

# Mathematical Model for Schistosomiasis Transmission Dynamics and Optimal Control in Uganda

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## ABSTRACT

Schistosomiasis, a parasitic disease spread by snails, is a major health and economic issue in tropical and sub-tropical areas, particularly in Pakwach District, Northern Uganda, where it significantly affects both humans and cattle. This study aims to develop a mathematical model to understand the transmission dynamics of schistosomiasis, with a focus on the role of varied intermediate hosts in the region. The model uses nonlinear differential equations and includes human, animal, and snail populations to explore the effectiveness of control measures like mass drug administration, snail control, and public health education. Key findings highlight that controlling the environment where infected snails thrive is crucial, and public health education is especially effective. Limitations include reliance on existing data, and future research should focus on refining the model, vaccine development, and evaluating the cost-effectiveness of control strategies. The study provides valuable insights for schistosomiasis control in Pakwach and similar regions globally.

**Keywords:** schistosomiasis, transmission, dynamics, Pakwach district and optimal control.

## 1.0 INTRODUCTION

Schistosomiasis, a disease caused by parasitic worms from the *Schistosoma* genus, remains a major public health concern worldwide, especially in tropical and subtropical areas. Sub-Saharan Africa is hit hardest, with the disease having a profound effect on both health and economic development (World Health Organization [WHO], 2023). Despite efforts to control it over the years, transmission continues due to the intricate interactions between the parasite, freshwater snails that serve as intermediate hosts, and humans (Vaughan et al., 2022). Snails from the *Biomphalaria* and *Bulinus* genera are essential to the spread of the disease (Ayoubi et al., 2022). Factors like snail population density, age, and how easily they get infected complicate efforts to model and control the transmission (Gauthier et al., 2023). Tackling these complexities is crucial for improving our understanding of how the disease spreads and for refining control measures (Sousa et al., 2021).

The transmission cycle of schistosomiasis has several stages. It begins when eggs from adult worms in human hosts hatch in water, releasing miracidia that infect snails. Inside the snails, the parasites develop and are later released as cercariae, which can penetrate human skin when people come into contact with contaminated water (Vaughan et al., 2022). Schistosomiasis affects more than 240 million people across 78 countries, causing serious health issues such as anemia, malnutrition, and stunted growth and cognitive development in children. The disease also results in substantial economic losses due to decreased productivity and higher healthcare costs (WHO, 2023).

Several studies have used mathematical models to explore different aspects of schistosomiasis transmission and control. Zhang and Zhao (2020) investigated how seasonal changes and water temperature impact parasite development, suggesting eradication is possible if the basic reproduction number ( $R_0$ ) is below 1, though they didn't include animal populations or environmental factors. Anderson et al. (2021) highlighted the importance of snail age structure in influencing human infection

rates, but their model didn't consider environmental or control factors. Madubueze et al. (2022) focused on acute and chronic human infections, emphasizing the role of snail death rates and parasite decay, without exploring specific control strategies. Kadaleka et al. (2022) incorporated both human and animal hosts and found molluscicides to be highly effective, though they did not examine detailed intervention strategies. Kalinda et al. (2019) applied optimal control theory and showed that low-cost strategies could significantly reduce human infections, but did not consider vector control or migration. Finally, Ding et al. (2017) included an exposed compartment for the disease's incubation period, but did not account for animal populations or a contaminated environment. These studies highlight the complexity of modeling schistosomiasis and underscore the need for more comprehensive models that integrate ecological, environmental, and control factors for better disease management (2001).

## The Mathematical Model

A model of schistosomiasis is developed using a deterministic compartmental modeling approach. At any given time  $t$ , the total human population denoted by  $N_h(t)$  is divided into three subgroups consist of the following classes: susceptible human

$S_h$ , exposed human  $E_h$  and infected human  $I_h$ . Hence,

$$N_h = S_h + E_h + I_h \quad (1)$$

Similarly, the total snail population denoted by  $N_v(t)$  is divided into three subgroups consist of the following classes: susceptible snail  $S_v$ , exposed snail  $E_v$  and infected snail  $I_v$ . Hence,

$$N_v = S_v + E_v + I_v \quad (2)$$

Also, the total Animals population denoted by  $N_a(t)$  is divided into three subgroups consist of the following classes: susceptible Animals  $S_a$ , exposed snail  $E_a$  and infected

Animals snail  $I_m$ . Hence,

$$N_a = S_a + E_a + I_a \quad (3)$$

Also, the schistosome population denoted by  $N_c$  is the concentration of Miracidia and Cercariae in the contaminated water.

$$N_c = C_e \quad (4)$$

The schistosomiasis model uses a deterministic compartmental approach to describe disease transmission among humans, snails, animals, and the contaminated environment. Each population—humans, snails, and animals—is divided into susceptible, exposed, and infected classes. Individuals move between these classes based on recruitment, infection through contact with contaminated water, progression of the disease, and natural or disease-related death. The aquatic environment becomes contaminated with schistosome parasites (miracidia and cercariae) through contributions from infected humans, snails, and animals. The parasite concentration in water drives new infections across all populations. This model captures the dynamics of transmission and highlights key points for intervention and disease control.

