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Mathematical Analysis and Optimal Control of Schistosomiasis Transmission Model

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Abstract

Schistosomiasis, a health challenge in many communities, is prevalent as the rate of infection is one in every thirty individuals. In this work, a deterministic model for schistosomiasis transmission dynamics is studied. The stability properties of equilibrium states, disease-free and endemic equilibria are established in terms of the basic reproduction number, R_0. The sensitivity analysis of R_0 with respect to the model parameters is carried out using Partial rank correlation coefficients (PRCCs). The optimal control model with control measures, public health education, diagnosis and treatment and snail control, is formulated and its optimality system is derived using Pontragyin's maximum Principle. Simulation results showed that simultaneous implementation of public health education, diagnosis and treatment and snail control will reduce the burden of the schistosomiasis infection in the population. However due to toxicity of some snail controls to other aquatic bodies and difficulty to single out the chemical control that will focus only on the snail population even though snails are special food in Africa, it is preferable to implement public health education and diagnosis and treatment simultaneously in order to eradicate schistosomiasis transmission in the affected regions.

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1 Introduction

Schistosomiasis is a public health challenge in many countries as the rate of infection is one in every thirty individuals seen [5]. This makes its control a challenging task in areas where the infection is endemic. There are two stages of the disease; acute and chronic stages [15]. The acute stage is prevalent among persons who are exposed to fresh water in endemic regions while chronic stage occurs as a result of continuous deposition of the parasite eggs in the body of those who had acute infection [5]. The prevention that is put in place for schistosomiasis control focuses on expanding the use of mass administration of praziquantel, an anti schistosomal drug, to minimize infection-induced sickness which reduces infection rate among school-age children and adults in high-risk populations [2].

Although, mass drug administration is determined by schistosomiasis prevalence in affected communities, re-infection can occur even after praziquantel is administered [15] because treatment cannot eliminate the disease as the recovery rate only changes the speed of approaching equilibrium and in the mean time prevalence can be reduced [20]. There are concerns that repeated usage of praziquantel could lead to a situation where the body develop resistance to the effectiveness of the drug [3]. According to King *et al.* [30], schistosoma transmission can be reduced if snail control strategies will be implemented properly and this would complement strategies of modern day mass drug delivery programs, resulting in much improved prevention of infection and re-infection of schistosomiasis.

Several mathematical models on schistosomiasis have been studied and recommendations were made arising from the results of the models. Guiro *et al.* [1] observed through the use of threshold analysis that public enlightenment campaign has a positive impact on controlling schistosomiasis. Diaby *et al.* [16] considered snail competition for the disease control. Ishikawa *et al.* [17] predicted that among various possible control measures on the effective elimination of schistosoma, there is little probability of the resurgence of an epidemic, Chen *et al.* [18] proposed that environmental factors should be included in the control and eradication of schistosomiasis while Gao *et al.* [7] and Dida *et al.* [6] observed that the use of molluscicides as snail control would be the most effective control measure to curtail schistosomiasis transmission. Furthermore, Abokwara and Madubueze [25] considered the impact of public health education and snail control while

Kanyi *et al.* [8] discussed the optimal control of schistosomiasis with early treatment, snail elimination, and chlorination of the water body as control measures.

From the aforementioned authors, it is only Kanyi *et al.* [8] that studied optimal control of schisotomiasis. Application of optimal control to schisotomiasis disease provides information on how the implementation of control measures minimize schisotomiasis transmission in an endemic population. Therefore, this research would consider the optimal control impact of public health education, snail control using molluscicides and early treatment on schistosomaisis transmission which is an extension of Abokwara and Madubueze [25] and Kanyi *et al.* [8]. Abokwara and Madubueze [25] did not consider the treatment of chronic stage of the disease and optimal control and sensitivity analysis of their model. For Kanyi *et al.* [8], the public health education impact and chronic stage of the disease and sensitivity analysis were not examined. The public health eduction is considered based on the result of Abokwara and Madubueze [25] and Sacolo *et al.* [22] that enlightening members of the society about schistosomiasis infection will help to reduce the burden of the disease.

The work will involve carrying out qualitative analysis and sensitivity analysis of the model parameters to identify the most influential parameter(s) to be targeted for intervention strategies. With the results of sensitivity analysis, an optimal control model will be formulated for effective decision making in controlling the spread of schisotosomiasis.

The rest of this paper consists model formulation in Section II, model analysis in Section III, Section IV is the optimal control analysis with Section V as the numerical simulation and discussion while conclusion is Section VI.

2 Model formulation

The model comprises two host populations, the human population and the snail population, the miracidia, $M(t)$ and the cercaria, $P(t)$ at any time, t. The human population is subdivided into susceptible human population, $S_H(t)$, acute infected human population, $I_{1H}(t)$, chronic infected human population, $I_{2H}(t)$, treatment compartment, $T_H(t)$. Individuals in the human population moves from one class to another as their status changes and the disease evolves. The infection occurs when the susceptible human

have contact with fresh water that has free living larva called cercariae. The susceptible individuals, $S_H(t)$ progress to $I_{1H}(t)$ as a result of infection at a rate, λ_H , where $\lambda_H = \frac{\beta_1 P}{P_0+\varepsilon P}$, with P_0, β_1 and ε defined in [12]. The acute infected human, I_{1H} progresses to Chronic stage when treatment is not given early or completed and this result to continue deposition of the parasite eggs that are trapped in the host (human) tissues [5].

The treatment class, $T_H(t)$, constitutes of infected humans, $I_{1H}(t)$ and $I_{2H}(t)$, undergoing treatment at respective rates, σ_1 and σ_2 and they can not shed the eggs due to their treatment. They recover and become susceptible again at a rate, e. The infected humans, $I_{1H}(t)$ and $I_{2H}(t)$, contribute to the life cycle of the schistosoma as they shed eggs when they come to swim or fetch water from river at rates, $N_E\gamma_1$ and $N_E\gamma_1\alpha$ respectively. These eggs find their way into fresh water supply and hatch into a free swimming ciliated larva called miracidium and this constitute the miracidia population, $M(t)$.

For the snail population, it is subdivided into susceptible snail population, $S_S(t)$ and infected snail population, $I_S(t)$ at any time, t. The susceptible snail comes in contact with miracidia at the rate, λ_S, where $\lambda_S = \frac{\beta_2 M}{M_0+\varepsilon M}$ with β_2, and ε, and M_0 defined in [12]. The infected snails release a free living larva called cercariae, $P(t)$. There is no direct transmission of the disease between human and snail population [15]. Furthermore, disease induced death, δ_{2H} for $I_{2H}(t)$, is assumed to be greater than disease induced death in δ_{1H} for $I_{1H}(t)$, climate variation do not affect the contact patterns and reproduction does not take place in I_S class as a result of infection. The descriptions of the parameters of the model and the model systematic diagram are presented in Table 1 and Figure 1 respectively.

With the Table 1 and Figure 1, we have the under listed differential equations as follows

$$\left.\begin{array}{ll}
\frac{dS_H}{dt} & = \Lambda_H + eT_H - \lambda_H S_H - \mu_H S_H \\
\frac{dI_{1H}}{dt} & = \lambda_H S_H - (k + \delta_{1H} + \sigma_1 + \mu_H) I_{1H} \\
\frac{dI_{2H}}{dt} & = kI_{1H} - (\sigma_2 + \mu_H + \delta_{2H}) I_{2H} \\
\frac{dT_H}{dt} & = \sigma_1 I_{1H} + \sigma_2 I_{2H} - eT_H - \mu_H T_H \\
\frac{dM}{dt} & = N_E\gamma_1(I_{1H} + \alpha I_{2H}) - \mu_M M \\
\frac{dS_S}{dt} & = \Lambda_S - \lambda_S S_S - \mu_S S_S - d_1 S_S \\
\frac{dI_S}{dt} & = \lambda_S S_S - (\mu_S + d_1 + \delta_S) I_S \\
\frac{dP}{dt} & = \gamma_2 I_S - \mu_P P
\end{array}\right\} \quad (1)$$

