Journal homepage http://revistas.unitru.edu.pe/index.php/SSMM

SELECCIONES MATEMÁTICAS
Universidad Nacional de Trujillo
ISSN: 2411-1783 (Online)
2025; Vol.12(2):309-325.

Special issue: XII International Congress of Applied and Computational Mathematics

Perturbation of a biological control model of malaria considering species competition

Marco Antonio Tamariz Milla and Roxana López-Cruz

Received, Jun. 10, 2025; *Accepted, Oct. 30, 2025;* *Published, Dec. 27, 2025*

How to cite this article:
Tamariz M, López-Cruz R. *Perturbation of a biological control model of malaria considering species competition*. Selecciones Matemáticas. 2025;12(2):309–325. https://doi.org/10.17268/sel.mat.2025.02.04

Abstract

This work studies a mathematical model describing the transmission dynamics of malaria, incorporating intraspecific competition in human and mosquito populations, and biological control through larvivorous fish. We prove the existence of a compact attracting set for the proposed system of differential equations. Through stability analysis, we characterize the disease-free and endemic equilibrium points, determining epidemiologically relevant thresholds. The basic reproduction number (R_0) is a key parameter: when $R_0 \leq 1$, the disease-free equilibrium is globally asymptotically stable, whereas for $R_0 > 1$, a stable endemic equilibrium emerges. Numerical simulations validate the theoretical results and reveal the regulatory effect of intraspecific competition on disease prevalence. Finally, we generalize previous models by incorporating competitive interactions and vector management strategies.

Keywords . Dynamical systems, stability, intraspecific competition, mathematical epidemiology.

1. Introduction. Malaria is caused by parasites of the genus *Plasmodium*, transmitted to humans through the bites of infected female mosquitoes of the genus *Anopheles* (vectors) [1]. It remains a major health concern, particularly in tropical regions. In 2017, there were 219 million cases and 435,000 deaths, underscoring malaria's significant global impact [2]. In response to the increasing resistance to insecticides and drugs, the use of biological control strategies, such as the introduction of larvivorous fish, offers a sustainable alternative to chemical interventions. However, existing models often overlook intraspecific competition effects, which can significantly influence disease dynamics.

2. Model. The classical Ross–Macdonald model laid the foundations for the mathematical modeling of malaria [3]. Subsequently, works such as those by Lou and Zhao [4] incorporated biological control through larvivorous fish. Ghosh et al. [5] extended these models by considering variable populations and identifying backward bifurcation phenomena. Based on Ghosh et al. [5], which focused on human–vector and predator–prey interactions (larvivorous fish and mosquito larvae), in this study we incorporate intraspecific competition via the coefficients m_h for humans and m_v for female mosquitoes. Therefore, we propose the following model:

*Universidad Nacional del Santa, Chimbote, Perú. **Correspondence author** (2018825032@uns.edu.pe).
†Universidad Nacional Mayor de San Marcos, Lima, Perú. (rlopezc@unmsm.edu.pe).

$$\frac{dL}{dt} = gN_v - dL - d_1L^2 - \lambda_v L - \alpha_1 vLP - \alpha_1\alpha_2(1-v)LqEP \tag{2.1}$$

$$\frac{dN_v}{dt} = \lambda_v L - d_v N_v - m_v N_v^2 \tag{2.2}$$

$$\frac{dP}{dt} = rP\left(1 - \frac{P}{K}\right) + \gamma\alpha_1 vLP - qeP \tag{2.3}$$

$$\frac{dI_v}{dt} = c\beta(N_v - I_v)\frac{I_h}{N_h} - d_v I_v - m_v I_v N_v \tag{2.4}$$

$$\frac{dI_h}{dt} = b\beta(N_h - I_h)\frac{I_v}{N_h} - (d_h + \rho + \mu)I_h - m_h I_h N_h \tag{2.5}$$

$$\frac{dN_h}{dt} = \Gamma - d_h N_h - \mu I_h - m_h N_h^2, \tag{2.6}$$

where the variables and parameters are defined in Tables 2.1 and 2.2 bellow.

Table 2.1: Model variables.

Symbol	**Description**	**Units**
$L(t)$	Total density of mosquito larvae per unit surface area	individuals
$N_v(t)$	Total population of female mosquitoes	individuals
$I_v(t)$	Population of infected female mosquitoes	individuals
$P(t)$	Density of the larvivorous fish population in region one per unit surface area	individuals
$N_h(t)$	Total human population	individuals
$I_h(t)$	Population of infected humans	individuals

Table 2.2: Model parameters.

Parameter	**Description**	**Units**
v	Fraction of total breeding sites that are ponds, lakes, and rivers considered as region one	dimensionless
g	Egg-laying rate of adult female mosquitoes	1/day
λ_v	Larval maturation rate	1/day
β	Average number of bites per mosquito per unit time	1/day
d	Natural mortality rate of mosquito larvae	1/day
c	Probability of transmission from infected humans to mosquitoes	dimensionless
b	Probability of transmission from infected mosquitoes to humans	dimensionless
d_1	Density-dependent mortality rate of mosquito larvae	1/(day·individual)
d_v	Mortality rate of mosquitoes	1/day
r	Intrinsic growth rate of the fish population	1/day
d_h	Constant mortality rate of the human population	1/day
K	Carrying capacity of the fish population in region one	individuals
Γ	Recruitment rate of the human population	1/day
α_1	Larval predation rate by fish	1/(day·individual)

Parameter	Description	Units
α_2	Fraction of fish harvested from region one that are introduced into region two	dimensionless
m_v	Mosquito competition coefficient	1/(day.individual)
m_h	Human competition coefficient	1/(day.individual)
ρ	Recovery rate in humans	1/day
μ	Malaria-induced mortality	1/day
q	Catchability coefficient of fish	dimensionless
γ	Conversion efficiency from consumed larval biomass to fish biomass	dimensionless
E	Harvesting effort of larvivorous fish from region one	1/day

3. Basic qualitative properties.

Theorem 3.1 (Positivity of solutions). *Let $U \subset \mathbb{R}^6$ be the biologically feasible region defined by*

$$U = \left\{(L, N_v, P, I_v, I_h, N_h) \in \mathbb{R}^6 : L \geq 0,\ P \geq 0,\ N_v \geq 0,\ I_v \geq 0,\ I_h \geq 0,\ N_h > 0\right\}.$$

If the initial conditions of system (2.1)–(2.6) are

$$\begin{aligned} L(0) = L^0 \geq 0, \qquad N_v(0) = N_v^0 \geq 0, \qquad P(0) = P^0 \geq 0, \\ I_v(0) = I_v^0 \geq 0, \qquad I_h(0) = I_h^0 \geq 0, \qquad N_h(0) = N_h^0 > 0, \end{aligned}$$

then the solutions of the system belong to the set U.

Proof: We first prove that $L(t) \geq 0$, $\forall t \geq 0$. By contradiction, suppose there exists $t_1 \geq 0$ and exists $\delta_1 > 0$ such that

$$L(t_1) = 0 \quad \text{and} \quad L'(t) < 0, \quad \forall t \in [t_1, t_1 + \delta_1],$$

and furthermore $N_v(t) \geq 0$, $\forall t \in [t_1, t_1 + \delta_1]$. Then we have:

$$\begin{aligned} \frac{dL(t_1)}{dt} &= gN_v(t_1) - dL(t_1) - d_1 L(t_1)^2 - \lambda_v L(t_1) \\ &\quad - \alpha_1 v L(t_1) P(t_1) - \alpha_1 \alpha_2 (1 - v) L(t_1) qe P(t_1) \\ &= gN_v(t_1) \geq 0, \end{aligned}$$

which is a contradiction. Now, we must prove that $N_v(t) \geq 0$, $\forall t \geq 0$. By contradiction, suppose there exist $t_2 \geq 0$ and $\delta_2 > 0$ such that

$$N_v(t_2) = 0 \quad \text{and} \quad N_v'(t) < 0, \quad \forall t \in [t_2, t_2 + \delta_2],$$

and furthermore $L(t) \geq 0$, $\forall t \in [t_2, t_2 + \delta_2]$. Then we have:

$$\begin{aligned} \frac{dN_v(t_2)}{dt} &= \lambda_v L(t_2) - d_v N_v(t_2) - m_v N_v(t_2)^2 \\ &= \lambda_v L(t_2) \geq 0, \end{aligned}$$

which is again a contradiction. Similarly, the result can be verified for each of the remaining variables. □

