

Mathematical Analysis of a Malaria–Schistosomiasis Co-infection Model with Immune Response, Environmental Transmission and Drug-Resistant Malaria

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Abstract

Malaria and schistosomiasis occur together in many tropical communities and may interact through shared exposure, immune response and treatment-related factors. This study formulates and analyses a deterministic model for malaria–schistosomiasis co-infection with malaria drug resistance, immune response and environmental transmission of schistosomiasis. The human population is divided into susceptible, malaria-infected, schistosomiasis-infected, co-infected, resistant-malaria and recovered classes. Two additional variables describe immune activity and environmental contamination. Positivity, boundedness, existence and uniqueness of solutions are established. The malaria-only and schistosomiasis-only submodels are analysed separately. Their disease-free and endemic equilibria are obtained, reproduction numbers are derived by the next-generation matrix method, and local and global stability conditions are established. The full model reproduction number is expressed in terms of the malaria and schistosomiasis transmission components. Numerical simulations show that malaria and schistosomiasis transmission rates increase infection burden, resistance progression increases resistant malaria, and environmental clearance reduces schistosomiasis contamination. With the higher-transmission initial transmission used for the numerical illustration, the combined reproduction number is greater than one. After the malaria transmission rate is reduced, the reproduction number falls below one and the infected classes decline. The results show the importance of simultaneous malaria control, resistance management and environmental control in co-endemic settings.

Index Terms– co-infection, drug resistance, environmental transmission, malaria, mathematical model, schistosomiasis.

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I. Introduction

Malaria and schistosomiasis remain important parasitic diseases in many tropical countries, and the burden is still highest in sub-Saharan Africa [1,2]. The World Health Organization estimated 282 million malaria cases and 610,000 deaths in 2024, with the African Region accounting for most of the deaths [1]. Malaria control depends on a combination of case management, vector control, chemoprevention and vaccination, but progress is slowed by weak health systems, insecticide resistance and antimalarial drug resistance [1–3]. These problems are important in countries with persistent transmission, including Nigeria and other high-burden African countries [1].

Schistosomiasis also remains widely distributed in tropical and subtropical areas [4]. At least 253.7 million people required preventive treatment in 2024, and the largest burden remains in Africa [4]. Transmission depends on human contact with freshwater in which the parasite passes through suitable snail hosts [4,5]. Infected people contaminate water with parasite eggs, while cercariae released from infected snails infect susceptible people during water contact [4]. For this reason, treatment with praziquantel alone may not be enough where unsafe water, poor sanitation and suitable snail habitats continue to maintain transmission [5,6].

The two diseases occur together in many African communities [7,8]. A recent meta-analysis reported that malaria–*Schistosoma mansoni* co-infection remains common in sub-Saharan Africa, although prevalence differs widely between locations [7]. A Nigerian review also reported substantial malaria–*Schistosoma haematobium* co-infection and showed that the burden varies across endemic settings [8]. Shared exposure patterns, age, occupation, water contact and local transmission conditions can place the same population at risk of both infections [7]. This overlap means that the two infections cannot always be treated as independent processes.

The biological interaction is also important. Helminth infections can modify immune regulation and may change the host response to malaria [9,10]. Studies of malaria–schistosomiasis co-infection have reported changes

in antimalarial antibody responses, cytokine profiles and parasite density [11,12]. In Nigerian children, variation in the interleukin-10 pathway has also been studied in malaria, schistosomiasis and co-infection [13]. The direction and size of the interaction are not identical in all populations, but the available evidence shows that immune response can influence infection outcome [9,11,12].

Malaria immunity is acquired gradually after repeated exposure and does not usually produce complete protection against infection [14,15]. Antibodies and several cellular responses contribute to control of parasite density and severe disease [14,16]. The protection may also decrease when exposure is reduced [17]. These observations support the inclusion of an immune-response variable in transmission models when the aim is to represent the feedback between infection and partial protection [18]. In the model, infection stimulates immune activity, while the immune response reduces the effective forces of infection.

Drug resistance is another major concern in malaria control. Resistance to older antimalarial drugs has already changed treatment policy in many endemic countries [19]. More recently, artemisinin partial resistance has emerged independently in parts of Africa [20]. Surveillance in Uganda has shown the spread of several *Pfkelch13* mutations associated with partial resistance [21]. The 2025 World Malaria Report identifies antimalarial drug resistance as a growing threat to malaria control [1]. This makes it useful to distinguish drug-sensitive malaria from resistant malaria in a mathematical model rather than placing all malaria infections in one class.

Environmental transmission is central to schistosomiasis. The parasite is not passed directly from one infected person to another; the aquatic stage and snail host are necessary parts of the transmission cycle [4,5]. Mathematical representation of an environmental contamination variable therefore gives a direct way to link infected humans, contamination and new schistosomiasis infections. It also allows environmental clearance to enter the reproduction number and numerical analysis. This is consistent with control programmes that combine preventive chemotherapy with water, sanitation, hygiene and snail control [5,6].

Mathematical models provide a useful framework for joining these mechanisms in one system [18,22]. The basic reproduction number gives a threshold for invasion, while equilibrium and stability analysis show the long-term behaviour associated with that threshold [23,24]. The next-generation matrix method is widely used when several infected states are present [23,24]. Numerical analysis is also useful because it shows which parameters have the strongest effect on the threshold quantity [25]. These methods have been applied to malaria and to co-infection systems to identify transmission and control parameters that deserve closer attention [22,25].

Earlier malaria–schistosomiasis models established that interaction between the two infections can change the conditions for persistence [22]. This model extends this direction by placing four mechanisms in one system: malaria–schistosomiasis co-infection, malaria drug resistance, an infection-induced immune response and an environmental contamination pathway for schistosomiasis. The analysis follows the order of the main model development. The basic properties of the full system are established first. The malaria-only model is then studied through its disease-free equilibrium, reproduction number, endemic equilibrium and stability. The schistosomiasis-only model follows with its disease-free equilibrium, endemic equilibrium, reproduction number and stability.

II. Model Description and Formulation

The total human population at time t is

$$N(t) = S(t) + I_m(t) + I_s(t) + I_{ms}(t) + I_m^r(t) + R(t). \quad (1)$$

The class $S(t)$ contains individuals who are susceptible to both infections. The class $I_m(t)$ contains individuals infected with malaria only, while $I_s(t)$ contains individuals infected with schistosomiasis only. Individuals carrying both infections are placed in $I_{ms}(t)$. The class $I_m^r(t)$ represents individuals with drug-resistant malaria, and $R(t)$ contains individuals who have recovered from malaria, schistosomiasis or co-infection.

Two further state variables are included, but they are not counted as human population classes. The variable $I(t)$ measures the level of infection-induced immune response in the population. The variable $W(t)$ measures environmental contamination associated with schistosomiasis transmission. Thus, the full system has eight state variables,

$$X(t) = (S, I_m, I_m^r, I_s, I_{ms}, R, I, W)^T. \quad (2)$$

The immune and environmental variables are important parts of the model. Infection stimulates $I(t)$, while the resulting immune response reduces the effective forces of infection. In contrast, schistosomiasis-infected individuals add contamination to $W(t)$, and the contaminated environment provides the indirect route through which susceptible individuals acquire schistosomiasis.

The malaria force of infection is denoted by η_m and is defined by

$$\eta_m = \frac{\beta_m(I_m + \xi_1 I_{ms} + \xi_2 I_m^r)}{N(1 + \theta I)}. \quad (3)$$

Here β_m is the effective malaria transmission rate. The parameters ξ_1 and ξ_2 modify the infectious contributions of co-infected and resistant-malaria individuals. The term $1 + \theta I$ represents suppression of malaria transmission by the immune response. Therefore, an increase in I reduces η_m when the other variables are fixed.

Schistosomiasis is transmitted through the environmental reservoir. Its force of infection is

$$\eta_s = \frac{\beta_s W}{N(1 + \phi I)}, \quad (4)$$

where β_s is the schistosomiasis transmission rate and ϕ measures the reduction in schistosomiasis acquisition caused by the immune response. Equation (4) links the human population to environmental contamination and makes $W(t)$ a direct part of schistosomiasis transmission.

Individuals enter the susceptible class at the constant recruitment rate Π and leave every human class through natural death at rate μ . A susceptible individual acquires malaria at rate η_m or schistosomiasis at rate η_s . Recovered individuals lose temporary protection at rate ω and return to S .

A malaria-only infected individual may recover at rate γ_m , develop drug resistance at rate ρ , or acquire schistosomiasis and move to the co-infected class at rate α_i . Malaria infection also stimulates immune response at rate κ_m . Resistant-malaria individuals recover at rate γ_r , enter the co-infected class at rate α_r , and experience the mortality term $\mu + \delta_m$.