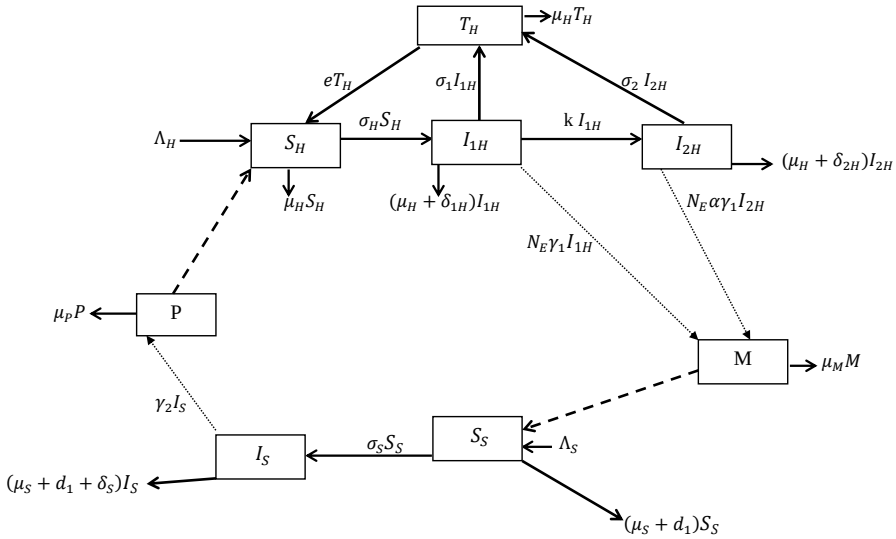

Figure 1: Schematic diagram of the Schistosomiasis disease

Table 1: Parameter descriptions and values

Parameter	Epidemiological Interpretation	Baseline(Range)	Sources
Λ_H	Recruitment rate for human population	$254(-)$	[25]
Λ_S	Recruitment rate for snail population	$3000(-)$	[12]
k	Progression rate from I_{1H} to I_{2H}	$0.0262(0.02-0.03)$	[25]
δ_{1H}	Disease induced death for I_{1H}	$2.74(2-90)\times 10^{-4}$	[12]
δ_{2H}	Disease induced death for I_{2H}	$9.13((80-100)\times 10^{-4}$	[25]
e	Re-susceptibility rate from T_H to S_H	$6.87(5-7)\times 10^{-4}$	[14]
μ_H	Natural death rate for human population	$4.379\times 10^{-4}(-)$	[12]
μ_S	Natural death rate for snail population	$0.000569(-)$	[12]
μ_M	Natural death rate for M	$0.9(0.6-0.95)$	[12]
μ_P	Natural death rate for P	$4(3-6)\times 10^{-3}$	[12]
P_0	Half saturation constant of cercariae	$9\times 10^{7}(-)$	[12]
δ_S	Disease induced death for snail population	$4.012(3.5-45)\times 10^{-4}$	[14]
M_0	Half saturation constant of Miracidia	1×10^{8}(-)	[12]
ε	Growth velocity limitation of P and M	0.2	[12]
β_1	Transmission rate for human population	$0.09753(0.07-0.12)$	[25]
β_2	Transmission rate for snail population	$0.616(0.5-0.8)$	[12]
α	Influential shedding rate for I_{2H}	$1.01(0.5-1.5)$	[25]
γ_1	Shedding rate for I_{1H}	$6.96(6.0-7.5)$	[25]
γ_2	Shedding rate for I_S	$2.6(2.0-3.5)$	[12]
d_1	Predation rate for snail population	$0.01(-)$	Assumed
N_E	Number of eggs secreted by humans	$300(250-350)$	[12]
σ_1	Treatment rate for I_{1H}	$0.05(0.04-0.06)$	[25]
σ_2	Treatment rate for I_{2H}	$0.03(0.02-0.04)$	[25]

subject to initial conditions $S_H(0) > 0$, $I_{1H}(0) \geq 0$, $I_{2H}(0) \geq 0$, $M(0) \geq 0$, $S_S(0) > 0$, $I_S(0) \geq 0$ and $P(0) \geq 0$ with $\lambda_H = \frac{\beta_1 P}{P_0+\varepsilon P}$, $\lambda_S = \frac{\beta_2 M}{M_0+\varepsilon M}$. The model parameters are assumed to be positive.

3 Model Analysis

3.1 Invariant region and positivity of the solutions

Let $N_H(t) = S_H(t) + I_{1H}(t) + I_{2H}(t) + T_H(t)$ and $N_S(t) = S_S(t) + I_S(t)$ be total human and snail populations at any time, t with initial conditions $N_H(0) = N_{H0}$ and $N_S(0) = N_{S0}$. We state the following lemma.

Lemma 1 (Invariant region)**.** *All feasible solutions of system (1) are uniformly bounded in a proper subset $D = D_H \times D_S \times D_M \times D_P$ with non-negative initial conditions where $D_H = \{(S_H, I_{1H}, I_{2H}, T_H)\epsilon\Re_+^4 : N_H(t) \leq \frac{\Lambda_H}{\mu_H}\}$, $D_S = \left\{(S_S, I_S)\epsilon\Re_+^2 : N_S(t) \leq \frac{\Lambda_S}{\mu_S+d_1}\right\}$, $D_P = \left\{P \leq \frac{\Lambda_S\gamma_2)}{(\mu_S+d_1)\mu_P}\right\}$ and $D_M = \left\{M \leq \frac{\Lambda_H\gamma_1 N_E(1+\alpha)}{\mu_H\mu_M}\right\}$ are the subsets for human population, snail population, cercariae and miracidia respectively.*

Proof. We have from the total human population that $\frac{dN_H}{dt} \leq \Lambda_H - \mu_H N_H$. Applying Birkhoff and Rota Theorem [31] on differential inequality and integrating with initial condition, $N_H(0)$, we have

$$N_H \leq \frac{\Lambda_H}{\mu_H} - \Big[\frac{(\Lambda_H - \mu_H N_H(0))}{\mu_H}\Big]e^{-\mu_H t}.$$

As $t \to \infty$, the population size, N_H approaches $N_H \leq \frac{\Lambda_H}{\mu_H}$.

For the Miracidia population, we have from the fifth equation of model equation (1) that

$$\frac{dM}{dt} = N_E\gamma_1 I_{1H} + N_E\gamma_1\alpha I_{2H} - \mu_M M.$$

But, $I_{1H} \leq N_H(t)$ and $I_{2H} \leq N_H(t)$, so that

$$\frac{dM}{dt} \leq \gamma_1 N_E(1+\alpha)N_H(t) - \mu_M M.$$

With $N_H \leq \frac{\Lambda_H}{\mu_H}$, we have

$$\frac{dM}{dt} \leq \gamma_1 N_E(1+\alpha)\frac{\Lambda_H}{\mu_H} - \mu_M M.$$

Applying the theorem in [31] with initial conditions, $M(0)$ yields

$$M \leq \frac{\Lambda_H \gamma_1 N_E(1+\alpha)}{\mu_M \mu_H} - \Big[\frac{\Lambda_H \gamma_1 N_E(1+\alpha)}{\mu_M \mu_H} - M(0)\Big] e^{-\mu_M t}.$$

As $t \to \infty$, the population size, $M(t) \leq \frac{\Lambda_H \gamma_1 N_E(1+\alpha)}{\mu_M \mu_H}$.

For the snail population with initial condition, $N_S(0)$, $\frac{dN_S}{dt} \leq \Lambda_S - (\mu_S + d_1)N_S$ so that

$$N_S \leq \frac{\Lambda_S}{(\mu_S + d_1)} - \Big[\frac{\Lambda_S - (\mu_S + d_1)N_S(0)}{(\mu_S + d_1)}\Big] e^{-(\mu_S + d_1)t}.$$

As $t \to \infty$, the population size, $N_S \leq \frac{\Lambda_S}{(\mu_S + d_1)}$.

Furthermore for cercariae concentration $P(t)$ of the eighth equation of model equation (1) with $I_S \leq N_S \leq \frac{\Lambda_S}{(\mu_S + d_1)}$, we have

$$\frac{dP}{dt} \leq \frac{\gamma_2 \Lambda_S}{(\mu_S + d_1)} - \mu_P.$$

With the theorem in [31] and initial conditions, $P(0)$, we get

$$P \leq \frac{\Lambda_S \gamma_2}{(\mu_S + d_1)\mu_P} - \Big[\frac{\Lambda_S \gamma_2}{(\mu_S + d_1)\mu_P} - P(0)\Big] e^{-\mu_P t}.$$

As $t \to \infty$, $P(t) \leq \frac{\Lambda_S \gamma_2}{(\mu_S + d_1)\mu_P}$.

Hence, the feasible solutions of model (1) will enter the positive invariant region $D = D_H \times D_S \times D_M \times D_P$. This completes the proof. □

Theorem 1 (Positivity of solutions)**.** *The solutions of system (1),S_H, I_{1H}, I_{2H}, T_H, M, S_S, I_S, P with non-negative initial data are non-negative for all time, $t > 0$.*

Proof. Let $\tau = \sup\{t > 0 : S_H(0) > 0, I_{1H}(0) > 0, I_{2H} > 0, T_H > 0, M(0) > 0, S_S(0) > 0, I_S(0) > 0, P(0) > 0\} \in [0, t]$.

