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Control of schistosomiasis by the selective competitive and predatory intervention of intermediate hosts: A mathematical modeling approach

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ABSTRACT

Schistosomiasis, a freshwater-borne neglected tropical disease, disproportionately affects impoverished communities mainly in the tropical regions. Transmission involves humans and intermediate host (IH) snails. This manuscript introduces a mathematical model to probe schistosomiasis dynamics and the role of non-host snail competitors and predators as biological control agents for IH snails. The numerical analyses include investigations into steady-state conditions and reproduction numbers associated with uncontrolled scenarios, as well as scenarios involving non-host snail competitors and/or predators. Sensitivity analysis reveals that increasing snail mortality rates is a key to reducing the IH snail population and control of the transmission. Results show that specific snail competitors and/or predators with strong competition/predation abilities reduce IH snails and the subsequent infectious cercaria populations, reduce the transmission, and possibly eradicate the disease, while those with weaker abilities allow disease persistence. Hence our findings advocate for the effectiveness of snail competitors with suitable competitive pressures and/or predators with appropriate predatory abilities as nature-based solutions for combating schistosomiasis, all while preserving IH snail biodiversity. However, if these strategies are implemented at insignificant levels, IH snails can dominate, and disease persistence may pose challenges. Thus, experimental screening of potential (native) snail competitors and/or predators is crucial to assess the likely behavior of biological agents and determine the optimal biological control measures for IH snails.

1. Introduction

Schistosomiasis, a prevalent disease in tropical regions, is caused by parasites known as *Schistosoma*. These parasitic trematode worms rely on two primary hosts: the humans, within whom it matures into an adult, leading to schistosomiasis infection, and intermediate host (IH) snails, which aid in the early stages of its development. The propagation of this ailment hinges on the interactions between these hosts, intricately connecting the life cycle of the parasite with both human and snail organisms [1,2]. This disease is closely associated with impoverished living conditions resulting from factors such as poverty, inadequate sanitation, and limited access to clean water sources [3]. Schistosomiasis imposes a considerable toll in terms of mortality and morbidity [4]. On a global scale, more than 700 million individuals are at risk of infection, and over 200,000 people lose their lives to it annually, with the highest burden experienced in sub-Saharan Africa, where it accounts for up to 90 % of infections worldwide [5,6].

In terms of disease control, it is important to note that there is currently no vaccine available to prevent schistosomiasis. However, the primary interventions for managing the transmission of this disease at the human population level involve mass drug administration and/or the implementation of WASH (water, sanitation, and hygiene) measures [7]. However, despite these efforts, schistosomiasis frequently re-emerges shortly after interventions, presenting significant challenges at the human level and limited impact in affected regions. In contrast, there is a growing body of evidence suggests that targeting IH snails as a control strategy could effectively manage their populations and curb the spread of schistosomiasis [8–11]. This approach has achieved substantial success in various Asian regions [9]. In support of this approach, the 65th World Health Assembly took a crucial step by passing a resolution

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advocating for the integration of biological controls in the management of IH snails [8]. Within this context, studies have revealed that coexisting with potential competitors and predators of various species plays a crucial role in regulating the distribution of these snails, emerging as a critical ecological factor in the development of diseases [12,13]. Significantly, numerous potential competitors to IH snails have been documented, including species such as *Thiara granifera and Physella acuta* [14]. In the presence of many competitors, snails intensify their feeding behavior, and species out-competition could potentially lead to colonization by non-host invasive species [15]. Previous research has reported instances of displacement and/or colonization of IH snails by non-host snails, as documented by Butler et al. [16], who observed the colonization of IH *Biomphalaria* species by the presence of *Thiara granifera*.

On the contrary, potential predators within the ecosystem encompass a diverse array of species, including crayfish, river prawns, the water bug *Sphaerodema urinator*, various insect species, selected leech species, Sciomyzidae flies in their larval stage, as well as several fish and crustacean species [15–19]. The presence of these snail predators exerts a remarkable influence on the IH snail population, inducing heightened stress and instigating observable behavioral modifications. For instance, IH snails possess the ability to detect the presence of these predators, prompting them to seek refuge. This behavioral shift subsequently leads to a decrease in their feeding rate, thereby leading to consequential alterations in the patterns of disease dissemination [15,20]. For example, the study conducted by Sokolow et al. [10] documented a decrease in human schistosomiasis transmission following the reintroduction of a native river prawn, known to prey on the IH snails. Furthermore, recent research by Mathers et al. [21] has unveiled considerable variations in the handling time and attack rate exhibited by predators when preying upon different gastropod species. These observed variations may lead to shifts in the predation dynamics, affecting the strategies and effectiveness of predators in capturing their prey.

Consequently, the roles played by both snail competitors and predators emerge as key components within the ecological framework. These elements play significant roles in the complex ecological interactions within the system, influencing the dynamics of *Schistosoma* transmission. Nonetheless, the development of mathematical models has significantly enhanced our comprehension of population dynamics and facilitated the evaluation of various control programs. The utilization of mathematical models for assessing the relative effectiveness of different biological interventions and the elimination of schistosomiasis transmission holds paramount importance in informing policy decisions. Several studies, including those conducted by Altizer et al. [22], Chiyaka and Garira [23], Gao et al. [24], and Abokwara and Madubueze [25], have explored the dynamics and control of schistosomiasis at the human population level. Additionally, research has provided valuable insights into temperature control for different aspects of the *Schistosoma* life cycle, as seen in studies conducted by Mangal et al. [26], Kalinda et al. [27] and Tabo et al. [28]. To the best of our knowledge, a few mathematical models have investigated biological control strategies for the management of IH snails and the control of schistosomiasis [29,30], but none have examined the influence of non-host competitor snails and snail predators concerning concurrent disease propagation. In our present model, we formulate ordinary differential equations (ODEs) to represent the principal stages of the *Schistosoma* life cycle. Within this model, we incorporate non-host competitors and predators as integral biological components. Consequently, we utilize this model as a tool to illuminate the control of schistosomiasis and quantify the anticipated impacts of snail competitors and predators in the effective management of IH snail vectors and the control of schistosomiasis.

2. Materials and methods

2.1. Model formulation

The primary stages of the *Schistosoma* spp. life and transmission cycle (Fig 1) served as the basis for our mathematical model. The key stages include the human host, free-living parasites (cercaria and miracidia), and the IH snail. Within the human population, individuals can be classified into two distinct groups: individuals who exhibit susceptibility denoted as $H(t)$, and those who have been infected, denoted as $I(t)$. The infected individuals represented by $I(t)$, expel parasite eggs $E(t)$ through feces or urine, which may either be directly excreted or introduced into freshwater sources. In favorable freshwater environments, the parasite eggs undergo hatching, giving rise to miracidia $\mathrm{M}(t)$, which function as hosts for IH snails. These IH snails can be classified as either susceptible, denoted as $S(t)$, or infected, labeled as $Is(t)$. Infected IH snails, $Is(t)$, release infectious cercaria parasites $C(t)$, which subsequently initiate infection in humans to complete the *Schistosoma* life cycle. Furthermore, the model incorporates biological control agents, denoted as $X(t)$, which represent potential competitor for IH snail, and $Y(t)$, which represent potential predator.