Individuals in I_s recover at rate γ_s or acquire malaria and enter I_{ms} at rate α_s . They stimulate immunity at rate κ_s and contribute to environmental contamination at rate σ . The co-infected class receives individuals from I_m , I_s and I_m^r through α_i , α_s and α_r , respectively. Co-infected individuals recover at rate γ_{ms} , stimulate immunity at rate κ_{ms} and experience natural and disease-induced mortality at rate $\mu + \delta_{ms}$.

The immune response grows through stimulation from I_m , I_s and I_{ms} and decreases at rate μ . Environmental contamination grows through shedding from the schistosomiasis-only class at rate σ and decays at rate μ_w . Thus, the immune response acts back on transmission, while the environmental variable keeps the indirect schistosomiasis pathway in the system.

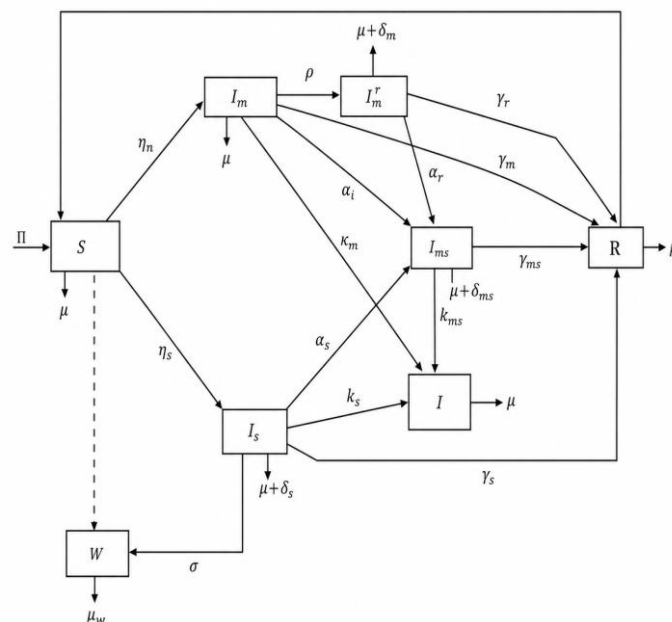


Figure 1: Flow diagram of the malaria–schistosomiasis co-infection model with immune response, environmental contamination and drug-resistant malaria.

The model equation is given by

$$\begin{aligned}
 \frac{dS}{dt} &= \Pi + \omega R - (\eta_m + \eta_s + \mu)S, \\
 \frac{dI_m}{dt} &= \eta_m S - (\alpha_i + \kappa_m + \rho + \gamma_m + \mu)I_m, \\
 \frac{dI_m^r}{dt} &= \rho I_m - (\alpha_r + \gamma_r + \mu + \delta_m)I_m^r, \\
 \frac{dI_s}{dt} &= \eta_s S - (\alpha_s + \gamma_s + \mu + \delta_s + \kappa_s + \sigma)I_s, \\
 \frac{dI_{ms}}{dt} &= \alpha_i I_m + \alpha_s I_s + \alpha_r I_m^r - (\gamma_{ms} + \mu + \delta_{ms} + \kappa_{ms})I_{ms}, \\
 \frac{dR}{dt} &= \gamma_m I_m + \gamma_r I_m^r + \gamma_s I_s + \gamma_{ms} I_{ms} - (\omega + \mu)R, \\
 \frac{dI}{dt} &= \kappa_m I_m + \kappa_s I_s + \kappa_{ms} I_{ms} - \mu I, \\
 \frac{dW}{dt} &= \sigma I_s - \mu_w W.
 \end{aligned} \tag{5}$$

All state variables have non-negative initial values. All model parameters are non-negative, while Π , μ and μ_w are strictly positive.

For later analysis, define

$$\begin{aligned}
 A_1 &= \alpha_i + \kappa_m + \rho + \gamma_m + \mu, \\
 A_2 &= \alpha_r + \gamma_r + \mu + \delta_m, \\
 A_3 &= \alpha_s + \gamma_s + \mu + \delta_s + \kappa_s + \sigma, \\
 A_4 &= \gamma_{ms} + \mu + \delta_{ms} + \kappa_{ms}.
 \end{aligned} \tag{6}$$

These quantities are the total removal rates from I_m , I_m^r , I_s and I_{ms} , respectively.

III. Basic Properties of the Model

3.1 Positivity of solutions

Theorem 1. For non-negative initial conditions, every solution of equation (5) remains non-negative for all $t \geq 0$.

Proof. From (5),

$$\frac{dS}{dt} \geq -(\eta_m + \eta_s + \mu)S, \tag{7}$$

so

$$S(t) \geq S(0) \exp \left[- \int_0^t (\eta_m(u) + \eta_s(u) + \mu) du \right] \geq 0. \tag{8}$$

Similarly,

$$\begin{aligned}
 I_m(t) &\geq I_m(0)e^{-A_1 t}, & A_1 &= \alpha_i + \kappa_m + \rho + \gamma_m + \mu, \\
 I_m^r(t) &\geq I_m^r(0)e^{-A_2 t}, & A_2 &= \alpha_r + \gamma_r + \mu + \delta_m, \\
 I_s(t) &\geq I_s(0)e^{-A_3 t}, & A_3 &= \alpha_s + \gamma_s + \mu + \delta_s + \kappa_s + \sigma, \\
 I_{ms}(t) &\geq I_{ms}(0)e^{-A_4 t}, & A_4 &= \gamma_{ms} + \mu + \delta_{ms} + \kappa_{ms}, \\
 R(t) &\geq R(0)e^{-(\omega + \mu)t}, & & \\
 I(t) &\geq I(0)e^{-\mu t}, & & \\
 W(t) &\geq W(0)e^{-\mu_w t}. & &
 \end{aligned} \tag{9}$$

Hence all state variables remain non-negative. ▀

3.2 Boundedness and invariant region

Summing the six human equations gives

$$\begin{aligned} \frac{dN}{dt} = & \Pi - \mu N - \delta_m I_m^r - \delta_s I_s - \delta_{ms} I_{ms} \\ & - \kappa_m I_m - \kappa_s I_s - \kappa_{ms} I_{ms} - \sigma I_s \leq \Pi - \mu N. \end{aligned} \quad (10)$$

Therefore

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Pi}{\mu}(1 - e^{-\mu t}), \quad (11)$$

and

$$\limsup_{t \rightarrow \infty} N(t) \leq \frac{\Pi}{\mu}. \quad (12)$$

A biologically feasible region is consequently

$$\Omega = \left\{ (S, I_m, I_m^r, I_s, I_{ms}, R, I, W) \in \mathbb{R}_+^8 : N \leq \frac{\Pi}{\mu} \right\}, \quad (13)$$

The human part is therefore bounded. The two auxiliary variables are also bounded. Since $I_m, I_s, I_{ms} \leq N \leq \frac{\Pi}{\mu}$ in the invariant region,

$$\dot{I} \leq \frac{\Pi}{\mu}(\kappa_m + \kappa_s + \kappa_{ms}) - \mu I. \quad (14)$$

Comparison with the corresponding linear equation gives

$$\limsup_{t \rightarrow \infty} I(t) \leq \frac{\Pi(\kappa_m + \kappa_s + \kappa_{ms})}{\mu^2}. \quad (15)$$

Likewise,

$$\dot{W} \leq \frac{\sigma \Pi}{\mu} - \mu_w W, \quad \limsup_{t \rightarrow \infty} W(t) \leq \frac{\sigma \Pi}{\mu \mu_w}. \quad (16)$$

Thus a closed bounded feasible set can be written as

$$\begin{aligned} \Omega^* = \{ X \in \mathbb{R}_+^8 : & N \leq \frac{\Pi}{\mu}, I \leq \Pi(\kappa_m + \kappa_s + \kappa_{ms})/\mu^2, \\ & W \leq \frac{\sigma \Pi}{\mu \mu_w} \}. \end{aligned} \quad (17)$$

This set is positively invariant for solutions that enter it.