Theorem 3.2 (Boundedness and biological feasibility of solutions). *The system (2.1)–(2.6) has a unique bounded solution with initial value*

$$x^0 = (L^0, N_v^0, P^0, I_v^0, I_h^0, N_h^0) \in W = \{(L, N_v, P, I_v, I_h, N_h) \in U : I_v \leq N_v,\ I_h \leq N_h\}.$$

Moreover, the compact set

$$\Omega = \left\{ \begin{array}{l} (L, N_v, P, I_v, I_h, N_h) \in W : \\ L \leq \dfrac{g\lambda_v}{d_1 d_v}, \quad P \leq \dfrac{K}{r}\left(\gamma\alpha_1 v \dfrac{g\lambda_v}{d_1 d_v} + (r - qE)\right), \\ N_v \leq \dfrac{g}{d_1}\left(\dfrac{\lambda_v}{d_v}\right)^2, \quad N_h \leq \dfrac{\Gamma}{d_h}, \quad I_h \leq N_h, \quad I_v \leq N_v \end{array} \right\},$$

attracts all positive solutions with initial conditions in W.

Proof: For any $x = (x_1, x_2, x_3, x_4, x_5, x_6) \in W$, since $f \in C^1(\text{int}(U))$, it follows that the system (2.1)–(2.6) has a unique solution $u(t, x)$ defined over its maximal interval of existence (a, b), with $0 \in (a, b)$ and $u(0) = x$, by Theorem 1 (Section 2.4) in Perko [6].

Let $u(t, x) = \big(u_1(t, x), u_2(t, x), u_3(t, x), u_4(t, x), u_5(t, x), u_6(t, x)\big) \in \mathbb{R}^6_+$, for all $t \in (a, b)$. By contradiction, it can be shown that

$$u_5(t, x) \leq u_6(t, x) \quad \text{and} \quad u_4(t, x) \leq u_2(t, x),$$

hence $u(t, x) \in W$ for all $t \in (a, b)$.

We now prove that the solution $u(t, x)$ is bounded for all $t \in (a, b)$, given the differential equations:

$$\frac{du_1}{dt} = gu_2 - d_1u_1^2, \qquad \frac{du_2}{dt} = \lambda_v u_1 - d_v u_2.$$

We define the function:

$$h(u_1, u_2) = \left(gu_2 - d_1u_1^2,\ \lambda_v u_1 - d_v u_2\right).$$

Then

$$Dh(u_1, u_2) = \begin{pmatrix} -2d_1u_1 & g \\ \lambda_v & -d_v. \end{pmatrix}$$

- The function h is cooperative on $\mathbb{R}^2_+$; that is,

$$\frac{\partial h_i}{\partial u_j} \geq 0, \quad \text{for } 1 \leq i, j \leq 2,\ i \neq j.$$

- The Jacobian matrix $Dh(u_1, u_2)$ is irreducible for all $(u_1, u_2) \in \mathbb{R}^2_+$.
- $h(0, 0) = (0, 0)$ and $h_i(u_1, u_2) \geq 0,\ \forall (u_1, u_2) \in \mathbb{R}^2_+$ with $u_i = 0;\ i = 1, 2$.
- h is strictly subhomogeneous on $\mathbb{R}^2_+$; that is, $h(p(u_1, u_2)) > ph(u_1, u_2)$ for any $p \in (0, 1)$ and $(u_1, u_2) \in \text{Int}(\mathbb{R}^2_+)$.

Evaluating the Jacobian matrix at $(0, 0)$:

$$Dh(0, 0) = \begin{pmatrix} 0 & g \\ \lambda_v & -d_v \end{pmatrix}.$$

The spectral radius of $Dh(0, 0)$ is defined as $\rho(Dh(0, 0)) = \max\{\text{Re}\,\lambda : \det(\lambda I - Dh(0, 0)) = 0\}$. Thus, the characteristic equation is:

$$x^2 + xd_v - \lambda_v g = 0 \Rightarrow x = \frac{-d_v \pm \sqrt{d_v^2 + 4\lambda_v g}}{2} \Rightarrow \rho(Dh(0, 0)) > 0.$$

Therefore, by Corollary 3.2 of Zhao and Jing [7], the system has an equilibrium point

$$\left(\frac{g\lambda_v}{d_v d_1},\ \frac{g}{d_1}\left(\frac{\lambda_v}{d_v}\right)^2\right),$$

which is globally asymptotically stable with respect to all initial values in $\mathbb{R}^2_+ \setminus \{(0, 0)\}$.

Now, we observe that:

$$\frac{dL}{dt} \leq gN_v - d_1L^2, \qquad \frac{dN_v}{dt} \leq \lambda_v L - d_v N_v.$$

By the comparison principle, there exist positive constants M_1 and M_2 such that:

$$L(t) \leq M_1, \qquad N_v(t) \leq M_2, \quad \forall t \in [0, \sigma_x).$$

$$\frac{dP}{dt} = rP\left(1 - \frac{P}{K}\right) + \gamma\alpha_1 vLP - qEP$$

$$K\frac{dP}{dt} \leq (K\gamma\alpha_1 v M_1 + K(r - qE) - rP)\, P.$$

First, suppose that:

$$0 \leq K\gamma\alpha_1 v M_1 + K(r - qE) - rP \Rightarrow P \leq \frac{K}{r}(\gamma\alpha_1 v M_1 + r - qE).$$

From which we directly obtain:

$$P \leq \frac{K}{r}\left(\gamma\alpha_1 v \frac{g\lambda_v}{d_v d_1} + r - qE\right).$$

Now, suppose that:

$$0 > K\gamma\alpha_1 v M_1 + K(r - qE) - rP \Rightarrow P > \frac{K}{r}\left(\gamma\alpha_1 v M_1 + r - qE\right).$$

$$K\frac{1}{(rP - K\gamma\alpha_1 v M_1 - K(r - qE))\, P}\frac{dP}{dt} \leq -1.$$

Then, we have:

$$\frac{rP - K\gamma\alpha_1 v M_1 - K(r - qE)}{P} \leq \exp\left(-t(\gamma\alpha_1 v M_1 + r - qE)\right)\cdot\frac{rP(0) - K\gamma\alpha_1 v M_1 - K(r - qE)}{P(0)}.$$

When $t \to \infty$, it follows that:

$$\frac{rP - K\gamma\alpha_1 v M_1 - K(r - qE)}{P} \leq 0 \quad \Rightarrow \quad rP \leq K\left(\gamma\alpha_1 v \frac{g\lambda_v}{d_v d_1} + r - qE\right).$$

Thus, in all cases we conclude that:

$$P \leq \frac{K}{r}\left(\gamma\alpha_1 v \frac{g\lambda_v}{d_v d_1} + r - qE\right).$$

From the previous arguments we have:

$$\limsup_{t\to\infty}(L_v(t), N_v(t)) \leq \left(\frac{g\lambda_v}{d_v d_1}, \frac{g}{d_1}\left(\frac{\lambda_v}{d_v}\right)^2\right).$$

Moreover

$$\limsup_{t\to\infty} P(t) \leq \frac{K}{r}\left(\gamma\alpha_1 v \frac{g\lambda_v}{d_v d_1} + (r - qE)\right)$$

$$\frac{dN_h(t)}{dt} = \Gamma - d_h N_h - \mu_h - m_h(N_h)^2 \leq \Gamma - d_h N_h.$$

Hence

$$N_h \leq \Gamma\left(\frac{1}{d_h} - \frac{e^{-d_h t}}{d_h}\right) + N_h(0)e^{-d_h t}.$$

When $t \to \infty$, we obtain $N_h \leq \frac{\Gamma}{d_h}$. Therefore, Ω is globally attractive, and since the solution $u(t, x)$ is bounded on $[0, b)$, we have $b = \infty$ by Corollary 2(Section 2.4) in Perko [6]. □

