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# On the mathematical modeling of schistosomiasis transmission dynamics with heterogeneous intermediate host

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Schistosomiasis is a neglected disease affecting almost every region of the world, with its endemicity mainly experience in sub-Saharan Africa. It remains difficult to eradicate due to heterogeneity associated with its transmission mode. A mathematical model of Schistosomiasis integrating heterogeneous host transmission pathways is thus formulated and analyzed to investigate the impact of the disease in the human population. Mathematical analyses are presented, including establishing the existence and uniqueness of solutions, computation of the model equilibria, and the basic reproduction number ( $R_0$ ). Stability analyses of the model equilibrium states show that disease-free and endemic equilibrium points are locally and globally asymptotically stable whenever  $R_0 < 1$  and  $R_0 > 1$ , respectively. Additionally, bifurcation analysis is carried out to establish the existence of a forward bifurcation around  $R_0 = 1$ . Using Latin-hypercube sampling, global sensitivity analysis was performed to examine and investigate the most significant model parameters in  $R_0$  which drives the infection. The sensitivity analysis result indicates that the snail's natural death rate, cercariae, and miracidia decay rates are the most influential parameters. Furthermore, numerical simulations of the model were done to show time series plots, phase portraits, and 3-D representations of the model and also to visualize the impact of the most sensitive parameters on the disease dynamics. Our numerical findings suggest that reducing the snail population will directly reduce Schistosomiasis transmission within the human population and thus lead to its eradication.

## KEYWORDS

schistosomiasis, heterogeneous host, mathematical analysis, sensitivity analysis, global stability

## 1. Introduction

Schistosomiasis which is also known as *Bilharzia*, *Bilharziasis*, and *snail fever*, is one of the neglected tropical diseases (NTD) common in some tropical and subtropical countries, in areas where there is no access to safe drinking water, adequate hygiene, and appropriate sanitation (sewage disposal). It is a water-based disease that is

transmitted through contact with water infested with cercariae and it affects about 240 million people worldwide and more than 700 million people live in an area where the disease is endemic [1]. Schistosomiasis infection is also known as a parasite disease that is caused by schistosomiasis organisms (blood fluke). The two main forms of schistosomiasis infection are urogenital and intestinal which are caused by five schistosomiasis species responsible for human or animal infections namely, *Schistosoma mansoni*, *Schistosoma intercalatum*, *Schistosoma haematobium*, *Schistosoma mekongi*, and *Schistosoma japonicum* [1]. There are two stages of schistosomiasis infection namely; acute and chronic stages. Acute schistosomiasis infection referred to as Katayama Syndrome is a clinical manifestation of infection caused by trematode worms [2].

When non-immune persons are exposed to schistosomiasis infection, there is an incubation period symptoms of infection include abdominal pain, blood in stool or urine, wasting, anemia, dysuria [2]. Chronic schistosomiasis infection is caused by schistosome, which has severe long-term implications [3]. The symptoms of the chronic stage are growth retardation, renal failure, ascites, grand mal epilepsy, decreased fitness, anemia, portal hypertension, transverse myelitis, bladder carcinoma, and infertility [2]. Normally, snails harbor cercariae and shed the infective stage (cercariae) of the parasite (schistosome) in the freshwater [4]. Hence, there is an increase in the prevalence of Schistosomiasis as a result of freshwater habitats such as dams and irrigation, which enhances snail habitation and thus, increases transmission. Despite the progressive achievements in schistosomiasis control over the years, Schistosomiasis is still one of the most devastating parasitic diseases ravaging the entire world. Guiro et al. [5] proposed a mathematical model for schistosomiasis control, considering two population hosts, humans, and snails with delays. Their results suggest that an effective education campaign and reasonable coverage level of drug treatment will help in the control of Schistosomiasis. A mathematical model for the human-cattle-snail transmission of Schistosomiasis in Hubei Province, China was studied by Chen et al. [6]. Their results show that to control or eliminate Schistosomiasis in Hubei Province, China. Environmental factors need to be considered to lower (narrow) cattle-snail transmission. Since chemotherapy alone cannot end the spread of the causative parasite (schistosome), additional interventions and control strategies must be combined to lower re-infection, reduce the prevalence, and move toward the elimination of Schistosomiasis [7]. Many researchers have considered the use of molluscicides and different kinds of environmental modifications to control snails to reduce or eliminate schistosomiasis prevalence [7–9]. Interested readers can also see the following references [10–20] for other published works that have used mathematical models to gain insight into the transmission dynamics of Schistosomiasis and other

infectious diseases in the human population and various suggestion for their control/elimination.

Given the aforementioned above, our study presents a mathematical modeling work that examines qualitative mathematical analysis for the prevention and control of the spread of Schistosomiasis through the control of snails. This is in addition to chemotherapy, molluscicides, and other environmental factors already studied in the previous research works and also the inclusion of the acute and chronic infected populations (compartments). The present study also investigates if mammals such as cattle contribute to the spread of Schistosomiasis infection within the population. The model described in this work considers three population hosts that is: humans, cattle, and snails.

The paper is organized as follows: Section 2 gives the Model formulation, Section 3 the Mathematical model analysis is presented, Section 4 presents the Sensitivity analysis of parameters in the model, Section 5 presents the Results of the numerical simulations and finally, Section 6 gives a brief Discussion and Conclusion.

## 2. Model formulation

The life cycle of the schistosome parasite is formulated mathematically. The populations considered are the human population, other mammal population, and snail population with the Cercariae  $C(t)$  and Miracida  $M(t)$ . Cercariae  $C(t)$  is a larva worm that the infected snails shed into the aquatic environment, while the Miracida  $M(t)$  are eggs that the infected mammals (human and other mammals) shed in the stream when they come to the river for human activities such as fishing, swimming, fetching water, and/or drinking of water by other mammals. In this study, other mammals include cattle, mice, and dogs that the schistosome parasite can infect. The total human population,  $N_h(t)$ , at time,  $t$ , is divided into the sub-populations; susceptible,  $S_h(t)$ , acute infected,  $I_A(t)$ , and chronic infected,  $I_{ch}(t)$  humans. The other mammal population,  $N_a(t)$ , is subdivided into susceptible mammals,  $S_a$  and infected mammals,  $I_a$ , while the snail population,  $N_s(t)$  is made up of susceptible snails,  $S_s(t)$  and infected snails  $I_s(t)$ . Human recruitment and natural death rates are assumed to be  $\Lambda_h$  and  $\mu_h$ , respectively. The susceptible humans,  $S_h(t)$ , become infected through contact with fresh water contaminated with Cercariae from infected snails. The force of infection is given by

$$\frac{\beta_h \alpha_h C}{C_0 + \varepsilon_1 C}, \quad (1)$$

where  $\beta_h$  is the human transmission rate,  $\alpha_h$  is the adult parasite production rate for humans,  $C_0$ , is the half-saturation constant of Cercariae within the environment and  $\varepsilon_1$  is the limitation of the growth velocity of the pathogens for Cercariae. Susceptible

humans become acutely infected upon infection and enter into class,  $I_A(t)$ . In the acutely infected sub-population, a proportion of them may recover with partial immunity depending on the mass drug administration program within the community and return to the susceptible class at a rate,  $\psi$ . On the other hand, some human may develop a chronic infection at a rate,  $k$  and move to class,  $I_{ch}$ . Chronically infected humans may succumb to infection at a rate,  $\delta_h$ . Shedding of infection within the environment by acute infected and chronic infected humans is assumed to occur at rate  $N_E\tau_1\gamma_h$  and  $N_E\tau_1\gamma_h\sigma$ , respectively. Here,  $N_E$  is the number of eggs shed by mammals (both human and other mammals),  $\tau_1$  is the probability of eggs developing into Miracidium,  $\gamma_h$  is the infected human shedding rate and  $\sigma$  is a parameter that influences the shedding rate of the chronically infected humans. For the other mammals, the recruitment rate is assumed to be  $\Lambda_a$  while the natural death rate is  $\mu_a$ . The susceptible mammals are infected through contact with fresh water contaminated with Cercariae from infected snails and the force of infection is given by

$$\frac{\beta_a\alpha_a C}{C_0 + \varepsilon_1 C}, \tag{2}$$

where  $\beta_a$  is the other mammal transmission rate,  $\alpha_a$ , is the adult parasite production probability for other mammals. The other mammals shed the eggs that hatch into the water at a rate,  $N_E\tau_1\gamma_a$  where  $\gamma_a$  is the other mammals shedding rate. Death due to infection among other infected mammals is assumed to be at a rate,  $\delta_a$ . Furthermore, susceptible snails are recruited at a rate,  $\Lambda_s$  and death due to natural causes of snails is at a rate,  $\mu_s$ . Susceptible snails become infected by contact with miracidia from the viral shedding of infected humans and other mammals and the force of infection is given by

$$\frac{\beta_s\alpha_s M}{M_0 + \varepsilon_2 M}, \tag{3}$$

where  $\beta_s$ , is the snail transmission rate,  $\alpha_s$  is the Miracidial penetration probability for snails,  $\varepsilon_2$ , is the limitation of the growth velocity for the Miracidium and  $M_0$ , is the half-saturation constant for Miracida within the environment. Death due to infection of snails is assumed to be at a rate,  $\delta_s$ . Infected snails shed larva worm in the environment at a rate,  $\tau_2\gamma_s$  where  $\tau_2$  is the density of Cercariae for snails and  $\gamma_s$  is the snail shedding rate. Finally, the decay rates for the Cercariae and Miracida are  $\mu_c, \mu_M$ , respectively. The study assumes that there is no immigration of infectious humans, other mammals, and snails within their populations. Also, susceptible humans, other mammals and snails are recruited at a constant rate. The dynamics of infection are presented in the model flow diagram in Figure 1. This leads to the following system of non-linear differential equations:

$$\left\{ \begin{aligned} \frac{dS_h}{dt} &= \Lambda_h - \frac{\beta_h\alpha_h S_h C}{C_0 + \varepsilon_1 C} - \mu_h S_h + \psi I_A, \\ \frac{dI_A}{dt} &= \frac{\beta_h\alpha_h S_h C}{C_0 + \varepsilon_1 C} - (k + \mu_h + \psi)I_A, \\ \frac{dI_{ch}}{dt} &= kI_A - (\mu_h + \delta_h)I_{ch}, \\ \frac{dS_a}{dt} &= \Lambda_a - \frac{\beta_c\alpha_a C S_a}{C_0 + \varepsilon_1 C} - \mu_a S_a, \\ \frac{dI_a}{dt} &= \frac{\beta_a\alpha_a C S_a}{C_0 + \varepsilon_1 C} - (\mu_a + \delta_a)I_a, \\ \frac{dM}{dt} &= N_E\tau_1(\gamma_h(I_A + \sigma I_{ch}) + \gamma_a I_a) - \mu_m M, \\ \frac{dS_s}{dt} &= \Lambda_s - \frac{\beta_s\alpha_s M S_s}{M_0 + \varepsilon_2 M} - \mu_s S_s, \\ \frac{dI_s}{dt} &= \frac{\beta_s\alpha_s M S_s}{M_0 + \varepsilon_2 M} - (\mu_s + \delta_s)I_s, \\ \frac{dC}{dt} &= \tau_2\gamma_s I_s - \mu_c C \end{aligned} \right. \tag{4}$$

with initial conditions

$$\begin{aligned} S_h(0) > 0, I_A(0) \geq 0, I_{ch}(0) \geq 0, S_a(0) > 0, I_a(0) \geq 0, \\ S_s(0) > 0, I_s(0) \geq 0, M(0) \geq 0, C(0) \geq 0. \end{aligned}$$

All the parameters are assumed to be non-negative over the modeling time frame.