3.3 Existence and uniqueness of solutions

Let

$$X = (x_1, x_2, \dots, x_8)^T = (S, I_m, I_m^r, I_s, I_{ms}, R, I, W)^T. \quad (18)$$

The system can be written in vector form as

$$\dot{X} = F(X), \quad X(0) = X_0, \quad (19)$$

where

$$F(X) = (F_1(X), F_2(X), \dots, F_8(X))^T, \quad (20)$$

with

$$\begin{aligned} F_1 &= \Pi + \omega R - (\eta_m + \eta_s + \mu)S, \\ F_2 &= \eta_m S - A_1 I_m, \\ F_3 &= \rho I_m - A_2 I_m^r, \\ F_4 &= \eta_s S - A_3 I_s, \\ F_5 &= \alpha_i I_m + \alpha_s I_s + \alpha_r I_m^r - A_4 I_{ms}, \\ F_6 &= \gamma_m I_m + \gamma_r I_m^r + \gamma_s I_s + \gamma_{ms} I_{ms} - (\omega + \mu)R, \\ F_7 &= \kappa_m I_m + \kappa_s I_s + \kappa_{ms} I_{ms} - \mu I, \\ F_8 &= \sigma I_s - \mu_w W. \end{aligned} \quad (21)$$

For existence and uniqueness, the vector field F is shown to be locally Lipschitz in the feasible region. Let

$$\Omega_M = \{X \in \Omega^*: \varepsilon \leq N \leq M_N, \quad 0 \leq I \leq M_I, \quad 0 \leq W \leq M_W\}, \quad (22)$$

where $\varepsilon > 0$ and

$$M_N = \frac{\Pi}{\mu}, \quad M_I = \frac{\Pi(\kappa_m + \kappa_s + \kappa_{ms})}{\mu^2}, \quad M_W = \frac{\sigma \Pi}{\mu \mu_w}. \quad (23)$$

The positive lower bound ε is taken on any finite time interval. Because $\Pi > 0$, a solution starting with $N(0) > 0$ cannot cross $N = 0$.

For the malaria force of infection, write

$$\eta_m = \frac{\beta_m Z_m}{N(1 + \theta I)}, \quad Z_m = I_m + \xi_1 I_{ms} + \xi_2 I_m^r. \quad (24)$$

Let

$$c_m = \max\{1, \xi_1, \xi_2\}. \quad (25)$$

Since $Z_m \leq c_m N$ in Ω_M , the partial derivatives of η_m satisfy

$$\begin{aligned} \left| \frac{\partial \eta_m}{\partial I_m} \right| &\leq \frac{\beta_m(1 + c_m)}{\varepsilon}, \\ \left| \frac{\partial \eta_m}{\partial I_{ms}} \right| &\leq \frac{\beta_m(\xi_1 + c_m)}{\varepsilon}, \\ \left| \frac{\partial \eta_m}{\partial I_m^r} \right| &\leq \frac{\beta_m(\xi_2 + c_m)}{\varepsilon}, \\ \left| \frac{\partial \eta_m}{\partial S} \right|, \left| \frac{\partial \eta_m}{\partial I_s} \right|, \left| \frac{\partial \eta_m}{\partial R} \right| &\leq \frac{\beta_m c_m}{\varepsilon}, \\ \left| \frac{\partial \eta_m}{\partial I} \right| &\leq \beta_m \theta c_m. \end{aligned} \quad (26)$$

Hence there is a constant $L_m > 0$ such that

$$|\eta_m(X) - \eta_m(Y)| \leq L_m \|X - Y\|, \quad X, Y \in \Omega_M. \quad (27)$$

Similarly,

$$\eta_s = \frac{\beta_s W}{N(1 + \phi I)}. \quad (28)$$

On Ω_M ,

$$\begin{aligned} \left| \frac{\partial \eta_s}{\partial S} \right|, \left| \frac{\partial \eta_s}{\partial I_m} \right|, \left| \frac{\partial \eta_s}{\partial I_m^r} \right|, \left| \frac{\partial \eta_s}{\partial I_s} \right|, \left| \frac{\partial \eta_s}{\partial I_{ms}} \right|, \left| \frac{\partial \eta_s}{\partial R} \right| &\leq \frac{\beta_s M_W}{\varepsilon^2}, \\ \left| \frac{\partial \eta_s}{\partial W} \right| &\leq \frac{\beta_s}{\varepsilon}, \\ \left| \frac{\partial \eta_s}{\partial I} \right| &\leq \frac{\beta_s \phi M_W}{\varepsilon}. \end{aligned} \quad (29)$$

Therefore there is $L_s > 0$ such that

$$|\eta_s(X) - \eta_s(Y)| \leq L_s \|X - Y\|, \quad X, Y \in \Omega_M. \quad (30)$$

The component functions are considered next. Since

$$0 \leq \eta_m \leq \beta_m c_m, \quad 0 \leq \eta_s \leq \frac{\beta_s M_W}{\varepsilon}, \quad (31)$$

define

$$B_m = \beta_m c_m, \quad B_s = \frac{\beta_s M_W}{\varepsilon}. \quad (32)$$

Using and

$$|S_X \eta_m(X) - S_Y \eta_m(Y)| \leq M_N L_m \|X - Y\| + B_m |S_X - S_Y|, \quad (33)$$

with the analogous inequality for $S\eta_s$, the eight components satisfy

$$\begin{aligned} |F_1(X) - F_1(Y)| &\leq L_1 \|X - Y\|, \\ |F_2(X) - F_2(Y)| &\leq L_2 \|X - Y\|, \\ |F_3(X) - F_3(Y)| &\leq L_3 \|X - Y\|, \\ |F_4(X) - F_4(Y)| &\leq L_4 \|X - Y\|, \\ |F_5(X) - F_5(Y)| &\leq L_5 \|X - Y\|, \\ |F_6(X) - F_6(Y)| &\leq L_6 \|X - Y\|, \\ |F_7(X) - F_7(Y)| &\leq L_7 \|X - Y\|, \\ |F_8(X) - F_8(Y)| &\leq L_8 \|X - Y\|, \end{aligned} \quad (34)$$

where one possible set of constants is

$$\begin{aligned} L_1 &= \omega + \mu + B_m + B_s + M_N(L_m + L_s), \\ L_2 &= A_1 + B_m + M_N L_m, \\ L_3 &= \rho + A_2, \\ L_4 &= A_3 + B_s + M_N L_s, \\ L_5 &= \alpha_i + \alpha_s + \alpha_r + A_4, \\ L_6 &= \gamma_m + \gamma_r + \gamma_s + \gamma_{ms} + \omega + \mu, \\ L_7 &= \kappa_m + \kappa_s + \kappa_{ms} + \mu, \\ L_8 &= \sigma + \mu_w. \end{aligned} \quad (35)$$

Hence,

$$\|F(X) - F(Y)\| \leq \left(\sum_{j=1}^8 L_j^2 \right)^{1/2} \|X - Y\| = L \|X - Y\|. \quad (36)$$

Thus F is locally Lipschitz on every bounded subset of the feasible region. The Picard–Lindelöf theorem gives a unique local solution. The positivity result keeps the solution in $\mathbb{R}_+^8$, while the invariant region gives uniform bounds for N , I and W . Hence finite-time blow-up cannot occur and the local solution extends to all $t \geq 0$. Therefore the model has a unique global non-negative solution for each non-negative initial state with $N(0) > 0$.
□

IV. Malaria-Only Submodel Analysis

To obtain the malaria-only system, set $I_s = I_{ms} = W = 0$ and remove schistosomiasis acquisition. The remaining variables are S, I_m, I_m^r, R and I . The force of infection becomes

$$\eta_m = \frac{\beta_m(I_m + \eta_2 I_m^r)}{N_m(1 + \theta I)}, \quad N_m = S + I_m + I_m^r + R. \quad (37)$$

The submodel is

$$\begin{aligned} \dot{S} &= \Pi + \omega R - (\eta_m + \mu)S, \\ \dot{I}_m &= \eta_m S - A_1 I_m, \\ \dot{I}_m^r &= \rho I_m - A_2 I_m^r, \\ \dot{R} &= \gamma_m I_m + \gamma_r I_m^r - (\omega + \mu)R, \\ \dot{I} &= \kappa_m I_m - \mu I. \end{aligned} \quad (38)$$

4.1 Disease-Free Equilibrium

At the disease-free state, $I_m = I_m^r = R = I = 0$. From $\dot{S} = 0$,

$$S_0 = \frac{\Pi}{\mu}, \quad (39)$$

so

$$E_{0m} = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0 \right). \quad (40)$$

4.2 Basic Reproduction Number

The basic reproduction number is obtained using the next-generation matrix method of van den Driessche and Watmough (n.d.-w). The infected compartments are first identified, and the infection subsystem is written in the form

$$\dot{x} = \mathcal{F}(x) - \mathcal{V}(x), \quad (41)$$

where $\mathcal{F}$ contains the rates at which new infections enter the infected compartments, while $\mathcal{V}$ contains all other transfers into and out of these compartments. At the disease-free equilibrium, the corresponding Jacobian matrices are denoted by F and V . The basic reproduction number is then

$$\mathcal{R}_0 = \rho(FV^{-1}), \quad (42)$$

where $\rho(\cdot)$ denotes the spectral radius. This procedure is applied first to the malaria-only and schistosomiasis-only submodels and later to the full co-infection model.