From the first equation of (1), we have

$$\frac{dS_H}{dt} = \Lambda_H + eT_H - \lambda_H S_H - \mu_H S_H \geq -(\lambda_H + \mu_H)S_H.$$

Using integrating factor method with initial condition, $S_H(0)$, it yields

$$S_H(t) \geq S_H(0) \exp\Big\{ - \int_0^t (\lambda_H(\tau) + \mu_H) d\tau \Big\} > 0.$$

Hence, S_H is always positive for $t > 0$.

In similar way for $t > 0$, $I_{1H} > 0$, $I_{2H} > 0$, $T_H > 0$, $M > 0$, $S_S > 0$, $I_S > 0$, $P > 0$. Therefore, the solutions $(S_H(t), I_{1H}(t), I_{2H}(t), T_H, M(t), S_S(t), I_S(t), P(t))$ of model equation (1) are non-negative for $t > 0$. This implies that the model is well posed and make biological meaning so it is possible to carry out mathematical analysis of the model. □

3.2 Existence of the disease-free equilibrium state and basic reproduction number, R_0

The disease-free equilibrium state, E_0, is an equilibrium state where there is no infection. It is given by

$$E_0 = (S_H^0, I_{1H}^0, I_{2H}^0, T_H^0, M^0, S_S^0, I_S^0, P^0) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, \frac{\Lambda_S}{\mu_S + d_1}, 0, 0\right). \tag{2}$$

The basic reproduction number, R_0, is the number of new cases reproduced in a wholly susceptible population when an infective individual is introduced into the population [10].

Applying the next generation matrix method [10], let $\mathcal{F}(x)$ be the rate of new infections and $\mathcal{V}(x)$ be the rate of transition by any other means with $x = (I_{1H}, I_{2H}, T_H, M, I_S, P, S_H, S_S)$. The model equation (1) can be written as

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x)$$

where

$$\mathcal{F} = \begin{pmatrix} \frac{\beta_1 P S_H}{P_0 + \varepsilon P} \\ 0 \\ 0 \\ 0 \\ \frac{\beta_2 M S_S}{M_0 + \varepsilon M} \\ 0 \\ 0 \\ 0 \end{pmatrix}, \qquad \mathcal{V} = \begin{pmatrix} fI_{1H} \\ -kI_{1H} + gI_{2H} \\ -\sigma_1 I_{1H} - \sigma_2 I_{2H} + hT_T \\ -N_E\gamma_1(I_{1H} + \alpha I_{2H}) + \mu_M M \\ qI_S \\ -\gamma_2 I_S + \mu_P P \\ -\Lambda_H - eT_H + \frac{\beta_1 P S_H}{P_0 + \varepsilon P} + \mu_H S_H \\ -\Lambda_S + \frac{\beta_2 M S_S}{M_0 + \varepsilon M} + \mu_S S_S \end{pmatrix},$$

where

$$\begin{aligned} f &= k + \sigma_1 + \delta_{1H} + \mu_H, \\ g &= \mu_H + \sigma_2 + \delta_{2H}, \\ n &= \mu_S + d_1, \\ h &= e + \mu_H, \\ q &= n + \delta_S. \end{aligned} \tag{3}$$

Taking the derivatives of $\mathcal{F}$ and $\mathcal{V}$ at E_0 give the Jacobian matrices, F and V, as follows

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & \frac{S_H^0 \beta_1}{P_0} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{S_S^0 \beta_2}{M_0} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} f & 0 & 0 & 0 & 0 & 0 \\ -k & g & 0 & 0 & 0 & 0 \\ -\sigma_1 & -\sigma_2 & h & 0 & 0 & 0 \\ -N_E\gamma_1 & -N_E\gamma_1\alpha & 0 & \mu_M & 0 & 0 \\ 0 & 0 & 0 & 0 & q & 0 \\ 0 & 0 & 0 & 0 & -\gamma_2 & \mu_P \end{pmatrix}.$$

The eigenvalues of matrix FV^{-1} are

$$0, 0, 0, 0, \pm\sqrt{\frac{S_H^0 S_S^0 \beta_2 \beta_1 \gamma_2 \gamma_1 N_E(\alpha k + g)}{M_0 P_0 \mu_M \mu_P f g q}}.$$

With the definition of basic reproduction number, R_0, as the maximum positive eigenvalue of FV^{-1}, we have

$$R_0 = \sqrt{\frac{S_H^0 S_S^0 \beta_2 \beta_1 \gamma_2 \gamma_1 N_E(\alpha k + g)}{M_0 P_0 \mu_M \mu_P f g q}} = \sqrt{R_{0S}(R_{0HA} + R_{0HC})} \tag{4}$$

where $R_{0S} = \frac{S_S^0 \beta_2 \gamma_2}{q P_0 \mu_P}$, $R_{0HA} = \frac{S_H^0 \beta_1 \gamma_1 N_E}{f M_0 \mu_M}$, $R_{0HC} = \frac{S_H^0 \beta_1 \gamma_1 N_E \alpha k}{f g M_0 \mu_M}$. Here, R_{0S}, R_{0HA}, R_{0HC} are the reproduction numbers for the snail and miracidia interaction, acute infected human and cercariae interaction, and chronic infected human and cercariae interaction respectively.

3.2.1 Stability of the disease-free equilibrium state

Theorem 2. *If E_0 is the DFE of the model, then E_0 is locally asymptotically stable if $R_0 < 1$, but unstable if $R_0 > 1$.*

Proof. This is done by linearizing equation (1) at DFE, E_0 to give a Jacobian matrix, $J(E_0)$ as

$$\begin{pmatrix} -\mu_H & 0 & 0 & e & 0 & 0 & 0 & -\frac{S_H^0\beta_1}{P_0} \\ 0 & -f & 0 & 0 & 0 & 0 & 0 & \frac{S_H^0\beta_1}{P_0} \\ 0 & k & -g & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_1 & \sigma_2 & -h & 0 & 0 & 0 & 0 \\ 0 & N_E\gamma_1 & N_E\gamma_1\alpha & 0 & -\mu_M & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\frac{S_S^0\beta_2}{M_0} & -n & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{S_S^0\beta_2}{M_0} & 0 & -q & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \gamma_2 & -\mu_P \end{pmatrix}. \tag{5}$$

The eigenvalues of the Jacobian matrix, $J(E_0)$ are $-\mu_H, -h, -n$ and the roots of the following characteristic equation

$$\lambda^5 + A_1\lambda^4 + A_2\lambda^3 + A_3\lambda^2 + A_4\lambda + A_5 = 0 \tag{6}$$

where

$$\begin{aligned} A_1 &= f + \mu_M + g + q + \mu_P, \\ A_2 &= f(g + q + \mu_M + \mu_P) + g(q + \mu_M + \mu_P) + q(\mu_M + h\mu_P) + \mu_P\mu_M, \\ A_3 &= fg(q + \mu_M + \mu_P) + q(f + g)(\mu_P + \mu_M) + \mu_P\mu_M(f + g + q), \\ A_4 &= fgq(\mu_P + \mu_M) + g\mu_M\mu_P(q + f) + fq\mu_M\mu_P(1 - R_{0S}R_{0HA}), \\ A_5 &= fgq\mu_M\mu_P(1 - R_0^2). \end{aligned}$$

Using [4], the roots of the polynomial (6) have negative real part solutions if A_1, A_2, A_3, A_4, A_5 are positive which is true if $R_0 < 1$. This means that the Jacobian matrix (5) has negative eigenvalues whenever $R_0 < 1$. Hence, the DFE, E_0 is locally asymptotically stable if $R_0 < 1$. However when the $R_0 > 1$, $A_5 < 0$. This implies that positive eigenvalue exists as such means that the DFE, E_0 is unstable if $R_0 > 1$. □