The above description culminates into the following system of differential equations

**Susceptible human:**

$$\frac{dS_h}{dt} = \Lambda_h - (\beta_1 C_e + \mu) S_h \quad (5)$$

**The exposed human:**

$$\frac{dE_h}{dt} = \beta_1 S_h C_e - (\beta_2 + \mu) E_h \quad (6)$$

**Infected human:**

$$\frac{dI_h}{dt} = \beta_2 E_h - (\mu + \mu_0 + \beta_3) I_h \quad (7)$$

**Susceptible Snail:**

$$\frac{dS_v}{dt} = \Lambda_v - \beta_8 S_v C_e - \mu_3 S_v \quad (8)$$

**The exposed snail:**

$$\frac{dE_v}{dt} = \beta_8 S_v C_e - (\mu_3 + \beta_9) E_v \quad (9)$$

**Infected snail:**

$$\frac{dI_v}{dt} = \beta_9 E_v - (\mu_3 + \mu_4 + \beta_9) I_v \quad (10)$$

**Susceptible Animals:**

$$\frac{dS_a}{dt} = \Lambda_a - (\beta_4 C_e + \mu_1) S_a \quad (11)$$

**The exposed Animals:**

$$\frac{dE_a}{dt} = \beta_4 S_a C_e - (\mu_1 + \beta_6) E_a \quad (12)$$

**Infected Animals:**

$$\frac{dI_a}{dt} = \beta_6 E_a - (\mu_1 + \mu_2 + \beta_7) I_a \quad (13)$$

**The Contaminated Environment:**

$$\frac{dC_e}{dt} = \beta_3 I_h + \beta_7 I_a + \beta_{11} I_v - \mu_5 C_e \quad (14)$$

The table 1 and 2 below describe both the state variables and parameter of the model

**Table 1: Description of the state variables .**

Parameter	Description
$S_h$	Susceptible humans
$E_h$	Exposed humans
$I_h$	Infected humans
$S_a$	Susceptible animals
$E_a$	Exposed animals
$I_a$	Infected animals
$S_v$	Susceptible Snail vector
$E_v$	Exposed snail vector
$I_v$	Infected snail vector
$C_e$	Contaminated environment

**Table 2: Description of the model parameters and symbols.**

Parameter	Description	Value	References
$\Lambda_h$	Recruitment rates of humans	4,157	Tabo et al (2023)
$\mu$	Natural death of humans	0.00004025	Tabo et al (2023)
$\beta_1$	Rate of infection of exposed to susceptible humans	0.09753	Kalida et al (29)

$\beta_2$	Rate of infection of humans in contact with Cercariae	0.133	Feng et al (2020)
$\beta_3$	Rate at which infected humans produce Miracidia in the water.	6.96	Madubueze et al (2022)
$\mu_0$	Death of humans due to infection	0.0039	Gao et al (2021)
C	Cercariae parasite – lava stage	$1 \times 10^6$	Assumed
M	Miracidia – parasite egg stage	$1 \times 10^8$	Kanyi et al (2023)
$\Lambda_m$	Recruitment rate of Animals	200	Dabo et al (2021)
$\beta_4$	Rate of infection of susceptible Animals with water	0.133	Feng et al (2022)
$\beta_5$	Rate of infection of exposed susceptible Animals	0.0406	Assumed
$\beta_6$	Rate of infection of Animals in contact with Cercariae	0.036	Assumed
$\beta_7$	Rate at which infected animals produce Miracidia in the water.	6.96	Assumed
$\mu_1$	Natural death of animals	0.000392	Xiang et al(2022)
$\mu_2$	Death of animals due to infection	0.0004012	Feng et al (2022)
$\Lambda_v$	Recruitment rate of snail vector	200	Dabo et al (18)
$\mu_3$	Natural death of snail	0.0286	Kanyi et al (2021)
$\beta_8$	Rate of infection of exposed susceptible snail	0.0286	Kanyi et al (2021)
$\beta_9$	Rate of interaction of snail vector in contact with Miracidia	7.618	Assumed
$\beta_{10}$	Rate of infection of infected snail vector with Water	2.6	Zhang et al(2022)
$\mu_4$	Death of Snails due to infection	0.026	Dabo (2023)
$\mu_5$	Rate of deterioration of water	0.007	Assumed

**Invariant Region**

The total population sizes  $N_h, N_v, N_a$  and  $N_w$  can be determined by

$N_h = S_h + E_h + I_h$ ,  $N_v = S_v + E_v + I_v$ ,  $N_a = S_a + E_a + I_a$  and  $N_c = C_e$  or from the differential equations

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dI_h}{dt}, \quad (15)$$

$$= \Lambda_h - \mu_h N_h - (\mu_0 + \beta_3) I_h$$

$$\frac{dN_v}{dt} = \frac{dS_v}{dt} + \frac{dE_v}{dt} + \frac{dI_v}{dt} \quad (16)$$

$$= \Lambda_v - \mu_v N_v - (\mu_4 + \beta_9) I_v$$

$$\frac{dN_a}{dt} = \frac{dS_a}{dt} + \frac{dE_a}{dt} + \frac{dI_a}{dt} \quad (17)$$

$$= \Lambda_a - \mu_a N_a - (\mu_2 + \beta_7) I_a$$

and

$$\frac{dN_c}{dt} = \beta_3 I_h + \beta_7 I_a + \beta_{11} I_v - \mu_5 C_e \quad (18)$$

Assuming the disease does not kill and the populations to do not generate the disease into the environment yields:

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - (\mu_0 + \beta_3) I_h \leq \Lambda_h - \mu_h N_h \quad (19)$$

Similarly

$$\frac{dN_v}{dt} = \Lambda_v - \mu_v N_v - (\mu_4 + \beta_9) I_v \leq \Lambda_v - \mu_v N_v \quad (20)$$

$$\frac{dN_a}{dt} = \Lambda_a - \mu_a N_a - (\mu_2 + \beta_7) I_a \leq \Lambda_a - \mu_a N_a \quad (21)$$

and

$$\frac{dN_c}{dt} = \beta_3 I_h + \beta_7 I_a + \beta_{11} I_v \leq \mu_5 C_e \quad (22)$$

**Lemma 1:** The model system (15) has solutions which are contained in the feasible  $\Omega = \Omega_h \times \Omega_v \times \Omega_a \times \Omega_c$ .