### 3. Mathematical methods

#### 3.1. Invariant region

A biologically feasible population model such as model system (4) should always have positive solutions. To show that all solutions of model system (4) are non-negative, we let  $N_h, N_a$  and  $N_s$  be the total human population, other mammals population and snail populations, respectively, where,  $N_h(t) = S_h(t) + I_A(t) + I_{ch}(t)$ ,  $N_a(t) = S_a(t) + I_a(t)$ , and  $N_s(t) = S_s(t) + I_s(t)$ , respectively with the initial conditions  $N_h(0), N_a(0), N_s(0), M(0)$ , and  $C(0)$ .

Adding the human, other mammals and snail populations give the respective total populations as  $N_h'(t) = \Lambda_h - \delta_h I_{ch} - N_h\mu_h, N_a'(t) = \Lambda_a - \delta_a I_a - N_a\mu_a$  and  $N_s'(t) = \Lambda_s - \delta_s I_s - N_s\mu_s$ . In the absence of infection which implies that the disease-related death rates for chronically infected human,  $\delta_h$ , other infected mammals,  $\delta_a$  and infected snail,  $\delta_s$  are negligible, we assume that the total populations,  $N_h, N_a$  and  $N_s$  are asymptotically stable

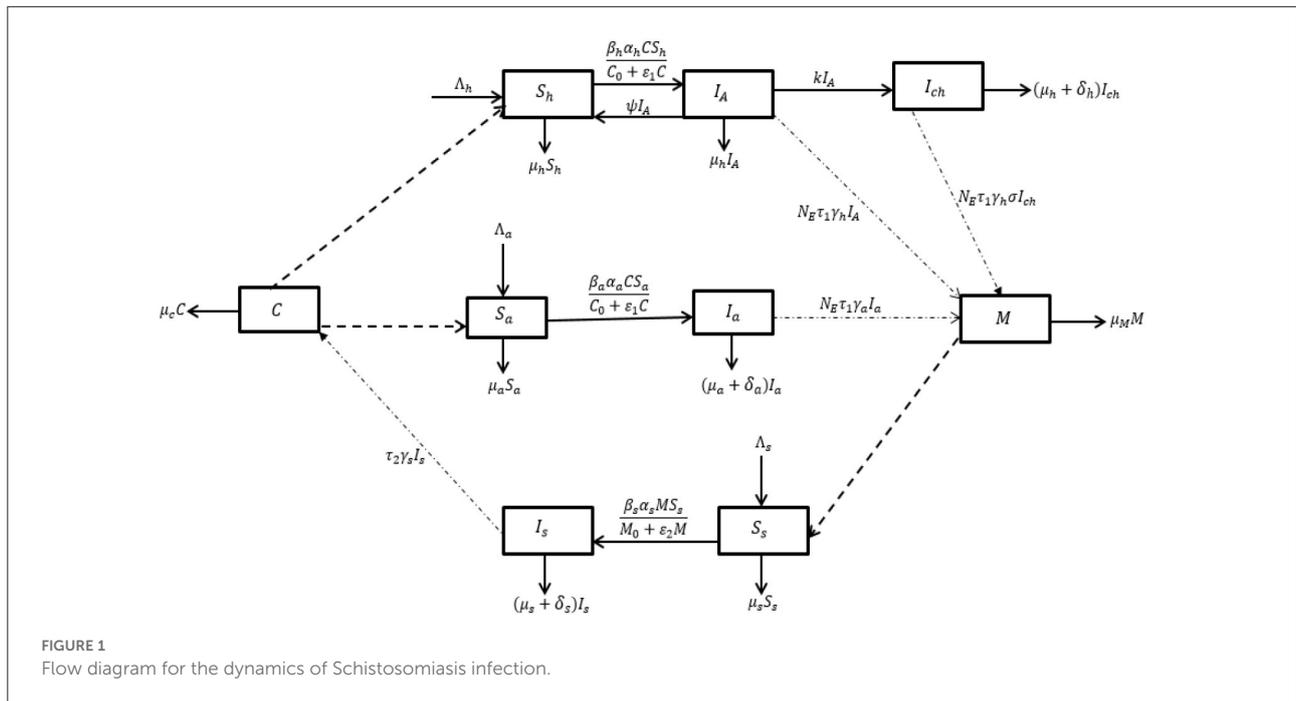


FIGURE 1  
Flow diagram for the dynamics of Schistosomiasis infection.

and yield

$$\begin{aligned}
 N_h'(t) &\leq \Lambda_h - N_h \mu_h, \\
 N_a'(t) &\leq \Lambda_a - N_a \mu_a, \\
 N_s'(t) &\leq \Lambda_s - N_s \mu_s.
 \end{aligned}
 \tag{5}$$

Solving (5) using the inequality theorem by Birkhoff and Rota [21] and integrating with the initial conditions  $N_h(0)$ ,  $N_a(0)$ ,  $N_s(0)$ ,  $C(0)$ , and  $M(0)$  gives

$$\begin{aligned}
 N_h &\leq \frac{\Lambda_h}{\mu_h} - \left( \frac{\Lambda_h - \mu_h N_{h0}}{\mu_h} \right) \exp(-\mu_h t), \\
 N_a &\leq \frac{\Lambda_a}{\mu_a} - \left( \frac{\Lambda_a - \mu_a N_{a0}}{\mu_a} \right) \exp(-\mu_a t), \\
 N_s &\leq \frac{\Lambda_s}{\mu_s} - \left( \frac{\Lambda_s - \mu_s N_{s0}}{\mu_s} \right) \exp(-\mu_s t).
 \end{aligned}$$

As  $t \rightarrow \infty$ , the total populations for humans, other mammals and snails approach  $N_h \leq \frac{\Lambda_h}{\mu_h}$ ,  $N_a \leq \frac{\Lambda_a}{\mu_a}$  and  $N_s \leq \frac{\Lambda_s}{\mu_s}$ , respectively. Since  $I_A, I_{ch} \leq N_h(t)$ ,  $I_a \leq N_a(t)$ , and  $I_s \leq N_s(t)$  it implies that the Cercariae ( $C$ ) and Miracidia ( $M$ ) populations can be written as

$$\begin{aligned}
 C'(t) &\leq \frac{\gamma_s \tau_2 \Lambda_s}{\mu_s} - \mu_c C, \\
 M'(t) &\leq N_E \tau_1 \left[ \frac{\gamma_h (1 + \sigma) \Lambda_h}{\mu_h} + \frac{\gamma_a \Lambda_a}{\mu_a} \right] - \mu_m M.
 \end{aligned}
 \tag{6}$$

Applying the inequality theorem by Birkhoff and Rota [21] with the initial conditions  $C(0) = C_{00}$  and

$M(0) = M_{00}$  yields

$$C(t) \leq \frac{\Lambda_s \gamma_s \tau_2}{\mu_s \mu_c} - \left[ \frac{\gamma_s \tau_2 \Lambda_s - \mu_s \mu_c C_{00}}{\mu_s \mu_c} \right] \exp(-\mu_c t),$$

and

$$\begin{aligned}
 M(t) &\leq N_E \tau_1 \left[ \frac{\Lambda_h \gamma_h (1 + \sigma)}{\mu_h \mu_m} + \frac{\Lambda_a \gamma_a}{\mu_a \mu_m} \right] \\
 &\quad - \left[ \frac{N_E \tau_1 (\Lambda_h \gamma_h \mu_a (1 + \sigma) + \Lambda_a \gamma_a \mu_h) - \mu_h \mu_a \mu_m M_{00}}{\mu_h \mu_a \mu_m} \right] \\
 &\quad \exp(-\mu_m t).
 \end{aligned}$$

As  $t \rightarrow \infty$ , the Cercariae and Miracidia populations tends to  $C(t) \leq \frac{\Lambda_s \gamma_s \tau_2}{\mu_s \mu_c}$  and  $M(t) \leq N_E \tau_1 \left[ \frac{\Lambda_h \gamma_h (1 + \sigma)}{\mu_h \mu_m} + \frac{\Lambda_a \gamma_a}{\mu_a \mu_m} \right]$  respectively.

This shows that all the feasible solutions  $N_h(t)$ ,  $N_a(t)$ ,  $N_s(t)$ ,  $C(t)$  and  $M(t)$  are bounded and the region,

$$\mathcal{Z} = \left\{ (S_h, I_A, I_{ch}, S_a, I_a, M, S_s, I_s, C) \in \mathbb{R}_+^9 \mid N_h \leq \frac{\Lambda_h}{\mu_h}, N_a \leq \frac{\Lambda_a}{\mu_a}, N_s \leq \frac{\Lambda_s}{\mu_s}, M \leq M_{max}, C \leq C_{max} \right\}$$

is positively invariant. Thus, we conclude that for all  $t > 0$ , model system (4) is biologically feasible and well-posed in  $\mathcal{Z}$ .

### 3.2. Positivity of the solution

We show that the solutions of the model system (4) are positive for all time by proving the following theorem:

**Theorem 3.1.** *With positive initial conditions, the solution set of model system (4),  $[S_h(t), I_A(t), I_{ch}(t), S_a(t), I_a(t), M(t), S_s(t), I_s(t), C(t)]$ , is positive for all time,  $t > 0$ .*

*Proof.* From the first equation of the model Equation (4), we have

$$S_h' = \Lambda_h - \frac{\beta_h \alpha_h C S_h}{C_0 + \varepsilon_c C} - \mu_h S_h + \psi I_A \geq - \left( \frac{\beta_h \alpha_h C}{C_0 + \varepsilon_c C} + \mu_h \right) S_h \tag{7}$$

Integrating with initial condition  $S_h(0)$ , we obtain

$$S_h(t) \geq S_h(0) \exp\left\{ \int_0^t -(y_h + \mu_h) dt \right\} > 0 \tag{8}$$

with  $y_h = \frac{\beta_h \alpha_h C}{C_0 + \varepsilon_1 C}$ .

In a similar way with  $y_a = \frac{\beta_a \alpha_a C}{C_0 + \varepsilon_1 C}$  and  $y_s = \frac{\beta_s \alpha_s M}{M_0 + \varepsilon_2 M}$ ,

$$\begin{aligned} I_A(t) &\geq I_A(0) \exp\{- (k + \mu_h + \psi)t\} > 0, \\ I_{ch}(t) &\geq I_{ch}(0) \exp\{- (\mu_h + \delta_h)t\} > 0, \\ S_a(t) &\geq S_a(0) \exp\left\{ \int_0^t -(y_a + \mu_a) dt \right\} > 0, \\ I_a(t) &\geq I_a(0) \exp\{- (\mu_a + \delta_a)t\} > 0, \\ S_s(t) &\geq S_s(0) \exp\left\{ \int_0^t -(y_s + \mu_s) dt \right\} > 0, \\ I_s(t) &\geq I_s(0) \exp\{- (\mu_s + \delta_s)t\} > 0, \\ C(t) &\geq C(0) \exp\{- \mu_c t\} > 0, \\ M(t) &\geq M(0) \exp\{- \mu_m t\} > 0. \end{aligned} \tag{9}$$

Therefore, we conclude that the solution set  $[S_h(t), I_A(t), I_{ch}(t), S_a(t), I_a(t), M(t), S_s(t), I_s(t), C(t)]$  of model Equation (4) is positive for all  $t > 0$ . This shows that the model is well-posed and biologically meaningful since the sub-population cannot be negative.  $\square$

### 3.3. Existence of disease-free equilibrium

We examine the stability of the disease-free equilibrium state of the schistosomiasis model system (4) in terms of basic reproduction number. The disease-free equilibrium,  $E_0$ , is the equilibrium state in the absence of schistosomiasis disease in the environment. At equilibrium state,

$$\begin{aligned} \frac{dS_h}{dt} = \frac{dI_A}{dt} = \frac{dI_{ch}}{dt} = \frac{dS_a}{dt} = \frac{dI_a}{dt} = \frac{dS_s}{dt} = \frac{dI_s}{dt} = \frac{dM}{dt} \\ = \frac{dC}{dt} = 0. \end{aligned}$$