3.3 Existence and stability of the endemic equilibrium state

Endemic equilibrium state, E_e, is the state where the infected state variables are not equal to zero. At equilibrium state, $\frac{dS_H}{dt} = 0, \frac{dI_{1H}}{dt} = 0, \frac{dI_{2H}}{dt} = 0, \frac{dM}{dt} = 0, \frac{dS_S}{dt} = 0, \frac{dI_S}{dt} = 0, \frac{dP}{dt} = 0$. Solving for non-zero infected state variables at equilibrium state gives the endemic equilibrium state, $E_e = (S_H^e, I_{1H}^e, I_{2H}^e, T_H^e, M^e, S_S^e, I_S^e, P^e)$ where

$$\left.\begin{aligned}
S_H^e &= \tfrac{\Lambda_H(nM_0\mu_M\mu_H fgh\varepsilon(R_0^2-1)+A+E\beta_1\mu_M M_0 n)}{\mu_H(nM_0\mu_M(ER_0^2\beta_1+\mu_H fgh\varepsilon(R_0^2-1))+A)},\\
I_{1H}^e &= \tfrac{\Lambda_H\beta_1\mu_M M_0 ghn(R_0^2-1)}{nM_0\mu_M(ER_0^2\beta_1+\mu_H fgh\varepsilon(R_0^2-1))+A},\\
I_{2H}^e &= \tfrac{k\Lambda_H\beta_1\mu_M M_0 hn(R_0^2-1)}{nM_0\mu_M(ER_0^2\beta_1+\mu_H fgh\varepsilon(R_0^2-1))+A},\\
T_H^e &= \tfrac{\Lambda_H\beta_1\mu_M M_0 n(g\sigma_1+k\sigma_2)(R_0^2-1)}{nM_0\mu_M(ER_0^2\beta_1+\mu_H fgh\varepsilon(R_0^2-1))+A},\\
M^e &= \tfrac{N_E\gamma_1\Lambda_H\beta_1 M_0 hn(g+\alpha k)(R_0^2-1)}{nM_0(ER_0^2\beta_1+\mu_H fgh\varepsilon(R_0^2-1))+A},\\
S_S^e &= \tfrac{fghM_0\mu_H\mu_M(P_0\mu_P+\Lambda_S\gamma_2\varepsilon)+B}{\gamma_2(nM_0\beta_1\mu_M E+A)},\\
I_S^e &= \tfrac{fghnP_0M_0\mu_P\mu_H\mu_M(R_0^2-1)}{\gamma_2(nM_0\beta_1\mu_M E+A)},\\
P^e &= \tfrac{fghnqP_0M_0\mu_P\mu_H\mu_M(R_0^2-1)}{q\mu_P(nM_0\beta_1\mu_M E+A)}.
\end{aligned}\right\}$$

with

$$\begin{aligned}
A &= M_0\varepsilon\mu_H\mu_M fghn + hN_E\gamma_1\beta_1\Lambda_H(k\alpha+g)(n\varepsilon+\beta_2),\\
E &= fgh - e(k\sigma_2+g\sigma_1) > 0,\\
B &= \Lambda_S\gamma_2(hN_E\gamma_1\beta_1\Lambda_H(k\alpha+g) + M_0\beta_1\mu_M E).
\end{aligned}$$

The endemic equilibrium state, E_e, exists whenever $R_0 > 1$.

3.3.1 Bifurcation Analysis

A dynamical system is said to exhibit bifurcation when its parameter value changes and causes a sudden qualitative change in its behaviour [29]. Bifurcation can be forward or backward. When a bifurcation is forward, it implies that the disease-free equilibrium and endemic equilibrium state are locally asymptotically stable if $R_0 < 1$ and $R_0 > 1$ respectively while in backward bifurcation, a coexistence of the disease-free equilibrium and endemic equilibrium states occur even when $R_0 < 1$. This implies that

when $R_0 < 1$ in the presence of control measures, it is not enough to control the spread of schistosomiasis. Thus, there may be need for more control measures that would make the bifurcation to be forward.

The bifurcation analysis is carried out using the Centre Manifold Theory by [19] that involves choosing a bifurcation parameter.

We state the Centre Manifold Theory as follows.

Theorem 3 (Centre Manifold Theory [19])**.** *Consider the following general system of ordinary differential equation with parameter* ϕ. $\frac{dx}{dt} = f(x, \phi), f : \Re^n \times \Re \longrightarrow \Re$ *and* $f \epsilon C^2(\Re^n \times \Re)$. *When 0 is an equilibrium point of the system (that is,* $f(0, \phi) \equiv 0$ *for all* ϕ*) and it implies that*

i. $N = D_x f(0,0) = \frac{\partial f_i}{\partial x_j}(0,0)$ *is the linearization matrix of the system around the equilibrium 0 with* ϕ *evaluated at 0.*

ii. Zero is a simple eigenvalue of N *and other eigenvalues of* N *have negative real parts.*

iii. Matrix N *has right eigenvector* z *and a left eigenvector* p *corresponding to the zero eigenvalue.*

Let f_k *be the kth component of* f *and*

$$r = \sum_{k,i,j=1}^{n} p_k z_i z_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$

$$s = \sum_{k,i=1}^{n} p_k z_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0).$$

The local dynamics of the system around the equilibrium point 0, is totally determined by the signs of r *and* r.

i. $r > 0$ *and* $s > 0$. *When* $\phi < 0$ *with* $|\phi| \ll 1, 0$ *is locally asymptotically stable, and there exist a positive unstable equilibrium; when* $0 < |\phi| \ll 1, 0$*, 0 is unstable and there exist a negative and locally asymptotically stable equilibrium.*

ii. If $r < 0$ *and* $s < 0$, *when* $\phi < 0$ *with* $|\phi| \ll 1, 0$ *unstable; when* $0 < |\phi| \ll 1$*, asymptotically stable, and there exist a positive unstable equilibrium.*

iii. If $r > 0$ and $s < 0$, when $\phi < 0$ with $|\phi| \ll 1,0$ unstable; and there exists a locally asymptotically stable negative equilibrium; when $0 < |\phi| \ll 1,0$ is stable and a positive unstable equilibrium appears.

iv. If $r < 0$ and $s > 0$, when ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly to a negative unstable equilibrium becomes positive and locally asymptotically stable.

Particularly, if $r > 0$ and $s > 0$, backward bifurcation occurs at $\phi = 0$ and a forward bifurcation occurs if $r < 0$ and $s > 0$.

Let β_1^* be the bifurcation parameter at $R_0 = 1$ and it is obtained by solving for β_1 at $R_0 = 1$ that is

$$\sqrt{\frac{S_H^0 S_S^0 \beta_2 \beta_1 \gamma_2 \gamma_1 N_E(\alpha k + g)}{M_0 P_0 \mu_M \mu_P f g q}} = 1.$$

This leads to

$$\beta_1^* = \beta_1 = \frac{f g q n \mu_H \mu_M \mu_P M_0 P_0}{\Lambda_S \Lambda_H \beta_2 \gamma_1 \gamma_2 N_E (g + \alpha k)}.$$

At $R_0 = 1$, the eigenvalues of the Jacobian matrix, $J(E_0)$ of equation (6) has simple zero eigenvalue and negative eigenvalues using Routh-Hurwitz criteria and [4].

Let $z_i s = (z_1, z_2, z_3, z_4, z_5, z_6, z_7, z_8)$, $p_i s = (p_1, p_2, p_3, p_4, p_5, p_6, p_7, p_8)$ be the right and left eigenvectors. The right eigenvalues $z_i s$ are determined by multiplying the Jacobian matrix, $J(E_0)$ with $z_i s$ and equate to zero. This is given as

$$\begin{aligned}
z_1 &= -\frac{E}{gh\mu_H} z_2, & z_3 &= \frac{k}{g} z_2, & z_4 &= \frac{g\sigma_1 + k\sigma_2}{gh} z_2, \\
z_5 &= \frac{P_0 M_0 f q \mu_P}{\beta_1 \beta_2 S_H^0 S_S^0 \gamma_2} z_2, & z_6 &= -\frac{P_0 f q \mu_P}{n \beta_1 S_H^0 \gamma_2} z_2, & & \\
z_7 &= \frac{f \mu_P P_0}{\gamma_2 \beta_1 S_H^0} z_2, & z_8 &= \frac{f P_0}{\beta_1 S_H^0} z_2, & z_2 &= z_2 > 0,
\end{aligned}$$

where $E = fgh - e(g\sigma_1 + k\sigma_2) > 0$.

