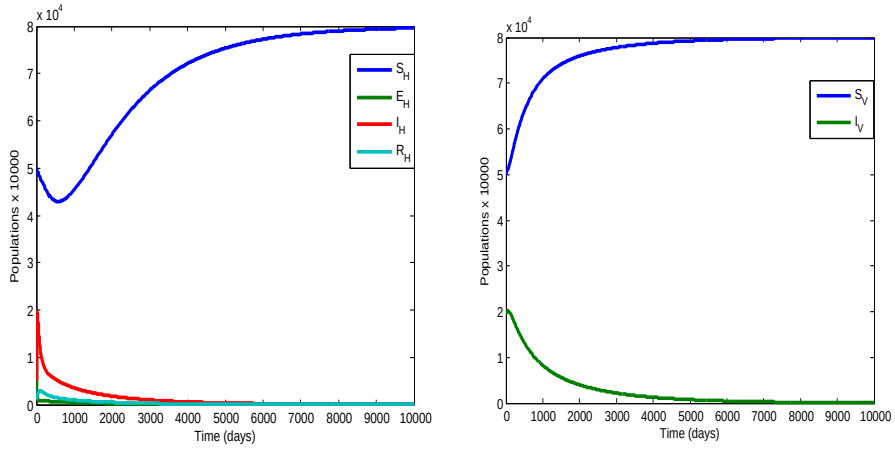

Figure 1: Numerical simulation of the model (1) made with data from the Table 1 for low transmission zone, in this case $\mathcal{R}_0 = 0.7144$.

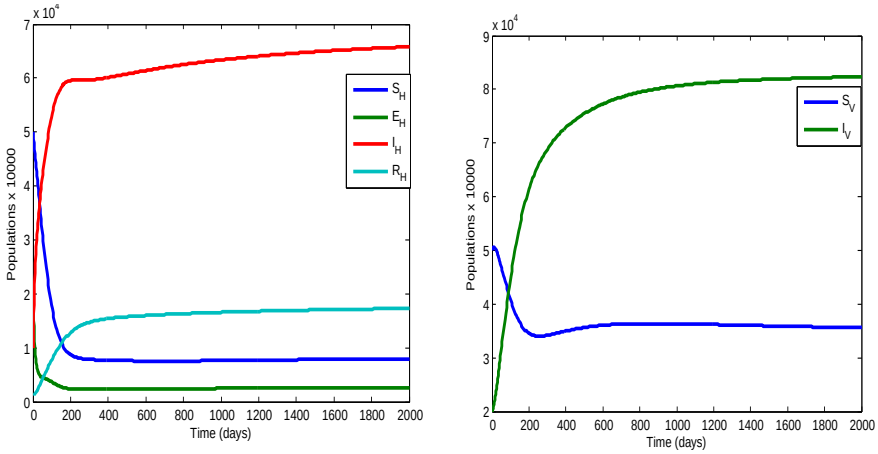

Figure 2: Numerical simulation of the model (1) made with data from the Table 1 for high transmission zone, in this case $\mathcal{R}_0 = 6.2638$.

transmission and the force of infection on the spread of malaria. The qualitative analysis was made in terms of the basic reproductive number $\mathcal{R}_0$ and the vegetative growth rate of infected individuals ρ^*, this revealed the existence of three steady states: the disease-free state E_0 and two endemic equilibria E_1 and E_2. When $\mathcal{R}_0 < 1$ the solutions approach the disease-free equilibrium which means that spread of malaria is controlled. If $\mathcal{R}_0 > 1$, malaria is not eliminated and different propagation scenarios are presented. The numerical simulations suggest that in the rural area the propagation of malaria can be controlled in a considerable time. However, in the urban area malaria has a very high level of incidence. In a future work we approach preventive control strategies that allow to reduce the incidence.

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