This is solved simultaneously for the schistosomiasis model Equation (4) to yield the disease-free equilibrium,

$$\begin{aligned} E_0 &= (S_h^0, I_A^0, I_{ch}^0, S_a^0, I_a^0, S_s^0, I_s^0, M^0, C^0) \\ &= \left( \frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_a}{\mu_a}, 0, \frac{\Lambda_s}{\mu_s}, 0, 0, 0 \right). \end{aligned} \tag{10}$$

#### 3.3.1. The basic reproduction number

The basic reproduction number of the schistosomiasis model system (4) denoted by  $R_0$ , is a threshold parameter that is interpreted biologically as the average number of infected individuals created by one infected individual introduced in a susceptible population during the course of its infectious period [22, 23]. We compute  $R_0$  using the next-generation matrix approach by Van den Driessche and Watmough [22]. Applying the approach, let  $\mathcal{F}$  and  $\mathcal{V}$  denote the Jacobian matrices for the rates of appearance of new infections and the transfer of infections in and out of the infected compartments, respectively at DFE,  $E_0$ , so that

$$\begin{aligned} \mathcal{F} &= \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & \frac{\beta_h \alpha_h S_h^0}{C_0} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{\beta_a \alpha_a S_a^0}{C_0} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta_s \alpha_s S_s^0}{M_0} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \text{ and} \\ \mathcal{V} &= \begin{pmatrix} f & 0 & 0 & 0 & 0 & 0 \\ -k & g & 0 & 0 & 0 & 0 \\ 0 & 0 & h & 0 & 0 & 0 \\ -A & -A\sigma & -B & \mu_m & 0 & 0 \\ 0 & 0 & 0 & 0 & q & 0 \\ 0 & 0 & 0 & 0 & -D & \mu_c \end{pmatrix}, \end{aligned} \tag{11}$$

where

$$\begin{aligned} f = k + \mu_h + \psi, h = \mu_a + \delta_a, g = \mu_h + \delta_h, q = \mu_s + \delta_s, \\ A = N_E \gamma_h \tau_1, B = N_E \gamma_a \tau_1, D = \gamma_s \tau_2. \end{aligned} \tag{12}$$

The basic reproduction number,  $R_0$ , for the model system (4) is defined as the spectral radius of the operator,  $\mathcal{F}\mathcal{V}^{-1}$ , where  $\mathcal{V}^{-1}$  is the inverse matrix of  $\mathcal{V}$ . This gives

$$\begin{aligned} R_0 &= \sqrt{\frac{\beta_s \alpha_s \Lambda_s D}{q \mu_c \mu_s M_0} \left( \frac{\beta_h \alpha_h \Lambda_h A (k\sigma + g)}{fg \mu_h C_0 \mu_m} + \frac{\beta_a \alpha_a \Lambda_a B}{h \mu_a C_0 \mu_m} \right)} \\ &= \sqrt{R_{sch} + R_{SA} + R_{sm}} \end{aligned} \tag{13}$$

for

$$R_{sch} = R_s \times R_{ch}, \quad R_{SA} = R_s \times R_A, \quad R_{sm} = R_s \times R_m,$$

$$\begin{aligned}
 R_s &= \frac{\beta_s \alpha_s D \Lambda_s}{\mu_c q M_0 \mu_s}, \\
 R_m &= \frac{\beta_a \alpha_a B \Lambda_a}{h \mu_m C_0 \mu_a}, \quad R_{ch} = \frac{\beta_h \alpha_h A k \sigma \Lambda_h}{f g \mu_m C_0 \mu_h}, \\
 R_A &= \frac{\beta_h \alpha_h A \Lambda_h}{f \mu_m C_0 \mu_h}.
 \end{aligned} \tag{14}$$

Here, the reproduction numbers,  $R_A$ ,  $R_{ch}$ ,  $R_m$ , and  $R_s$ , are the reproduction numbers for the acutely infected humans, chronic infected humans, other infected mammals and infected snails contribute, respectively. This shows that the basic reproduction number,  $R_0$  of the schistosomiasis model system (4) comprises of four parts, that is,  $R_A$ ,  $R_{ch}$ ,  $R_m$ ,  $R_s$ . Importantly, whenever  $R_0 < 1$ , it implies that the disease will fizzle out in the population and whenever  $R_0 > 1$ , it means that the disease will persist in the population.

### 3.4. Local stability of the disease-free equilibrium

We prove the local stability of the disease-free equilibrium,  $E_0$ , for the Schistosomiasis model system (4) using the Jacobian method.

**Theorem 3.2.** *The disease-free equilibrium state,  $E_0$ , of the schistosomiasis model system (4) is locally asymptotically stable when  $R_0 < 1$  and unstable for  $R_0 > 1$ .*

*Proof.* We compute the Jacobian matrix of the model system (4) at the equilibrium state,  $E_0$ , as follows.

$$J(E_0) = \begin{pmatrix}
 -\mu_h & \psi & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_h \alpha_h S_h^0}{C_0} \\
 0 & -f & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_h \alpha_h S_h^0}{C_0} \\
 0 & k & -g & 0 & 0 & 0 & 0 & 0 & 0 \\
 0 & 0 & 0 & -\mu_a & 0 & 0 & 0 & 0 & -\frac{\beta_a \alpha_a S_a^0}{C_0} \\
 0 & 0 & 0 & 0 & -h & 0 & 0 & 0 & \frac{\beta_a \alpha_a S_a^0}{C_0} \\
 0 & A & \sigma A & 0 & B & -\mu_m & 0 & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & -\frac{\beta_s \alpha_s S_s^0}{M_0} & -\mu_s & 0 & 0 \\
 0 & 0 & 0 & 0 & 0 & \frac{\beta_s \alpha_s S_s^0}{M_0} & 0 & -q & 0 \\
 0 & 0 & 0 & 0 & 0 & 0 & 0 & D & -\mu_c
 \end{pmatrix}, \tag{15}$$

where  $f, g, h, q, A, B, D$  are defined in (12). The eigenvalues of (15) are  $\lambda_1 = -\mu_h, \lambda_2 = -\mu_a, \lambda_3 = -\mu_s$  and the roots of the characteristics equation

$$\lambda^6 + P\lambda^5 + Q\lambda^4 + R\lambda^3 + U\lambda^2 + V\lambda + W = 0 \tag{16}$$

where

$$\begin{cases}
 P = \mu_c + \mu_m + q + h + g + f, \\
 Q = \mu_c(\mu_m + f + g + h + q) + \mu_m(f + g + h + q) \\
 \quad + f(g + h + q) + g(h + q) + hq, \\
 R = \mu_m\mu_c(f + g + h + q) + (\mu_c f + \mu_m f)(g + h + q) \\
 \quad + (\mu_m g + \mu_c g + fg)(h + q) + hq(\mu_c + f + g), \\
 U = \mu_m\mu_c q f(1 - R_{sA}) + \mu_m\mu_c q h(1 - R_{sm}) \\
 \quad + \mu_m\mu_c f(g + h) \\
 \quad + (\mu_m\mu_c g + \mu_c f g + \mu_m f g)(h + q) \\
 \quad + (\mu_c h q + \mu_m h q)(f + g) + f g h q, \\
 V = \mu_m\mu_c g q(1 - R_{sch} - R_{sA}) \\
 \quad + \mu_m\mu_c f h q(1 - R_{sA} - R_{sm}) \\
 \quad + \mu_m\mu_c q h g(1 - R_{sm}) + f g h(\mu_m\mu_c \\
 \quad + \mu_c q + \mu_m q), \\
 W = f g h q \mu_m \mu_c M_0 C_0 \mu_s \mu_a \mu_h (1 - R_0^2).
 \end{cases} \tag{17}$$

With the definitions of reproduction numbers in (14), we have by Routh-Hurwitz criteria in Kim et al. [24] and the conditions in Heffernan et al. [25] that the polynomial in (16) has negative real roots provided that  $P > 0, Q > 0, R > 0, U > 0, V > 0, W > 0, PQ > R, QU > W, UV > RW$  and this happens when  $R_0 < 1$ . With negative real eigenvalues, it implies that the disease-free equilibrium state,  $E_0$ , for Schistosomiasis model system (4) is locally asymptotically stable if  $R_0 < 1$  otherwise it is unstable.  $\square$

### 3.5. Existence of endemic equilibrium

For the endemic equilibrium, let

$$\lambda_c^* = \frac{C^*}{C_0 + \varepsilon_1 C^*}, \quad \lambda_m^* = \frac{M^*}{M_0 + \varepsilon_2 M^*}, \tag{18}$$

in model system (4). We have at equilibrium state that

$$\begin{aligned}
 S_h^* &= \frac{\Lambda_h - (\mu_h + k)I_A^*}{\mu_h}, \quad I_A^* = \frac{\lambda_c^* \beta_h \alpha_h \Lambda_h}{\beta_h \alpha_h (\mu_h + k) \lambda_c^* + f \mu_h}, \\
 I_{ch}^* &= \frac{k}{g} I_A^*, \quad S_a^* = \frac{\Lambda_a - h I_a^*}{\mu_a}, \\
 I_a^* &= \frac{\lambda_c^* \beta_a \alpha_a \Lambda_a}{\beta_a \alpha_a \lambda_c^* h + h \mu_a}, \quad S_s^* = \frac{\Lambda_s - q I_s^*}{\mu_s}, \\
 I_s^* &= \frac{\beta_s \alpha_s \Lambda_s \lambda_m^*}{\beta_s \alpha_s q \lambda_m^* + \mu_s q}, \quad C^* = \frac{D}{\mu_c} I_s^*, \\
 M^* &= \frac{A(I_A^* + \sigma I_{ch}^*) + B I_a^*}{\mu_m}.
 \end{aligned} \tag{19}$$

Substituting  $S_h^*, I_A^*, I_{ch}^*, S_a^*, I_a^*, S_s^*, I_s^*, M^*$ , and  $C^*$  into  $\lambda_c^* = \frac{C^*}{C_0 + \varepsilon_1 C^*}$  and  $\lambda_m^* = \frac{M^*}{M_0 + \varepsilon_2 M^*}$  and solve simultaneously, we obtain

$$\lambda_m^* = \frac{\mu_c \mu_s q C_0 \lambda_c^*}{\beta_s \alpha_s (D \Lambda_s - \mu_c q C_0 \lambda_c^* - D \Lambda_s \varepsilon_1 \lambda_c^*)}$$

with  $\lambda_c^*$  as solution of the polynomial

$$\lambda_c^*(X\lambda_c^{*2} + Y\lambda_c^* + Z) = 0, \tag{20}$$

where

$$\begin{aligned} X &= Ah\Lambda_h\beta_a\beta_h\alpha_h\alpha_a(k\sigma + g)[\mu_c\mu_sqC_0\varepsilon_1 + \beta_s\alpha_s\mu_cqC_0 \\ &\quad + \beta_s\alpha_sD\Lambda_s\varepsilon_1] + \beta_a\beta_h\alpha_h\alpha_ag(\mu_h + k) \\ &\quad \times [\mu_c\mu_sqC_0(\mu_mhM_0 + \varepsilon_2B\Lambda_a) \\ &\quad + \beta_s\alpha_sB\Lambda_a(D\Lambda_s\varepsilon_1 + \mu_cqC_0)], \\ Y &= (g + k\sigma)\beta_h\alpha_hAh\Lambda_h[\Lambda_s\beta_s\alpha_sD(\varepsilon_1\mu_a - \beta_a\alpha_a) \\ &\quad + \mu_cqC_0\mu_a(\beta_s\alpha_s + \mu_s\varepsilon_2)] + (\mu_h + k)\beta_h\alpha_hg \\ &\quad \times (\mu_c\mu_sqC_0\mu_mhM_0\mu_a - \beta_s\alpha_sBD\Lambda_a\Lambda_s\beta_a\alpha_a) \\ &\quad + \mu_c\mu_sqfgC_0\beta_a\alpha_a\mu_h(\mu_mhM_0 + \varepsilon_2B\Lambda_a) \\ &\quad + \beta_s\alpha_sBfg\Lambda_a\Lambda_s\beta_a\beta_h\alpha_h\alpha_a \\ &\quad + \mu_c\mu_sqfgC_0\beta_a\alpha_a\mu_h(\mu_mhM_0 + \varepsilon_2B\Lambda_a) \\ &\quad + \beta_s\alpha_sBfg\Lambda_a\beta_a\alpha_a\mu_h \\ &\quad \times (D\Lambda_s\varepsilon_1 + \mu_cqC_0), \\ Z &= \mu_c\mu_sqC_0\mu_mfghM_0\mu_a\mu_h(1 - R_0^2). \end{aligned} \tag{21}$$

Solving (20) for  $\lambda_c^*$ , it can be seen that one of the solutions is  $\lambda_c^* = 0$  which represents the disease-free equilibrium state, while the other solution of the polynomial (20) is the endemic equilibrium state of the model system (4) that is given by

$$\lambda_c^* = \frac{-Y + \sqrt{Y^2 - 4XZ}}{2X},$$

and it is positive whenever  $R_0 > 1$ . We further use Descartes's rule of signs to discuss the existence of possible positive roots of Equation (20) especially when  $R_0 < 1$ . The results are summarized in Table 1. Hence, the following theorem.