Take the infected vector $x_m = (I_m, I_m^r)^T$. At E_{0m} ,

$$F_m = \begin{pmatrix} \beta_m & \beta_m \eta_2 \\ 0 & 0 \end{pmatrix}, \quad V_m = \begin{pmatrix} A_1 & 0 \\ -\rho & A_2 \end{pmatrix}. \quad (43)$$

Since

$$V_m^{-1} = \begin{pmatrix} A_1^{-1} & 0 \\ \rho(A_1 A_2)^{-1} & A_2^{-1} \end{pmatrix}, \quad (44)$$

we obtain

$$F_m V_m^{-1} = \begin{pmatrix} \frac{\beta_m}{A_1} + \frac{\beta_m \eta_2 \rho}{A_1 A_2} & \frac{\beta_m \eta_2}{A_2} \\ 0 & 0 \end{pmatrix}. \quad (45)$$

Therefore

$$\mathcal{R}_{0m} = \rho(F_m V_m^{-1}) = \frac{\beta_m (A_2 + \eta_2 \rho)}{A_1 A_2}. \quad (46)$$

4.3 Endemic Equilibrium

Let $E_m^* = (S^*, I_m^*, I_r^*, R^*, I^*)$ with $I_m^* > 0$. From the last three equilibrium equations,

$$\begin{aligned} I_r^* &= \frac{\rho}{A_2} I_m^*, \\ I^* &= \frac{\kappa_m}{\mu} I_m^*, \\ R^* &= \frac{\gamma_m + \gamma_r \frac{\rho}{A_2}}{\omega + \mu} I_m^*. \end{aligned} \quad (47)$$

Also, from $\dot{I}_m = 0$,

$$\eta_m^* S^* = A_1 I_m^*, \quad S^* = \frac{A_1 I_m^*}{\eta_m^*}. \quad (48)$$

Using the equilibrium force of infection,

$$\eta_m^* = \frac{\beta_m I_m^* (1 + \eta_2 \frac{\rho}{A_2})}{N_m^* (1 + \theta \frac{\kappa_m I_m^*}{\mu})}, \quad (49)$$

which, together with the susceptible balance

$$\Pi + \omega R^* = (\eta_m^* + \mu) S^*, \quad (50)$$

reduces the endemic state to a scalar algebraic equation in I_m^* . A positive root determines all other equilibrium components through the expressions above. The positive branch exists when the malaria invasion threshold exceeds unity.

4.4 Stability Analysis

Local stability of the disease-free equilibrium. The Jacobian at E_{0m} is

$$J_m(E_{0m}) = \begin{pmatrix} -\mu & -\beta_m & -\beta_m \eta_2 & \omega & 0 \\ 0 & \beta_m - A_1 & \beta_m \eta_2 & 0 & 0 \\ 0 & \rho & -A_2 & 0 & 0 \\ 0 & \gamma_m & \gamma_r & -(\omega + \mu) & 0 \\ 0 & \kappa_m & 0 & 0 & -\mu \end{pmatrix}. \quad (51)$$

Three eigenvalues are immediately $-\mu$, $-\mu$ and $-(\omega + \mu)$. The remaining two come from

$$B_m = \begin{pmatrix} \beta_m - A_1 & \beta_m \eta_2 \\ \rho & -A_2 \end{pmatrix}. \quad (52)$$

Its determinant is

$$\det(B_m) = A_1 A_2 - \beta_m (A_2 + \eta_2 \rho) = A_1 A_2 (1 - \mathcal{R}_{0m}). \quad (53)$$

When $\mathcal{R}_{0m} < 1$, $\det(B_m) > 0$ and $\text{tr}(B_m) = \beta_m - A_1 - A_2 < 0$. Hence both roots have negative real parts.

Theorem 2. *The malaria-only DFE E_{0m} is locally asymptotically stable if $\mathcal{R}_{0m} < 1$ and unstable if $\mathcal{R}_{0m} > 1$.*

Global stability of the disease-free equilibrium. The local result only describes solutions that start close to the disease-free state. A global result is needed to show what happens for every admissible initial condition in the malaria feasible region.

Theorem 3. *If $\mathcal{R}_{0m} < 1$, the malaria-only disease-free equilibrium E_{0m} is globally asymptotically stable in the feasible region.*

Proof. Since $0 \leq \frac{S}{N_m} \leq 1$ and $0 < (1 + \theta I)^{-1} \leq 1$, the malaria force of infection satisfies

$$\eta_m S \leq \beta_m (I_m + \eta_2 I_m^r). \quad (54)$$

Therefore the infected equations satisfy

$$\begin{aligned} \dot{I}_m &\leq (\beta_m - A_1) I_m + \beta_m \eta_2 I_m^r, \\ \dot{I}_m^r &= \rho I_m - A_2 I_m^r. \end{aligned} \quad (55)$$

Let $Z_m = (I_m, I_m^r)^T$. Then

$$\dot{Z}_m \leq B_m Z_m, \quad B_m = \begin{pmatrix} \beta_m - A_1 & \beta_m \eta_2 \\ \rho & -A_2 \end{pmatrix}. \quad (56)$$

The characteristic polynomial of B_m is

$$\begin{aligned} p_m(\lambda) &= \det(\lambda I - B_m) \\ &= \lambda^2 + c_1 \lambda + c_0, \end{aligned} \quad (57)$$

where

$$c_1 = A_1 + A_2 - \beta_m, \quad c_0 = A_1 A_2 - \beta_m (A_2 + \eta_2 \rho). \quad (58)$$

Using [Rm],

$$c_0 = A_1 A_2 (1 - \mathcal{R}_{0m}). \quad (59)$$

If $\mathcal{R}_{0m} < 1$, then $c_0 > 0$. Also $\mathcal{R}_{0m} < 1$ gives

$$\beta_m < \frac{A_1 A_2}{A_2 + \eta_2 \rho} < A_1, \quad (60)$$

so $c_1 > A_2 > 0$. The Routh–Hurwitz conditions for a quadratic polynomial are therefore satisfied. Hence B_m is Hurwitz and every solution of the linear comparison system $\dot{Y} = B_m Y$ tends to zero. The comparison theorem gives

$$I_m(t) \rightarrow 0, \quad I_m^r(t) \rightarrow 0 \quad \text{as } t \rightarrow \infty. \quad (61)$$

The immune equation can be written with an integrating factor as

$$I(t) = I(0)e^{-\mu t} + \kappa_m \int_0^t e^{-\mu(t-s)} I_m(s) ds. \quad (62)$$

Since $I_m(t) \rightarrow 0$ and is bounded, the integral term tends to zero. Thus $I(t) \rightarrow 0$. In the same way,

$$R(t) = R(0)e^{-(\omega+\mu)t} + \int_0^t e^{-(\omega+\mu)(t-s)} [\gamma_m I_m(s) + \gamma_r I_m^r(s)] ds, \quad (63)$$

which gives $R(t) \rightarrow 0$. The susceptible equation then approaches

$$\dot{S} = \Pi - \mu S, \quad (64)$$

and hence

$$S(t) \rightarrow \frac{\Pi}{\mu}. \quad (65)$$

Thus every component approaches its disease-free value, so E_{0m} is globally asymptotically stable. ▀

V. Schistosomiasis-Only Submodel Analysis

Set $I_m = I_m^r = I_{ms} = 0$. The schistosomiasis force of infection is

$$\eta_s = \frac{\beta_s W}{N_s(1 + \phi I)}, \quad N_s = S + I_s + R. \quad (66)$$

The submodel becomes

$$\begin{aligned} \dot{S} &= \Pi + \omega R - (\eta_s + \mu)S, \\ \dot{I}_s &= \eta_s S - A_3 I_s, \\ \dot{R} &= \gamma_s I_s - (\omega + \mu)R, \\ \dot{I} &= \kappa_s I_s - \mu I, \\ \dot{W} &= \sigma I_s - \mu_w W. \end{aligned} \quad (67)$$

5.1 Disease-Free Equilibrium

The disease-free equilibrium is

$$E_{0s} = \left(\frac{\Pi}{\mu}, 0, 0, 0 \right). \quad (68)$$

5.2 Endemic Equilibrium

For an endemic equilibrium $E_s^* = (S^*, I_s^*, R^*, I^*, W^*)$ with $I_s^* > 0$,

$$\begin{aligned} R^* &= \frac{\gamma_s}{\omega + \mu} I_s^*, \quad I^* = \frac{\kappa_s}{\mu} I_s^*, \quad W^* = \frac{\sigma}{\mu_w} I_s^*, \\ S^* &= \frac{A_3 I_s^*}{\eta_s^*}, \quad \eta_s^* = \frac{\beta_s \frac{\sigma I_s^*}{\mu_w}}{N_s^* (1 + \phi \frac{\kappa_s I_s^*}{\mu})}. \end{aligned} \quad (69)$$

Substitution into the susceptible equilibrium equation gives a scalar equation for I_s^* . A positive solution gives the endemic equilibrium.