We assume that newborns are not recruited untill a certain age (τ) when they can interact or be washed in water. The IH snail and human host are infected through contact with miracidia and cercariae, respectively, in an infested freshwater environment. The competitor snail is not an infectious host and is not a vector. Snails grow logistically in the absence of the disease. The infected snails do not reproduce as a side effect of miracidia infection and due to infection, their mortality is potentially higher than for susceptible snails. The competitor snail is a superior competitor with the advantage of impacting more strongly on available resources than the IH snails. There are various potential predators with specific capabilities, and the only food available in the environment is the IH snails, especially the infected IH snails. The effort and time required by the predator to handle infected IH snails is less than that required time to handle the susceptible IH snails. Thus, the model equations, which depict the dynamic interactions of schistosomiasis in the presence of biological control agents, are presented in Eqs. (1)-(9):

$$\dot{H}(t) = \Lambda_1 - \frac{\beta_1 HC}{C_o + \varepsilon C} - \upsilon_1 H \tag{1}$$

$$\dot{I}(t) = \frac{\beta_1 HC}{C_o + \varepsilon C} - (\upsilon_1 + \delta_1) I \tag{2}$$

$$\dot{E}(t) = \rho\theta_h I\left(1 - \frac{E}{K}\right) - (\omega_1 + \upsilon_3) E \tag{3}$$

$$\dot{\mathrm{M}}(t) = \omega_1 E - \upsilon_4 \mathrm{M} \tag{4}$$

$$\dot{S}(t) = r_1 S\left(1 - \frac{S + e_1(I_s + X)}{K_1}\right) - \frac{\beta_2 MS}{M_o + \varepsilon M} - f_1(S)Y \tag{5}$$

$$\dot{I}_s(t) = \frac{\beta_2 MS}{M_o + \varepsilon M} - (\upsilon_2 + \delta_2 + e_2(S + X + I_s))I_s - f_2(I_s)Y \tag{6}$$

$$\dot{C}(t) = \omega_2 I_s - \upsilon_5 C \tag{7}$$

$$\dot{X}(t) = r_2 X\left(1 - \frac{X + e_3(S + I_s)}{K_2}\right) \tag{8}$$

$$\dot{Y}(t) = \eta_1 f_1(S)Y + \eta_2 f_2(I_s) - \upsilon_6 Y \tag{9}$$

Here, $\Lambda_1 = \lambda e^{-\upsilon_1 \tau}$ represents human recruitment through birth, which follows an exponentially distributed waiting time function. It has a maximum per capita birth rate of λ, a natural death rate of υ_1, and τ represents the age of first schistosomiasis infection in children. The term $e^{-\upsilon_1 \tau}$ represents the fraction of children waiting to be recruited at time τ,

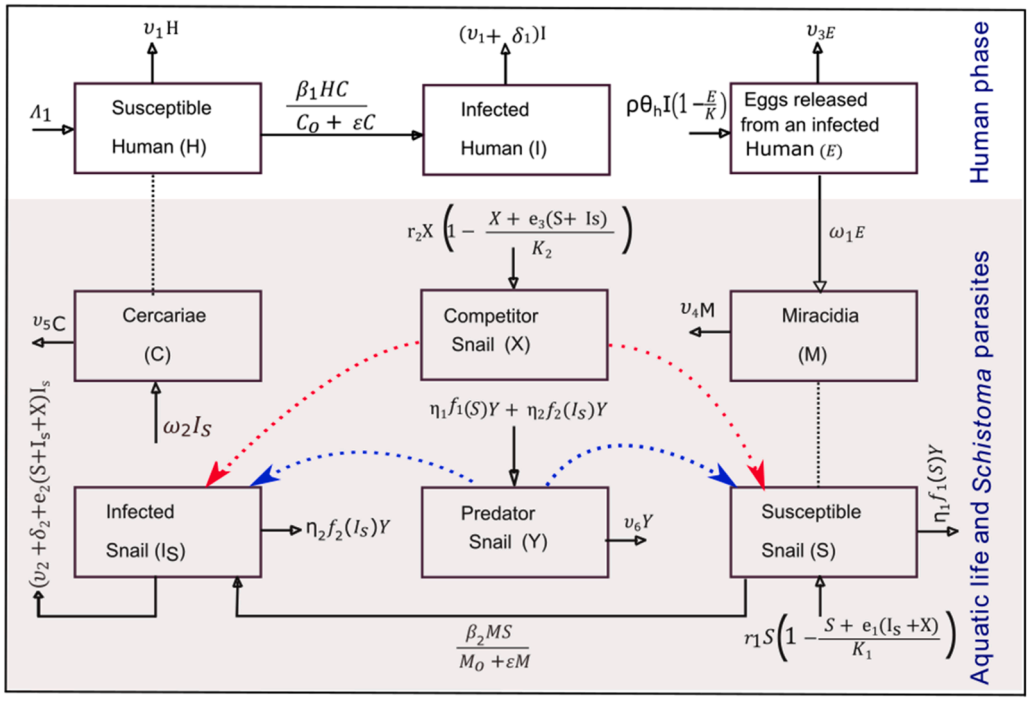

Fig. 1. A schematic description of the schistosomiasis transmission cycle. Interactions between snail competitors and predators with intermediate host snails are shown by the dashed arrows in red and blue, respectively. The parameter descriptions are provided in Table 1. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

with a $\frac{1}{\upsilon_1}$ chance of survival. The non-linear infection incidences in the model are denoted as $\beta_1 HC/(C_o + \varepsilon C)$ for human hosts and $\beta_2 MS/(M_o + \varepsilon M)$ for snail hosts. Here, β_1 and β_2 represent the infection rates of human and snail hosts. ε represents the crowding effect which encapsulates environmental and ecological constraints (temperature, competition, predation, water quality, etc.) that may limit the effective contact rate between hosts (human and snail) and parasites (cercaria and miracidia). C_o and M_o are saturation coefficients for cercariae and miracidia infectivity, respectively, and ensure that the incidence rate increases with C and M, but asymptotically approaches a maximum value. Thus, C_o and M_o represent the carrying capacity of the environment and the biological limits of the humans and snails that can be infected. Furthermore, infected humans excrete an average of ρ stool (volume of urine) per day, containing an average number of parasite eggs θ_h per gram of stool (*S. mansoni*) or per liter of urine per day (*S. haematobium*). Miracidia emerge from parasite eggs at an average rate of N_E miracidia per egg, and ω_1 represents the rate of parasite egg hatching. The populations of IH snails and competitor snails grow intrinsically at rates r_1 and r_2, respectively. Meanwhile, the predator population increases due to conversion factors η_1 and η_2, which represent susceptible snail-to-predator conversion and infected snail-to-predator conversion, respectively. Note that the intrinsic growth of parasite egg, IH snails, and competitor snails populations are constrained by their respective carrying capacity of K, $K_1$and K_2 respectively. Predators feed on host snails with a saturating Holling type II functional response to susceptible snail density, represented by $f_1(S) = \frac{t_1 S}{1+at_1 S}$, and to infected snail density, represented by $f_2(I_s) = \frac{t_2 I_s}{1+bt_2 I_s}$. The parameters (a, b), and (t_1, t_2) are handling times and attack rates of the susceptible and infected IH snails, respectively. Additionally, the cercaria population is shed from infected snails at a rate of ω_2. The snail, parasite egg, miracidia, cercaria, and predator populations naturally die at rates υ_2, υ_3, υ_5, and υ_6, respectively. Disease-related mortality rates for humans and snails are indicated by δ_1 and δ_2.