**Theorem 3.3.** System (4) has the following endemic equilibrium:

- If  $R_0 > 1$ , then the system has one positive unique endemic equilibrium state.
- If  $R_0 < 1$ , then the system has zero or two endemic equilibria.

In case (4), there is possibility of backward bifurcation of the model system (4) as it may contain endemic equilibrium with

TABLE 1 No. of possible positive real roots for (20).

Cases	X	Y	Z	$R_0$	No. of possible positive real roots
1	+	+	-	$R_0 > 1$	1
2	+	-	-	$R_0 > 1$	1
3	+	+	+	$R_0 < 1$	0
4	+	-	+	$R_0 < 1$	0, 2

DFE when  $R_0 < 1$ . This is further determine by carrying out a bifurcation analysis in the following subsection.

### 3.6. Bifurcation analysis

The stability of endemic equilibrium state is established using Center Manifold Theory by Chavez et al. [26]. The theory shows the direction of bifurcation, whether it is a forward or backward bifurcation. A forward bifurcation indicates that the disease-free and endemic equilibrium states are locally asymptotically stable if  $R_0 < 1$  and  $R_0 > 1$ , respectively, which implies the existence of global stability of the equilibrium states. A backward bifurcation means there is a co-existence of disease-free and endemic equilibrium when  $R_0 < 1$ . Therefore, we apply the theory in Chavez et al. [26] by re-writing the state variables as follows

$$\begin{aligned} S_h &= x_1, I_A = x_2, I_{ch} = x_3, M = x_4, S_c = x_5, I_c = x_6, \\ S_s &= x_7, I_s = x_8, C = x_9 \end{aligned}$$

such that the model system (4) can be rewritten as

$$\frac{dX}{dt} = F(X)$$

where  $F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8, f_9)^T$  is the right hand side of model system (4).

Let the bifurcation parameter be  $\beta_s = \beta_s^*$  at  $R_0 = 1$  so that the Jacobian matrix,  $J(E_0)$ , of (15) will have a simple zero eigenvalue and negative real eigenvalues. The left and right eigenvectors associated with a simple zero eigenvalue of the Jacobian matrix (15) are  $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9)$  and  $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9)$ , respectively, where

$$\begin{aligned} w_1 &= -\frac{(\mu_h + k)}{\mu_h}w_2, & w_3 &= \frac{k}{g}w_2, \\ w_4 &= -\frac{\beta_a\alpha_aS_a^0f}{\mu_a\beta_h\alpha_hS_h^0}w_2, & w_5 &= \frac{f\beta_a\alpha_aS_a^0}{h\beta_h\alpha_hS_h^0}w_2, \end{aligned}$$

$$\begin{aligned}
 w_6 &= -\frac{qfC_0\mu_c}{\beta_h\alpha_h\mu_s D S_h^0} w_2, & w_7 &= \frac{fC_0\mu_c}{\beta_h\alpha_h S_h^0 D} w_2, \\
 w_8 &= \frac{qM_0fC_0\mu_c}{\beta_h\beta_s\alpha_h\alpha_s D S_h^0 S_s^0} w_2, & w_9 &= \frac{fC_0}{\beta_h\alpha_h S_h^0} w_2, \\
 v_1 &= v_4 = v_6 = 0, & v_3 &= \frac{\sigma f}{(k\sigma + g)} v_2, \\
 v_5 &= \frac{Bfg}{hA(k\sigma + g)} v_2, & v_7 &= \frac{D}{q} v_9, & v_8 &= \frac{fg}{A(k\sigma + g)} v_2, \\
 v_9 &= \frac{\beta_h\alpha_h S_h^0 hA(k\sigma + g) + Bfg\beta_a\alpha_a S_a^0}{C_0 hA(k\sigma + g)} v_2.
 \end{aligned} \tag{22}$$

We state and prove the following theorem

**Theorem 3.4.** *Model system (4) undergoes a forward bifurcation at  $R_0 = 1$ .*

*Proof.* To determine the direction of the bifurcation, Theorem 4.1 in Castillo-Chavez and Song [27] is used to compute the bifurcation coefficients,  $m$  and  $n$ , of model system (4). The non-zero associated partial derivatives of  $F$  at the disease-free equilibrium,  $E_0$ , are

$$\begin{aligned}
 \frac{\partial^2 f_2(E_0)}{\partial x_1 \partial x_9} &= \frac{\beta_h\alpha_h}{C_0}, & \frac{\partial^2 f_2(E_0)}{\partial x_9^2} &= -\frac{2\beta_h\alpha_h\epsilon_1 S_h^0}{C_0^2}, \\
 \frac{\partial^2 f_5(E_0)}{\partial x_4 \partial x_9} &= \frac{\beta_a\alpha_a}{C_0}, & \frac{\partial^2 f_5(E_0)}{\partial x_9^2} &= -\frac{2\beta_a\alpha_a\epsilon_1 S_a^0}{C_0^2}, \\
 \frac{\partial^2 f_7(E_0)}{\partial x_6 \partial x_8} &= \frac{\beta_s\alpha_s}{M_0}, & \frac{\partial^2 f_7(E_0)}{\partial x_8^2} &= -\frac{2\beta_s\alpha_s\epsilon_2 S_s^0}{M_0^2}, \\
 \frac{\partial^2 f_7}{\partial \beta_s \partial x_8} &= \frac{\alpha_s S_s^0}{M_0}.
 \end{aligned}$$

Now applying the concept of Castillo-Chavez and Song [27], the value of  $m$  gives

$$\begin{aligned}
 m &= v_2 \left[ w_1 w_9 \frac{\partial^2 f_2(E_0)}{\partial x_1 \partial x_9} + w_9^2 \frac{\partial^2 f_2(E_0)}{\partial x_9^2} \right] \\
 &+ v_5 \left[ w_4 w_9 \frac{\partial^2 f_5(E_0)}{\partial x_4 \partial x_9} + w_9^2 \frac{\partial^2 f_5(E_0)}{\partial x_9^2} \right] \\
 &+ v_7 \left[ w_6 w_8 \frac{\partial^2 f_7(E_0)}{\partial x_6 \partial x_8} + w_8^2 \frac{\partial^2 f_7(E_0)}{\partial x_8^2} \right].
 \end{aligned} \tag{23}$$

Upon substituting the eigenvectors and partial derivatives, we obtain

$$m = -v_2 w_2^2 G \tag{24}$$

where

$$\begin{aligned}
 G &= \frac{(\mu_h + k)f}{\mu_h} + \frac{2\epsilon_1 f^2}{\beta_h\alpha_h S_h^0} \\
 &+ \frac{Bfg}{hA(k\sigma + g)} \left[ \frac{\beta_a^2 \alpha_a^2 f^2 S_a^0}{\mu_h \beta_h^2 \alpha_h^2 S_h^0} + \frac{2\beta_a \alpha_a \epsilon_1 f^2 S_a^0}{\beta_h^2 \alpha_h^2 S_h^0} \right] \\
 &+ \left[ \frac{\beta_h \alpha_h S_h^0 hA(k\sigma + g) + Bfg\beta_a \alpha_a S_a^0}{hA(k\sigma + g)} \right]
 \end{aligned}$$

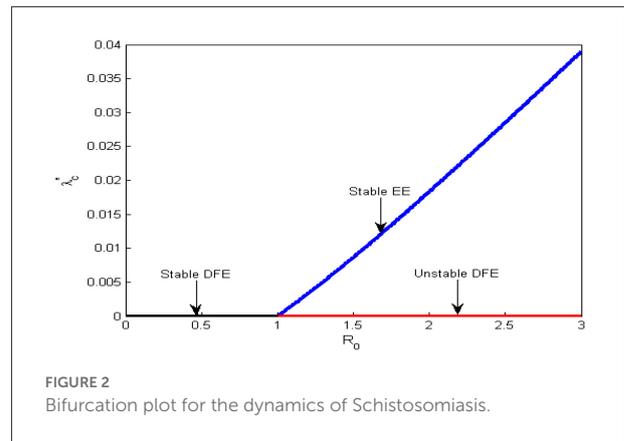


FIGURE 2 Bifurcation plot for the dynamics of Schistosomiasis.

$$\left[ \frac{qC_0 f^2 \mu_c^2 M_0}{\mu_s \beta_h^2 \alpha_h^2 f S_h^0} + \frac{2\epsilon_2 f \mu_c}{M_0} \right]. \tag{25}$$

Also for  $n$ , we have

$$\begin{aligned}
 n &= v_7 w_8 \frac{\partial^2 f_7(E_0)}{\partial \beta_s \partial x_8} \\
 &= \frac{f \mu_c}{\beta_h \beta_s \alpha_h \alpha_s S_s^0} \left[ \frac{\beta_h \alpha_h hA(k\sigma + g) S_h^0 + Bfg\beta_a \alpha_a S_a^0}{hA(k\sigma + g)} \right] v_2 w_2 > 0.
 \end{aligned} \tag{26}$$

A forward bifurcation exists since  $m < 0$  in (24) and  $n > 0$  in (26). Therefore, a forward bifurcation exists for the model (4) at  $R_0 = 1$ . This completes the proof. □

With the existence of forward bifurcation, a unique endemic equilibrium,  $E_1$ , exists and is locally asymptotically stable whenever  $R_0 > 1$  and unstable when  $R_0 < 1$ . Hence, there is possibility of global stability of the equilibrium states. A forward bifurcation plot is shown in Figure 2.

### 3.7. Global stability of the model

In this subsection, the global behavior of the disease-free and endemic equilibria of the system (4) is established by constructing a suitable Lyapunov functions and applying LaSalle’s invariance principle.

#### 3.7.1. Global stability of disease-free equilibrium, $E_0$

**Theorem 3.5.** *The disease-free equilibrium,  $E_0$ , of the schistosomiasis model system (4) is globally asymptotically stable when  $R_0 < 1$  otherwise unstable.*

*Proof.* Constructing a Lyapunov function using a matrix-theoretic method by [28] gives

$$L = \frac{A(k\sigma + g)}{\mu_m f g} I_A + \frac{A\sigma}{g\mu_m} I_{ch} + \frac{B}{h\mu_m} I_a + \frac{D}{q\mu_c} I_s + \frac{1}{\mu_m} M + \frac{1}{\mu_c} C \tag{27}$$

where the letters,  $A$ ,  $B$ , and  $D$  are defined in (12).