**Proof:** Let  $(S_h, E_h, I_h, S_v, E_v, I_v, S_a, E_a, I_a, C_e) \in \mathfrak{R}^{10}$  be any solution of the system with non-negative initial conditions. Since

$$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h \quad (23)$$

And using Birkhoff and Rota (1989) Theorem on differential inequality, we have

$$0 \leq N_h \leq \frac{\Lambda_h}{\mu_h}, \text{ hence} \\ \Lambda_h - \mu_h N_h \geq k e^{-\mu_h t} \quad \text{Where } k \text{ is constant} \quad (24)$$

Therefore, all feasible solutions of the human population only of the model system (15) are in the region

$$\Omega_h = \left\{ (S_h, E_h, I_h) \in \mathfrak{R}^3 : N_h \leq \frac{\Lambda_h}{\mu_h} \right\} \quad (25)$$

Similarly, the feasible solutions of the snail population only of the model system (15) are in the region

$$\Omega_v = \left\{ (S_v, E_v, I_v) \in \mathfrak{R}^3 : N_v \leq \frac{\Lambda_v}{\mu_v} \right\} \quad (26)$$

and

Similarly, the feasible solutions of the animal's population only of the model system (15) are in the region

$$\Omega_a = \left\{ (S_a, E_a, I_a) \in \mathfrak{R}^3 : N_a \leq \frac{\Lambda_a}{\mu_a} \right\} \quad (27)$$

The feasible solution set of the schistosome population of the model system (15) enters the region

$$\Omega_c = \left\{ C_e \in \mathfrak{R}_+, N_c \leq \frac{\beta_3 I_h + \beta_7 I_a + \beta_{11} I_v}{\mu_5 C_e} \right\}. \quad (28)$$

Thus, the feasible set for model system (15) is given by

$$\Omega = \left\{ (S_h, E_h, I_h, S_v, E_v, I_v, S_a, E_a, I_a, C_e) \in \mathfrak{R}_+^{11} : S_h, E_h, I_h, S_v, E_v, I_v, S_a, E_a, I_a, C_e \geq 0; N_h \leq \frac{\Lambda_h}{\mu_h}; N_v \leq \frac{\Lambda_v}{\mu_v}; N_a \leq \frac{\Lambda_a}{\mu_a}; N_w \leq \frac{\Lambda^*}{\mu_5} \right\} \quad (29)$$

$$\text{Where } \Lambda^* = \beta_3 I_h + \beta_7 I_a + \beta_{11} I_v \quad (30)$$

Which is a positively invariant set under the flow induced by the model (15). Hence the system (15) is epidemiologically meaningful and mathematically well-posed in the domain  $\Omega$ . Therefore, in this domain it is sufficient to consider the dynamics of the flow generated by the model (15). In addition, the usual existence, uniqueness and continuation of results hold for the system.

### Positivity of Solutions

**Lemma 2:** Let the initial data be

$$\{S_h(0), S_v(0), S_a(0), C_e(0) > 0, (E_h(0), I_h(0), E_h(0), I_h(0), E_v(0), I_v(0), E_a(0), I_a(0)) \geq 0\} \in \Omega \text{ Then}$$

the solution set  $(S_h, E_h, I_h, S_v, E_v, I_v, S_a, E_a, I_a, C_e)(t)$  of the model system (3.15) is positive for all  $t > 0$ .

**Proof:** The first equation of the model (15) gives

$$\begin{aligned}
 \frac{dS_h}{dt} &= \Lambda_h - (\beta_1 C_e + \mu) S_h \\
 &\geq -(\beta_1 C_e + \mu) S_h \\
 \int \frac{1}{S_h} dS_h &\geq -\int (\beta_1 C_e + \mu) \\
 S_h(t) &\geq S(0) e^{-(\mu t)} \geq 0.
 \end{aligned} \tag{31}$$

Similarly, the remaining equations are solved in the same manner.

Furthermore, we need to show that the region  $\Omega$  is positively invariant so that it suffices the dynamics of the above system. The right hand sides of equations (21)-(23) are both bounded by  $\Lambda_h - \mu_h N_h$ ,  $\Lambda_v - \mu_v N_v$ ,  $\Lambda_a - \mu_a N_a$  and  $\Lambda^* - \mu_5 C_e$  respectively, it follows that

$$\frac{dN_h}{dt} < 0 \text{ if } N_h(t) > \frac{\Lambda_h}{\mu_h}, \quad \frac{dN_v}{dt} < 0 \text{ if } N_v(t) > \frac{\Lambda_v}{\mu_v}, \quad \frac{dN_a}{dt} < 0 \text{ if } N_a(t) > \frac{\Lambda_a}{\mu_a} \text{ and } \frac{dN_c}{dt} < 0 \text{ if } N_c(t) > \frac{\Lambda_w}{\mu_w}. \tag{32}$$

Using a standard comparison theorem (see Amiru & Abdullahi 2022), it has been shown above that

$$\begin{aligned}
 N_h(t) &\leq \frac{\Lambda_h}{\mu_h} (1 - e^{(-\mu_h)t}) + N_h(0) e^{(-\mu_h)t} \\
 N_v(t) &\leq \frac{\Lambda_v}{\mu_v} (1 - e^{(-\mu_v)t}) + N_v(0) e^{(-\mu_v)t} \\
 N_a(t) &\leq \frac{\Lambda_a}{\mu_a} (1 - e^{(-\mu_a)t}) + N_a(0) e^{(-\mu_a)t}
 \end{aligned} \tag{33}$$

and

$$N_c(t) \leq \frac{\Lambda^*}{\mu_w} (1 - e^{(-\mu_5)t}) + N_c(0) e^{(-\mu_5)t}$$