2.2. Positivity of the solution and biologically invariant region

We show that our model is of epidemiological interest and can be analyzed in a region $\Omega \subset R^6_+$, if all parameters and state variables are non-negative for all future time, $\forall t > 0$ and, given non-negative initial conditions, all solutions remain non-negative and bounded.

Theorem 1. *Let* $\pounds(t) = [H(t), I(t), E(t), M(t), S(t), I_s(t), C(t), X(t), Y(t)]^T \subset \mathscr{R}^9_+$, *for initial conditions* $\pounds(0) \geq 0$, *then the solution set* $\pounds(t)$ *of the model equations Eqs. (1)-(9) is non-negative for* $\forall t > 0$ *and eventually enters the invariant attracting region* $\Omega \subset R^9_+$.

Proof. Consider equation (Eq. (1)) of the model equations Eqs. (1)-(9). It is obvious that $\dot{H}(t) \geq -\left(\frac{\beta_1 C}{C_0+\varepsilon C} - \upsilon_1\right)H$. By integration, we get $H(t) \geq H(0)$. This implies that as t approaches negative infinity, the lower bound of $H(t)$ tends towards zero, i.e., $\lim_{t\to-\infty} Inf(H(t)) \geq 0$. Consequently, the solution H(t) remains non-negative. Similarly, through analogous reasoning, we can show that $I(t) \geq 0$, $E(t) \geq 0$, $M(t) \geq 0$, $S(t) \geq 0$, $I_s(t) \geq 0$, $C(t) \geq 0$, $X(t) \geq 0$, $Y(t) \geq 0$. Thus, all state variables maintain non-negative values. This confirms that the feasible region Ω is positively invariant, since the solution trajectory of the system always remains within this non-negative region over time. This leads to the conclusion that the solution set $\pounds(t)$ of the model equations Eqs. (1)-(9) eventually enters and remains in the attracting invariant region $\Omega \subset R^9_+$.

Theorem 2. *Let the initial conditions* $\pounds(0) \geq 0$, *then all solutions to the set* $\pounds(t)$ *remain bounded for all future time*

Proof. Let's consider the total human population $N_h(t) = H(t) + I(t)$, $\forall t > 0$. The differential equation governing this population is given by: $\frac{dN_h}{dt} = \Lambda_1 - \upsilon_1 N_h - \delta_1 I$. If we have $\frac{dN_h}{dt} \leq \Lambda_1 - \upsilon_1 N_h$, integrating this inequality yields $N_h(t) \leq \frac{\Lambda_1}{\upsilon_1} - \left(\frac{\Lambda_1}{\upsilon_1} - N_h(0)\right)e^{-\upsilon_1 t}$. As t approaches

infinity, the supremum of $N_h(t)$ becomes bounded by $max\left\{N_h(o),\ \frac{\Lambda_1}{\upsilon_1}\right\}$, thus ensuring that $N_h(t)$ remains bounded above.

Now, consider the total host snail population $N_s(t) = S(t) + I_s(t)$ for $\forall\ t > 0$. The differential equation governing this population is given by: $\frac{dN_s(t)}{dt} = r_1 N_s\left(1 - \frac{N_s}{K_1}\right)$. Solving this equation gives $N_s(t) = \frac{K_1 N_s(o)}{N_s(o) + ((K_1 - N_s(0))e^{-r_1 t}}$, where $N_s(0) = S(0) + I_s(0)$. By setting $\underset{t\to\infty}{limSup}(N_s(t)) \le Q_1$, for $\forall t > 0$ where $Q_1 = max\{N_s(o), K_1\}$, we deduce that $N_s(t) \le Q_1$ for $\forall t > 0$. Additionally, by introducing the variable $Z = S + I_s + Y$, where Y represents some other components, we find that $\frac{dZ}{dt} + \gamma Z \le \delta$, where $\delta = (1 + r_1)Q_1$ and $\gamma = min\{1,\ (\upsilon_1 + \delta_1),\ \upsilon_5\}$. Solving this inequality gives $Z(t) \le Q_2 = \frac{\delta}{\gamma} + \left(Z(0) - \frac{\delta}{\gamma}\right)e^{-\delta t}$ for $\forall t > 0$. Hence $Q_2 = max\left\{Z(0),\ \frac{\delta}{\gamma}\right\}$. This guarantees that the solutions for $S(t)$, $I_s(t)$ and $Y(t)$ remain bounded above. Furthermore, this bound can be extended to $E(t)$, $M(t)$, $C(t)$ and $X(t)$, ensuring the boundedness of all variables. Consequently, feasible solutions converge within the region Ω. Thus, Theorems 1 and 2 together demonstrate that the system of model equations is both mathematically and epidemiologically well-posed. The well-posed nature of the model enables us to proceed with additional mathematical analyses of the model.

3. Analysis of the model (Eqs. (1)-(9))

In this section, we conduct our analyses and offer insights into the disease-free equilibria, including associated reproduction numbers for the model without control measures, with one control implemented separately and with the two controls implemented simultaneously. Furthermore, we present the endemic equilibrium for a complete model system and carry out a bifurcation analysis.

3.1. Disease-free equilibrium points

When all the populations are disease free, we assumed that $I(t) = E(t) = M(t) = I_s(t) = C(t) = 0$. By equating the left-hand side (LHS) of the model equation (Eqs. (1)-(9)) to zero, we present the disease-free equilibria in the following scenarios:

(i) We start with the scenario where no biological control measures are applied, resulting in a model with no competitor snails and no predators. In this case, we derive two disease-free equilibrium points:

$$D_{fe1} = \left(\frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1},\ 0,0,0,0,0,0\right) \text{ and } D_{fe2} = \left(\frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1}, 0,0,0,\ K_1, 0, 0\right)$$

(ii) Next, we consider the scenario where there are no snail predators, and competitor snails continue to exist. In this competition-only model, we obtain four disease-free equilibrium points categorized as follows:

(a) Human-trial equilibrium: $\left(\frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1}\ 0,0,0,0,0,0,\ 0\right)$,

(b) Two boundary equilibria: $\left(\frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1}, 0,0,0,\ K_1, 0,0,\ 0\right)$ and $\left(\frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1},\ 0,0,0,0,0,\ 0,\ K_2\right)$,

(c) Interior equilibrium point: $E_o^X = \left(\frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1},\ 0,\ 0,\ 0,\ \frac{K_1 - e_1 K_2}{1 - e_1 e_3},\ 0,\ 0,\ \frac{K_2 - e_3 K_1}{1 - e_1 e_3}\right)$, provided $\frac{K_1}{e_1 K_2} > 1$ for $e_1, e_3 \in [0,1]$.

(iii) In the absence of competitor snails and the presence of snail predators, we describe a predation-only model. In this scenario, we obtain up to four disease-free equilibrium points:

(a) Human-trivial equilibrium $\left(\frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1},\ 0,0,0,0,0,0,0\right)$,

(b) Two boundary equilibria $\left(\frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1}, 0,0,0,\ K_1,\ 0,0,\ 0\right)$ and $\left(\frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1},\ 0,0,0,0,0,\ 0,\ \frac{r_1}{t_1}\right)$,

(c) Interior equilibrium point $E_o^Y = \left(\frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1},\ 0,0,0, \frac{u_6}{t_1(\eta_1 - au_6)},\ 0,\ 0,\ \frac{r_1 \eta_1 K_1 t_1 (\eta_1 - au_6) - u_6}{t_1 K_1}\right)$ provided $\frac{\eta_1}{au_6} > 1$ and $\frac{r_1 \eta_1 K_1 t_1 (\eta_1 - au_6)}{u_6} > 1$.