For the eigenvector $p_i s$, we transpose the Jacobian matrix and multiply with $p_i s$ which is equate to zero to yield

$$p_1 = 0, \qquad p_3 = \frac{\gamma_1\gamma_2 N_E \alpha\beta_1\beta_2 S_H^0 S_S^0}{P_0 M_0 g q \mu_M \mu_P} p_2, \qquad p_4 = 0,$$
$$p_5 = \frac{\gamma_2\beta_1\beta_2 S_H^0 S_S^0}{P_0 M_0 q \mu_P \mu_M} p_2, \qquad p_6 = 0,$$
$$p_7 = \frac{\beta_1 S_H^0 \gamma_2}{P_0 h \mu_P} p_2, \qquad p_8 = \frac{\beta_1 S_H^0}{P_0 \mu_P} p_2, \qquad p_2 = p_2 > 0.$$

Let $S_H = x_1, I_{1H} = x_2, I_{2H} = x_3, T_H = x_4, M = x_5, S_S = x_6, I_S = x_7$, $P = x_8$. The functions

$$f_2 = \frac{\beta_1 x_8 x_1}{P_0 + \varepsilon x_8} - f x_2, f_7 = \frac{\beta_2 x_5 x_6}{M_0 + \varepsilon x_5} - q x_7$$

give non-zero partial derivative(s) at DFE, E_0 as

$$\frac{\partial^2 f_2(E_0)}{\partial x_1 \partial x_8} = \frac{\beta_1}{P_0}, \qquad \frac{\partial^2 f_2(E_0)}{\partial x_8^2} = -\frac{2\varepsilon S_H^0 \beta_1}{P_0^2},$$
$$\frac{\partial^2 f_7(E_0)}{\partial x_5 \partial x_6} = \frac{\beta_2}{M_0}, \qquad \frac{\partial^2 f_7(E_0)}{\partial x_5^2} = -\frac{2\varepsilon S_S^0 \beta_2}{M_0^2}.$$

Using the theorem in [19], the bifurcation coefficient, r, is given by

$$r = p_2 \left[z_1 z_8 \frac{\partial^2 f_2(E_0)}{\partial x_1 \partial x_8} + z_8^2 \frac{\partial^2 f_2(E_0)}{\partial x_8^2} \right] + p_7 \left[z_5 z_6 \frac{\partial^2 f_7(E_0)}{\partial x_5 \partial x_6} + z_5^2 \frac{\partial^2 f_7(E_0)}{\partial x_5^2} \right].$$

Upon substitution, we have

$$r = -p_2 z_2^2 \frac{f}{S_H^0} \Big[\frac{E}{g h \mu_H} + \frac{2 f \varepsilon}{\beta_1} + \frac{f q^2 P_0 \mu_P}{h \beta_1 \beta_2 S_S^0 \gamma_2} (M_0 + 2\varepsilon) \Big].$$

For the bifurcation coefficient s, we have

$$s = p_2 \Big[z_8 \frac{\partial^2 f_2(E_0)}{\partial \beta_1 \partial x_8} \Big] = \frac{f}{\beta_1} p_2 z_2.$$

Since $r < 0$ and $s > 0$, it implies that a forward bifurcation exists at $R_0 = 1$. This is display graphically in Figure 2. We have the following theorem.

Theorem 4. *The model (1) exhibits a forward bifurcation at* $R_0 = 1$.

This means that the endemic equilibrium bifurcates forward and exists only when $R_0 > 1$.

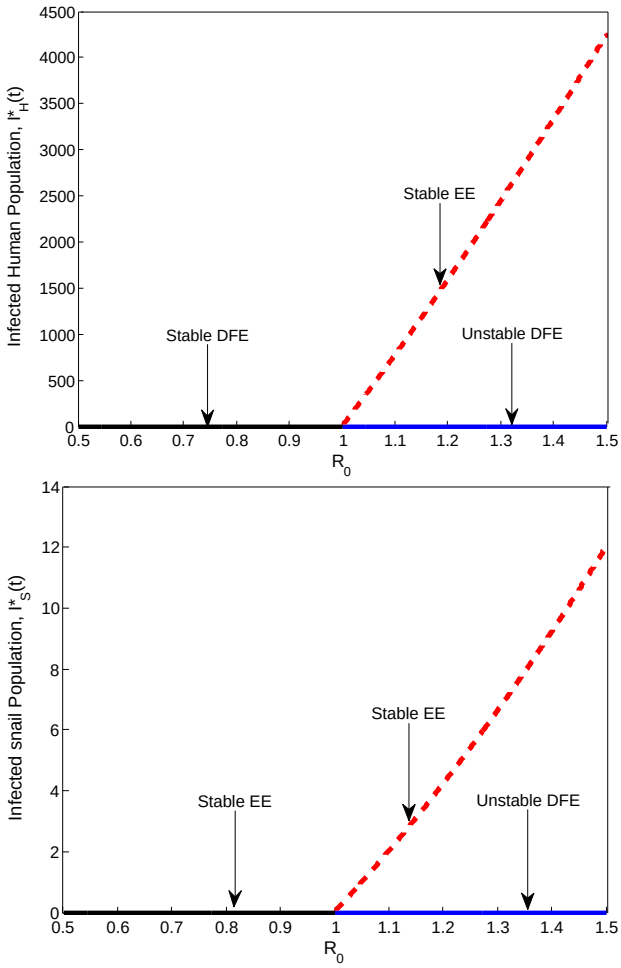

Figure 2: Forward bifurcation diagrams for I_{1H} and I_S as functions of R_0. All parameters are in Table 1.

3.4 Sensitivity Analysis

Sensitivity analysis investigates the robustness of model prediction to its parameters given that there are mostly errors in collecting data and parameter values are presumed [9]. In disease modelling, it tells how important each parameter of model is to disease eradication as a result of its impact on the basic reproduction number, R_0. Since basic reproduction number, R_0 is a threshold quantity that determines the eradication and persistence of the disease, sensitivity analysis is carried out on R_0 to find out the most influential model parameters to be targeted for intervention strategies. Some of the nominal values and ranges of the parameters are obtained from [12] and [14] while some are assumed. For each of these parameters range with an assumption of statistical independence, the simulations are evaluated using the partial rank correlation coefficients (PRCCs) of the parameters of interest. The Tornado plot of the PRCC is displayed in Fig. 3 while the relationship of R_0 and the most influential parameters is given in Fig. 4.

For the Tornado plot, the parameters with negative PRCCs reduce the spread of the disease if they are increased while the parameters with positive PRCCs promote the spread of disease whenever they are increased. Hence in Fig. 4, increasing mortality rates of cercariae, (μ_P), miracidia, (μ_M), as well as the rates at which acute infective and chronic infective human are transferred to the treatment class, (σ_1, σ_2) will reduce the spread of schistosomiasis in the population. Still in Fig. 4, it could be observed that the transmission rates for human population and snail population (β_1, β_2), the number of eggs secreted by infected humans (N_E), shedding rate for infected human and infected snails (γ_1, γ_2) and the parameter that influences the shedding rate for chronic infected class (α) contribute to the burden of schistosomiasis in the population.

4 Optimal control analysis

Optimal control deals with finding a control law for a given system in order to obtain a certain optimality criterion.

With the results of the sensitivity analysis in Fig. 4, we formulate an optimal control model of model (1) to determine optimal prevention in terms of public health education $(u(t))$, diagnosis and treatment $(v(t))$ and snail control $(w(t))$ strategies that will reduce the burden of schistosomiasis

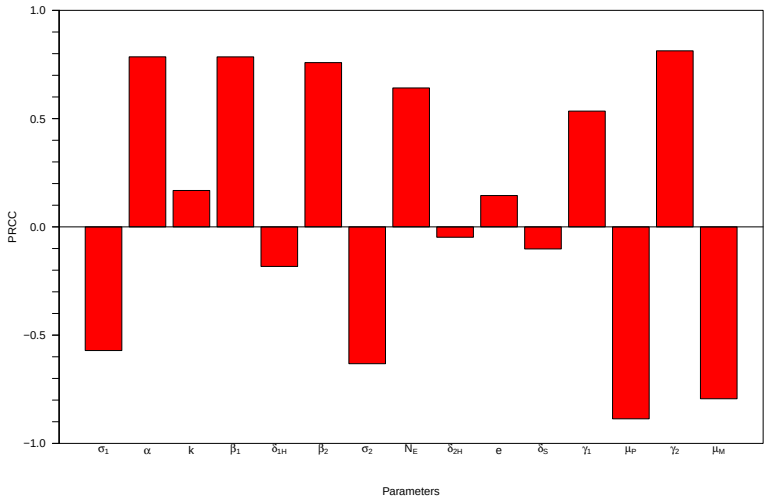

Figure 3: Tornado plot showing the impact of model parameters on the dynamics of the schistosomiasis model.