5.3 Basic Reproduction Number

For the schistosomiasis-only system, the same next-generation matrix procedure is used. The infected variables are selected and the matrices F_s and V_s are formed at the disease-free equilibrium.

Choose $x_s = (I_s, W)^T$. Then

$$F_s = \begin{pmatrix} 0 & \beta_s \\ 0 & 0 \end{pmatrix}, \quad V_s = \begin{pmatrix} A_3 & 0 \\ -\sigma & \mu_w \end{pmatrix}. \quad (70)$$

Hence

$$V_s^{-1} = \begin{pmatrix} A_3^{-1} & 0 \\ \sigma(A_3\mu_w)^{-1} & \mu_w^{-1} \end{pmatrix} \quad (71)$$

and

$$F_s V_s^{-1} = \begin{pmatrix} \frac{\beta_s \sigma}{A_3 \mu_w} & \frac{\beta_s}{\mu_w} \\ 0 & 0 \end{pmatrix}. \quad (72)$$

Therefore

$$\mathcal{R}_{0s} = \frac{\beta_s \sigma}{A_3 \mu_w}. \quad (73)$$

5.4 Stability Analysis

Local stability of the disease-free equilibrium. Using the variable order (S, I_s, R, I, W) , the Jacobian matrix at E_{0s} is

$$J_s(E_{0s}) = \begin{pmatrix} -\mu & 0 & \omega & 0 & -\beta_s \\ 0 & -A_3 & 0 & 0 & \beta_s \\ 0 & \gamma_s & -(\omega + \mu) & 0 & 0 \\ 0 & \kappa_s & 0 & -\mu & 0 \\ 0 & \sigma & 0 & 0 & -\mu_w \end{pmatrix}. \quad (74)$$

Three eigenvalues are obtained directly from the non-infected directions:

$$\lambda_1 = -\mu, \quad \lambda_2 = -\mu, \quad \lambda_3 = -(\omega + \mu). \quad (75)$$

The remaining eigenvalues are determined by the infected block

$$B_s = \begin{pmatrix} -A_3 & \beta_s \\ \sigma & -\mu_w \end{pmatrix}. \quad (76)$$

Its characteristic polynomial is

$$\lambda^2 + (A_3 + \mu_w)\lambda + A_3\mu_w - \beta_s\sigma = 0, \quad (77)$$

or

$$\lambda^2 + (A_3 + \mu_w)\lambda + A_3\mu_w(1 - \mathcal{R}_{0s}) = 0. \quad (78)$$

The coefficients are positive when $\mathcal{R}_{0s} < 1$, so both roots have negative real parts.

Theorem 4. *The schistosomiasis-only DFE is locally asymptotically stable when $\mathcal{R}_{0s} < 1$ and unstable when $\mathcal{R}_{0s} > 1$.*

Global stability of the disease-free equilibrium.

Theorem 5. *The schistosomiasis-only disease-free equilibrium E_{0s} is globally asymptotically stable in the feasible region whenever $\mathcal{R}_{0s} < 1$.*

Proof. For every solution in the feasible region,

$$0 \leq \frac{S}{N_s} \leq 1, \quad 0 < \frac{1}{1 + \phi I} \leq 1. \quad (79)$$

Thus,

$$\eta_s S = \frac{\beta_s W S}{N_s(1 + \phi I)} \leq \beta_s W. \quad (80)$$

Hence

$$\begin{aligned} \dot{I}_s &\leq -A_3 I_s + \beta_s W, \\ \dot{W} &= \sigma I_s - \mu_w W. \end{aligned} \quad (81)$$

With $Z_s = (I_s, W)^T$,

$$\dot{Z}_s \leq B_s Z_s, \quad B_s = \begin{pmatrix} -A_3 & \beta_s \\ \sigma & -\mu_w \end{pmatrix}. \quad (82)$$

The characteristic polynomial is

$$\begin{aligned} p_s(\lambda) &= \lambda^2 + (A_3 + \mu_w)\lambda + (A_3\mu_w - \beta_s\sigma) \\ &= \lambda^2 + (A_3 + \mu_w)\lambda + A_3\mu_w(1 - \mathcal{R}_{0s}). \end{aligned} \quad (83)$$

When $\mathcal{R}_{0s} < 1$, both coefficients required by the Routh–Hurwitz criterion are positive. Thus B_s is Hurwitz. The linear comparison system tends to the origin, and therefore

$$I_s(t) \rightarrow 0, \quad W(t) \rightarrow 0. \quad (84)$$

Next,

$$I(t) = I(0)e^{-\mu t} + \kappa_s \int_0^t e^{-\mu(t-u)} I_s(u) du, \quad (85)$$

so $I(t) \rightarrow 0$. Also,

$$R(t) = R(0)e^{-(\omega+\mu)t} + \gamma_s \int_0^t e^{-(\omega+\mu)(t-u)} I_s(u) du, \quad (86)$$

which gives $R(t) \rightarrow 0$. Finally, since $\eta_s(t) \rightarrow 0$ and $R(t) \rightarrow 0$,

$$\dot{S} = \Pi + \omega R - (\eta_s + \mu)S \quad (87)$$

approaches $\dot{S} = \Pi - \mu S$. Therefore $S(t) \rightarrow \frac{\Pi}{\mu}$. Hence every solution in the feasible region approaches E_{0s} . ■

VI. Combined Model Analysis

The next-generation matrix method is also used for the full co-infection system. The infected compartments that can contribute directly or indirectly to new infection are collected in the infected-state vector. For the full system, use the infected vector

$$x = (I_m, I_m^r, I_s, I_{ms}, W)^T. \quad (88)$$

At the DFE $E_0 = (\frac{\Pi}{\mu}, 0, 0, 0, 0, 0, 0)$, the new-infection matrix is

$$F = \begin{pmatrix} \beta_m & \beta_m \eta_2 & 0 & \beta_m \eta_1 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_s \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad (89)$$

while the transition matrix is

$$V = \begin{pmatrix} A_1 & 0 & 0 & 0 & 0 \\ -\rho & A_2 & 0 & 0 & 0 \\ 0 & 0 & A_3 & 0 & 0 \\ -\alpha_i & -\alpha_r & -\alpha_s & A_4 & 0 \\ 0 & 0 & -\sigma & 0 & \mu_w \end{pmatrix}. \quad (90)$$

The spectral radius of FV^{-1} separates into malaria and schistosomiasis pathways. The malaria component is

$$\mathcal{R}_{0m}^c = \frac{\beta_m [A_2 A_4 + \eta_2 \rho A_4 + \eta_1 (A_2 \alpha_i + \alpha_r \rho)]}{A_1 A_2 A_4}, \quad (91)$$

and the schistosomiasis component is

$$\mathcal{R}_{0s} = \frac{\beta_s \sigma}{A_3 \mu_w}. \quad (92)$$

Therefore

$$\mathcal{R}_0 = \max\{\mathcal{R}_{0m}^c, \mathcal{R}_{0s}\}. \quad (93)$$

This expression shows that the dominant transmission pathway determines whether infection can invade the disease-free population.

VII. Results

The numerical analysis uses the parameter values in Table 1. Values taken directly from published studies are cited, while values without a reported estimate are stated as assumptions used for the numerical illustration.

Table 1: Parameter values and literature basis used in the numerical analysis.