Taking the derivative of Equation (27) along the trajectory yields

$$\begin{aligned} L' = & \frac{A(k\sigma + g)}{\mu_m f g} \left( \frac{\beta_h \alpha_h C S_h}{C_0 + \varepsilon_1 C} - f I_A \right) + \frac{A\sigma}{g\mu_m} (k I_A - g I_{ch}) \\ & + \frac{B}{h\mu_m} \left( \frac{\beta_a \alpha_a C S_a}{C_0 + \varepsilon_1 C} - h I_a \right) \\ & + \frac{D}{q\mu_c} \left( \frac{\beta_s \alpha_s S_s M}{M_0 + \varepsilon_2 M} - q I_s \right) \\ & + \frac{1}{\mu_m} (A I_A + A\sigma I_{ch} + B I_a - \mu_m M) + \frac{1}{\mu_c} (D I_s - \mu_c C). \end{aligned} \tag{28}$$

Expanding Equation (28) and simplifying in terms of reproduction numbers ( $R_A, R_{ch}, R_m, R_s$ ) gives

$$\begin{aligned} L' = & (R_A + R_{ch} + R_m - 1)C + (R_s - 1)M \\ & - \frac{\beta_h \alpha_h A(k\sigma + g)}{\mu_m f g} \left( \frac{(S_h^0 - S_h)C_0 + \varepsilon_1 S_h^0 C}{C_0(C_0 + \varepsilon_1 C)} \right) \\ & - \frac{B\beta_a \alpha_a}{h\mu_m} \left( \frac{(S_a^0 - S_a)C_0 + \varepsilon_1 S_a^0 C}{C_0(C_0 + \varepsilon_1 C)} \right) C \\ & - \frac{D\beta_s \alpha_s}{q\mu_c} \left( \frac{(S_s^0 - S_s)M_0 + \varepsilon_2 S_s^0 M}{M_0(M_0 + \varepsilon_2 M)} \right) M. \end{aligned} \tag{29}$$

With  $S_h^0 \geq S_h, S_a^0 \geq S_a, S_s^0 \geq S_s$ , we have

$$L' \leq (R_A + R_{ch} + R_m - 1)C + (R_s - 1)M.$$

Since  $R_A + R_{ch} + R_m \leq 1, R_s \leq 1$  from the definition of the reproduction number in (13) and (14), this yields  $L' \leq 0$  if  $R_0 < 1$ . Hence by LaSalle’s invariance principle, the disease-free equilibrium is globally asymptotically stable if  $R_0 \leq 1$ . This completes the proof.  $\square$

### 3.7.2. Global stability of endemic equilibrium, $E_1$

To prove the stability of the endemic equilibrium ( $E_1$ ) we state and prove the following theorem

**Theorem 3.6.** *The endemic equilibrium,  $E_1$ , of the schistosomiasis model system (4) is globally asymptotically stable whenever  $R_0 > 1$  and  $\psi = 0$ .*

TABLE 3 PRCC values and significance.

Symbol	PRCC	P-value	Keep?
$\beta_s$	0.513775674	0.0000	TRUE
$\beta_h$	0.481893775	0.0000	TRUE
$\beta_a$	0.024026121	0.4503	FALSE
$\gamma_a$	0.001059474	0.9734	FALSE
$\gamma_h$	0.532874887	0.0000	TRUE
$\gamma_s$	0.513588913	0.0000	TRUE
$\mu_c$	-0.526427244	0.0000	TRUE
$\mu_m$	-0.516304961	0.0000	TRUE
$\mu_s$	-0.776046728	0.0000	TRUE
$\tau_1$	0.433204750	0.0000	TRUE
$\tau_2$	0.444561365	0.0000	TRUE

TABLE 2 Parameter values used for the numerical simulation of model system (4).

Symbol	Parameter value (day <sup>-1</sup> )	References	Symbol	Parameter value (day <sup>-1</sup> )	References
$\beta_h$	0.09753	Kalinda et al. [29]	$\delta_a$	0.0039	[30]
$\beta_a$	0.133	Feng et al. [30]	$\delta_s$	0.0004012	[31]
$\beta_s$	0.001	Assumed	$\sigma$	1.01	[32]
$\Lambda_h$	254	Kanyi et al. [33]	$\gamma_a$	0.00232	[34]
$\Lambda_a$	200	Assumed	$\gamma_h$	6.96	[35]
$\Lambda_s$	3,000	Zhang et al. [10]	$\gamma_s$	2.6	[31, 34]
$k$	0.0262	Abokwara and Madubueze[32]	$\tau_1$	0.00232	[34]
$\mu_h$	0.0000384	Feng et al. [30]	$\tau_2$	0.0026	[34]
$\mu_a$	0.000392	Xiang et al. [36]	$N_E$	300	[31]
$\mu_c$	0.004	Chiyaka and Garira [31]	$\varepsilon_1$	0.2	[31]
$\mu_s$	0.000569	Chiyaka and Garira [31]	$\varepsilon_2$	0.2	[31]
$\mu_m$	0.9	Chiyaka and Garira [31]	$M_0$	$1 \times 10^6$	Assumed
$\delta_h$	0.0039	Gao et al. [37]	$C_0$	$9 \times 10^7$	[31]
$\psi$	0.2546	Ding et al. [38]	$\alpha_h$	0.406	Assumed
$\alpha_a$	0.0406	Assumed	$\alpha_s$	0.615	[34]

*Proof.* The global stability of the endemic equilibrium is proved by constructing the Volterra-Lyapunov function

$$\mathcal{H} = \frac{I_A^* + \sigma I_{ch}^*}{\sigma I_{ch}^*} \left[ S_h - S_h^* - S_h^* \ln \frac{S_h}{S_h^*} \right] + \frac{I_A^* + \sigma I_{ch}^*}{\sigma I_{ch}^*} \left[ I_A - I_A^* - I_A^* \ln \frac{I_A}{I_A^*} \right] + \frac{\beta_h \alpha_h S_h^* \lambda_c^*}{k I_A^*} \times \left[ I_{ch} - I_{ch}^* - I_{ch}^* \ln \frac{I_{ch}}{I_{ch}^*} \right] + \frac{B I_a^* \beta_h \alpha_h S_h^* \lambda_m^*}{\beta_a \alpha_a S_a^* \lambda_c^* A \sigma I_{ch}^*} \left[ S_a - S_a^* - S_a^* \ln \frac{S_a}{S_a^*} \right] + \frac{B I_a^* \beta_h \alpha_h S_h^* \lambda_m^*}{\beta_a \alpha_a S_a^* \lambda_c^* A \sigma I_{ch}^*} \times \left[ I_a - I_a^* - I_a^* \ln \frac{I_a}{I_a^*} \right] + \left[ S_s - S_s^* - S_s^* \ln \frac{S_s}{S_s^*} \right]$$

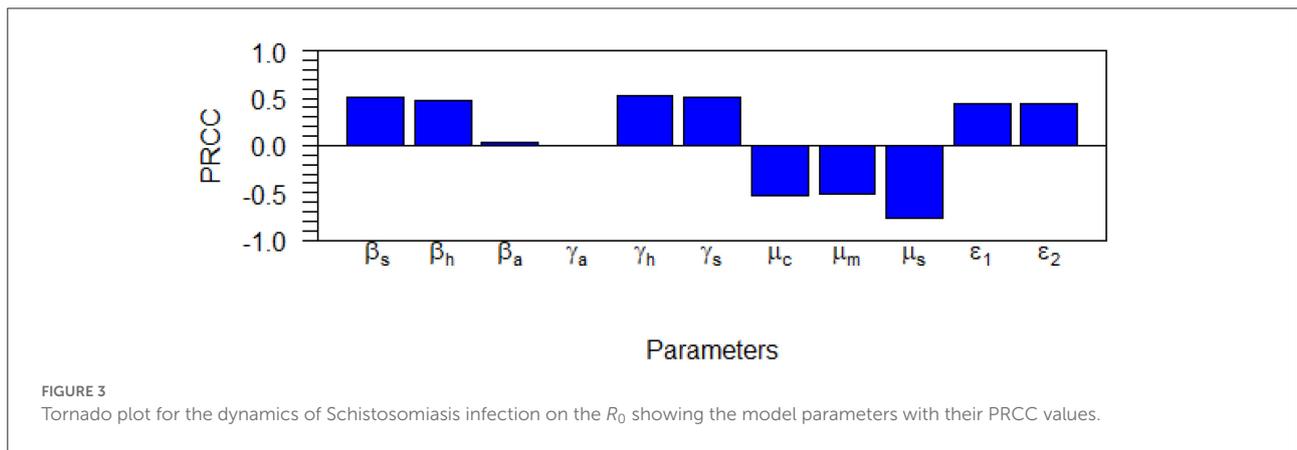


TABLE 4 Pairwise PRCC comparisons (Unadjusted  $P$ -values).

	$\beta_s$	$\beta_h$	$\gamma_h$	$\gamma_s$	$\mu_c$	$\mu_m$	$\mu_s$	$\tau_1$	$\tau_2$
$\beta_s$		0.3463	0.559	0.9955	0	0	0	0.02086	0.0457
$\beta_h$			0.1269	0.3492	0	0	0	0.1711	0.2909
$\gamma_h$				0.5552	0	0	0	0.003792	0.009809
$\gamma_s$					0	0	0	0.02117	0.04632
$\mu_c$						0.7575	0	0	0
$\mu_m$							0	0	0
$\mu_s$								0	0
$\tau_1$									0.7547
$\tau_2$									

TABLE 5 Pairwise PRCC comparisons (FDR adjusted  $P$ -values).

	$\beta_s$	$\beta_h$	$\gamma_h$	$\gamma_s$	$\mu_c$	$\mu_m$	$\mu_s$	$\tau_1$	$\tau_2$
$\beta_s$		0.4055	0.6098	0.9955	0	0	0	0.03176	0.06414
$\beta_h$			0.1692	0.4055	0	0	0	0.22	0.3611
$\gamma_h$				0.6098	0	0	0	0.006501	0.01605
$\gamma_s$					0	0	0	0.03176	0.06414
$\mu_c$						0.7791	0	0	0
$\mu_m$							0	0	0
$\mu_s$								0	0
$\tau_1$									0.7791
$\tau_2$									

$$\begin{aligned}
 & + \left[ I_s - I_s^* - I_s^* \ln \frac{I_s}{I_s^*} \right] + \frac{\beta_h \alpha_h S_h^* \lambda_c^*}{A \sigma I_{ch}^*} \\
 & \times \left[ M - M^* - M^* \ln \frac{M}{M^*} \right] \\
 & + \frac{\beta_s \alpha_s \lambda_m^* S_s^*}{D I_s^*} \left[ C - C^* - C^* \ln \frac{C}{C^*} \right], \tag{30}
 \end{aligned}$$

where  $\lambda_c^*$ ,  $\lambda_m^*$ ,  $A$ ,  $B$ , and  $D$  are defined in (12) and (18).