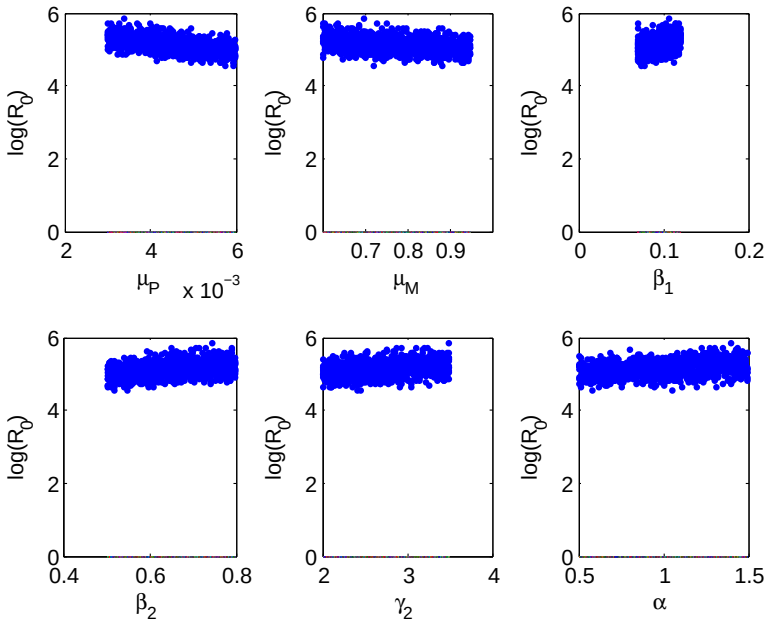

Figure 4: Monte Carlo simulations for the six parameters with the highest significant PRCC values, generated using the parameter values in Table 1. In each simulations run, 1000 simulations of the randomly selected parameters were used.

with minimal implementation cost. This is given by

$$\left.\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H + eT_H - \frac{(1-mu(t))\beta_2 PS_H}{P_0+\varepsilon P} - \mu_H S_H \\
\frac{dI_{1H}}{dt} &= \frac{(1-mu(t))\beta_1 PS_H}{P_0+\varepsilon P} - ((1-v(t))(k+\delta_{1H}) \\
&\quad + \sigma_1 + \mu_H + \eta_1 v(t))I_{1H} \\
\frac{dI_{2H}}{dt} &= (1-v(t))kI_{1H} - ((1-v(t))\delta_{2H} + \sigma_2 + \mu_H + \eta_2 v(t))I_{2H} \\
\frac{dT_H}{dt} &= (\sigma_1 + \eta_1 v(t))I_{1H} + (\sigma_2 + \eta_2 v(t))I_{2H} - (e+\mu_H)T_H \\
\frac{dM}{dt} &= (1-mu(t))N_E\gamma_1(I_{1H} + \alpha I_{2H}) - (\mu_M + bdw(t))M \\
\frac{dS_S}{dt} &= \Lambda_S - \frac{\beta_2 MS_H}{M_0+\varepsilon M} - \mu_S S_S - d_1 S_S - bdw(t)S_S \\
\frac{dI_S}{dt} &= \frac{\beta_2 MS_H}{M_0+\varepsilon M} - (\mu_S + d_1 + bdw(t) + \delta_S)I_S \\
\frac{dP}{dt} &= (1-bw(t))\gamma_2 I_S - (\mu_P + bdw(t))P
\end{aligned}\right\} \quad (7)$$

and is subject to initial conditions of the autonomous system (1). Here, η_1 and η_2 are the rates of diagnosis and treatment of infected humans, I_{1H} and I_{2H} respectively, m is the compliance rate of public health education, b as the efficacy rate of the snail control and d the death rate due to snail control strategy.

Using Pontryagin's Maximum Principle [26] with time interval $[0, t_f]$, where t_f is the final time on the control functions, we determine the duration for disease elimination for equation (7) with initial conditions. The objective functional is given by

$$\begin{aligned}
\Gamma(u,v,w) = \int_0^{t_f} &(\rho_1 I_{1H} + \rho_2 I_{2H} + \rho_3 N_S + \rho_4 M + \rho_5 P \\
&+ \frac{1}{2}C_1 u^2(t) + \frac{1}{2}C_2 v^2(t) + \frac{1}{2}C_3 w^2(t))dt
\end{aligned} \quad (8)$$

subject to the system of differential equations (7). Here, C_1, C_2 and C_3 are the weights associated with the costs of control programs $(u(t), v(t), w(t))$, $\rho_1, \rho_2, \rho_3, \rho_4$ and ρ_5 are positive weights to balance the factors of the acute infected humans, chronic infected humans, total snail population, miracidia

and cercariae respectively while t_f is the final time for implementation of the control functions.

To minimise the number of infected humans, snail population, miracidia and cercariae populations while minimising the cost of implementing these controls $u(t), v(t)$ and $w(t)$, we seek an optimal control, $u^*(t), v^*(t)$ and $w^*(t)$, such that

$$\Gamma(u^*, v^*, w^*) = \min_{(u,v,w)\in\Psi} \Gamma(u, v, w)$$

where $\Psi = \{u(t), v(t), w(t) | 0 \leq u(t) \leq 1, 0 \leq v(t) \leq 1, 0 \leq w(t) \leq 1, 0 \leq t \leq t_f\}$ is a bounded Lebesgue measurable control set subject to equation (7) and initial conditions.

To determine the duration for disease elimination for equation (7) with initial conditions using the Pontryagin's maximum principle [26] with time interval $[0, t_f]$, the Hamiltonian, $\mathcal{H}$, is defined by

$$\left.\begin{aligned}
&\mathcal{H} = \rho_1 I_{1H} + \rho_2 I_{2H} + \rho_3 N_S + \rho_4 M + \rho_5 P \\
&+\tfrac{1}{2}C_1 u^2(t) + \tfrac{1}{2}C_2 v^2(t) + \tfrac{1}{2}C_3 w^2(t) \\
&+\Omega_1\Big(\Lambda_H + eT_H - \tfrac{(1-mu(t))\beta_1 P S_H}{P_0+\varepsilon P} - \mu_H S_H\Big) \\
&+\Omega_2\Big(\tfrac{(1-mu(t))\beta_1 P S_H}{P_0+\varepsilon P} \\
&-((1-v(t))(k+\delta_{1H}) + \sigma_1 + \mu_H + \eta_1 v(t))I_{1H}\Big) \\
&+\Omega_3((1-v(t))kI_{1H} - ((1-v(t))\delta_{2H} + \sigma_2 + \mu_H + \eta_2 v(t))I_{2H}) \\
&+\Omega_4((\sigma_1 + \eta_1 v(t))I_{1H} + (\sigma_2 + \eta_2 v(t))I_{2H} - (e+\mu_H)T_H) \\
&+\Omega_5((1-mu(t))N_E\gamma_1(I_{1H} + \alpha I_{2H}) - (\mu_M + bdw(t))M) \\
&+\Omega_6(\Lambda_S - \tfrac{\beta_2 M S_S}{M_0+\varepsilon M} - (\mu_S + d_1 + bdw(t))S_S) \\
&+\Omega_7\Big(\tfrac{\beta_2 M S_S}{M_0+\varepsilon M} - (\mu_S + d_1 + bdw(t) + \delta_S)I_S\Big) \\
&+\Omega_8((1-bw(t))\gamma_2 I_S - (\mu_P + bdw(t))P)
\end{aligned}\right\} \quad (9)$$

where $\Omega_1, \Omega_2, \Omega_3, \Omega_4, \Omega_5, \Omega_6, \Omega_7, \Omega_8$ are the adjoint variables of the state variables, $S_H, I_{1H}, I_{2H}, T_H, M, S_S, I_S, P$ respectively. The control set Ψ is closed and convex following the approach in [27, 13].

Theorem 5. *Given the optimal control* $(u^*(t), v^*(t), w^*(t))$ *and solutions* $S^*_H(t), I^*_{1H}\ (t), I^*_{2H}(t), T^*_H(t), M^*(t), S^*_S(t), I^*_S(t), P^*(t)$ *of the corresponding state system (7) that minimises* $\Gamma(u(t), v(t), w(t))$ *over* Ψ*, there exist adjoint variables* $\Omega_1, \Omega_2, \Omega_3, \Omega_4, \Omega_5, \Omega_6, \Omega_7, \Omega_8$ *that satisfy the following systems of equations,*

$$\left.\begin{aligned}
\frac{d\Omega_1}{dt} &= \frac{(1-mu(t)^*)\beta_1 P^*}{P_0+\varepsilon P^*}(\Omega_1-\Omega_2)+\Omega_1\mu_H \\
\frac{d\Omega_2}{dt} &= -\rho_1+(\Omega_2-\Omega_3)k(1-v^*(t))+(\Omega_2-\Omega_4)(\sigma_1+\eta_1 v^*(t)) \\
&\quad +\Omega_2((1-v^*(t))\delta_{1H}+\mu_H)-\Omega_5(1-mu^*(t))N_E\gamma_1 \\
\frac{d\Omega_3}{dt} &= -\rho_2+\Omega_3((1-v^*(t))\delta_{2H}+\mu_H) \\
&\quad +(\Omega_3-\Omega_4)(\sigma_2+\eta_2 v^*(t))-\Omega_5(1-mu^*(t))N_E\gamma_1\alpha \\
\frac{d\Omega_4}{dt} &= (\Omega_4-\Omega_1)e+\Omega_4\mu_H \\
\frac{d\Omega_5}{dt} &= -\rho_4+\Omega_5(\mu_M+bdw^*(t))+(\Omega_6-\Omega_7)\frac{\beta_2 S_S^* M_0}{(M_0+\varepsilon M^*)^2} \\
\frac{d\Omega_6}{dt} &= -\rho_3+\Omega_6(\mu_S+d_1+bdw^*(t))+(\Omega_6-\Omega_7)\frac{\beta_2 M^*}{M_0+\varepsilon M^*} \\
\frac{d\Omega_7}{dt} &= -\rho_3+\Omega_7(\mu_S+\delta_S+d_1+bdw^*(t))-\Omega_8(1-bw^*(t))\gamma_2 \\
\frac{d\Omega_8}{dt} &= -\rho_5+\Omega_8(\mu_P+bdw^*(t))+(\Omega_1-\Omega_2)\frac{(1-mu^*(t))\beta_1 S_H^* P_0}{(P_0+\varepsilon P^*)^2}
\end{aligned}\right\} \quad (10)$$

with transversality conditions

$$\begin{aligned}
\Omega_1(t_f)=\Omega_2(t_f)=\Omega_3(t_f)=\Omega_4(t_f)=\Omega_5(t_f) \\
=\Omega_6(t_f)=\Omega_7(t_f)=\Omega_8(t_f)=0.
\end{aligned} \quad (11)$$