Especially if,

$$\begin{aligned}
 N_h(0) < \frac{\Lambda_h}{\mu_h} \text{ then } N_h(0) &\leq \frac{\Lambda_h}{\mu_h}, \quad N_v(0) < \frac{\Lambda_v}{\mu_v} \text{ then } N_v(0) &\leq \frac{\Lambda_v}{\mu_v}, \quad N_a(0) < \frac{\Lambda_a}{\mu_a} \text{ then } N_a(0) &\leq \frac{\Lambda_a}{\mu_a}, \\
 N_c(0) < \frac{\Lambda^*}{\mu_5} \text{ then } N_c(0) &\leq \frac{\Lambda^*}{\mu_5}.
 \end{aligned} \tag{34}$$

Therefore,  $\Omega$  is positively invariant.  $N_h(0) > \frac{\Lambda_h}{\mu_h}, N_v(0) > \frac{\Lambda_v}{\mu_v}, N_a(0) > \frac{\Lambda_a}{\mu_a}$ , and  $N_c(0) > \frac{\Lambda^*}{\mu_5}$ ,

then either the solution enter  $\Omega$  in finite time, or

$$N_h(t) \text{ approaches } \frac{\Lambda_h}{\mu_h}, \quad N_v(t) \text{ approaches } \frac{\Lambda_v}{\mu_v}, \quad N_a(t) \text{ approaches } \frac{\Lambda_a}{\mu_a}, \quad N_c(t) \text{ approaches } \frac{\Lambda^*}{\mu_5},$$

asymptotically, and the infected state variables approaches zero.

#### Existence and Stability of Steady-State Solutions

The  $E = (S_m^*, E_h^*, I_h^*, S_v^*, E_v^*, I_v^*, S_a^*, E_a^*, I_a^*, C_e^*)$  is the steady-state of the system (15) which can be calculated by setting the right hand side of the model (15) to zero, giving us the following

#### The Existence of the Trivial Equilibrium Point

For as long as the human recruitment term  $\Lambda$  and the mosquito recruitment term  $\pi$  are not zero, the population will not be extinct. This implies that there is no trivial equilibrium point, thus

$$(S_h^*, E_h^*, I_h^*, S_v^*, E_v^*, I_v^*, S_a^*, E_a^*, I_a^*, C_e^*) \neq (0, 0, 0, 0, 0, 0, 0, 0, 0, 0) \tag{35}$$

#### 3.4.2 Disease-free Equilibrium Point $E_0$

Disease-free equilibrium points (DFE) are steady-state solutions where there is no disease (schistosomiasis). We define the "diseased" classes as the human, Snail or Animal populations and the Water body that are either exposed, or infected, that is;

$E_h, I_h, E_v, I_v, E_m, I_m, C_e$  in the system (3.15). Hence, the DFE of the basic schistosomiasis model (15) is given by

$$(S_h^*, E_h^*, I_h^*, S_v^*, E_v^*, I_v^*, S_a^*, E_a^*, I_a^*, C_e^*) = \left( \frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0, \frac{\Lambda_a}{\mu_a}, 0, 0, 0 \right) \quad (36)$$

That represents the state in which there is no infection in the society and is known as the disease-free equilibrium point (DFE)

The next-generation matrix method has been used to demonstrate the rate of appearance of new infections in the compartments,

#### The basic reproduction number

$$\mathcal{R}_0 = \frac{\mu_1 \mu_3 J_{10} \Lambda_h \beta_1 + \mu J_{12} \Lambda_a \beta_4 \mu_3 + \mu J_{14} \Lambda_v \beta_8 \mu_1}{\mu \mu_1 \mu_3} \quad (37)$$

The basic reproduction number can be utilized to assess the local stability of the disease-free equilibrium point.

#### Sensitivity Analysis of Model Parameters

Understanding the key factors driving disease transmission is essential for reducing human and animal fatalities and alleviating suffering. The transmission dynamics of the disease are primarily governed by the basic reproduction number ( $\mathcal{R}_0$ ) (Amiru &

Abdullahi, 2022). By analyzing the sensitivity of model parameters in relation  $\mathcal{R}_0$ , the most influential factors in disease spread can be identified. Modifying these parameters can help control transmission, either by mitigating or amplifying disease spread. This sensitivity analysis is crucial for experimental design, data assimilation, and simplifying complex nonlinear models (Abdullahi & Amiru, 2023a). Table 2 provides a detailed description of the parameters used in the schistosomiasis model.

**Table 3. Sensitivity Analysis of Parameters.**

S/NO	Parameter	Sensitivity Index
1	$\mu_5$	-1.00
2	$C$	0.54
3	$\Lambda_h$	0.51
4	$\beta_1$	0.51
5	$M$	0.46
6	$\Lambda_v$	0.46
7	$\beta_8$	0.46
8	$\beta_{10}$	0.17
9	$\mu_3$	-0.09
10	$\mu_4$	-0.08
11	$\Lambda_a$	0.03
12	$\beta_4$	0.03
13	$\mu_0$	-0.0003
14	$\beta_3$	-0.0003
15	$\beta_7$	0.000002

The sensitivity analysis identifies key factors influencing the spread of schistosomiasis. The most critical factor is the rate of water deterioration, with a strong negative sensitivity index (-1.00), indicating that poor water quality disrupts the parasite's life cycle by affecting infected snails. Highly sensitive factors (sensitivity > 0.5) include human exposure to contaminated water (0.54) and snail population dynamics (0.51), suggesting that improving sanitation, reducing water contamination, and controlling snails can significantly curb transmission. Moderately sensitive factors (sensitivity between 0.17 and 0.46) such as snail reproduction rates and secondary host interactions also influence transmission but are less critical. Low sensitivity parameters (sensitivity < 0.1), including the natural mortality rates of humans and snails, have minimal effect on disease spread. One parameter shows an almost negligible impact (0.000002), indicating it is irrelevant for intervention efforts.