Taking the time derivative of  $\mathcal{H}$  along the solutions of the schistosomiasis model Equation (4) yields

$$\begin{aligned}
 \mathcal{H}' = & \frac{I_A^* + \sigma I_{ch}^*}{\sigma I_{ch}^*} \left[ 1 - \frac{S_h^*}{S_h} \right] (\Lambda_h - \beta_h \alpha_h S_h \lambda_c - \mu_h S_h + \psi I_A) \\
 & + \frac{I_A^* + \sigma I_{ch}^*}{\sigma I_{ch}^*} \left[ 1 - \frac{I_A^*}{I_A} \right] (\beta_h \alpha_h S_h \lambda_c - f I_A) \\
 & + \frac{\beta_h \alpha_h S_h^* \lambda_c^*}{k I_A^*} \left[ 1 - \frac{I_{ch}^*}{I_{ch}} \right] (k I_A - g I_{ch}) \\
 & + \frac{B I_a^* \beta_h \alpha_h S_h^* \lambda_m^*}{\beta_a \alpha_a S_a^* \lambda_c^* A \sigma I_{ch}^*} \left[ 1 - \frac{S_a^*}{S_a} \right] (\Lambda_a - \beta_a \alpha_a \lambda_c S_a - \mu_a S_a) \\
 & + \frac{B I_a^* \beta_h \alpha_h S_h^* \lambda_m^*}{\beta_a \alpha_a S_a^* \lambda_c^* A \sigma I_{ch}^*} \left[ 1 - \frac{I_a^*}{I_a} \right] (\beta_a \alpha_a \lambda_c S_a - h I_a) \\
 & + \left[ 1 - \frac{S_s^*}{S_s} \right] (\Lambda_s - \beta_s \alpha_s \lambda_m S_s - \mu_s S_s) + \left[ 1 - \frac{I_s^*}{I_s} \right] \\
 & \times (\beta_s \alpha_s \lambda_m S_s - q I_s) \\
 & + \frac{\beta_h \alpha_h S_h^* \lambda_c^*}{A \sigma I_{ch}^*} \left[ 1 - \frac{M^*}{M} \right] (A I_A + A \sigma I_{ch}) \\
 & + B I_a - \mu_m M + \frac{\beta_s \alpha_s \lambda_m^* S_s^*}{D I_s^*} \\
 & \times \left[ 1 - \frac{C^*}{C} \right] (D I_s - \mu_c C). \tag{31}
 \end{aligned}$$

At endemic equilibrium state, we have

$$\Lambda_h = \beta_h \alpha_h S_h^* \lambda_c^* + \mu_h S_h^* - \psi I_A^*, \quad f = \frac{\beta_h \alpha_h S_h^* \lambda_c^*}{I_A^*},$$

$$\begin{aligned}
 g = & \frac{k I_A^*}{I_{ch}^*}, \quad \Lambda_c = \beta_a \alpha_a \lambda_c^* S_a^* + \mu_a S_a^*, \\
 h = & \frac{\beta_a \alpha_a \lambda_c^* S_a^*}{I_a^*}, \quad \Lambda_s = \beta_s \alpha_s \lambda_m^* S_s^* + \mu_s S_s^*, \\
 q = & \frac{\beta_s \alpha_s \lambda_m^* S_s^*}{I_s^*}, \quad \mu_m = \frac{A I_A^*}{M^*} + \frac{A \sigma I_{ch}^*}{M^*} + \frac{B I_a^*}{M^*}, \\
 \mu_c = & \frac{D I_s^*}{C^*}. \tag{32}
 \end{aligned}$$

Substituting Equation (32) into (31) and expanding and simplifying gives

$$\begin{aligned}
 \mathcal{H}' = & \frac{\mu_h S_h^* (I_A^* + \sigma I_{ch}^*)}{\sigma I_{ch}^*} \left[ 2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*} \right] \\
 & + \frac{B I_a^* \beta_h \alpha_h \lambda_m^* S_h^* \mu_a}{\beta_a \alpha_a A \sigma \lambda_c^* I_{ch}^*} \left[ 2 - \frac{S_a^*}{S_a} - \frac{S_a}{S_a^*} \right] \\
 & + \mu_s S_s^* \left[ 2 - \frac{S_s^*}{S_s} - \frac{S_s}{S_s^*} \right] \\
 & + \psi I_A^* \left( \frac{I_A^* + \sigma I_{ch}^*}{\sigma I_{ch}^*} \right) \left[ \frac{S_h^*}{S_h} + \frac{I_A}{I_A^*} - 1 - \frac{I_A}{I_A^*} \frac{S_h^*}{S_h} \right] \\
 & + \frac{\beta_h \alpha_h S_h^* \lambda_c^* I_A^*}{\sigma I_{ch}^*} \left[ 3 + \frac{\lambda_c}{\lambda_c^*} - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*} \frac{\lambda_c}{\lambda_c^*} \frac{I_A^*}{I_A} - \frac{M}{M^*} \right. \\
 & \left. - \frac{I_A}{I_A^*} \frac{M^*}{M} \right] + \beta_h \alpha_h S_h^* \lambda_c^* \left[ 4 + \frac{\lambda_c}{\lambda_c^*} - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*} \frac{\lambda_c}{\lambda_c^*} \frac{I_A^*}{I_A} \right. \\
 & \left. - \frac{I_A}{I_A^*} \frac{I_{ch}^*}{I_{ch}} - \frac{M}{M^*} - \frac{I_{ch}}{I_{ch}^*} \frac{M^*}{M} \right] + \frac{B I_a^* \beta_h \alpha_h \lambda_m^* S_h^*}{A \sigma I_{ch}^*} \\
 & \times \left[ 3 + \frac{\lambda_c}{\lambda_c^*} - \frac{S_a^*}{S_a} - \frac{S_a}{S_a^*} \frac{\lambda_c}{\lambda_c^*} \frac{I_a^*}{I_a} - \frac{M}{M^*} - \frac{I_a}{I_a^*} \frac{M^*}{M} \right] \\
 & + \beta_s \alpha_s \lambda_m^* S_s^* \left[ 3 + \frac{\lambda_m}{\lambda_m^*} - \frac{S_s^*}{S_s} - \frac{S_s}{S_s^*} \frac{\lambda_m}{\lambda_m^*} \frac{I_s^*}{I_s} - \frac{C}{C^*} \right. \\
 & \left. - \frac{C^*}{C} \frac{I_s}{I_s^*} \right]. \tag{33}
 \end{aligned}$$

TABLE 6 Parameters different after FDR adjustment.

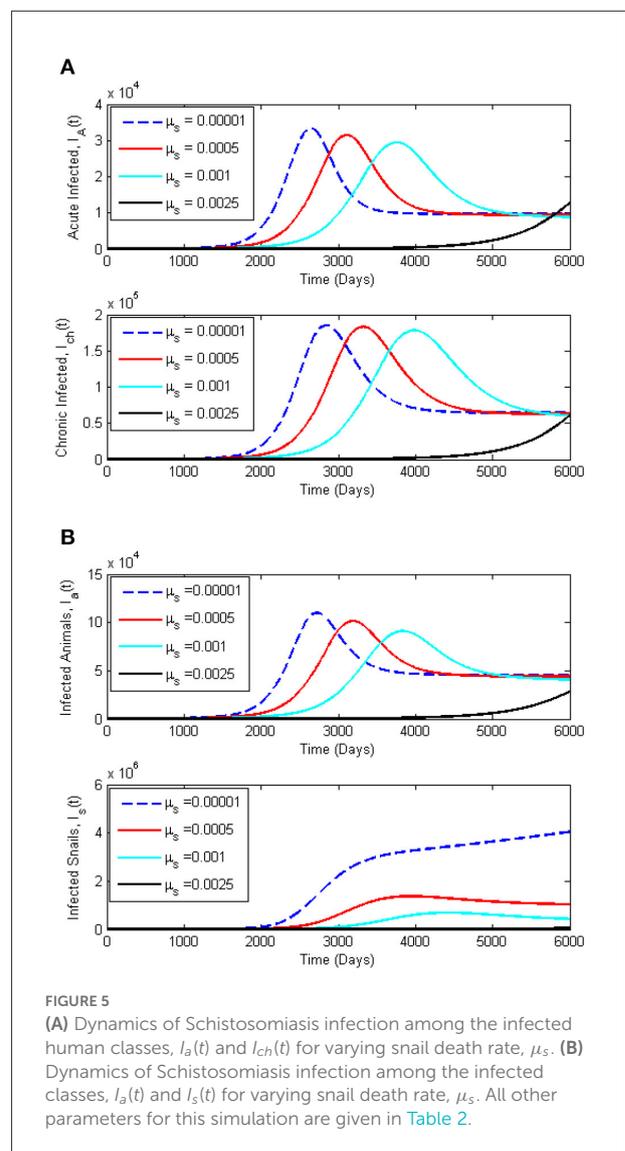
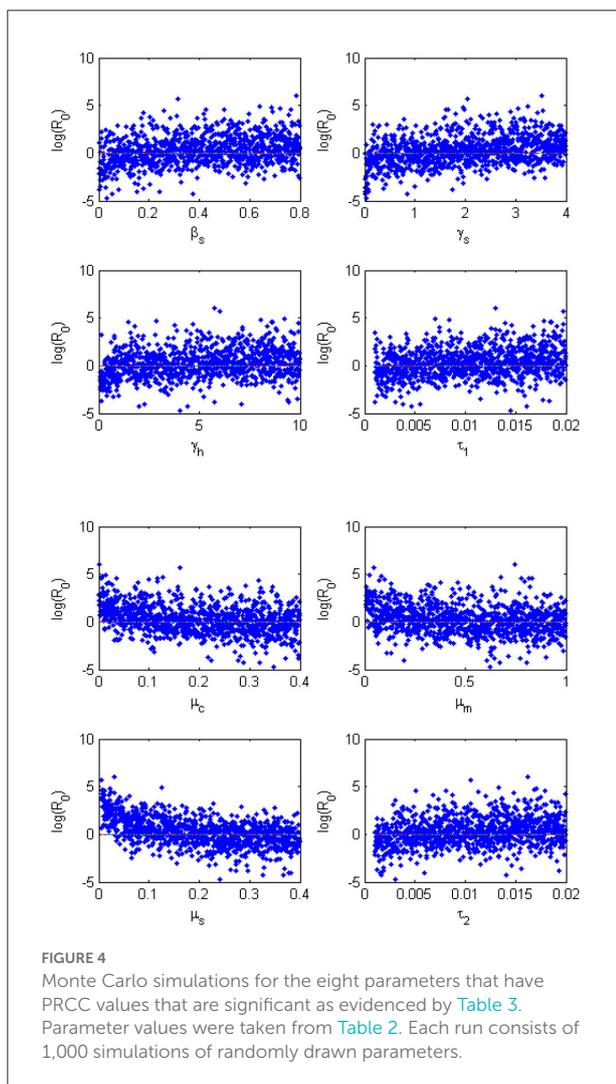
	$\beta_s$	$\beta_h$	$\gamma_h$	$\gamma_s$	$\mu_c$	$\mu_m$	$\mu_s$	$\tau_1$	$\tau_2$
$\beta_s$		FALSE	FALSE	FALSE	TRUE	TRUE	TRUE	TRUE	FALSE
$\beta_h$			FALSE	FALSE	TRUE	TRUE	TRUE	FALSE	FALSE
$\gamma_h$				FALSE	TRUE	TRUE	TRUE	TRUE	TRUE
$\gamma_s$					TRUE	TRUE	TRUE	TRUE	FALSE
$\mu_c$						FALSE	TRUE	TRUE	TRUE
$\mu_m$							TRUE	TRUE	TRUE
$\mu_s$								TRUE	TRUE
$\tau_1$									FALSE
$\tau_2$									