4. Existence of equilibria and the basic number R_0.

Theorem 4.1 (Disease-Free Equilibrium). *The system (2.1)–(2.6) admits a unique disease-free equilibrium point*

$$D = (L^*, N_v^*, P^*, 0, 0, N_h^*) \quad \textit{with} \quad N_v^* \neq 0, \quad P^* \neq 0,$$

under the conditions:

$$\frac{d_v r(d + \lambda_v + AK)}{AKqEd_v + rg\lambda_v} < 1,$$

$$d_v N_v^* + m_v(N_v^*)^2 > \frac{(qE - r)\lambda_v}{\gamma\alpha_1 v},$$

where N_v^* is the positive root of the cubic equation:

$$a_3x^3 + a_2x^2 + a_1x + a_0 = 0,$$

with:

$$\begin{aligned}
A &= \alpha_1 v + \alpha_1\alpha_2(1-v)qE,\\
a_3 &= d_1 m_v^2 + \frac{AK}{r}\gamma\alpha_1 v m_v^2,\\
a_2 &= 2d_1 d_v m_v + 2\frac{AK}{r}\gamma\alpha_1 v m_v d_v,\\
a_1 &= d_1 d_v^2 + (d+\lambda_v)\lambda_v m_v + AK\lambda_v m_v + \frac{AK}{r}\gamma\alpha_1 v d_v^2 - \frac{AK}{r}\alpha_1 qE m_v,\\
a_0 &= -g\lambda_v^2 + d\lambda_v d_v + \lambda_v^2 d_v + AK\lambda_v d_v - \frac{AK}{r}\lambda_v qE d_v.
\end{aligned}$$

The expressions for the other variables in the disease-free equilibrium are:

$$P^* = \frac{K}{r}\left(r + \gamma\alpha_1 v L^* - qE\right), \quad L^* = \frac{d_v N_v^* + m_v (N_v^*)^2}{\lambda_v}, \quad N_h^* = \frac{-d_h + \sqrt{d_h^2 + 4m_h\Gamma}}{2m_h}.$$

Proof:

Setting $I_h = 0$ and $I_v = 0$ in the system (2.1)–(2.6) and equating the right-hand sides to zero, we obtain the expressions for P^*, L^* and N_h^*.

In addition, we obtain the equation:

$$q(x) = a_3x^3 + a_2x^2 + a_1x + a_0 = 0,$$

whose positive real solution is N_v^*.

It can be noted that:

$$q''(x) > 0, \quad \forall x \in [0,\infty),$$

that is, the polynomial is convex on $[0,\infty)$. For the polynomial $q(x)$ to have a positive real root, the condition $q(0) < 0$ must hold, which is equivalent to:

$$\frac{d_v r(d+\lambda_v + AK)}{AKqed_v + rg\lambda_v} < 1$$

□

4.1. Basic Reproduction Number R_0.

The subsystem describing the dynamics of the infected compartments is given by:

$$\begin{aligned}
\frac{dI_v(t)}{dt} &= c\beta(N_v - I_v)\frac{I_h}{N_h} - d_v I_v - m_v I_v N_v,\\
\frac{dI_h(t)}{dt} &= b\beta(N_h - I_h)\frac{I_v}{N_h} - (d_h + \rho + \mu)I_h - m_h I_h N_h.
\end{aligned}$$

The basic reproduction number, following the epidemiological definition in Basáñez and Rodríguez [3], is defined as the number of secondary infections generated by a typical infected individual in a disease-free population.

Following the next-generation matrix approach [8], where $\mathcal{F}$ represents the rate of appearance of new infections, and $\mathcal{V}$ represents the rate of transitions and removals:

$$\mathcal{F} = \begin{pmatrix} c\beta(N_v - I_v)\dfrac{I_h}{N_h} \\ b\beta(N_h - I_h)\dfrac{I_v}{N_h} \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} d_v I_v + m_v I_v N_v \\ (d_h + \rho + \mu)I_h + m_h I_h N_h \end{pmatrix}.$$

Matrices F and V are the Jacobians of $\mathcal{F}$ and $\mathcal{V}$ with respect to I_v and I_h, respectively, evaluated at the disease-free equilibrium D:

$$F = \begin{pmatrix} 0 & \dfrac{c\beta N_v^*}{N_h^*} \\ b\beta & 0 \end{pmatrix}, \quad V = \begin{pmatrix} d_v + m_v N_v^* & 0 \\ 0 & d_h + \rho + \mu + m_h N_h^* \end{pmatrix}.$$

The next-generation matrix is given by:

$$FV^{-1} = \begin{pmatrix} 0 & \dfrac{c\beta N_v^*}{(d_h+\rho+\mu+m_h N_h^*)N_h^*} \\ \dfrac{b\beta}{d_v+m_v N_v^*} & 0 \end{pmatrix}.$$

Then, the spectral radius of the matrix FV^{-1} is the basic reproduction number R_0:

$$R_0 = \sqrt{\frac{bc\beta^2 N_v^*}{(d_v+m_v N_v^*)(d_h+\rho+\mu+m_h N_h^*)N_h^*}}. \tag{4.1}$$

Theorem 4.2 (Endemic Equilibrium). *The system (2.1)–(2.6) has at least one endemic equilibrium point*

$$E_E = (\widehat{L}, \widehat{N}_v, \widehat{P}, \widehat{I}_v, \widehat{I}_h, \widehat{N}_h),$$

with $\widehat{N}_v \neq 0$ and $\widehat{P} \neq 0$, under the following conditions:

$$\frac{d_v r(d+\lambda_v+AK)}{AKqEd_v + rg\lambda_v} < 1,$$
$$d_v\widehat{N}_v + m_v(\widehat{N}_v)^2 > \frac{(qE-r)\lambda_v}{\gamma\alpha_1 v},$$

and

$$\frac{-(d_h+\mu)+\sqrt{(d_h+\mu)^2+4m_h\Gamma}}{2m_h} < \widehat{N}_v \leq \frac{-d_h+\sqrt{d_h^2+4m_h\Gamma}}{2m_h}.$$

The variable $\widehat{N}_v$ is the unique positive root of the cubic equation:

$$a_3x^3 + a_2x^2 + a_1x + a_0 = 0,$$

and $\widehat{N}_h$ is one of the positive roots of the quartic equation:

$$b_4x^4 + b_3x^3 + b_2x^2 + b_1x + b_0 = 0.$$

The coefficients are defined as:

$$A = \alpha_1 v + \alpha_1\alpha_2(1-v)qE,$$
$$a_3 = d_1m_v^2 + \frac{AK}{r}\gamma\alpha_1 v m_v^2,$$
$$a_2 = 2d_1d_vm_v + 2\frac{AK}{r}\gamma\alpha_1 v m_v d_v,$$
$$a_1 = d_1d_v^2 + (d+\lambda_v)\lambda_v m_v + AK\lambda_v m_v + \frac{AK}{r}\gamma\alpha_1 v d_v^2 - \frac{AK}{r}\alpha_1 qe m_v,$$
$$a_0 = -g\lambda_v^2 + d\lambda_v d_v + \lambda_v^2 d_v + AK\lambda_v d_v - \frac{AK}{r}\lambda_v qE d_v,$$
$$b_4 = c\beta m_h^2,$$
$$b_3 = m_h c\beta(d_h+\rho+\mu) + d_h c\beta m_h - m_h\mu(d_v+m_v\hat{N}_v),$$
$$b_2 = m_h bc\beta^2\hat{N}_v + d_h c\beta(d_h+\rho+\mu) - \mu(d_v+m_v\hat{N}_v)(d_h+\rho+\mu) - \Gamma c\beta m_h,$$
$$b_1 = \mu bc\beta^2\hat{N}_v + d_h bc\beta^2\hat{N}_v - \Gamma c\beta(d_h+\rho+\mu),$$
$$b_0 = -\Gamma bc\beta^2\hat{N}_v$$

The expressions for the other variables at the endemic equilibrium are:

$$\hat{L} = \frac{d_v\hat{N}_v + m_v\hat{N}_v^2}{\lambda_v}, \quad \hat{P} = \frac{K}{r}(r - qE + \gamma\alpha_1 v\hat{L}), \quad \hat{I}_v = \frac{(d_h+\rho+\mu+m_h\hat{N}_h)\hat{I}_h\hat{N}_h}{b\beta(\hat{N}_h-\hat{I}_h)},$$

and $\hat{I}_h = \frac{\Gamma - d_h\hat{N}_h - m_h\hat{N}_h^2}{\mu}$.