Also, the optimality conditions $u^*(t)$, $v^*(t)$ and $w^*(t)$ are given by

$$\left.\begin{aligned}
u^*(t) &= \max\{0, min(1, u_*^*(t))\} \\
v^*(t) &= \max\{0, min(1, v_*^*(t))\} \\
w^*(t) &= \max\{0, min(1, w_*^*(t))\}
\end{aligned}\right\}, \quad (12)$$

where

$$\begin{aligned}
u_*^*(t) &= \frac{1}{C_1}\left[\frac{(\Omega_2-\Omega_1)S_H^* m\beta_1 P^*}{P_0+\varepsilon P^*}+m\gamma_1 N_E\Omega_5(I_{1H}^*+\alpha I_{2H}^*)\right], \\
v_*^*(t) &= \frac{1}{C_2}\left[(\Omega_3-\Omega_2)kI_{1H}^*+(\Omega_2-\Omega_4)\eta_1 I_{1H}^*+(\Omega_3-\Omega_4)\eta_2 I_{2H}^*\right. \\
&\quad \left.-\Omega_2\delta_{1H}I_{1H}^*-\Omega_3\delta_{2H}I_{2H}^*\right], \\
w_*^*(t) &= \frac{1}{C_3}\left[(\Omega_5 M^*+\Omega_6 S_S^*+\Omega_7 I_S^*+\Omega_8 P^*)bd+b\Omega_8 I_S^*\gamma_2\right].
\end{aligned} \quad (13)$$

Proof. The adjoint equation (10) is determined by differentiating Hamiltonian function, $\mathcal{H}$, with respect to $S_H, I_{1H}, I_{2H}, T_H, M, S_S, I_S$, and P and multiply by minus, that is

$$\frac{d\Omega_1}{dt} = -\frac{\partial \mathcal{H}}{\partial S_H}, \qquad \frac{d\Omega_2}{dt} = -\frac{\partial \mathcal{H}}{\partial I_{1H}}, \qquad \frac{d\Omega_3}{dt} = -\frac{\partial \mathcal{H}}{\partial I_{2H}},$$
$$\frac{d\Omega_4}{dt} = -\frac{\partial \mathcal{H}}{\partial T_H}, \qquad \frac{d\Omega_5}{dt} = -\frac{\partial \mathcal{H}}{\partial M}, \qquad \frac{d\Omega_6}{dt} = -\frac{\partial \mathcal{H}}{\partial S_S},$$
$$\frac{d\Omega_7}{dt} = -\frac{\partial \mathcal{H}}{\partial I_S}, \qquad \frac{d\Omega_8}{dt} = -\frac{\partial \mathcal{H}}{\partial P}$$

with respective transversality conditions of equation (8).

For the optimality conditions, $\frac{\partial \mathcal{H}}{\partial u} = 0$, $\frac{\partial \mathcal{H}}{\partial v} = 0$ and $\frac{\partial \mathcal{H}}{\partial w} = 0$ is used to solve for $u^*(t), v^*(t), w^*(t)$ respectively on the interior of the control set, $\Psi = \{u(t), v(t), w(t) | 0 \leq u(t) \leq 1, 0 \leq v(t) \leq 1, 0 \leq w(t) \leq 1, 0 \leq t \leq t_f\}$ and this yield the optimal control characterization (12).

The optimality system comprises of equations (7), the adjoint system (10), initial conditions at $t = 0$, boundary conditions (11) and the characterisation of the optimal control (12) with (13). Thus, the optimal control can be computed using the optimality system. Hence, using the fact that the second derivatives of the Hamiltonian with respect to u, v and w respectively are positive indicates that the optimal problem is minimum at control u^*, v^* and w^*. □

5 Numerical simulations and discussion

Numerical simulations are carried out using parameter values in Table 1 to illustrate the behaviour of the schistosomiasis transmission dynamics with or without controls. The values for weight constants and cost constants used in the simulations are $\rho_1 = 0.6$, $\rho_2 = 0.4$, $\rho_3 = \rho_4 = \rho_5 = 0.2$, $C_1 = 2 \times 10^6$, $C_2 = C_3 = 2 \times 10^4$ with $d = 0.0000369$, $d = m = \eta_1 = \eta_2 = 0.5$. The simulations are implemented for the first 1000 days.

5.1 Discussion

The impact of implementing one control, two controls and all the three controls on infected classes of model (1) are depicted in Figs. 5, 6 and 7 respectively. The controls are public health education ($u(t)$), diagnosis and

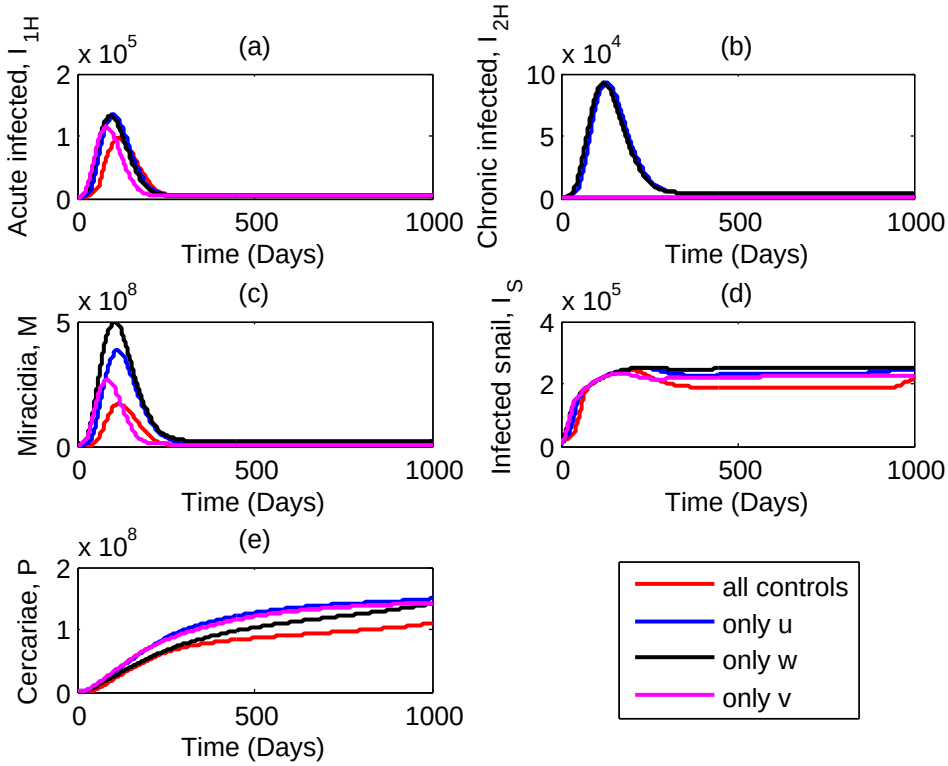

Figure 5: Simulation results of the infected classes, $I_{1H}(t)$, $I_{2H}(t)$, $M(t)$, $I_S(t)$, $P(t)$ with only one control. All parameter values of Table 1 are used.

treatment ($v(t)$) and snail control ($w(t)$). In Fig. 5(a)-5(e), there is a significant reduction in the number of infected human, miracidia and infected snail when only $v(t)$ is carried out when compared with only $u(t)$ and only $w(t)$ (see Fig. 5(a)-5(d)). This effect is more on the chronic infected population as it reduces them drastically to a minimum (Fig. 5b). Meanwhile, the implementation of only $w(t)$ and only $u(t)$ behave the same for infected human population (see Fig. 5(a)-5(b)) while for the miracidia and infected snail, implementing only $u(t)$ is preferable to only $w(t)$. Applying only $w(t)$ in the population reduces cercariae population more than as seen in other infected compartments. This reduction does not occur in infected snails population because the number of infected snail depends on the amount of miracidia in the population which also depends on the number of infected