Using the assumption that  $\psi = 0$  and  $\frac{\lambda_c}{\lambda_c^*} \frac{\lambda_m}{\lambda_m^*} \leq 1$ , we have by Arithmetic-Geometric Theorem that

$$\begin{aligned}
 &2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*} \leq 0, 2 - \frac{S_a^*}{S_a} - \frac{S_a}{S_a^*} \leq 0, 2 - \frac{S_s^*}{S_s} - \frac{S_s}{S_s^*} \leq 0, \\
 &3 + \frac{\lambda_c}{\lambda_c^*} - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*} \frac{\lambda_c}{\lambda_c^*} \frac{I_A^*}{I_A} - \frac{M}{M^*} - \frac{I_A}{I_A^*} \frac{M^*}{M} \leq 4 - \frac{S_h^*}{S_h} \\
 &\quad - \frac{S_h}{S_h^*} \frac{I_A^*}{I_A} - \frac{M}{M^*} - \frac{I_A}{I_A^*} \frac{M^*}{M} \leq 0, \\
 &4 + \frac{\lambda_c}{\lambda_c^*} - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*} \frac{\lambda_c}{\lambda_c^*} \frac{I_A^*}{I_A} - \frac{I_A}{I_A^*} \frac{I_{ch}^*}{I_{ch}} - \frac{M}{M^*} - \frac{I_{ch}}{I_{ch}^*} \frac{M^*}{M} \leq 5 \\
 &\quad - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*} \frac{I_A^*}{I_A} - \frac{I_A}{I_A^*} \frac{I_{ch}^*}{I_{ch}} - \frac{M}{M^*} - \frac{I_{ch}}{I_{ch}^*} \frac{M^*}{M} \leq 0, \\
 &3 + \frac{\lambda_c}{\lambda_c^*} - \frac{S_a^*}{S_a} - \frac{S_a}{S_a^*} \frac{\lambda_c}{\lambda_c^*} \frac{I_a^*}{I_a} - \frac{M}{M^*} - \frac{I_a}{I_a^*} \frac{M^*}{M} \leq 4 - \frac{S_a^*}{S_a} \\
 &\quad - \frac{S_a}{S_a^*} \frac{I_a^*}{I_a} - \frac{M}{M^*} - \frac{I_a}{I_a^*} \frac{M^*}{M} \leq 0,
 \end{aligned}$$

$$\begin{aligned}
 &3 + \frac{\lambda_m}{\lambda_m^*} - \frac{S_s^*}{S_s} - \frac{S_s}{S_s^*} \frac{\lambda_m}{\lambda_m^*} \frac{I_s^*}{I_s} - \frac{C}{C^*} - \frac{C^*}{C} \frac{I_s}{I_s^*} \leq 4 - \frac{S_s^*}{S_s} \\
 &\quad - \frac{S_s}{S_s^*} \frac{I_s^*}{I_s} - \frac{C}{C^*} - \frac{C^*}{C} \frac{I_s}{I_s^*} \leq 0.
 \end{aligned} \tag{34}$$

This implies that  $L' \leq 0$  in the region,  $Z$ . Applying LaSalle’s invariance principle, the endemic equilibrium state of the schistosomiasis model system (4) is globally asymptotically stable in  $Z$  if  $R_0 > 1$  and  $\psi = 0$ . This means that with different initial conditions at  $\psi = 0$ , the global stability of the schistosomiasis model system (4) will always converge to the disease-free and endemic equilibrium states whenever  $R_0 < 1$  and  $R_0 > 1$ , respectively. The numerical simulations are carried out in the next section. □



## 4. Sensitivity analysis

### 4.1. Parameter values

To perform numerical simulations, parameters for the model are mainly sourced from literature, as shown in [Table 2](#). Parameters that are represented by probabilities are assumed to be within the range of  $[0 - 1]$ . Any parameters that are outside the prescribed range are properly sampled from a uniform distribution within ranges defined in [Table 2](#).

### 4.2. Sensitivity analysis results

Sensitivity analysis examines the relative change of the mathematical model output (variable) when the model

parameter(s) are altered or changed. It helps in determining and identifying the model parameters that require special attention. To carry out the sensitivity analysis of the basic reproduction number  $R_0$ , we adopt the Latin Hypercube Sampling (LHS) scheme and Partial Rank Correlation Coefficients (PRCCs) technique used by Blower and Dowlatbadi [39] to compute and identify the biological implication of each model parameter on  $R_0$ . See also the following published articles [40–45] with similar analyses for different epidemic models where the LHS techniques have been applied. We performed 1,000 simulations per run and examined and evaluated the PRCC of the model parameters concerning  $R_0$ . The PRCC values indicate the degree of monotonicity between the parameters of the model and  $R_0$ . Thus, juxtaposing the PRCC values gives us a clear picture on the contribution of each parameter of the model on  $R_0$ . [Table 3](#) and the Tornado plot of [Figure 3](#) give the model parameters with their PRCCs. The parameters with the potential of increasing(decreasing) the value of  $R_0$ , are those with positive(negative) PRCCs. Thereby, increasing parameters,  $\beta_s, \beta_h, \beta_a, \gamma_h, \gamma_a, \gamma_s, \tau_1, \tau_2$ , increases the value of  $R_0$  which in turn increase the spread of schistosomiasis disease within the population (see [Table 2](#) and [Figure 3](#)). On the other hand, parameters  $\mu_c, \mu_m, \mu_s$  with negative PRCCs influence the reduction of the burden of schistosomiasis disease within the population. When these parameters are increased, we observe a reduction in the value of  $R_0$ , hence the endemicity of the Schistosomiasis within the population is also reduced. The natural death rate for the snail population is the most sensitive parameter with respect to  $R_0$  with a PRCC value of 0.776046728. The implication of this is that increasing snail death rate reduces the spread of Schistosomiasis within the population. To examine the significance of the model parameters, we compute the  $p$ -value of PRCC of each model parameter using the Fisher Transformation method as shown in [Table 3](#). [Table 3](#) reveals that the parameters,  $\beta_s, \beta_h, \gamma_h, \gamma_s, \mu_c, \mu_m, \mu_s, \tau_1, \tau_2$  have significant  $p$ -values while the parameters,  $\beta_a, \gamma_a$ , have insignificant  $p$ -values. This implies that the shedding and transmission rates of the other mammals,  $\gamma_a$  and  $\beta_a$ , are non-monotonically related to  $R_0$ , though they can still produce changes in the transmission dynamics of schistosomiasis infection.

The pairwise comparison of the significant parameters of [Table 3](#), whose  $p < 0.05$  is given in [Tables 5, 6](#). The pairwise comparison is carried out to observe the model parameters with notable influence on increasing the burden of the schistosomiasis infection in the population, as well as to establish whether there exists any difference between the processes describing the compared parameters. We presented the outcome of the pairwise PRCC comparison between the unadjusted  $p$ -values ([Table 4](#)) and the false discovery rate (FDR) adjusted  $p$ -values in [Tables 5, 6](#). According to [Table 6](#), whenever the  $p$ -values of the compared pair of significant parameters are  $< 0.05$ , it is interpreted to be significant (TRUE) otherwise it is insignificant (FALSE). It is also important to note from

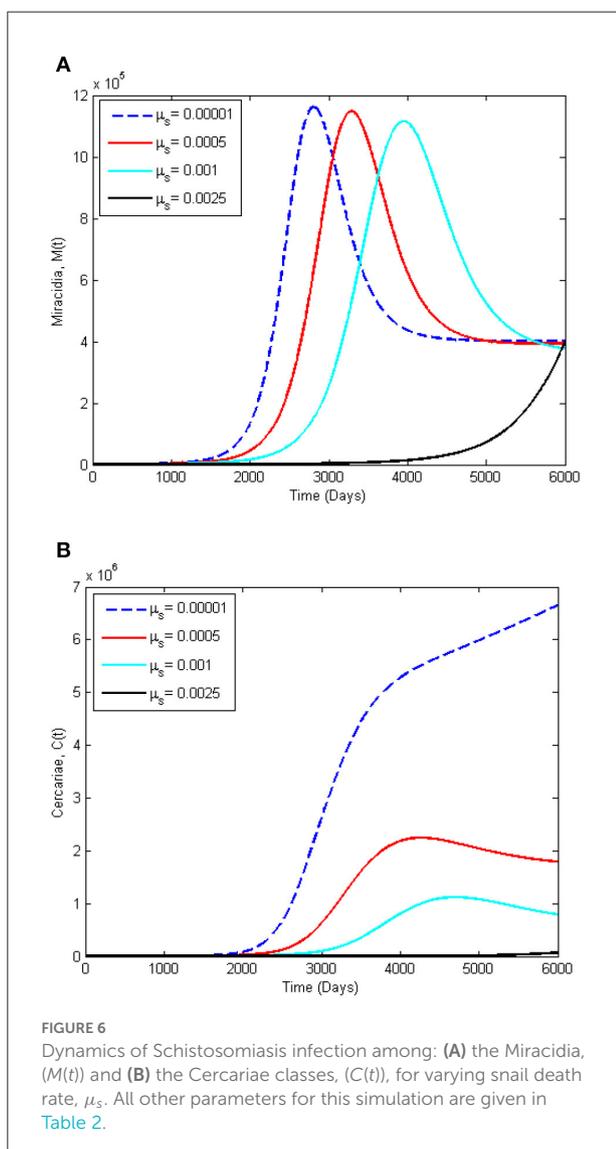
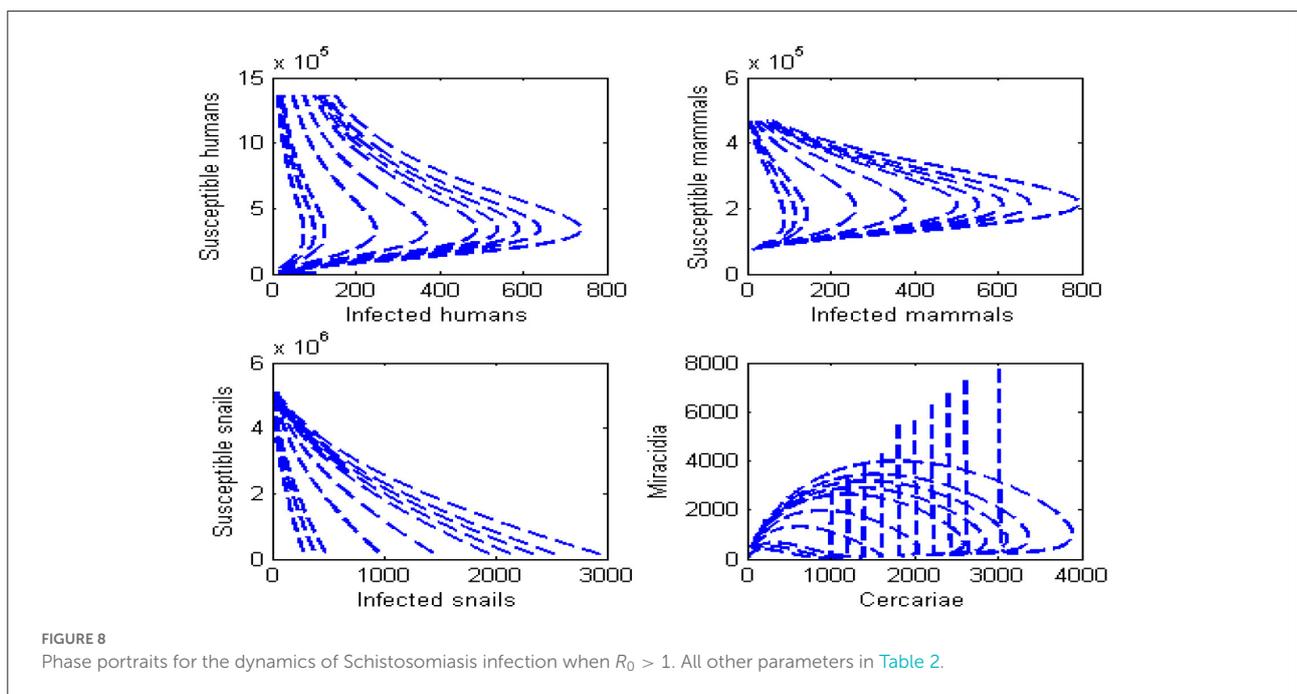
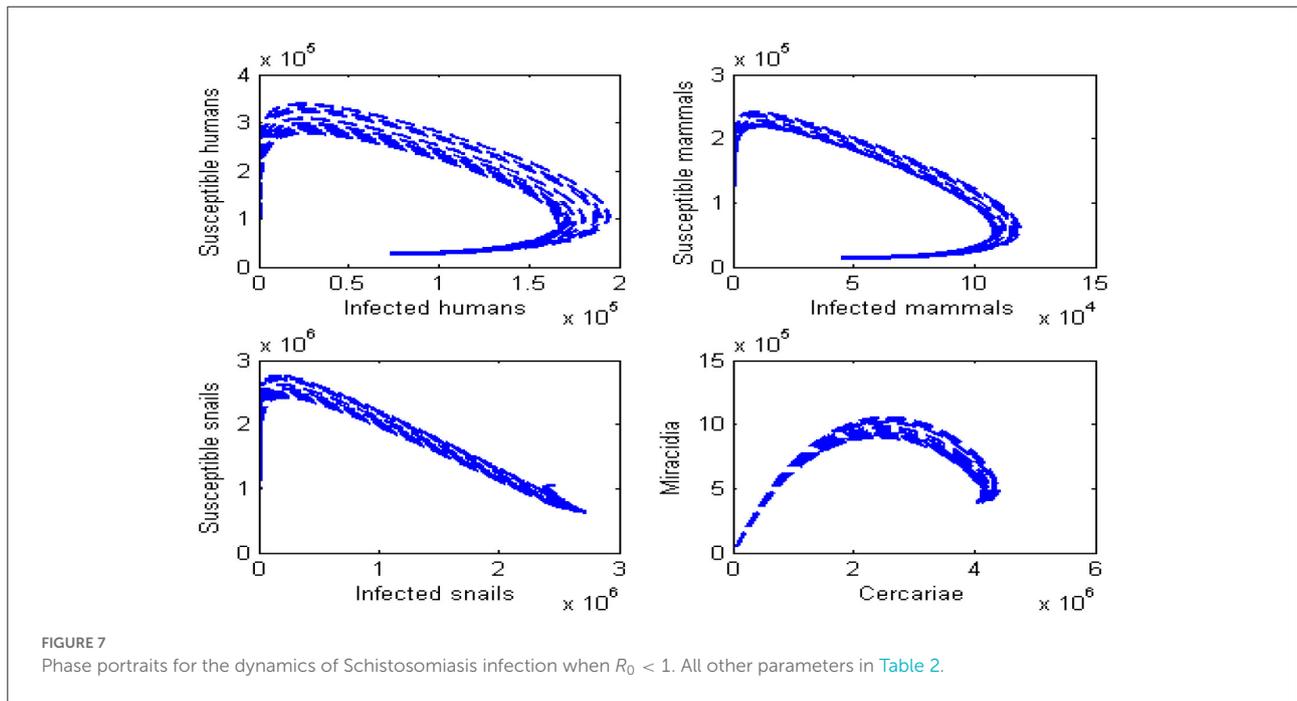