Moreover, if $R_0 > 1$ *and*

$$\begin{aligned}3c^2\beta^2(d_h+\rho+\mu)^2 + 3\mu^2(d_v+m_v\hat{N}_v)^2 + 3c^2\beta^2 d_h^2 + 2c^2\beta^2 d_h(d_h+\rho+\mu)\\ + 4m_h\Gamma c^2\beta^2 < 2c\beta\mu(d_h+\rho+\mu)(d_v+m_v\hat{N}_v) + 4m_h bc^2\beta^3\hat{N}_v\\ + 2c\beta\mu d_h(d_v+m_v\hat{N}_v)m_h,\end{aligned}$$

then, $\hat{N}_h$ *has a unique positive real value; that is,* E_E *is unique.*

Proof:

The differential system (2.1)–(2.6), when each equation is set to zero, yields the expressions for $\widehat{L}, \widehat{P}, \widehat{I}_v$, and $\widehat{I}_h$.

From the expression for $\widehat{I}_v$, its positivity requires $\widehat{N}_v$ to satisfy the following bounds:

$$0 < \frac{-(d_h+\mu)+\sqrt{(d_h+\mu)^2+4m_h\Gamma}}{2m_h} < \widehat{N}_h \leq \frac{-d_h+\sqrt{d_h^2+4m_h\Gamma}}{2m_h} = N_h^*.$$

Furthermore, the equation:

$$q(x) = a_3x^3 + a_2x^2 + a_1x + a_0 = 0,$$

has the unique positive root $\widehat{N}_v$, and the equation:

$$p(x) = b_4x^4 + b_3x^3 + b_2x^2 + b_1x + b_0 = 0,$$

where $\widehat{N}_h$ is one of its positive real roots.

Since $\widehat{N}_h \in (0, N_h^*]$, evaluating $p(x)$ gives:

$$p(0) = -\Gamma bc\beta^2\widehat{N}_v = b_0 < 0, \quad p(N_h^*) = \mu N_h^*(R_0^2 - 1),$$

As $p(0) < 0$, the following cases may occur:

- If $R_0 \geq 1$, then $p(N_h^*) \geq 0$. Therefore, the polynomial $p(x)$ intersects the x-axis at some $\widehat{N}_h \in (0, N_h^*]$.
- If $R_0 < 1$, then $p(N_h^*) < 0$, implying that there is no positive root of $p(x)$ in $[0, N_h^*]$, and the solution would be found for $x > N_h^*$.

To guarantee the uniqueness of the positive real root of $p(x)$ in the first case, the graph of $p(x)$ must be convex on $(0, \infty)$, which holds if:

$$p''(x) > 0, \quad \forall x \in (0, \infty).$$

The second derivative of the polynomial is:

$$\begin{aligned}p''(x) = 12c\beta(m_h)^2x^2 + 6\left[m_hc\beta(2d_h+\rho+\mu) - m_h\mu(d_v+m_v\widehat{N}_v)\right]x\\ + \left[m_hbc\beta^2\widehat{N}_v + d_hc\beta(d_h+\rho+\mu) - \mu(d_v+m_v\widehat{N}_v)(d_h+\rho+\mu) - \Gamma c\beta m_h\right].\end{aligned}$$

Since $p''(x)$ is a quadratic polynomial, to be strictly positive on $(0, \infty)$ its discriminant Δ must be negative. That is, the following must hold:

$$\begin{aligned}3c^2\beta^2(d_h+\rho+\mu)^2 + 3\mu^2(d_v+m_v\widehat{N}_v)^2 + 3c^2\beta^2d_h^2 + 2c^2\beta^2d_h(d_h+\rho+\mu) + 4m_h\Gamma c^2\beta^2\\ < 2c\beta\mu(d_h+\rho+\mu)(d_v+m_v\widehat{N}_v) + 4m_hbc^2\beta^3\widehat{N}_v + 2c\beta\mu d_h(d_v+m_v\widehat{N}_v)m_h.\end{aligned}$$

This condition ensures the uniqueness of the positive real root of the polynomial $p(x)$.

□

Remark 1. *It is observed that at the disease-free equilibrium* D*, the densities of larvae, fish, and mosquito population coincide with those at the endemic equilibrium* E_E*, that is,* $L^* = \widehat{L},\ P^* = \widehat{P},\ N_v^* = \widehat{N}_v$ *and* $\widehat{N}_h \leq N_h^*$.

5. Stability.

5.1. Local Stability.

If $x = (x_1, x_2, x_3, x_4, x_5, x_6)$ and we define the vector function f as

$$f(x) = (f_1(x), f_2(x), f_3(x), f_4(x), f_5(x), f_6(x)),$$

where

$$\begin{aligned}
f_1(x) &= gx_2 - dx_1 - d_1x_1^2 - \lambda_v x_1 - \alpha_1 v x_1 x_3 - \alpha_1\alpha_2(1-v)qEx_1x_3,\\
f_2(x) &= \lambda_v x_1 - d_v x_2 - m_v x_2^2,\\
f_3(x) &= rx_3\left(1 - \frac{x_3}{K}\right) + \gamma\alpha_1 v x_1 x_3 - qEx_3,\\
f_4(x) &= \beta\frac{(x_2 - x_4)x_5}{x_6} - d_v x_4 - m_v x_4 x_2,\\
f_5(x) &= b\beta\frac{(x_6 - x_5)x_4}{x_6} - (d_h + \rho + \mu)x_5 - m_h x_6 x_5,\\
f_6(x) &= \Gamma - d_h x_6 - \mu x_5 - m_h x_6^2.
\end{aligned}$$

Thus, the system (2.1)–(2.6) can be written as

$$\frac{dx}{dt} = f(x) \tag{5.1}$$

Theorem 5.1. *The disease-free equilibrium D is locally asymptotically stable if $R_0 < 1$ and locally unstable if $R_0 > 1$, where $N_v^* \neq 0$ and $P^* \neq 0$.*

Proof: From the equivalent system (5.1), the characteristic polynomial of the Jacobian matrix $Jf(D)$ is:

$$P(x) = P_1(x)\, P_2(x)\, P_3(x),$$

where:

$$\begin{aligned}
P_1(x) &= -d_v - 2N_v^* m_v - x,\\
P_2(x) &= (b\beta)\left(-\frac{c\beta N_v^*}{N_h^*}\right) + (d_h + \rho + \mu + m_h N_h^* + x)(d_v + N_v^* m_v + x),\\
P_3(x) &\text{ is the characteristic polynomial of the matrix } J_3,
\end{aligned}$$

with:

$$J_3 = \begin{pmatrix}
-d - 2d_1L^* - \lambda_v - AP^* & -AL^* & g\\
\gamma\alpha_1 v P^* & r\left(1 - \dfrac{2P^*}{K}\right) + \gamma\alpha_1 v L^* - qe & 0\\
\lambda_v & 0 & -d_v - 2m_v N_v^*
\end{pmatrix}.$$

We observe that $P_1(x)$ has a negative real root. Now, expanding $P_2(x)$, we obtain:

$$P_2(x) = x^2 + (d_h + \rho + \mu + m_h N_h^* + d_v + N_v^* m_v)\, x + (d_h + \rho + \mu + m_h N_h^*)(d_v + N_v^* m_v)\,[1 - R_0^2].$$

- If $R_0 < 1$, then $P_2(x)$ has two negative real roots.
- If $R_0 > 1$, then $P_2(x)$ has one positive real root (instability).

As for $P_3(x)$, all its coefficients are positive, which implies that all its roots are negative real numbers.