Parameter	Value	Source
Π	20	[30, 26]
μ	0.02	[31]
β_m	0.10	[30]
β_s	0.10	[32]
ξ_1	0.60	[26]
ξ_2	0.50	[30]
θ	0.10	[5, 7]
φ	0.10	[10, 11]
α_i	0.05	[26]
α_s	0.04	[26]
α_r	0.03	[30]
ρ	0.02	[30]
γ_m	0.10	[30, 26]
γ_s	0.08	[32, 31]

γ_r	0.07	[30]
γ_{ms}	0.06	[26]
δ_m	0.01	[30, 26]
δ_s	0.015	[26]
δ_{ms}	0.02	[26]
κ_m	0.03	[5, 7]
κ_s	0.025	[10, 11]
κ_{ms}	0.02	[10, 11]
σ	0.20	[32, 31]
μ_w	0.30	[32, 31]
ω	0.05	[8, 5]

7.1 Transmission and control scenarios

The threshold calculations are first shown for an initial transmission situation in which both component reproduction numbers are greater than one. The values $\beta_m = 0.20$ and $\beta_s = 0.60$ are used for this initial transmission scenario, while the remaining parameters take the values in Table 1. Thus,

$$\mathcal{R}_{0m}^c = 1.2273 > 1, \quad \mathcal{R}_{0s} = 1.0526 > 1, \quad (94)$$

and

$$\mathcal{R}_0 = \max\{1.2273, 1.0526\} = 1.2273 > 1. \quad (95)$$

Both infections can therefore maintain transmission in this situation.

The control scenarios then change only the parameters shown in Table 2. A malaria-control scenario reduces β_m ; a schistosomiasis-control scenario reduces β_s ; the environmental-control scenario increases μ_w ; and the combined-control scenario applies the reductions together. This shows the change from persistence to elimination without changing the structure of the model.

Table 2: Reproduction numbers under the numerical scenarios.

Scenario	β_m	β_s	μ_w	$\mathcal{R}_{0m}^c$	$\mathcal{R}_{0s}$	$\mathcal{R}_0$
Initial transmission	0.20	0.60	0.30	1.2273	1.0526	1.2273
Malaria control	0.10	0.60	0.30	0.6136	1.0526	1.0526
Schistosomiasis control	0.20	0.10	0.30	1.2273	0.1754	1.2273
Environmental control	0.20	0.60	0.60	1.2273	0.5263	1.2273
Combined control	0.10	0.10	0.60	0.6136	0.0877	0.6136

The malaria-control scenario gives $\mathcal{R}_{0m}^c = 0.6136 < 1$, but $\mathcal{R}_{0s} = 1.0526 > 1$. Malaria is pushed below its threshold, while schistosomiasis can still persist. Under the schistosomiasis-control scenario, $\mathcal{R}_{0s} = 0.1754 < 1$, but $\mathcal{R}_{0m}^c = 1.2273 > 1$, so malaria remains above its threshold. Increasing environmental clearance to $\mu_w = 0.60$ gives $\mathcal{R}_{0s} = 0.5263 < 1$, but malaria is still above one. In the combined-control scenario,

$$\mathcal{R}_{0m}^c = 0.6136 < 1, \quad \mathcal{R}_{0s} = 0.0877 < 1, \quad (96)$$

so

$$\mathcal{R}_0 = 0.6136 < 1. \quad (97)$$

The two transmission pathways are therefore below their threshold values only when the controls are applied together.

7.2 Scenario simulations

The full model was solved numerically for the scenarios in Table 2. The same initial conditions were used in all four comparisons,

$$(S, I_m, I_m^r, I_s, I_{ms}, R, I, W)(0) = (900, 50, 10, 30, 10, 0, 0, 10). \quad (98)$$

The same initial values are used in each case so that the effect of the parameter changes can be compared directly.

Figure 2 compares the malaria-infected class under the initial transmission and malaria-control scenarios. Reducing β_m lowers the malaria trajectory and moves the malaria reproduction number below one.

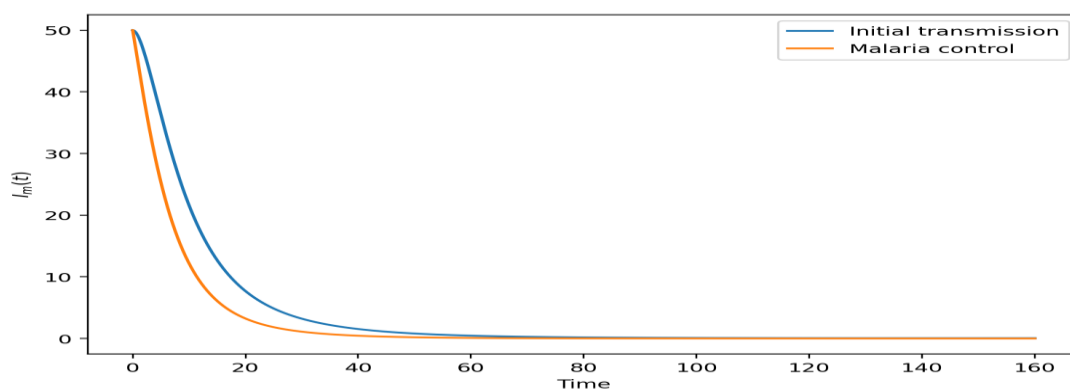


Figure 2: Malaria-infected population under the initial transmission and malaria-control scenarios.

Figure 3 compares schistosomiasis infection under the initial transmission, reduced schistosomiasis transmission and increased environmental clearance. Both controls reduce $I_s(t)$, but they act through different parts of the model.

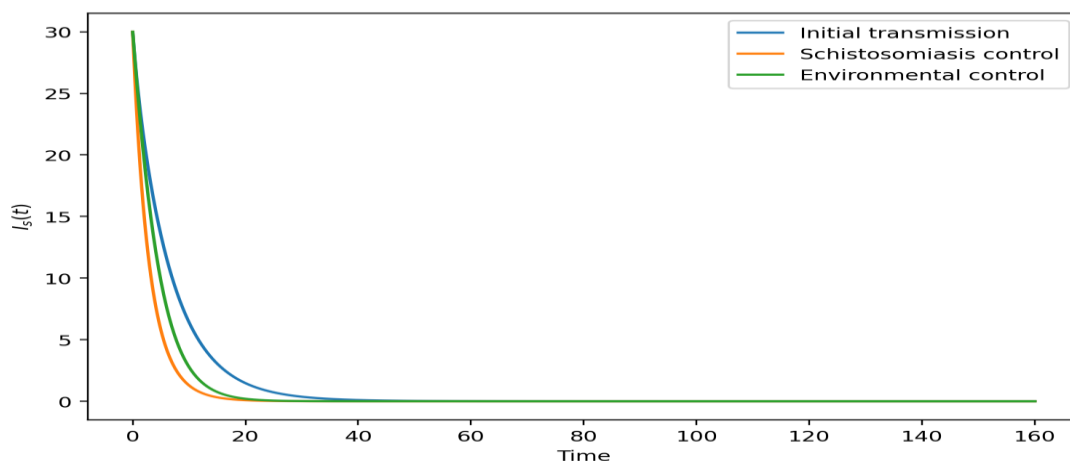


Figure 3: Schistosomiasis infection under the initial transmission, schistosomiasis-control and environmental-control scenarios.

The effect of environmental control on the contamination variable is shown in Figure 4. A larger μ_w removes contamination faster, while the combined-control scenario produces the strongest decline.

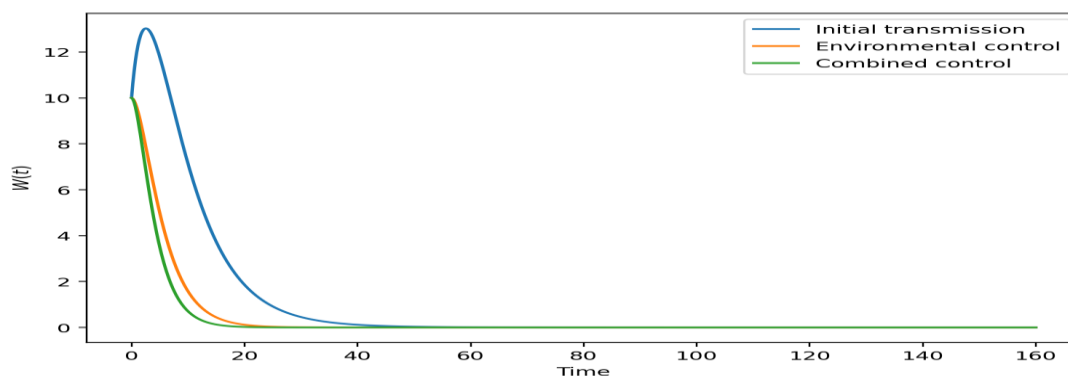


Figure 4: Environmental contamination under the initial transmission, environmental-control and combined-control scenarios.

Figure 5 compares the co-infected class under the initial transmission and combined-control scenarios. When both component reproduction numbers are below one, the co-infected population falls toward zero.