(iv) Lastly, in the presence of both snail competitor and snail predator, we have a competition-predation model. In this case, we identify a single disease-free equilibrium point denoted as:

$E_o^{XY} = \left(\frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1},\ 0,\ 0,\ 0,\ \frac{u_6}{t_1(\eta_1 - au_6)},\ 0,\ 0,\ \frac{K_1 t_1 (\eta_1 - au_6) - e_3 u_6}{t_1(\eta_1 - au_6)},\ \eta_1 \left[\frac{r_1 t_1 (\eta_1 - au_6)(K_1 - e_1 K_2) - (1 - e_1 e_2)u_6}{K_1 (t_1(\eta_1 - au_6)^2)}\right]\right)$, provided $\frac{\eta_1}{au_6} > 1$, $\frac{K_1 t_1 (\eta_1 - au_6)}{e_3 u_6} > 1$, $\frac{K_1}{e_1 K_2} > 1$, and $\frac{r_1 t_1 (\eta_1 - au_6)(K_1 - e_1 K_2)}{(1 - e_1 e_2)u_6} > 1$.

The interior equilibrium points, specifically E_o^X, E_o^Y, and E_o^{XY}, play a significant role in our study as they signify the presence of IHs and the dynamic interactions between the hosts and control agents across different scenarios. These equilibrium points hold biological significance in our assessment of control strategies. In contrast, the trivial and boundary equilibrium points typically indicate scenarios where both snail and control agent populations either go extinct or reach their maximum carrying capacity. These points are not suitable for evaluating control strategies.

3.1.1. Reproduction number for model Eqs. (1)-(9)

This represents the number of new secondary schistosomiasis cases caused by an infected human/snail with both biological interventions in place. It is the effective reproduction number (R_{XY}) used to assess the impact of interventions on reducing disease transmission. We use the next-generation approach according to Diekmann et al. [31], Castillo-Chavez et al. [32], and Van den Driessche and Watmough [33], to calculate R_{XY}. Let $E_{XY} = (H^*,\ 0,0,0,\ S^*,\ 0,\ 0,\ X^*, Y^*)$ represent the disease-free equilibrium point for the model incorporating biological interventions. The result R_{XY} can be expressed as follows:

$$R_{XY} = \sqrt{\left(\frac{\beta_1 \omega_2 H^*}{C_o \upsilon_5 (\upsilon_2 + \delta_2 + e_2(S^* + X^*) + t_2 Y^*)}\right) \cdot \left(\frac{\beta_2 \omega_1 \rho \theta_h S^*}{M_o \upsilon_4 (\upsilon_1 + \delta_1)(\omega_1 + \upsilon_3)}\right)} \tag{10}$$

In this equation (Eq. (10)), $R_{XY}^H = \frac{\beta_1 \omega_2 H^*}{C_o \upsilon_5 (\upsilon_2 + \delta_2 + e_2(S^* + X^*) + t_2 Y^*)}$ and $R_{XY}^S = \frac{\beta_2 \omega_1 \rho \theta_h S^*}{M_o \upsilon_4 (\upsilon_1 + \delta_1)(\omega_1 + \upsilon_3)}$ represent the number of new human infections per infected IH snail and the number of new snail infections per infected human, respectively, under both biological interventions. Epidemiologically, schistosomiasis can emerge and spread in the population depending on the value of the bifurcation parameter, which corresponds to the threshold value $R_0 = 1$. Taking the snail infection rate $\beta_2 = \beta_{XY}^*$ as the bifurcation parameter with $R_{XY} = 1$, the critical value β_{XY}^* can be expressed as:

$$\beta_{XY}^* = \frac{\upsilon_4 \upsilon_5 C_o M_o (\upsilon_1 + \delta_1)(\omega_1 + \upsilon_3)(\upsilon_2 + \delta_2 + e_2(S^* + X^*) + t_2 Y^*)}{\beta_1 \omega_1 \omega_2 \rho \theta_h H^* S^*} \tag{11}$$

This critical value (Eq. (11)) represents the threshold below which the susceptible snails are not infected by miracidia and the infection

cannot progress in the presence of both the competitor snail and the predator, and vice versa. In particular, increasing the competition term $e_2(S^* + X^*)$ and the predation term $t_2 Y^*$ in Equation (Eq. (11)), increases β^*_{XY}. This, in turn, limits how far the infection can spread. Furthermore, in Equation Eq. (10), R_{XY} is a decreasing function of $e_2(S^* + X^*)$ and $t_2 Y^*$, suggesting that R_{XY} decreases with increase in the intervention.

In addition, we substitute for (H^*, S^*, X^*, Y^*) at the equilibrium point E_o^{XY} with both biological interventions to obtain

$$R_{XY} = \sqrt{\left(\frac{\beta_1\omega_2 K_1\lambda(t_1(\eta_1 - au_6))^2 e^{-\upsilon_1\tau}}{C_o\upsilon_1\upsilon_5((\upsilon_2+\delta_2)K_1(t_1\eta_1 - au_6))^2 + e_2t_1((\eta_1 - au_6)q_1 + t_2\eta_1 q_2)}\right)\cdot\left(\frac{\beta_2\omega_1\rho\theta_h u_6}{M_o\upsilon_4 t_1(\upsilon_1+\delta_1)(\omega_1+\upsilon_3)(\eta_1 - au_6)}\right)}$$

stands as the critical value for the rate of miracidial infection on susceptible snails.

3.1.3. Reproduction number with a snail competitor

This is an effective reproduction number for the model with a snail competitor intervention. It is used to assess the impact of the snail competitor intervention on reducing disease transmission. Similarly, in the presence of the snail competitor and when the predator goes extinct ($Y^* = 0$), we introduce the equilibrium terms of the variables (H^*, S^*, X^*) at E_X into equation Eq. (10), resulting in:

$$R_X = \sqrt{\left(\frac{(1-e_1e_3)\beta_1\omega_2\lambda e^{-\upsilon_1\tau}}{\upsilon_1\upsilon_5 C_o[(1-e_1e_3)(\upsilon_2+\delta_2) + e_2(K_1+K_2-e_3K_1-e_1K_2)]}\right)\cdot\left(\frac{\beta_2\omega_1\rho\theta_h(K_1-e_1K_2)}{\upsilon_4 M_o(1-e_1e_3)(\upsilon_1+\delta_1)(\omega_1+\upsilon_3)}\right)} \tag{13}$$

where,

$q_1 = K_1(K_1t_1(\eta_1 - au_6)) + (1-e_3)u_6$

$q_2 = r_1t_1(\eta_1 - au_6)(K_1 - e_1K_2) - (1-e_1e_2)u_6.$

Consequently, by substituting the variables (H^*, S^*, X^*, Y^*) into Equation (Eq. (10)) at distinct disease-free equilibrium points E_o, E_o^X, and E_o^Y, we obtain the corresponding reproduction numbers R_o, R_X, and R_Y for the sub-models as described in the Sections 3.1.2-3.1.4.