□

Theorem 5.2. *The endemic equilibrium E_E is locally asymptotically stable if $R_0 > 1$ and $\frac{\Gamma}{\mu} \geq \widehat{N}_h$, with $\widehat{N}_v \neq 0, \widehat{P} \neq 0$.*

Proof:

The characteristic polynomial of the Jacobian matrix in E_E is

$$P(x) = P_1(x)P_2(x).$$

Here $P_2(x)$ is the characteristic polynomial of the matrix

$$\begin{pmatrix} -d - 2d_1\hat{L} - \lambda_v - A\hat{P} & -A\hat{L} & g \\ \gamma\alpha_1 v\hat{P} & r\left(1 - \dfrac{2\hat{P}}{K}\right) + \gamma\alpha_1 v\hat{L} - qE & 0 \\ \lambda_v & 0 & -d_v - 2m_v\hat{N}_v \end{pmatrix}$$

previously analyzed in the proof of the disease-free equilibrium, which determined that it always has three negative solutions.

Now we analyze the polynomial

$$P_1(x) = x^3 + a_1x^2 + a_2x + a_3,$$

where:

$$\begin{aligned}
a_1 &= \left(d_v + m_v\hat{N}_v + \frac{c\beta\hat{I}_h}{\hat{N}_h}\right) + \left(d_h + 2m_h\hat{N}_h\right) + \left(d_h + \rho + \mu + \hat{N}_h m_h + \frac{b\beta\hat{I}_v}{\hat{N}_h}\right), \\
a_2 &= -\frac{bc\beta^2(\hat{N}_v - \hat{I}_v)(\hat{N}_h - \hat{I}_h)}{\hat{N}_h^2} + \left(d_h + 2m_h\hat{N}_h\right)\left(d_v + m_v\hat{N}_v + \frac{c\beta\hat{I}_h}{\hat{N}_h}\right) \\
&\quad + \left(d_v + m_v\hat{N}_v + \frac{c\beta\hat{I}_h}{\hat{N}_h}\right)\left(d_h + \rho + \mu + \hat{N}_h m_h + \frac{b\beta\hat{I}_v}{\hat{N}_h}\right) \\
&\quad + \left(d_h + \rho + \mu + \hat{N}_h m_h + \frac{b\beta\hat{I}_v}{\hat{N}_h}\right)\left(d_h + 2m_h\hat{N}_h\right) \\
&\quad + \mu\left(\frac{b\beta\hat{I}_v\hat{I}_h}{\hat{N}_h^2} - m_h\hat{I}_h\right), \\
a_3 &= -\left(d_h + 2m_h\hat{N}_h\right)\frac{bc\beta^2(\hat{N}_v - \hat{I}_v)(\hat{N}_h - \hat{I}_h)}{\hat{N}_h^2} \\
&\quad + \left(d_h + \rho + \mu + \hat{N}_h m_h + \frac{b\beta\hat{I}_v}{\hat{N}_h}\right)\left(d_v + m_v\hat{N}_v + \frac{c\beta\hat{I}_h}{\hat{N}_h}\right)\left(d_h + 2m_h\hat{N}_h\right) \\
&\quad + \mu\left(d_v + \hat{N}_v m_v + \frac{c\beta\hat{I}_h}{\hat{N}_h}\right)\left(\frac{b\beta\hat{I}_v\hat{I}_h}{\hat{N}_h^2} - m_h\hat{I}_h\right) \\
&\quad - \mu\frac{bc\beta^2(\hat{N}_v - \hat{I}_v)(\hat{N}_h - \hat{I}_h)\hat{I}_h}{\hat{N}_h^3}.
\end{aligned}$$

Applying the Routh–Hurwitz criterion given in Kiseliov et al. [9] to the polynomial P_1, for it to have three roots with negative real part (two complex conjugate roots and one real root) the following must hold:

$$\Delta_1 = a_1 > 0, \quad \Delta_2 = a_1a_2 - a_3 > 0, \quad \text{and} \quad a_3 > 0.$$

Using that $\Gamma/\mu \geq \hat{N}_h$ and $R_0 > 1$, the required conditions are satisfied. This completes the proof.

□

5.2. Global stability of the disease-free equilibrium.

Theorem 5.3. *If $R_{00} \leq R_0 \leq 1$, then the disease-free equilibrium D is globally asymptotically stable for the system (2.1)–(2.6) in* $\mathrm{int}(W)$.

Moreover, the epidemiological threshold R_{00} is given by:

$$R_{00} = \sqrt{\frac{d_h\, bc\, \beta^2\, N_v^*}{d_v\, \Gamma\, (d_h + \rho + \mu)}}.$$

The complete proof of Theorem 5.3 is provides by Tamariz [10] in Theorem 4.6.3, but it follows from the decomposition of the full system into two invariant subsystems and the application of global stability results from the literature:

- First three equations of (2.1)-(2.6). Global stability is established via the Lyapunov stability theorem (Perko [6], Barreira [11]).
- Last three equations. Global stability is obtained by combining the threshold R_{00} with Corollary 3.2 of Zhao [7], Lemma 1.2.1' of Zhao [12], and Theorem 3.2 with Remark 4.6 of Hirsch et al. [13].

6. Numerical Simulations.

Recall that the vector $(L, N_v, P, I_v, I_h, N_h)$ represents, respectively, the populations of larvae, fish, infected mosquitoes, total mosquito population, infected humans, and total human population for each component of the vector.

6.1. Population Evolution when $\mathbf{R_0 < 1}$ and $\mathbf{R_0 > 1}$. We analyze the evolution of the populations L, P, N_v, and N_h for the cases $R_0 < 1$ and $R_0 > 1$. The parameter values given in Table 6.1 correspond to a disease-free equilibrium point D. All parameter values are taken from Ghosh et al.[5], except for the intra-specific competition coefficients m_h and m_v, which are proposed in this study.

Table 6.1: Parameter values for the disease-free equilibrium D.

Parameter	Value	Parameter	Value
g	60	K	2000
d	0.05	c	0.3
d_1	0.02	β	0.2
λ_v	0.0625	d_h	0.00003913
v	0.2	d_v	0.05
α_1	0.2	ρ	0.005
α_2	0.5	μ	0.00005
q	0.3	b	0.3
e	0.5	Γ	0.8
r	0.2	m_v	0.002
γ	0.1	m_h	0.004

From these values, we obtain $R_0 \approx 0.79 < 1$ ($R_{00} \approx 0.09$) and the disease-free equilibrium point $D = (12.46,\ 10.86,\ 998.53,\ 0,\ 0,\ 14.14)$, which is locally asymptotically stable. Figure 6.1 shows the evolution, according to the model, of the involved populations over a period of 50 days. It is observed that both the infected human and infected mosquito populations decrease and tend toward the disease-free equilibrium. Initially, a larger quantity of larvivorous fish is required so that, by consuming larvae, this population ceases to grow. Subsequently, as the larval population decreases, a smaller number of larvivorous fish will be needed.

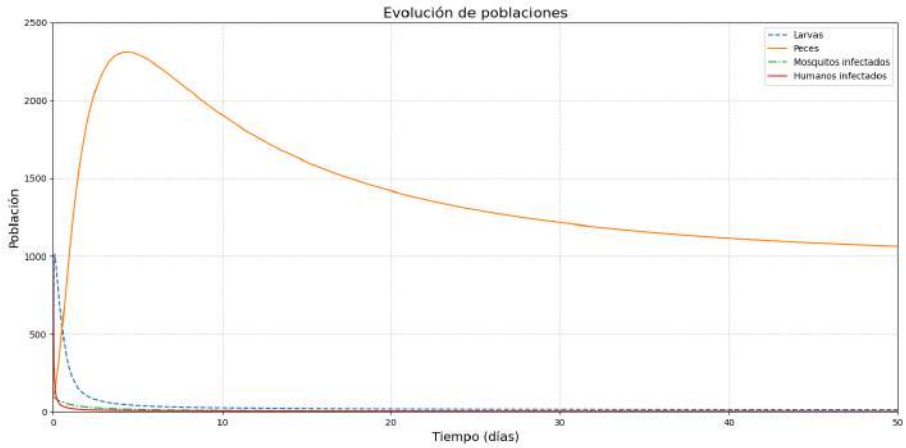
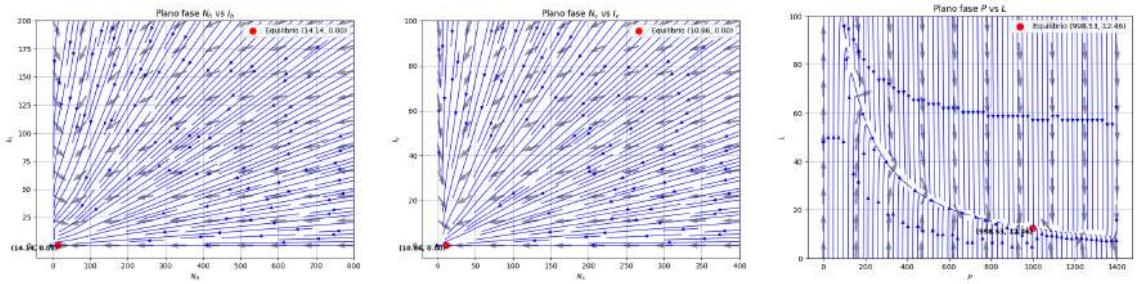