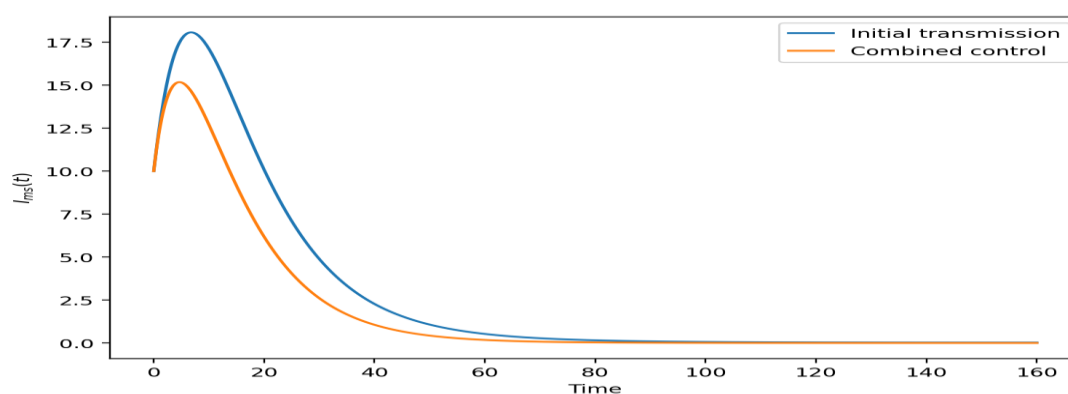


Figure 5: Co-infected population under the initial transmission and combined-control scenarios.

Figure 6 shows the malaria-only infected class for the values of β_m used in the main work. Larger values produce a higher malaria burden.

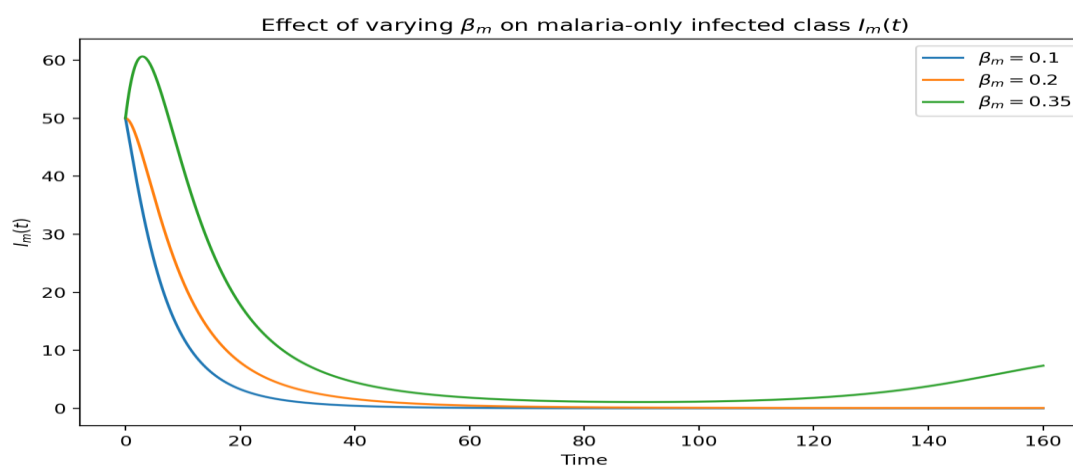


Figure 6: Effect of varying the malaria transmission rate β_m on the malaria-infected class.

Figure 7 shows the response of the schistosomiasis-infected class to changes in β_s . Higher transmission gives a larger infected population.

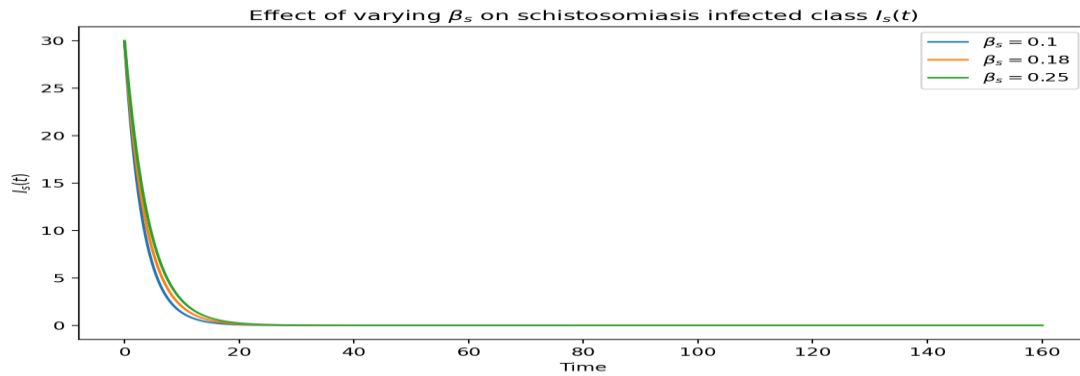


Figure 7: Effect of varying the schistosomiasis transmission rate β_s on $I_s(t)$.

Figure 8 shows that increasing ρ increases the resistant-malaria class. Faster progression to resistant infection therefore increases the resistant burden.

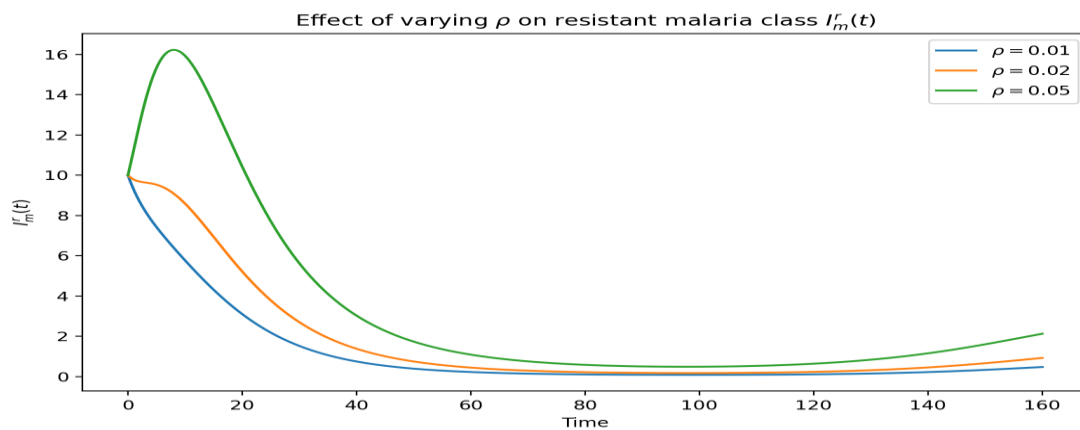


Figure 8: Effect of the resistance progression rate ρ on resistant malaria.

Figure 9 shows that larger μ_w removes environmental contamination more quickly.

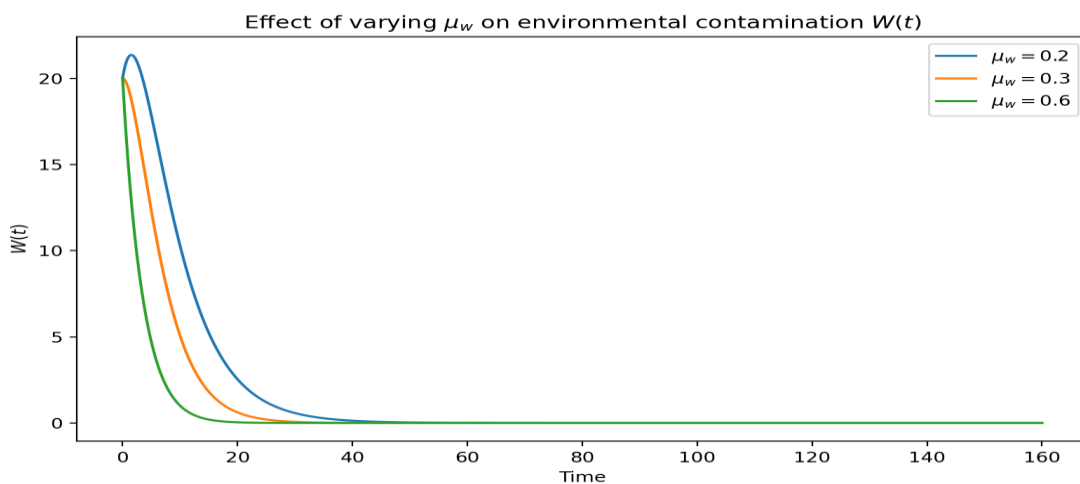


Figure 9: Effect of environmental clearance μ_w on $W(t)$.