3.1.2. Basic reproduction number with no control

This provides insight into the potential for disease spread in a population without any interventions. It quantifies the initial disease transmission, allowing us to understand the inherent transmissibility of a disease. This value is obtained when we substitute $X^* = Y^* = 0$ and the equilibrium terms for the variables (H^*, S^*) at the disease free equilibrium with no control, E_o into Equation (Eq. (10)). The result is expressed as:

$$R_0 = \sqrt{\left(\frac{\beta_1\omega_2\lambda e^{-\upsilon_1\tau}}{\upsilon_1\upsilon_5 C_o(\upsilon_2+\delta_2)}\right)\cdot\left(\frac{\beta_2\omega_1\rho\theta_h K_1}{\upsilon_4 M_o(\upsilon_1+\delta_1)(\omega_1+\upsilon_3)}\right)} \tag{12}$$

Here, $R_H = \frac{\beta_1\omega_2\lambda e^{-\upsilon_1\tau}}{\upsilon_1\upsilon_5 C_o(\upsilon_2+\delta_2)}$ represents the number of human infections caused by one infectious snail and $R_S = \frac{\beta_2\omega_1\rho\theta_h K_1}{\upsilon_4 M_o(\upsilon_1+\delta_1)(\omega_1+\upsilon_3)}$ represents the number of snail infections caused by one infectious human, assuming no interventions are in place. If we assume $\beta_2 = \beta^*$ as the bifurcation parameter for model, then $R_0 = 1$ and $\beta^* = \frac{\upsilon_1\upsilon_4\upsilon_5 C_o M_o K(\upsilon_1+\delta_1)(\upsilon_2+\delta_2)(\omega_1+\upsilon_3)}{\beta_1\omega_1\omega_2\rho\theta_h K_1\lambda e^{-\upsilon_1\tau}}$

where $R_X^H = \frac{(1-e_1e_3)\beta_1\omega_2\lambda e^{-\upsilon_1\tau}}{\upsilon_1\upsilon_5 C_o[(1-e_1e_3)(\upsilon_2+\delta_2)+e_2(K_1+K_2-e_3K_1-e_1K_2)]}$ and $R_X^S = \frac{\beta_2\omega_1\rho\theta_h(K_1-e_1K_2)}{\upsilon_4 M_o(1-e_1e_3)(\upsilon_1+\delta_1)(\omega_1+\upsilon_3)}$ is the number of possible new human infections per infected snail and the number of possible new snail infections per infected human, respectively if the snail competitor survives. Considering $\beta_2 = \beta_X^*$ as the bifurcation parameter and setting $R_X = 1$, the critical value $\beta_2 = \beta_X^*$ is determined as $\beta_X^* = \frac{\upsilon_1\upsilon_4\upsilon_5 C_o M_o[(1-e_1e_3)(\upsilon_2+\delta_2)+e_2(K_1+K_2-e_3K_1-e_1K_2)](\upsilon_1+\delta_1)(\omega_1+\upsilon_3)}{\beta_1\omega_1\omega_2\rho\theta_h\lambda(K_1-e_1K_2)e^{-\upsilon_1\tau}}$. This critical value represents the rate at which susceptible snails become infected by miracidia in the presence of only a snail competitor.

3.1.4. Reproduction number with a predator

This represents the effective reproduction number within the model involving a snail predator intervention, and isused to evaluate the impact of introducing predatory snails in reducing disease transmission. In this scenario, the competition term in Eq. (10), specifically $e_2(S^* + X^*)$, is set to zero. The resulting specific reproduction number, denoted as R_Y, is determined at the equilibrium point E_o^Y for the predator-only model, and it is given by:

$$R_Y = \sqrt{\left(\frac{\beta_1\omega_2\lambda e^{-\upsilon_1\tau}}{C_o\upsilon_1\upsilon_5(\upsilon_2+\delta_2+r_1\eta_1K_1t_1t_2(\eta_1 - au_6) - (t_2u_6/t_1K_1))}\right)\cdot\left(\frac{\beta_2\omega_1\rho\theta_h u_6}{M_o\upsilon_4 t_1(\upsilon_1+\delta_1)(\omega_1+\upsilon_3)(\eta_1 - au_6)}\right)} \tag{14}$$

Here, $R_Y^H = \frac{\beta_1\omega_2\lambda e^{-\upsilon_1\tau}}{C_o\upsilon_1\upsilon_5(\upsilon_2+\delta_2+r_1\eta_1K_1t_1t_2(\eta_1-au_6)-(t_2u_6/t_1K_1))}$ represents the potential number of new human infections per infected snail, and $R_Y^S = \frac{\beta_2\omega_1\rho\theta_h u_6}{M_o\upsilon_4 t_1(\upsilon_1+\delta_1)(\omega_1+\upsilon_3)(\eta_1-au_6)}$ represents the potential number of new snail infections per infected human, specifically in case where snail predation

is the only control strategy. If we consider $\beta_2 = \beta_Y^*$ as the bifurcation parameter with $R_Y = 1$, then $\beta_Y^* = \frac{\upsilon_1\upsilon_4\upsilon_5 C_o M_o t_1(\upsilon_2+\delta_2+r_1\eta_1 K_1 t_1 t_2(\eta_1-a\upsilon_6)-(t_2\upsilon_6/t_1K_1))(\upsilon_1+\delta_1)(\omega_1+\upsilon_3)(\eta_1-a\upsilon_6)}{\beta_1\omega_1\omega_2\upsilon_6\rho\theta_h\lambda e^{-\upsilon_1\tau}}$ represents the rate at which susceptible snails become infected by miracidia when only a snail predator is present in the environment.

$$J(XY) = \begin{bmatrix} -\upsilon_1 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_1 H^*}{C_o} & 0 & 0 \\ 0 & -(\upsilon_1+\delta_1) & 0 & 0 & 0 & 0 & \frac{\beta_1 H^*}{C_o} & 0 & 0 \\ 0 & \rho\theta_h & -(\omega_1+\upsilon_3) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \omega_1 & -\upsilon_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\frac{\beta_2 S^*}{M_o} & g_1 & \frac{-r_1 S^*}{K_1} & 0 & \frac{-r_1 S^*}{K_1} & \frac{-t_1 S^*}{1+at_1 S^*} \\ 0 & 0 & 0 & \frac{\beta_2 S^*}{M_o} & 0 & g_2 & 0 & 0 & t_2 Y^* \\ 0 & 0 & 0 & 0 & 0 & \omega_2 & -\upsilon_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{-r_2 X^*}{K_2} & \frac{-r_2 X^*}{K_2} & 0 & g_3 & 0 \\ 0 & 0 & 0 & 0 & \frac{\eta_1 t_1 S^*}{(1+at_1 S^*)^2} & \eta_2 t_2 Y^* & 0 & 0 & g_4 \end{bmatrix}$$

3.2. The local stability of the disease-free equilibrium E_o^{XY} for model Eqs. (1)-(9)

We present Theorem 3, which addresses the stability of the disease-free equilibrium for the complete model system.