Figure 6.1: Population evolution with $R_0 < 1$.

In Figure 6.2, the phase planes N_h–I_h, N_v–I_v, and P–L are shown, where the direction fields for each case and the location of the equilibrium point (in red) can be observed. In each phase plane of system (2.1)–(2.6), for the parameter values given in Table 6.1, the asymptotic stability of the infection-free equilibrium point D is illustrated.



(a) L-I_v plane (b) I_v-I_h plane (c) I_h-P plane

Figure 6.2: Phase planes showing convergence to the disease-free equilibrium ($R_0 < 1$).

We now consider the parameter values given in Table 6.2, which correspond to the endemic equilibrium point E_E. All parameter values are taken from Ghosh et al. [5], except for the parameters m_h and m_v, which are specific to this study.

Table 6.2: Parameter values for the endemic equilibrium point E.

Parameter	Value	Parameter	Value
g	60	K	2000
d	0.05	c	0.5
d_1	0.02	β	0.3
λ_v	0.0625	d_h	0.00003913
v	0.2	d_v	0.05
α_1	0.2	ρ	0.005
α_2	0.5	μ	0.00005
q	0.3	b	0.5
e	0.5	Γ	0.3
r	0.2	m_v	0.000002
γ	0.1	m_h	0.000004

We obtain $R_0 \approx 2.8 > 1$, and the endemic equilibrium point is

$$E_E(23.24,\ 29.01,\ 1429.53,\ 19.03,\ 168.76,\ 265.14),$$

which is locally asymptotically stable. Figure 6.3 shows the evolution, according to the model, of the populations involved over a 50-day period. It can be observed that both the infected human and infected mosquito populations decrease and converge to the endemic equilibrium at a slower rate than in the case of the disease-free equilibrium. Consequently, a larger initial number of larvivorous fish will be required to consume the larvae and halt their population growth, after which the larval population will decrease, reducing the required number of larvivorous fish.

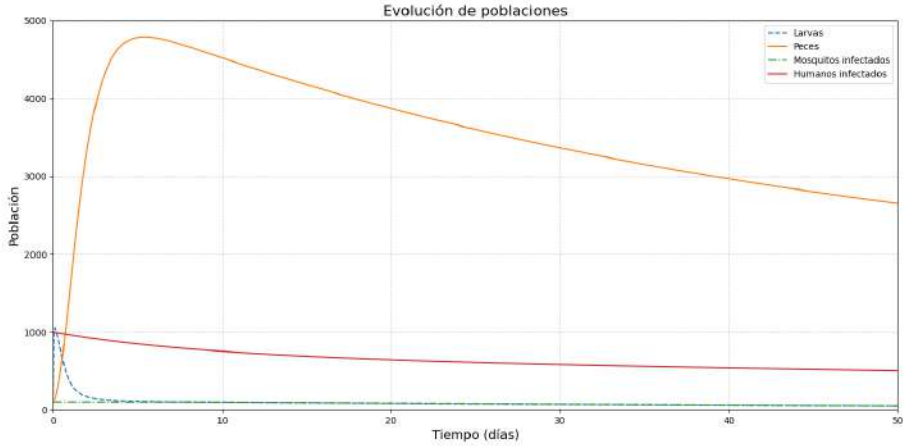
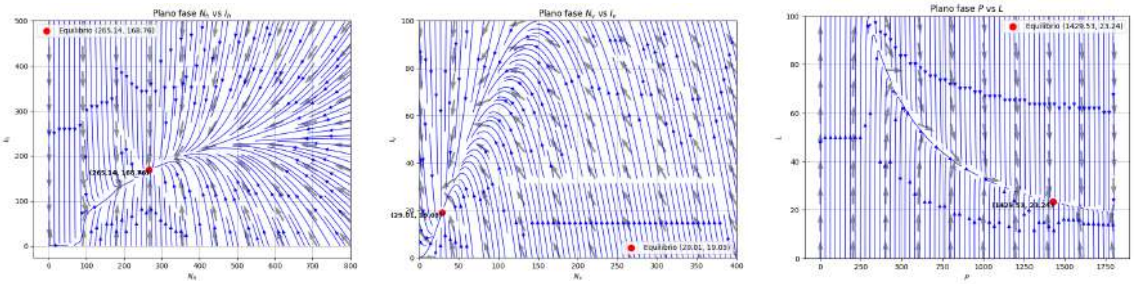


Figure 6.3: Population evolution in the endemic regime ($R_0 > 1$).

Figure 6.4 shows the phase planes $N_h - I_h$, $N_v - I_v$, and $P - L$, where the direction field for each and the location of the equilibrium point, marked in red, can be observed. In each phase plane of system (2.1)–(2.6), for the values given in Table 6.2, the asymptotic stability of the endemic equilibrium point E_E is shown.



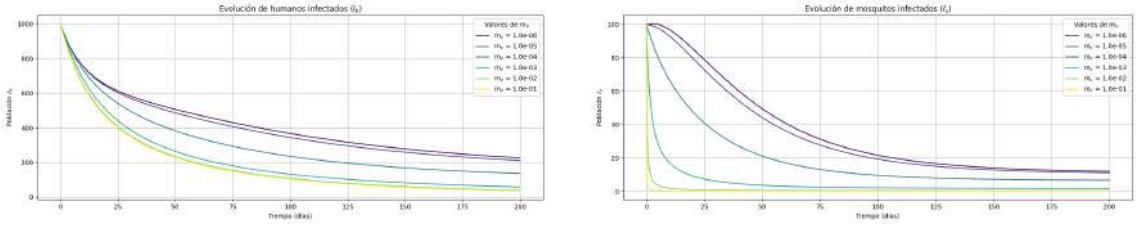
(a) L-I_v plane (b) I_v-I_h plane (c) I_h-P plane

Figure 6.4: Trajectories towards the endemic equilibrium in different phase planes ($R_0 > 1$).

6.2. of the Infected Populations I_v and I_h Varying $\mathbf{m_v}$ and/or $\mathbf{m_h}$. In this analysis, the parameters of system (2.1)–(2.6) take the same values as those given in Table 6.2, except for m_v and m_h, which vary within the interval $[0.000001,\ 0.1]$.

Figures 6.5 display the curves for the populations of infected humans and infected mosquitoes, respectively, when varying the intraspecific competition coefficient among mosquitoes, m_v, while keeping the intraspecific competition coefficient among humans, m_h, constant. Results indicate that as m_v increases—that is, as competition among mosquitoes becomes stronger—the infected mosquito population decreases, and the infected human population also decreases. This suggests that intraspecific competition among mosquitoes limits the growth of the vector population, thereby reducing the spread of the parasite.