#### Implications for Schistosomiasis Control

To effectively reduce schistosomiasis cases, control efforts should focus on the most highly sensitive parameters. This includes increasing Rate of deterioration of water (-1.00), reducing human exposure to contaminated water (0.54), and managing snail populations (0.51). Environmental factors (0.46 - 0.17) should be addressed as supportive measures, but low-sensitivity parameters should not be a priority, as they have minimal impact.

### Optimal Model Formulation

To effectively control schistosomiasis, time-dependent strategies are considered based on sensitivity analysis. These include mass drug administration to reduce human infections, snail control to target the parasite's intermediate host, and public health education with improved sanitation to prevent transmission and promote healthy behaviors. Susceptible humans and animals get infected following their contact with Cercariae parasite in the water at a rate  $(1-u_3)\beta_1 C_e S_h$  and  $(1-u_3)\beta_1 C_e S_a$ ,  $u_3$  ( $0 \leq u_3 \leq 1$ )

is the control on the use of Public Health Education. The treatment of both humans and animals  $u_1$  ( $0 \leq u_1 \leq 1$ ) is the use of the

Mass Drug Administration (MDA). Susceptible snails ( $S_v$ ) get infected following effective contacts Miracidia – parasite egg stage

at a rate  $(1-u_2)\beta_8 S_v C_e$ . Thus  $(1-u_2)$  is the use of the Snail control.

Therefore, putting the above formulations and assumptions together gives the following human-Snail – Animal model, represented by system of ordinary differential equations below,

$$\left. \begin{aligned} \frac{dS_h}{dt} &= \Lambda_h - (1-u_3)\beta_1 C_e S_h - \mu S_h \\ \vdots \\ \frac{dC_e}{dt} &= \beta_3 I_h + \beta_7 I_a + \beta_{10} I_v - (\mu_5 + \mu_2) C_e \end{aligned} \right\} \quad (38)$$

In investigating optimal control efforts to effectively manage the disease, it's crucial to frame the problem as an optimal control problem. The objective, or cost functional, typically aims to minimize certain measures related to the disease burden and control efforts. Here's a general formulation of the objective functional for the optimal control problem in the context of managing schistosomiasis:

$$J(u_1, u_2, u_3) = \int_0^T [A_1 I_h + A_2 I_a + A I_v + B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2] \quad (39)$$

Where  $T$  the final time and the coefficients is  $A_1, A_2, B_1, B_2, B_3$  are positive weights to balance the factors. With the given

objective functional  $J(\cup)$ , it is aimed at minimizing the number of infected human  $I_h(t)$ , infected animals  $I_a$  and the infected

snails  $I_v$  while minimizing the cost of control  $u_1(t), u_2(t), u_3(t)$ . Therefore, an optimal control  $u_1^*, u_2^*, u_3^*$  is obtained such that

$$J(u_1^*, u_2^*, u_3^*) = \min_{u_1, u_2, u_3} \{J(u_1, u_2, u_3) | u_1, u_2, u_3 \in \cup\} \quad (40)$$

where the control set  $U = \{(u_1^*, u_2^*, \text{and } u_3^*) | u_i : [0, T] \rightarrow [0, 1], \text{lebesgue measurable } i = 1, 2, 3\}$  (41)

### Pontryagin's Maximum Principle

The essential states that an optimal control should fulfill come from the Pontryagin's Maximum Principle (Abdullahi et al 2023).

The principle converts (39) – (40) into a problem of minimizing point wise a Hamiltonian  $H$ , with respect to  $(u_1, u_2, u_3)$ .

Using the optimal control theory, let  $\lambda_i(t)$  be adjoint variables with  $i = 1, \dots, 10$ . The Hamiltonian for the present optimal control problem is given by,



$$\begin{aligned}
 H = & A_1 I_h + A_2 I_a + A I_v + B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2 + \lambda_1 \left\{ \Lambda_h - ((1-u_3) \beta_1 C_e + \mu) S_h \right\} \\
 & + \lambda_2 \left\{ (1-u_3) \beta_1 S_h \beta_1 C_e - (\beta_2 + \mu) E_h \right\} + \lambda_3 \left\{ \beta_2 E_h - (\mu + \mu_0 + \beta_3 + u_1) I_h \right\} \\
 & + \lambda_4 \left\{ \Lambda_a - ((1-u_3) \beta_4 \beta_1 C_e + \mu_1) S_a \right\} + \lambda_5 \left\{ (1-u_3) \beta_4 S_a \beta_1 C_e - (\mu_1 + \beta_6) E_a \right\} \\
 & + \lambda_6 \left\{ \beta_6 E_a - (\mu_1 + \mu_2 + \beta_7 + u_1) I_a \right\} + \lambda_7 \left\{ \Lambda_v - (1-u_2) \beta_8 S_v \beta_1 C_e - \mu_3 S_v \right\} \\
 & + \lambda_8 \left\{ (1-u_2) \beta_8 S_v \beta_1 C_e - (\mu_3 + \beta_9) E_v \right\} + \lambda_9 \left\{ \beta_9 E_v - (\mu_3 + \mu_4 + \beta_{10}) I_v \right\} \\
 & + \lambda_{10} \left\{ \beta_3 I_h + \beta_7 I_a + \beta_{10} I_v - (\mu_5 + u_2) C_e \right\}
 \end{aligned} \quad (42)$$