Theorem 3. *The disease-free equilibrium point E_o^{XY} is locally asymptotically stable if $R_{XY} < 1$ and unstable if $R_{XY} > 1$*

Proof. We show that Jacobian matrix $J(XY)$ of the model equation Eqs. (1)-(9) has negative eigenvalue. The $J(XY)$ at E_o^{XY} is given by

Here $g_1 = r_1\left(1 - \frac{2S^*+X^*}{K_1}\right) - \frac{t_1\alpha_1 Y^*}{(1+at_1S^*)^2}$, $g_2 = -(\upsilon_2 + \delta_2 + e_1(S^* + X^*) + t_2Y^*)$, $g_3 = r_2\left(1 - \frac{2X^*+S^*}{K_2}\right)$, and $g_4 = \frac{\eta_1 t_1 S^*}{1+at_1S^*} - \upsilon_6$. The negative eigenvalues of $J(XY)$ are: $-\upsilon_1, -(\upsilon_1+\delta_1), -(\omega_1+\upsilon_3), -\upsilon_4$ and $-\upsilon_5$. The remaining eigenvalues can be obtained from a reduced 4×4 matrix which results from $J(XY)$, and its characteristic equation is expressed as:

$$\lambda^4 - d_1\lambda^3 + d_2\lambda^2 + d_3\lambda + d_4 = 0 \tag{15}$$

Table 1
Model parameters and their descriptions.

Symbol	Definition	Symbol	Definition
λ	Human reproduction rate	C_0	Saturation coefficient for cercaria infectivity
β_1	Human infection rate	M_0	Saturation coefficient for miracidia infectivity
β_2	Snail infection rate	ρ	Portion of stool per person
τ	Initial age of infection in children	θ_h	Number of eggs per gram of stool
δ_1	Human mortality due to infection	K	Egg carrying capacity
δ_2	IH snail mortality due to infection	K_1	Carrying capacity of host snails
υ_1	Human mortality rate	K_2	Carrying capacity of competitor snail
υ_3	Parasite egg mortality rate	r_1	Intrinsic growth rate of snails
υ_2	IH snail mortality rate	r_2	Intrinsic growth rate of competitor snails
υ_4	Miracidia death rate	ε	Crowding effect of miracidia/cercaria
b_s	IH snail birth rate	t_1	Attack rate of susceptible snail by a predator
υ_5	Cercaria mortality rate	t_2	Attack rate of infected snail by a predator
ω_1	Miracidia production rate	a	Handling time for susceptible snails by a predator
ω_2	Cercariae emergence rate	b	Handling time of infected snail by a predator
e_1	Competition factor against susceptible snails	η_1	Susceptible snail-to-predator conversion factor
e_2	Competition factor against infected snails	η_2	Infected snail-to-predator conversion factor
e_3	Competition factor against snail competitor	υ_6	Death rate of the predator

where,

$$
\begin{cases}
d_1 = \left(r_1\left(1-\dfrac{2S^*+X^*}{K_1}\right)+\dfrac{t_1Y^*}{(1+at_1S^*)^2}\right)+\left(r_2\left(1-\dfrac{2X^*+S^*}{K_2}\right)\right)+\left(\dfrac{\eta_1t_1S^*}{1+at_1S^*}+\upsilon_6\right)-(\upsilon_2+\delta_2+e_1(S^*+X^*)+t_2Y^*),\\
d_2 = \left[\left(r_1\left(1-\dfrac{2S^*+X^*}{K_1}\right)+\dfrac{t_1Y^*}{(1+at_1S^*)^2}\right)+\left(r_2\left(1-\dfrac{2X^*+S^*}{K_2}\right)\right)\right]\left[\left(\dfrac{\eta_1t_1S^*}{1+at_1S^*}+\upsilon_6\right)-(\upsilon_2+\delta_2+e_1(S^*+X^*)+t_2Y^*)\right]\\
-\left[\left(\dfrac{\eta_1t_1S^*}{1+at_1S^*}+\upsilon_6\right)\left(\upsilon_2+\delta_2+e_1(S^*+X^*)+\dfrac{t_2Y^*}{\alpha_2}\right)+\eta_2t_2^{*2}Y^{*2}\right],\\
d_3 = \left[\left(r_1\left(1-\dfrac{2S^*+X^*}{K_1}\right)+\dfrac{t_1\alpha_1Y^*}{(1+at_1S^*)^2}\right)+\left(r_2\left(1-\dfrac{2X^*+S^*}{K_2}\right)\right)\right]\left[\left(\dfrac{\eta_1t_1S^*}{1+at_1S^*}+\upsilon_6\right)(\upsilon_2+\delta_2+e_1(S^*+X^*)+t_2Y^*)+\eta_2t_2^{*2}Y^{*2}\right]\\
-\left[\left(\dfrac{\eta_1t_1S^*}{1+at_1S^*}+\upsilon_6\right)-\left(\upsilon_2+\delta_2+e_1(S^*+X^*)+\dfrac{t_2Y^*}{\alpha_2}\right)\right]\left[\left(r_1\left(1-\dfrac{2S^*+X^*}{K_1}\right)+\dfrac{t_1\alpha_1Y^*}{(1+at_1S^*)^2}\right)\left(r_2\left(1-\dfrac{2X^*+S^*}{K_2}\right)\right)-\dfrac{r_1r_2X^*}{K_1K_2}\right]\\
+\dfrac{\eta_1\alpha_1t_1S^*}{(\alpha_1+S^*)^2}\left(\dfrac{r_1t_2S^*Y^*}{K_1\alpha_2}-\dfrac{t_1S^*}{1+at_1S^*}\right),\\
d_4 = \dfrac{\eta_1\alpha_1r_1t_1t_2X^*YS^{*2}}{K_1K_2\alpha_2(1+at_1S^*)^2}+\dfrac{r_1r_2X^*}{K_1K_2}\left(\dfrac{\eta_1t_1S^*}{\alpha_1+S^*}+\upsilon_6\right)(\upsilon_2+\delta_2+e_1(S^*+X^*)+t_2Y^*)-\dfrac{\eta_1\alpha_1r_1t_1t_2X^*YS^{*2}}{K_1K_2\alpha_2(1+at_1S^*)^2}r_2\left(1-\dfrac{2X^*+S^*}{K_2}\right)-\\
\dfrac{\eta_1\alpha_1(t_1S^*)^2}{(1+at_1S^*)^3}\left(r_2\left(1-\dfrac{2X^*+S^*}{K_2}\right)\right)-\\
\left(r_1\left(1-\dfrac{2S^*+X^*}{K_1}\right)+\dfrac{t_1\alpha_1Y^*}{(1+at_1S^*)^2}\right)\left(r_2\left(1-\dfrac{2X^*+S^*}{K_2}\right)\right)\left[\left(\dfrac{\eta_1t_1S^*}{1+at_1S^*}+\upsilon_6\right)(\upsilon_2+\delta_2+e_1(S^*+X^*)+t_2Y^*)+\eta_2t_2^{*2}Y^{*2}\right].
\end{cases}
$$

The Ruth-Hurwitz criterion serves as both the necessary and sufficient condition for establishing the asymptotic stability of E_o^{XY}. This criterion ensures that all roots of the characteristic polynomial (Eq. (15)) have negative real components. This condition is fulfilled if the inequality $(d_1d_2+d_3)d_3+d_1^2d_4\le 0$ is satisfied. Consequently, for the matrix J(XY) to have all eigenvalues negative, it is essential that $R_{XY}\le 1$, and E_o^{XY} achieves local asymptotic stability if $R_{XY}<1$.

Table 2
Model parameters, possible values and their sources.