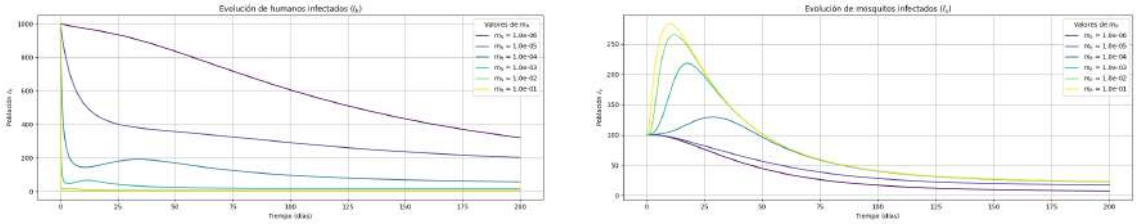
The I_h curve exhibits a much steeper decline than the I_v curve, indicating that the dynamics of transmission to humans are more sensitive to changes in m_v. This is possibly because even a small change in the mosquito population has a large impact on the rate of infectious bites to humans.



(a) Evolution of I_h (b) Evolution of I_v

Figure 6.5: Evolution of I_h and I_v under variations in m_v.

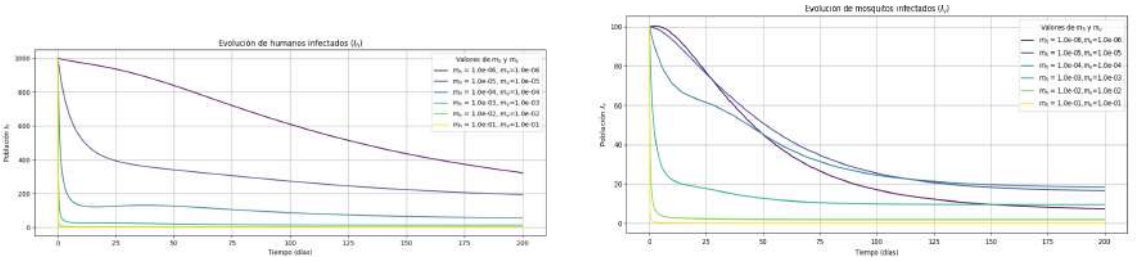
Figures 6.6 show the curves for the infected mosquito population and the infected human population, respectively, when varying the intraspecific competition coefficient among humans while keeping constant the intraspecific competition coefficient among mosquitoes. It can be observed that as m_h increases, the infected human population does not decrease abruptly when $m_h = 10^{-6}$; however, for successive larger values of m_h, the decline in the infected human population becomes much more pronounced, tending towards the endemic equilibrium. This may be interpreted as higher competition among humans for treatments, medical care, hospital beds, and related resources, which could prolong the infection period, increase malaria-induced mortality, or raise vulnerability. In contrast, the infected mosquito population decreases smoothly for smaller values of m_h, but as m_h becomes larger, the number of infected mosquitoes exhibits growth peaks within the first 30 days before declining, due to the reduction in the infected human population, ultimately approaching the endemic equilibrium.



(a) Evolution of I_h (b) Evolution of I_v

Figure 6.6: Evolution of I_h and I_v under variations in m_h.

Subsequently, by varying m_v and m_h simultaneously, with $m_v = m_h$, Figure 6.7 shows that increasing both coefficients leads to a concurrent decrease in the populations of infected humans and infected mosquitoes, with both tending toward extinction.



(a) Evolution of I_h (b) Evolution of I_v

Figure 6.7: Evolution of I_h and I_v for simultaneous variations in m_h and m_v.

6.3. Evolution of $\mathbf{R_0}$ by varying $\mathbf{m_v}$ and/or $\mathbf{m_h}$.

In this analysis, the parameters of (2.1)–(2.6) take the same values as those given in Table 6.2, except for m_v and m_h, which will vary. Figure 6.8 shows the behavior of R_0 when the values of m_h vary while m_v remains constant. Results indicate that greater competition among humans leads R_0 to approach a value greater than 1, implying that the disease will become endemic. This indicates the importance of implementing public policies that reduce competition for consumption

resources among humans, such as improving access to healthcare facilities and medications for malaria treatment.

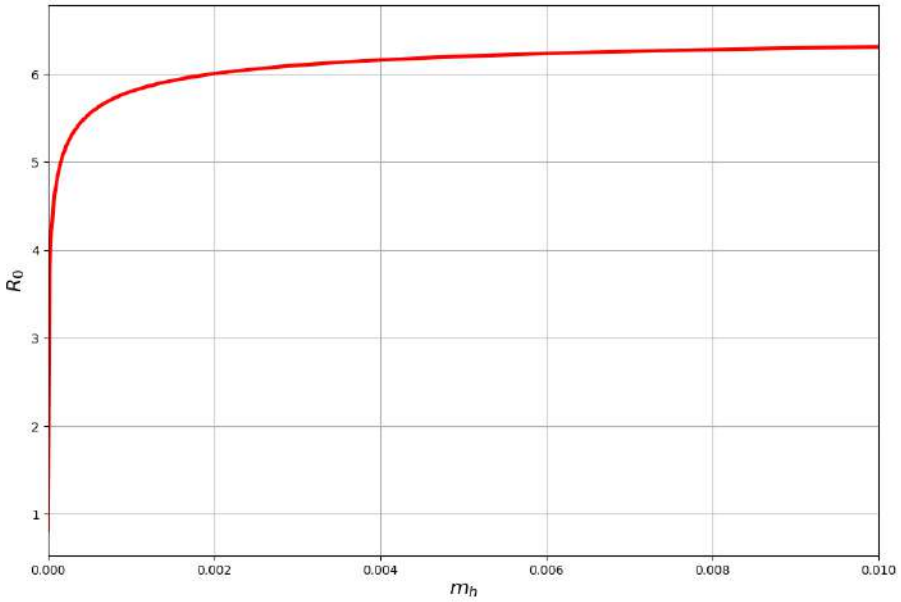


Figure 6.8: Relationship between R_0 and m_h.

In Figure 6.9, the behavior of R_0 is shown when the values of m_v vary while the value of m_h remains constant. It can be observed that greater competition among vectors causes R_0 to tend toward a value less than one, which implies that the disease will tend to disappear. In this case, public policies aimed at increasing competition among vectors would lead to the disappearance of the infection in the human population. Examples of such policies include the elimination of mosquito breeding sites, the use of bed nets, and similar measures.

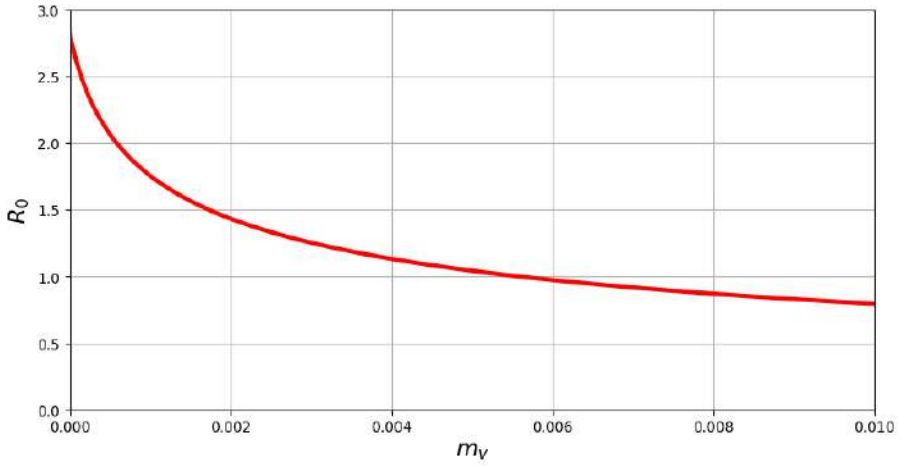


Figure 6.9: Relationship between R_0 and m_v.

In the figure 6.10 the surface of R_0 as a function of m_v and m_h reveals an inverse relationship of R_0 with m_v, where higher vector intraspecific competition (or mortality) reduces R_0 and thus the transmission potential. In contrast, the effect of m_h is nonlinear: initial increases in human intraspecific competition raise R_0, but this effect slows down and may stabilize at higher levels of m_h. Biologically, these results indicate that malaria dynamics depend on the joint interaction of both human and vector competition, suggesting that effective control strategies must integrate both components to limit disease spread.