**Theorem 1** Given an optimal control  $u_1, u_2$ , and  $u_3$  and solutions  $S_h^*, E_h^*, I_h^*, S_v^*, E_v^*, I_v^*, S_a^*, E_a^*, I_a^*, C_e^*$  of the corresponding state system (50) that minimizes  $J(u_1, u_2, u_3)$  over  $\mathcal{U}$ . Then there exists adjoint variable  $\lambda_i = 0$  with  $i = 1, \dots, 10$ . Satisfying, Adjoint System, Transversality conditions and Stationary values.

The Adjoint System computed

$$\begin{aligned}
 \frac{\partial \lambda_1}{\partial t} &= -C_e (\lambda_1 - \lambda_2) (u_3 - 1) \beta_1 + \mu \lambda_1 \\
 &\vdots \\
 \frac{\partial \lambda_{10}}{\partial t} &= -(\beta_1 (\lambda_1 - \lambda_2) S_h + \beta_4 (S_a \lambda_4 - \lambda_5 E_h)) (u_3 - 1) C_e - S_v C_e \beta_8 (\lambda_7 - \lambda_8 (u_2 - 1) + (\mu_5 + u_2)) \lambda_{10}
 \end{aligned} \quad (43)$$

As the optimal control just has initial conditions, it is necessary to find the transversality conditions that corresponds to a terminal condition in the adjoint equation,

**Transversality conditions**

$$\lambda_i = 0 \text{ with } i = 1, \dots, 10. \quad (44)$$

**Stationary values**

the control  $u_1, u_2$ , and  $u_3$  satisfy the optimality condition

$$\begin{aligned}
 u_1^* &= \max \left\{ 0, \min \left( 1, \frac{\lambda_6 I_a + \lambda_2 I_h}{2B_1} \right) \right\} \\
 u_2^* &= \max \left\{ 0, \min \left( 1, \frac{-S_v C_e \beta_8 \lambda_7 + S_v C_e \beta_8 \lambda_8 + C_e \lambda_{10}}{2B_2} \right) \right\} \\
 u_3^* &= \max \left\{ 0, \min \left( 1, \frac{C_e (E_a \beta_4 \lambda_5 - S_a \beta_4 \lambda_4 - S_h \beta_1 \lambda_1 - S_h \beta_1 \lambda_2)}{2B_3} \right) \right\}
 \end{aligned} \quad (45)$$

**Proof:** Corollary 4.1 of (Fleming & Rishel 1975) as cited by Abdullahi & Amiru (2023b) present the existence of an optimal control as a result of the convexity of the integrand of  $J$  with respect to  $u_1$  and  $u_2$ , a priori boundedness of the state solutions, and the Lipschitz property of the state system with respect to the state variables. The governing equations of the adjoints variables are solved via differentiation of the Hamiltonian function, estimated at the optimal control. Then the adjoint system can be written as

$$\begin{aligned}
 \frac{\partial \lambda_1}{\partial t} &= -C_e (\lambda_1 - \lambda_2) (u_3 - 1) \beta_1 + \mu \lambda_1 \\
 &\vdots \\
 \frac{\partial \lambda_{10}}{\partial t} &= -(\beta_1 (\lambda_1 - \lambda_2) S_h + \beta_4 (S_a \lambda_4 - \lambda_5 E_h)) (u_3 - 1) C_e - S_v C_e \beta_8 (\lambda_7 - \lambda_8 (u_2 - 1) + (\mu_5 + u_2)) \lambda_{10}
 \end{aligned} \quad (46)$$

and with transversality conditions



$$\lambda_i = 0 \text{ with } i = 1, \dots, 10 \quad (47)$$

On the interior of the control set, where  $0 \leq u_i \leq 1$ , for  $i = 1, 2, 3$  yields

Thus, it is obtained that,

$$\begin{cases} u_1^* = \max \left\{ 0, \min \left( 1, \frac{\lambda_6 I_a + \lambda_2 I_h}{2B_1} \right) \right\} \\ u_2^* = \max \left\{ 0, \min \left( 1, \frac{C_e (-S_v \beta_8 \lambda_7 + S_v \beta_8 \lambda_8 + \lambda_{10})}{2B_2} \right) \right\} \\ u_3^* = \max \left\{ 0, \min \left( 1, \frac{C_e (E_a \beta_4 \lambda_5 - S_a \beta_4 \lambda_4 - S_h \beta_1 \lambda_1 - S_h \beta_1 \lambda_2)}{2B_3} \right) \right\} \end{cases}$$

where

$$u_1^* = \min \{1, \xi_1^*\}, u_2^* = \min \{1, \xi_2^*\}, u_3^* = \min \{1, \xi_3^*\}. \quad (48)$$

Subsequently, the numerical solutions of the optimality system and the corresponding optimal control pair, the parameter choices, and the interpretations from various cases are discussed.