Figure 10 shows the effect of σ on the schistosomiasis-infected class. The environmental pathway remains important because infected humans contribute contamination that later supports new infection.

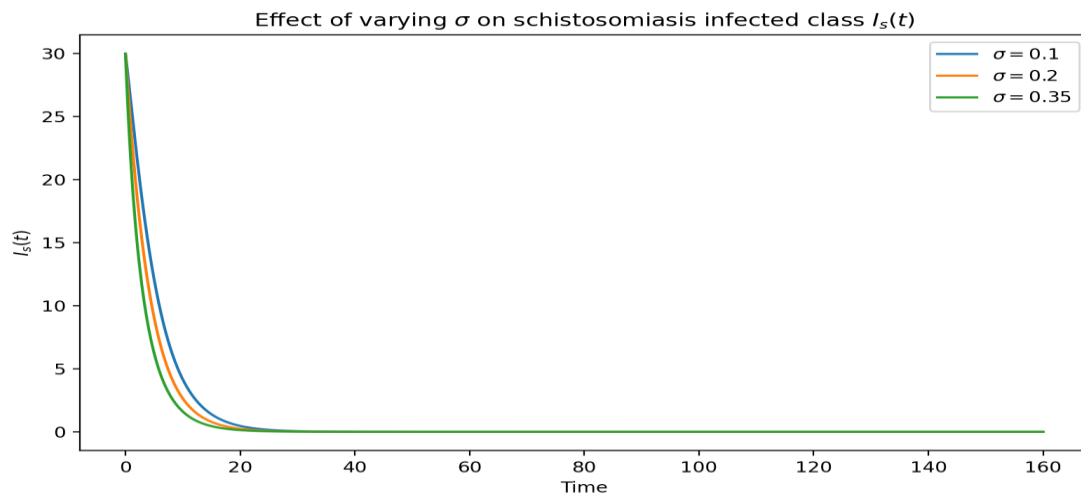


Figure 10: Effect of the environmental contamination parameter σ on schistosomiasis infection.

Figure 11 compares the co-infected class under lower and higher transmission conditions.

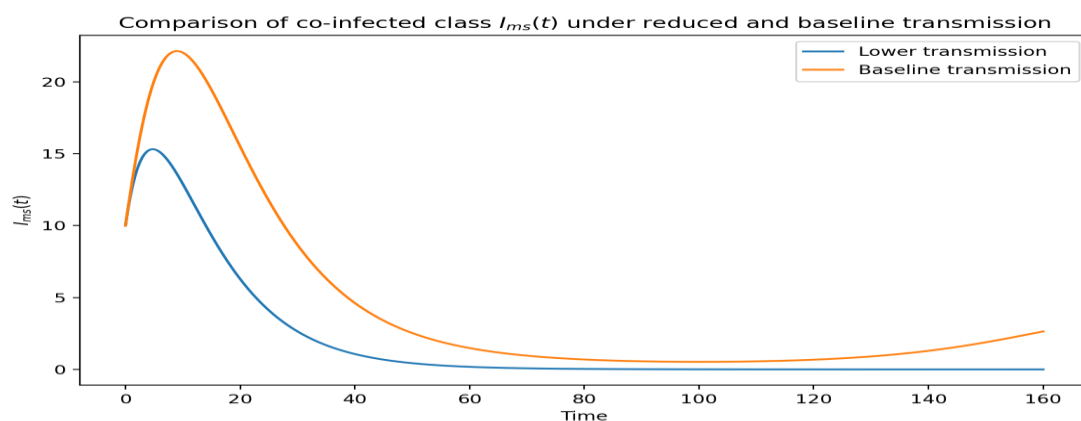


Figure 11: Co-infected population under reduced and higher transmission conditions.

VIII. Discussion

The malaria-only and schistosomiasis-only analyses give separate threshold conditions for the two infections. The disease-free equilibrium of the malaria submodel is locally and globally stable when $\mathcal{R}_{0m} < 1$, while the disease-free equilibrium of the schistosomiasis submodel is locally and globally stable when $\mathcal{R}_{0s} < 1$. Thus, each infection dies out when its reproduction number is below one. When the threshold is above one, the corresponding endemic equilibrium becomes relevant and the infection can remain in the population.

The malaria reproduction number contains the sensitive-malaria and resistant-malaria pathways. In particular, the term containing $\eta_2\rho$ shows how progression to resistant malaria can add to malaria transmission. In the full model, the co-infected class also enters the malaria transmission component through the term containing η_1 . This means that malaria transmission in a co-endemic population is not determined by the malaria-only class alone. Resistant infection and co-infection can also contribute to the force of infection.

For schistosomiasis, the reproduction number contains β_s , σ and μ_w . The parameters β_s and σ support transmission through exposure and environmental contamination, while μ_w removes contamination from the environment. This agrees with the numerical results. Higher schistosomiasis transmission and contamination increase the infected population, whereas faster environmental clearance reduces $W(t)$ and lowers schistosomiasis transmission. The result also agrees with the importance of safe water, sanitation and environmental control in endemic areas

The numerical scenarios give a clear threshold pattern. At the initial transmission level,

$$\mathcal{R}_{0m}^c = 1.2273, \quad \mathcal{R}_{0s} = 1.0526, \quad (99)$$

so both infections can persist. Malaria control alone reduces $\mathcal{R}_{0m}^c$ below one, but $\mathcal{R}_{0s}$ remains above one. In the same way, schistosomiasis or environmental control alone reduces the schistosomiasis component but leaves the malaria component above one. Under combined control,

$$\mathcal{R}_{0m}^c = 0.6136, \quad \mathcal{R}_{0s} = 0.0877, \quad (100)$$

and therefore $\mathcal{R}_0 = 0.6136 < 1$. The simulations show the same pattern: control of only one pathway does not remove the other infection, while the combined scenario produces a stronger decline in the infected and co-infected classes.

The main contribution of this study is the construction and analysis of one malaria–schistosomiasis co-infection model that brings together four features that are usually treated separately: resistant malaria, immune response, co-infection and environmental schistosomiasis transmission. The model also separates the malaria and schistosomiasis reproduction components before combining them as

$$\mathcal{R}_0 = \max\{\mathcal{R}_{0m}^c, \mathcal{R}_{0s}\}. \quad (101)$$

This form makes it possible to identify which infection remains above its threshold when only one control pathway is strengthened. The separate submodel analysis, the full-model threshold and the numerical control scenarios therefore give a direct link between the mathematical results and the two transmission routes.

The model is theoretical, and some parameter values are assumed. The numerical curves describe model behaviour under the stated parameter sets rather than predictions for a particular community. Estimation with malaria, schistosomiasis, treatment, resistance and environmental data would be needed for location-specific prediction. The equilibrium and threshold results obtained here also provide the basis for a separate study of bifurcation behaviour near the critical reproduction number.

IX. Conclusion

This study developed and analysed a deterministic model for malaria–schistosomiasis co-infection with drug-resistant malaria, immune response and environmental transmission of schistosomiasis. The model was shown to have non-negative and bounded solutions, and existence and uniqueness were established. The malaria-only and schistosomiasis-only submodels were then studied separately. Their disease-free and endemic equilibria were obtained, their reproduction numbers were derived, and the local and global stability conditions of the disease-free equilibria were established.

For the full system, the reproduction number is determined by the larger of the malaria and schistosomiasis transmission components. This result is useful because control of one infection does not automatically place the whole co-infection system below the epidemic threshold. The numerical scenarios illustrate this point. When both component reproduction numbers are greater than one, malaria and schistosomiasis can persist. Reducing only the malaria component leaves schistosomiasis above its threshold, while reducing only the schistosomiasis component leaves malaria above its threshold. When the two transmission pathways are controlled together, both component reproduction numbers fall below one and the infected classes decline.

The contribution of the work lies in linking malaria drug resistance, immune response, environmental schistosomiasis transmission and co-infection within the same mathematical framework. The model does not only give a single overall threshold; it also shows the separate malaria and schistosomiasis contributions to that threshold. This makes the effect of malaria control, schistosomiasis control and environmental clearance easier to interpret. The simulations further show the roles of malaria transmission, schistosomiasis transmission, resistance progression, environmental contamination and environmental clearance.

The results support a combined control approach in places where malaria and schistosomiasis occur together. Malaria transmission and drug resistance need to be reduced at the same time that schistosomiasis transmission and environmental contamination are addressed. Since some parameter values used here are assumed, future application should estimate the parameters from field or surveillance data. The analytical results obtained

in this paper also provide a suitable starting point for further work on bifurcation and other nonlinear behaviour of the co-infection system.

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