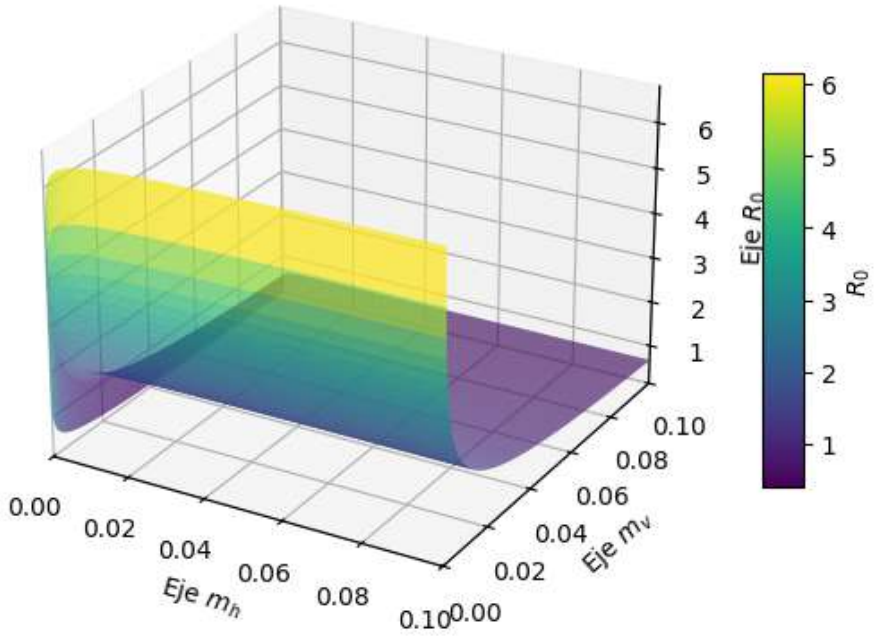


Figure 6.10: 3D surface of R_0 as a function of m_v and m_h.

6.4. Evolution of R_0 and I_h at the Equilibrium Point by Varying β. Figure 6.11 presents the plot of the equilibrium values of I_h for system (2.1)–(2.6) as the parameter β varies, showing a forward bifurcation from the disease-free point at $R_0 = 1$. This result indicates that, for instance, if public policies are implemented to keep the intraspecific competition parameters favorably controlled, it will be sufficient for the infection to converge to the disease-free equilibrium when $R_0 < 1$.

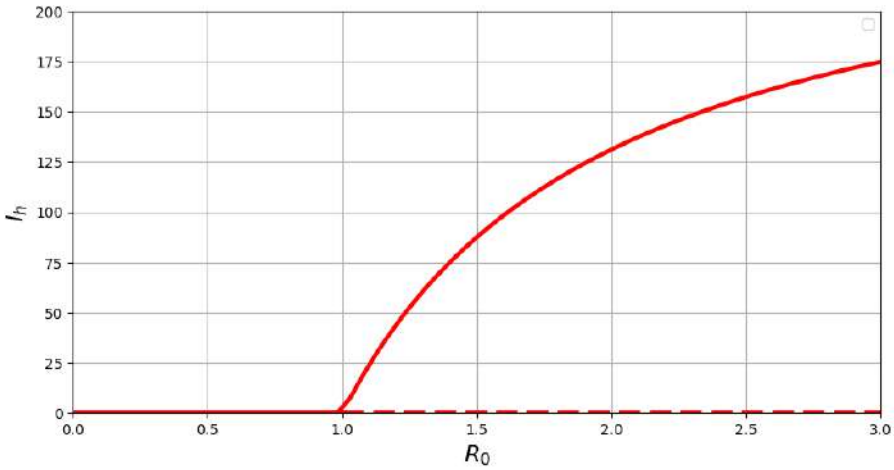


Figure 6.11: Forward bifurcation at $R_0 = 1$.

7. Conclusions.
Based on the results, the following conclusions were reached:

- The model with biological control and intraspecific competition presents feasible equilibrium points (disease-free and endemic), whose existence and uniqueness depend on the system parameters and the basic reproduction number R_0.
- To ensure the local and global stability of the disease-free equilibrium point, it is necessary that the basic reproduction number satisfies $R_0 < 1$. Furthermore, to guarantee the local stability of the endemic equilibrium point, it is necessary that $R_0 > 1$.

- Numerical simulations show that if competition among humans increases (e.g., competition for treatments, medical care, hospital beds, etc.), while competition among mosquitoes remains constant, then the disease will become endemic.
- Numerical simulations show that if competition among vectors increases (e.g., competition for food, mating, etc.), while competition among humans remains constant, then the disease will be eradicated.
- In scenarios where both intraspecific competition among humans (m_h) and vectors (m_v) increase, the simulations suggest a complex dynamic in which the disease may undergo a transient expansion phase within the population, and subsequently, under certain conditions of high competition, tend either to stabilize (endemic state) or to die out. This highlights the importance of integrated control strategies that address both types of competition.

Author contributions. The work carried out by the authors was equally distributed.

Funding. Did not receive financing.

Acknowledgment. Marco T. would like to express his sincere gratitude to Dr. Roxana López Cruz for her invaluable guidance, insightful feedback, and continuous support throughout the development of this research work.

Conflicts of interest. "The authors declare no conflict of interest".

ORCID and License
Marco Antonio Tamariz Milla https://orcid.org/0000-0001-7316-9016
Roxana López-Cruz https://orcid.org/0000-0002-7703-5784

References

[1] Centers for Disease Control and Prevention. About Malaria [Internet]. 2021 [cited 2024 Sep 01]. Available from: https://www.cdc.gov/malaria/about/index.html

[2] World Health Organization. World malaria report 2018 [Internet]. 2018 [cited 2024 Sep 01]. Available from: https://apps.who.int/iris/bitstream/handle/10665/275867/9789241565653-eng.pdf

[3] Basáñez M, Rodríguez D. Dinámica de transmisión y modelos matemáticos en enfermedades transmitidas por vectores. Entomotrópica: Revista internacional para el estudio de la entomología tropical. 2004;19(3):1–22. Available from: http://hdl.handle.net/1807/5787

[4] Lou Y, Zhao X. Modelling malaria control by introduction of larvivorous fish. Bulletin of Mathematical Biology. 2011;73(10):2384–2407. doi:http://dx.doi.org/10.1007/s11538-011-9628-6

[5] Ghosh M, Lashari A, Li X. Biological control of malaria: a mathematical model. Applied Mathematics and Computation. 2013;219:7923–7939. doi:http://dx.doi.org/10.1016/j.amc.2013.02.053

[6] Perko L. Differential equations and dynamical systems (Vol. 7). Springer Science & Business Media; 2013.

[7] Zhao X, Jing Z. Global asymptotic behavior in some cooperative systems of functional differential equations. Canadian Applied Mathematics Quarterly. 1996;4(4):421–444. Disponible en: https://www.math.mun.ca/~zhao/Selectpapers/ZhaoJingCAMQ96.pdf

[8] Van den Driessche P, Watmough J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Mathematical Biosciences. 2002;180(1–2):29–48. doi:http://dx.doi.org/10.1016/S0025-5564(02)00108-6

[9] Kiseliov A, Krasnov M, Makarenko G, Bernardo E. Problemas de ecuaciones diferenciales ordinarias. Mir; 1968.

[10] Tamariz M. Perturbación de un modelo de control biológico de la malaria considerando competencia de especies [Doctoral Dissertation]. Chimbote, Peru: Universidad Nacional del Santa; 2025. 168 p. [cited 2025 Sep 20]. Available from: https://repositorio.uns.edu.pe/handle/20.500.14278/5157

[11] Barreira L, Valls C. Ordinary differential equations: Qualitative theory. American Mathematical Soc; 2012.

[12] Zhao X. Dynamical systems in population biology. Springer, New York; 2003.

[13] Hirsch M, Smith H, Zhao X. Chain transitivity, attractivity, and strong repellors for semidynamical systems. Journal of Dynamics and Differential Equations. 2001; 13:107–131. doi:http://dx.doi.org/10.1023/A:1009044515567