FIGURE 6 Dynamics of Schistosomiasis infection among: (A) the Miracidia,  $M(t)$  and (B) the Cercariae classes,  $C(t)$ , for varying snail death rate,  $\mu_s$ . All other parameters for this simulation are given in [Table 2](#).

Table 6 that the pairs with significantly different (TRUE) are more sensitive parameters.

The effect of the most sensitive parameters on  $R_0$  is presented in Figures 3, 4 as scatter plots to support results in Table 2. These plots show how parameters,  $\beta_s, \gamma_s, \gamma_h, \tau_1, \mu_c, \mu_m, \mu_s, \tau_2$  monotonically increase or decrease  $R_0$ . The most noticeable result is the scatter plot for snail death  $\mu_s$  which is the most sensitive parameter. The

sensitivity result reveals that the spread of schistosomiasis infection will be controlled and eradicated in the population if more snails die. If the Miracidia and Cercariae ( $\mu_m, \mu_c$ ) are cleared within the population, we will observe a reduction in the contact rates of human to Cercariae ( $\beta_h$ ), snail to Miracidia ( $\beta_s$ ), probability of egg developing into a Miracidium ( $\tau_1$ ) and the density of Cercariae in the environment ( $\tau_2$ ).



## 5. Numerical simulation results

### 5.1. Time series trajectories

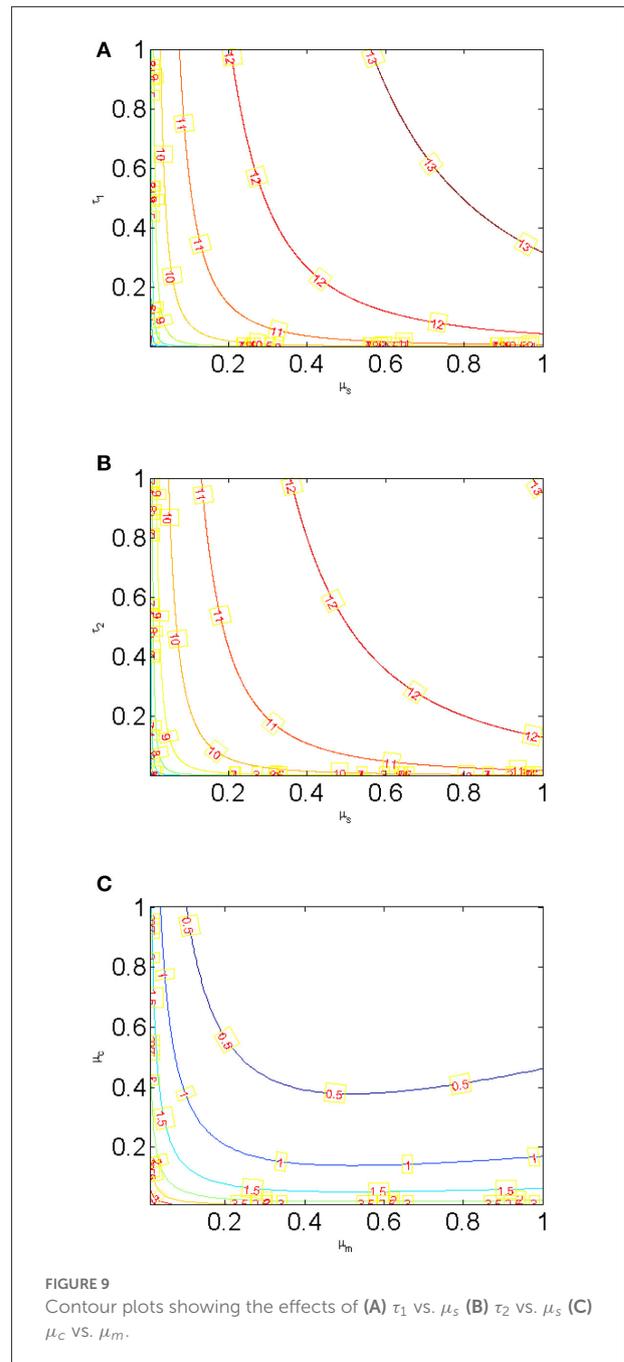
To support the analytical results presented in this paper, we present numerical simulations in the form of time series plots and phase plots on the dynamics of Schistosomiasis. The simulations presented are produced using MATLAB software. Figures 5A,B, present the time series plots for the acute infected class  $I_A(t)$ , chronic infected class,  $I_{ch}(t)$ , infected animals  $I_a(t)$ , and infected snails,  $I_s(t)$ , while varying the snail death rate,  $\mu_s$ . The snail death rate,  $\mu_s$  is considered for simulation as it is the most sensitive parameter. It is observed that when the death rate of the snail population is increasing, the peak of  $I_A(t)$ ,  $I_{ch}(t)$ , and  $I_a(t)$ , shifts to the right in a reduced manner. On the other hand, Figures 6A,B present the dynamics of Miracidia,  $M(t)$  and Cercariae,  $C(t)$ . We observe that an increase in the snail death rates leads to a decrease in the population of the Miracidia. Also, the populations of  $I_s(t)$  and  $C(t)$  reduce drastically as the snail population's death rate increases. This indicates that reducing the snail population reduces the time it will take to infect human and mammal populations, including Cercariae that will be cleared in the population. This eventually reduces the number of Schistosomiasis cases within the population.

### 5.2. Phase portraits

Figures 7, 8 present the phase portraits of the infection sub-populations when  $R_0 > 1$  and  $R_0 < 1$ , respectively. When  $R_0 < 1$ , it can be seen that solutions tend to disease-free equilibrium over time. This means that Schistosomiasis infection can be contained within the population over time provided that  $R_0 < 1$ . On the other hand when  $R_0 > 1$ , the solutions lead to an endemic equilibrium as seen by a decrease in the susceptible population and an increase in the infected population. It is also observed that as Miracidia initially increase, Cercariae increases as well and reaches a peak. Thereafter, Miracidia decreases gradually as Cercariae continues to increase. This means that Schistosomiasis infection will persist within the population whenever  $R_0 > 1$ .

### 5.3. Effects of parameters on $R_0$

Using contour plots, Figures 9A,B depict that  $R_0$  attains a lower value when the probability of eggs developing into Miracidium is minimal and the snail death rate is high as shown by the contour plot and the 3-D representation. Figure 9C shows  $R_0$  vs. varying the natural decay rates of Miracidium, ( $\mu_m$ ) and Cercariae ( $\mu_c$ ). It can be seen that if the decay rates are effectively increased, the reproduction number,  $R_0$



can be reduced to below one, implying that the disease-free equilibrium can be achieved. The implication of Figures 9A–C is that there is a possibility of approaching a disease-free equilibrium if these parameters are controlled accordingly. However, this may be difficult to attain due to the lack of proper control measures that can simultaneously increase the decay rates. Generally, it can be seen that the control of snails is very important in the reduction of the spread of Schistosomiasis within the population. Therefore, measures

need to be put in place so that all breeding areas for snails are identified and dealt with appropriately without harming other aquatic organisms.

## 6. Conclusion

In this paper, a deterministic mathematical model of transmission dynamics of Schistosomiasis disease that incorporates acute and chronic infected humans is formulated. The main objective of the model is to study the impact of snail control on the dynamics of Schistosomiasis. The model analysis comprised of establishing the invariant region, the positivity of solutions, the existence of disease-free and endemic equilibrium, bifurcation analysis and computation of the basic reproduction number,  $R_0$  using the next-generation approach. We proved that whenever  $R_0 < 1$ , the disease-free equilibrium is globally asymptotically stable, which means that the disease will fizzle out within the population over time. The endemic equilibrium was found to be globally asymptotically stable implying that if not controlled, Schistosomiasis will persist within the population. We also carried out a sensitivity analysis of the parameters governing the basic reproduction number,  $R_0$ , using the Latin Hypercube Sampling (LHS) scheme-Partial Rank Correlation Coefficient (PRCC) technique. The results of the sensitivity analysis revealed that the spread of schistosomiasis infection can be controlled and eradicated in the population by increasing the natural death rate of the snail population. It also established that there is need to increase the decay rates of Miracidia and Cercariae within the environment by applying a control measure. Therefore, the results of our study effectively conclude that the most effective method to control the transmission dynamics and spread of schistosomiasis disease is by rapidly killing of infected snails and applying control measures that will clear the parasite, miracidia and cercariae, in the environment.

One limitation of our model is that it was not fitted to real-life data. Instead, our model was parameterized using accurate parameter estimates from existing literature on Schistosomiasis dynamics with inclusive heterogeneity in the disease transmission. Secondly, the assumption of mammals could be made specific by considering only cattle in the modeling framework. To bypass this complexity, we carried out a global sensitivity analysis to vary the parameter values and establish the uncertainty in the model parameters. Notwithstanding this limitation, we maintain that our findings remain valid and

applicable if followed by policymakers to help eradicate the spread of Schistosomiasis.

Future work may look at an optimal control analysis of the model presented in the presence of different control strategies. The model may also be extended to include infection dynamics in the presence of snail age stratification.

## Data availability statement

The original contributions presented in the study are included in the article/supplementary material, further inquiries can be directed to the corresponding author/s.

## Author contributions

CM and IO contributed to conception and model formulation of the study. CM, ZC, IO, CC, and FF contributed in the analysis and write-up of sections of the manuscript. All authors contributed to manuscript revision, read, and approved the final version of the manuscript.

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## Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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