### Numerical analysis

This section analyzes the impact of optimal control strategies on schistosomiasis using a numerical approach. The model solves state and adjoint equations iteratively with the Runge-Kutta method, updating control strategies until results converge. Four strategies are tested: combining two controls at a time—Mass Drug Administration, Snail Control, and Public Health Education—and finally applying all three together to evaluate their effectiveness in reducing disease transmission. For the numerical simulation

the following weight factors are use  $A_i, i = 1, 2, 3$  and  $B_i, i = 1, 2, 3 = 0$  and use the parameter values from Table.2. Initial states variables are chosen as  $S_h(0) = 100,000, I_h(0) = 1, E_h(0) = 0, S_v(0) = 100,000, I_v(0) = 1, E_v(0) = 0, S_a(0) = 20,000, I_a(0) = 1, E_a(0) = 0$  (Zaliki 2023). Other parameter values are in Table 2 to illustrate the effect of different optimal.

### Optimal use of the Combination of Mass Drug Administration and snail control (Strategy A)

The Mass Drug Administration ( $u_1$ ) and snail control ( $u_2$ ) are both used to optimize the objective functional  $J$ , while the control

public health education  $u_3$  is set to zero. In Figures 1 to 8, the results demonstrate a significant difference in the transmission dynamics of schistosomiasis when optimal control strategies are applied compared to when no control measures are in place. Specifically, the inclusion of optimal control interventions leads to a noticeable reduction in transmission rates, highlighting the effectiveness of these strategies in managing schistosomiasis within the model framework.

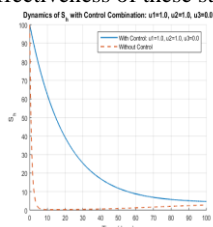


Figure 1: effect of the control strategy A on susceptible humans

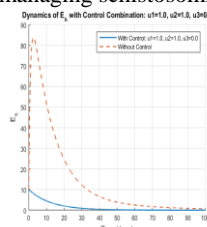


Figure 2: effect of the control strategy A on expose humans

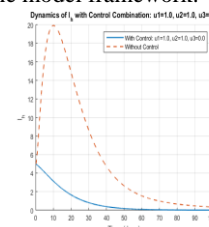


Figure 3: effect of the control strategy A on infected humans

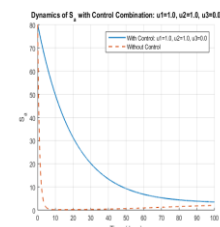


Figure 4: effect of the control strategy A on susceptible Animals

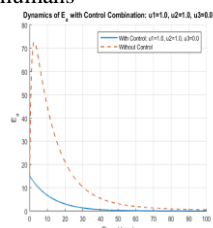


Figure 5: effect of the control strategy A on exposed Animals

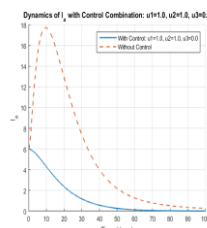


Figure 6: effect of the control strategy A on infected Animals

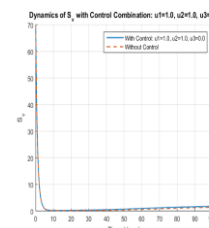


Figure 7: effect of the control strategy A on Susceptible Snails

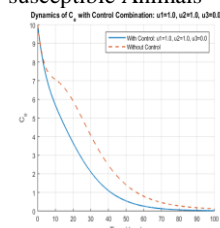


Figure 8: effect of the control strategy A on

### Optimal use of the Mass Drug Administration and Public Health Education (strategy B)

The Mass Drug Administration  $u_1$  and Public Health Education  $u_3$  are both used to optimize the objective functional  $J$ , while the control Snail control  $u_2$  is set to zero. In Figures 10 to 17, the results demonstrate a significant difference in the transmission dynamics of schistosomiasis when optimal control strategies are applied compared to when no control measures are in place. Specifically, the inclusion of optimal control interventions leads to a noticeable reduction in transmission rates, highlighting the effectiveness of these strategies in managing schistosomiasis within the model framework

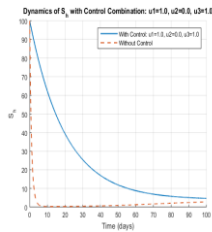


Figure 9: : effect of the control strategy B on Susceptible Humans

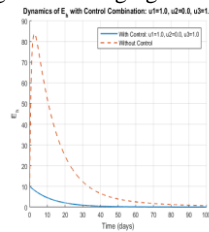


figure 10: : effect of the control strategy B on exposed Humans

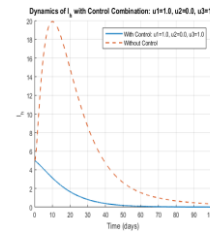


Figure 11: : effect of the control strategy B on Infected Humans

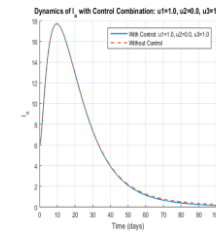


Figure 12: : effect of the control strategy B on Infected Animals

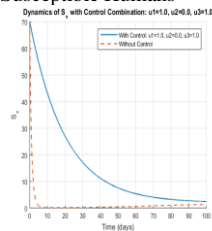


Figure 13: : effect of the control strategy B on Susceptible snails

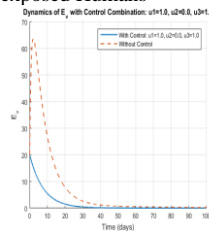


Figure 15: : effect of the control strategy B on Exposed snails

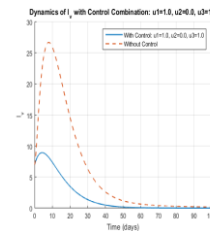


Figure 16: : effect of the control strategy B on infected Snails

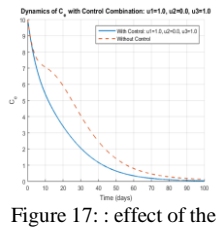


Figure 17: : effect of the control strategy B on contaminated environment

### Optimal use of the Combination of Snail Control and Public Health Education. (Strategy C)

The Snail Control  $u_2$  and Public Health Education  $u_3$  are both used to optimize the objective functional  $J$ , while the control Mass Drug Administration  $u_1$  is set to zero. In Figures 18 to 22, the results demonstrate a significant difference in the transmission dynamics of schistosomiasis when optimal control strategies are applied compared to when no control measures are in place. Specifically, the inclusion of optimal control interventions leads to a noticeable reduction in transmission rates, highlighting the effectiveness of these strategies in managing schistosomiasis within the model framework