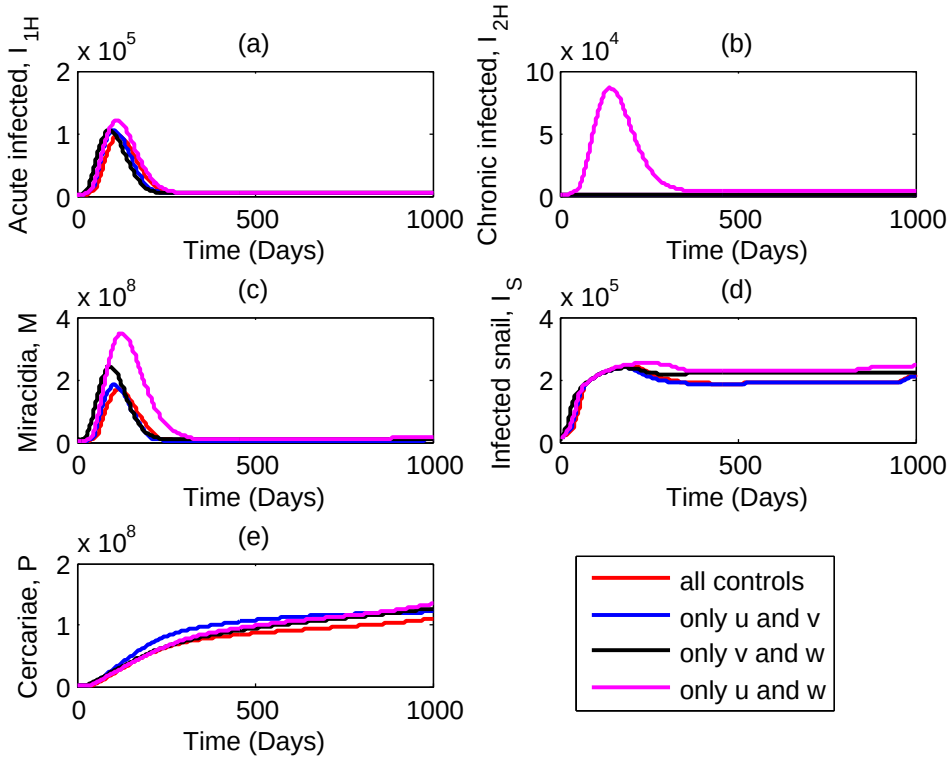

Figure 6: Simulation results of the infected classes, $I_{1H}(t)$, $I_{2H}(t)$, $M(t)$, $I_S(t)$, $P(t)$ with two controls. All parameter values of Table 1 are used.

humans.

The application of two combined control measures on infected compartments are displayed in Fig. 6(a)-6(e). It is observed that implementation of $(u(t), (v(t))$ reduces the number of infected classes $(I_{1H}(t), I_{2H}(t), M(t), I_S(t), P(t))$ in the population when compared with other two combined controls, $(u(t), w(t))$ and $(v(t), w(t))$ while the implementation of $v(t), w(t))$ lowers the infected classes more than $(u(t), w(t))$.

Furthermore, Fig. 7(a)-7(e) show a clear difference between the implementation of all three controls and without control. There is a reduction in the population of infected compartments when the three controls are implemented. Fig. 7(f) shows the control profile for the three controls and their influence in reducing the disease prevalence. The control, u is kept at

maximum, 100%, for the first 50 days and declines gradually to 50% where it remains for the next 150 days before increasing to the upper bound which is maintained for 750 days and then gradually reduces to the lower bound at the final time of implementation. For the control, v, it declines from the 100% to 45% for the first 50 days before gradually increases and peaks at 50% where it gradually reduces to the lower bound at the final time of implementation. The control, w is maintained at 100% for the duration of implementation.

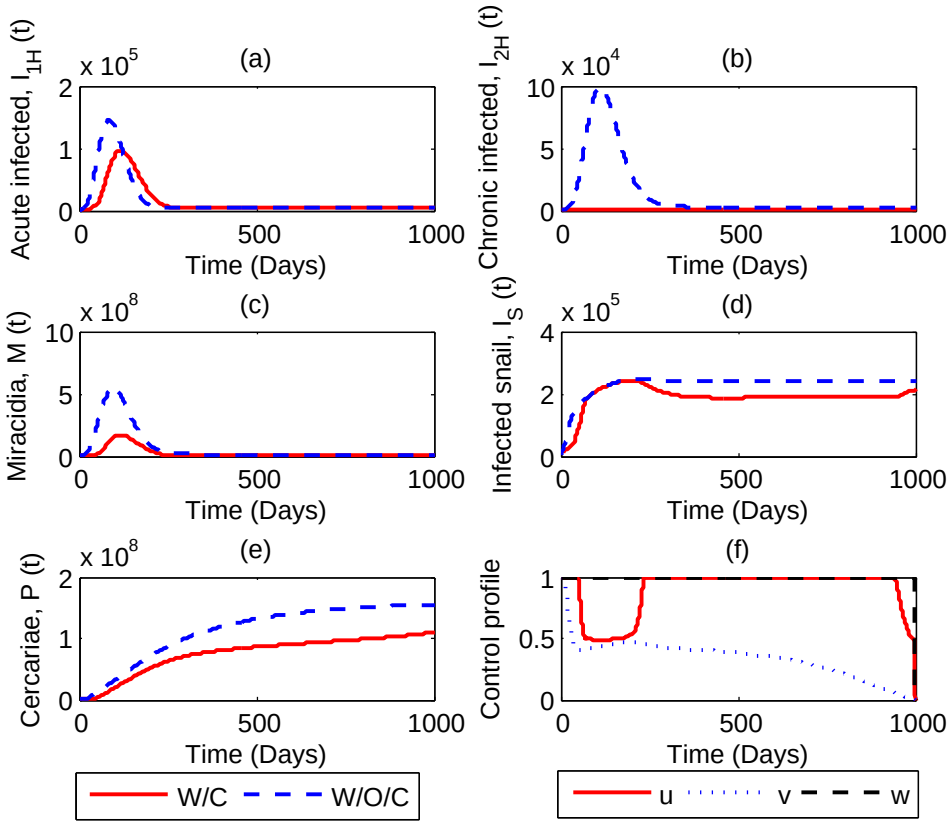

Figure 7: Simulation results of the infected classes, $I_{1H}(t)$, $I_{2H}(t)$, $M(t)$, $I_S(t)$, $P(t)$ with or without control measures and the control profiles. W/C means with control measures while W/O/C means without control measures. All parameter values of Table 1 are used.

It could be observed from Figs. 5 and 6 that combined controls with inclusion of diagnosis and treatment, $v(t)$, is important in the control of schistosomiasis transmission which is supported by Kanyi *et al.* [8]. Diagnosis and treatment for the infected human will prevent the mortality rate of this disease but this have to be complemented with other controls. According to the WHO [15], complementing control strategies is important in eradicating schistosomiasis disease in the population especially where is endemic. So, implementing public health education ($u(t)$), diagnosis and treatment ($v(t)$) and snail control ($w(t)$) simultaneously will reduce the burden of the disease. However, some snail controls such as chemical control are toxic to other aquatic bodies and it may be difficult to single out the chemical control that will focus only on the snail population even though snails are special food in Africa. Hence, it is preferable to implement public health education and diagnosis and treatment ($u(t), v(t)$) simultaneously in order to eradicate schistosomiasis transmission in the affected area. This is support by work of Chiyaka and Garira [12] that advised that intervention strategies should target snail to human transmission.

6 Conclusion

The dynamics of schistosomiasis disease transmission is analysed in this research. The model has eight compartments divided into susceptible human, acute infected human, chronic infected human, treatment compartment, miracidia, susceptible snail, infected snail and cercariae populations respectively. The stability analysis for the disease-free and endemic equilibria are investigated in terms of basic reproduction number, R_0 and the model exhibits a forward bifurcation. The sensitivity analysis for influence of the parameters on schistosomiasis is examined using the PRCC. With the result of sensitivity analysis, the optimal control model with public health education, diagnosis and treatment and snail control as controls is developed and analysed. Through the numerical simulations, the implementation of public health education, diagnosis and treatment and snail control simultaneously reduce the transmission of the schistosomiasis in the population. However due to the toxicity of some snail controls to water bodies, it will be advisable to implement public health education and diagnosis and treatment of the infected humans together.

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