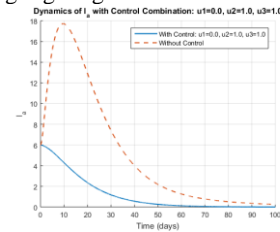


Figure 18: : effect of the control strategy C on Infected Animals

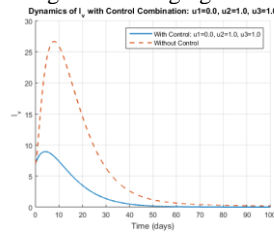


Figure 19: : effect of the control strategy C on Infected snails

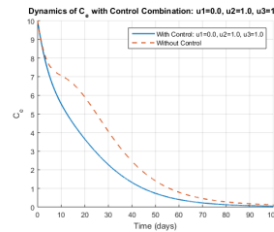


Figure 20: : effect of the control strategy C on contaminated environment

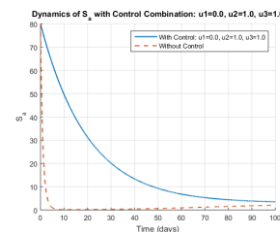


Figure 21: : effect of the control strategy C on susceptible Animals

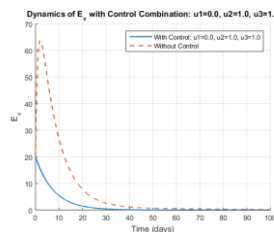


Figure 22: : effect of the control strategy C on exposed snails

**Mass Drug Administration, Snail Control, and Public Health education.**

Here, all the control strategies  $u_1, u_2$ , and  $u_3$  are used to optimize the objective functional  $J$ . In Figures 23 to 29, the results demonstrate a significant difference in the transmission dynamics of schistosomiasis when optimal control strategies are applied compared to when no control measures are in place. Specifically, the inclusion of optimal control interventions leads to a noticeable reduction in transmission rates, highlighting the effectiveness of these strategies in managing schistosomiasis within the model framework

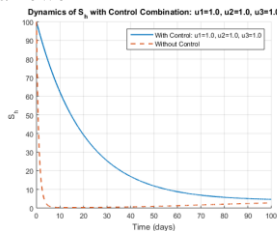


Figure 24 : effect of the control strategy D on Susceptible Humans

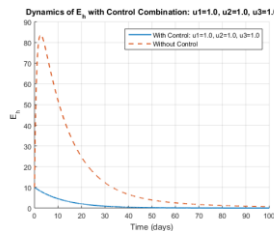


Figure 25 : effect of the control strategy D on exposed Humans

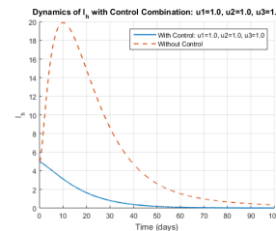


figure 26: effect of the control strategy D on infected Humans

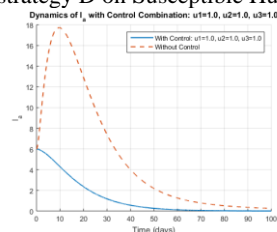


Figure 27 : effect of the control strategy D on infected Animals

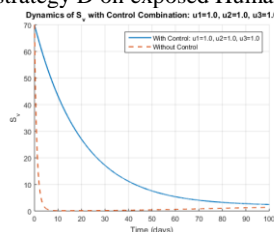


Figure 28 : effect of the control strategy D on Susceptible snails

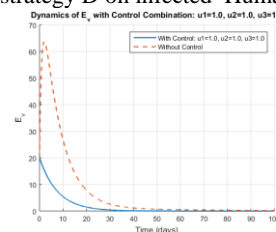


Figure 29 : effect of the control strategy D on exposed snails

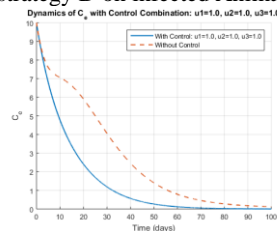


Figure 30 : effect of the control strategy D on contaminated environment

**Numerical Analysis and Optimal Control Strategies**

Numerical simulations showed that combining control measures is effective in reducing schistosomiasis. Strategies involving two interventions—such as drug administration, snail control, or public health education—reduced infection rates, but the most significant impact was achieved when all three were used together. This highlights the importance of a comprehensive approach, and the model helps evaluate the cost-effectiveness of each strategy for better resource planning..

**CONCLUSION**

This study developed a mathematical model to understand the transmission dynamics of schistosomiasis in Pakwach District, Northern Uganda, focusing on the role of freshwater snails as intermediate hosts. The model showed that managing both snail populations and environmental factors, such as water sanitation, along with public health education, could significantly reduce disease transmission. An integrated approach combining mass drug administration (MDA), snail control, and education was identified as the most effective strategy. However, the model's reliance on existing literature rather than local data is a limitation. Future research could refine the model with real-world data, explore vaccine development, and assess the cost-effectiveness of control strategies. The study offers valuable insights for policymakers, providing a framework for targeted

interventions, and has broader applications in public health planning, vector control, and global health initiatives.

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