Symbol	Baseline value	Ranges	References
λ	8000/**day**	6000–10,000	[39]
β_1	0.075/ **day**	0.028–0.122	[26]
β_2	0.0006635/ **day**	0.000127–0.0012	[26]
τ	730**days**	730	[28]
δ_1	0.0227/**d ay**	0.00039- 0.0227	[27,40]
δ_2	0.018805/**day**	0.00122–0.03639	[27]
υ_1	0.0028/**day**	0–0.5	[40]
υ_3	0.004/**day**	0.004–0.0182	[26]
υ_2	0.000569/**day**	0.0001–0.04	[23]
υ_4	2 /**day**	2–10	[26]
b_s	0.08/**day**	0.08–0.118	[26]
υ_5	1/**day**	1–5	[26,41]
ω_1	500/**day**		[26]
ω_2	4615/**day**	829–8400	[26,27]
e_1	0–0.5/**day**	0–0.5	Varied
e_2	0–0.5/**day**	0–0.5	Varied
e_3	0.001/**day**	0.001	Estimated
C_o	1000,000	1000,000	[24]
M_o	1000,000	1000,000	[28]
ρ	115 grams/**day**	70–160	[42]
θ_h	262/grams/**day**	10–513	[42]
K	1×10^5	1×10^5	[28]
K_s	1×10^5	1×10^5	[27]
K_2	1×10^5	1×10^5	Estimated
r_1	0.16 /**day**	0–0.5	Estimated
r_2	1.5 r_1/**day**	0–0.5	Estimated
ε	0.2	0.2–0.3	[24,39]
t_1	0–4/**day**	0–4	Varied
t_2	0–4/**day**	0–4	Varied
a	0- 3/**day**	0- 3	Varied
b	0–3/**day**	0- 3	Varied
η_1	0.07	0–1	[10]
η_2	0.09	0–1	[10]
υ_6	0.02/**day**	0.0001–0.04	[23,30]

3.3. Endemic equilibrium point of model Eqs. (1)-(9)

This represents a steady state solution that occurs when the disease persists in the community, and is calculated by equating the derivatives of the model equations (Eqs. (1)-(9)) to zero. Let $E_1^{XY}=\left(H_1^*,I_1^*,E^*,M^*,S_1^*,\ I_s^*,\ C^*,X_1^*,\ Y_1^*\right)$ be the endemic equilibrium with variables expressed in terms of I_s^* as follows:

$$
\begin{cases}
H^*\left(I_s^*\right)=\dfrac{\lambda\left(\upsilon_5C_o+\varepsilon\omega_2I_s^*\right)e^{-\upsilon_1\tau}}{\upsilon_1^2\upsilon_5C_o+\upsilon_1(\omega_2\beta_1+\varepsilon\upsilon_1\omega_2)I_s^*},\\
I^*\left(I_s^*\right)=\dfrac{\beta_1\omega_1\lambda e^{-\upsilon_1\tau}I_s^*}{\upsilon_1(\upsilon_1+\delta_1)\left[\upsilon_1\upsilon_5C_o+(\beta_1\omega_2+\varepsilon\upsilon_1\omega_2)I_s^*\right]}\\
\#\\
E^*\left(I_s^*\right)=\dfrac{\beta_1\omega_1\rho\theta_hK\lambda e^{-\upsilon_1\tau}I_s^*}{\upsilon_1\upsilon_5C_oK(\upsilon_1+\delta_1)(\omega_1+\upsilon_3)+(\beta_1\omega_1\rho\theta_hK\lambda e^{-\upsilon_1\tau}+\beta_1\omega_2+\varepsilon\upsilon_1\omega_2)I_s^*},\\
\#\\
M^*\left(I_s^*\right)=\dfrac{\beta_1\omega_1^2\rho\theta_hK\lambda e^{-\upsilon_1\tau}I_s^*}{\upsilon_1\upsilon_4\upsilon_5C_oK(\upsilon_1+\delta_1)(\omega_1+\upsilon_3)+\beta_1\omega_1\rho\theta_hK\lambda e^{-\upsilon_1\tau}+\omega_2\upsilon_4(\beta_1+\varepsilon\upsilon_1)I_s^*},\\
S_1^*\left(I_s^*\right)=\dfrac{\upsilon_6+t_2(b\upsilon_6-\eta_2)I_s^*}{t_1(\eta_1-a\upsilon_6)+t_1t_2(\eta_1+a\eta_2-ab\upsilon_6)I_s^*},\\
C^*\left(I_s^*\right)=\dfrac{\omega_2}{\upsilon_5}I_s^*,\\
X_1^*\left(I_s^*\right)=K_2-e_3\left(\dfrac{\upsilon_6+t_2(b\upsilon_6-\eta_2)I_s^*}{t_1(\eta_1-a\upsilon_6)+t_1t_2(\eta_1+a\eta_2-ab\upsilon_6)I_s^*}+I_s^*\right)\\
Y_1^*\left(S_1^*,I_s^*,X_1^*\right)=\dfrac{\beta_2S_1^*I_1^*-\left(I_{0s}+\varepsilon_1I_s^*\right)\left(\upsilon_2+\delta_2+e_2\left(S^*+I_s^*+X^*\right)\right)I_s^*}{\left(1+bt_2I_s^*\right)\left(I_{0s}+\varepsilon_1I_s^*\right)}
\end{cases}
$$

To determine I_s^*, we use Eq. (5) of model (Eqs. (1)-(9)) at endemic equilibrium when $dX/dt=0$. Substituting $S_1^*\left(I_s^*\right)$ and $X_1^*\left(I_s^*\right)$, we obtain

an equation in the form

$$A_1 I_s^{*2} + A_2 I_s^{*} - A_3 = 0 \tag{16}$$

where

$$A_1 = e_3 t_1 t_2 (1 - e_3)(\eta_1 + a\eta_2 - ab\upsilon_6),$$

$$A_2 = t_1 t_2 K_2 (1 + e_3)(\eta_1 + a\eta_2 - ab\upsilon_6) + e_3 t_1 (1 - e_3)(\eta_1 - a\upsilon_6) - t_2 (1 + e_3^2)(b\upsilon_6 - \eta_2)$$

$$A_3 = t_1 K_2 (1 + e_3)(\eta_1 - a\upsilon_6) - (1 + e_3^2)\upsilon_6.$$

The positive root of Eq. (14) provides the solution for I_s^*, specifically:

$$I_s^* = \frac{-A_2 \pm \sqrt{{A_2}^2 + 4A_1A_3}}{2A_1}.$$

This solution is valid and the endemic equilibrium point exists if $I_s^* \in R_{+0}$, the set of non-negative real numbers.

3.4. Bifurcation analysis

To investigate both the type of bifurcation exhibited and the local stability of the endemic equilibrium, E_1^{XY} of the model (Eqs. (1)-(9)), we use the center manifold theory described by Castillo-Chavez and Song [34]. To apply this theory, a change of variables is introduced for the normalized version of model (Eqs. (1)-(9)). i.e., introducing $H = x_1$, $I = x_2$, $E = x_3$, $M = x_4$, $S = x_5$, $I_s = x_6$, $X = x_8$, $Y = x_9$, the model is transformed to:

$$\left.\begin{aligned}
\dot{x}_1 &= \Lambda_1 - \frac{\beta_1 x_1 x_7}{C_o + \varepsilon x_7} - \upsilon_1 x_1 := f_1,\\
\dot{x}_2 &= \frac{\beta_1 x_1 x_7}{C_o + \varepsilon x_7} - (\upsilon_1 + \delta_1)x_2 := f_2\\
\dot{x}_3 &= \rho\theta_h x_2\left(1 - \frac{x_3}{K}\right) - (\omega_1 + \upsilon_3)x_3 := f_3,\\
\dot{x}_4 &= \omega_1 x_3 - \upsilon_4 x_4 := f_4\\
\dot{x}_5 &= r_1 x_5\left(1 - \frac{x_5 + e_1(x_6 + x_8)}{K_1}\right) - \frac{\beta_2 x_4 x_5}{M_o + \varepsilon x_4} - \frac{t_1 x_5 x_9}{1 + at_1 x_5} := f_5,\\
\dot{x}_6 &= \frac{\beta_2 x_4 x_5}{M_o + \varepsilon x_4} - (\upsilon_2 + \delta_2 + e_2(x_5 + x_8) + x_6)x_6 - \frac{t_2 x_6 x_9}{1 + bt_2 x_6} := f_6\\
\dot{x}_7 &= \omega_2 x_6 - \upsilon_5 x_7 := f_7,\\
\dot{x}_8 &= r_2 x_8\left(1 - \frac{x_8 + e_3(x_5 + x_6)}{K_2}\right) := f_8\\
\dot{x}_9 &= \frac{\eta_1 t_1 x_5 x_9}{1 + at_1 x_5} + \frac{\eta_2 t_2 x_6 x_9}{\alpha_2 + x_6} - \upsilon_6 x_9 := f_9
\end{aligned}\right\} \tag{17}$$

The transformed model has the disease-free equilibrium given as $E_0^d = \left({x_1}^* = \frac{\lambda e^{-\upsilon_1 \tau}}{\upsilon_1},\ {x_2}^* = 0,\ {x_3}^* = 0,\ {x_4}^* = 0,\ {x_5}^* = {x_5}^*,\ {x_6}^* = 0,\ {x_7}^* = 0,\ {x_8}^* = {x_8}^*,\ {x_9}^* = {x_9}^*\right)$. Let $\beta_2 = \beta^{**}$ be the bifurcation parameter, with $R_{XY} = 1$, Subsequently, we have $\beta^{**} = \frac{\upsilon_1\upsilon_4\upsilon_5 C_o M_o K(\upsilon_1+\delta_1)(\upsilon_2+\delta_2+e_2(S^*+X^*)+t_2Y^*)}{\beta_1\omega_2\rho\theta_h\lambda e^{-\upsilon_1\tau}}$ and the corresponding Jacobian matrix of the model (Eq. (17)) at the E_0^d is:

$$J(E_0^d) = \begin{bmatrix}
-\upsilon_1 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_1 {x_1}^*}{C_o} & 0 & 0\\
0 & -(\upsilon_1 + \delta_1) & 0 & 0 & 0 & 0 & \frac{\beta_1 {x_1}^*}{C_o} & 0 & 0\\
0 & \rho\theta_h & -(\omega_1 + \upsilon_3) & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & \omega_1 & -\upsilon_4 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & -\frac{\beta^{**} {x_5}^*}{M_o} & g_1 & \frac{-r_1 {x_5}^*}{K_1} & 0 & \frac{-r_1 {x_5}^*}{K_1} & \frac{-t_1 {x_5}^*}{1 + at_1 {x_5}^*}\\
0 & 0 & 0 & \frac{\beta^{**} {x_5}^*}{M_o} & 0 & g_2 & 0 & 0 & t_2 {x_9}^*\\
0 & 0 & 0 & 0 & 0 & \omega_2 & -\upsilon_5 & 0 & 0\\
0 & 0 & 0 & 0 & \frac{-r_2 {x_8}^*}{K_2} & \frac{-r_2 {x_8}^*}{K_2} & 0 & g_3 & 0\\
0 & 0 & 0 & 0 & \frac{\eta_1 t_1 {x_5}^*}{(1 + at_1 {x_5}^*)^2} & \eta_2 t_2 {x_9}^* & 0 & 0 & g_4
\end{bmatrix}$$

where $g_1 = r_1\left(1 - \frac{2{x_5}^* + {x_8}^*}{K_1}\right) - \frac{t_1 {x_9}^*}{(1+at_1 {x_5}^*)^2}$, $g_2 = -(\upsilon_2 + \delta_2 + e_2({x_5}^* + {x_8}^*) + t_2 {x_9}^*)$, $g_3 = r_2\left(1 - \frac{2{x_8}^* + {x_5}^*}{K_2}\right)$, and $g_4 = \frac{\eta_1 t_1 {x_5}^*}{1+at_1 {x_5}^*} - \upsilon_6$. It is clear

that zero is a simple eigenvalue of the Jacobian matrix, $J(E_0^d)$. The corresponding eigenvectors represent an approximate rate of infected human/snails in an endemic state when the disease-free state is unstable and the asymptotic distribution of the infected human/snails as the disease dies out. Thus $J(E_0^d)$ has a right eigenvector $u = (u_1, u_2, u_3, u_4, u_5, u_6, u_7, u_8, u_9)$ and a left eigenvector $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9)$ associated with zero eigenvalue satisfying the condition $u.v = 1$, where

$$\begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \\ u_5 \\ u_6 \\ u_7 \\ u_8 \\ u_9 \end{pmatrix} = \begin{pmatrix} -\frac{\beta_1 {x_1}^*}{C_o} u_7, \\ \frac{\beta_1 {x_1}^*}{C_o(v_1+\delta_1)} u_7, \\ \frac{\beta_1 \rho \theta_h {x_1}^*}{C_o(v_1+\delta_1)(\omega_1+v_3)} u_7, \\ \frac{\beta_1 \omega_1 \rho \theta_h {x_1}^*}{C_o v_4(v_1+\delta_1)(\omega_1+v_3)} u_7, \\ -\frac{r_1 r_2 K_1 v_5 {x_8}^*}{r_1 \omega_2 (r_1 K_2 {x_5}^* + r_2 K_1 {x_8}^*)} u_7, \\ \frac{v_5}{\omega_2} u_7, \\ u_7 > 0 \\ -\frac{r_1 r_2 K_1 v_5 (1+ \mathrm{at}_1 {x_5}^*)^2 {x_5}^* {x_8}^*}{\omega_2 g_3 \eta_1 t_1 {x_5}^* (r_1 K_2 {x_5}^* + r_2 K_1 {x_8}^*)} u_7, \\ -\frac{r_1 r_2 K_1 v_5 (1+ \mathrm{at}_1 {x_5}^*)^2 {x_5}^* {x_8}^*}{\omega_2 g_3 \eta_1 t_1 {x_5}^* (r_1 K_2 {x_5}^* + r_2 K_1 {x_8}^*)} u_7, \end{pmatrix} = \begin{pmatrix} 0 \\ \frac{C_o v_5}{\beta_1 {x_1}^*} v_7 \\ \frac{C_o v_5 (v_1+\delta_1)}{\rho \theta_{h\beta_1} {x_1}^*} v_7 \\ \frac{C_o v_5 (v_1+\delta_1)(\omega_1+v_3)}{\omega_1 \rho \theta_{h\beta_1} {x_1}^*} v_7 \\ \frac{g_3 C_o M_o v_5^2 (v_1+\delta_1)(\omega_1+v_3)}{K_1 \omega_1 \rho \theta_{h\beta_1 (r_1 {x_5}^* - g_3 K_1) {x_1}^*}} v_7 \\ \frac{\omega_2 {x_5}^*}{(v_2+\delta_2)} v_7 \\ v_7 > 0 \\ \frac{C_o M_o v_5^2 (v_1+\delta_1)(\omega_1+v_3)}{r_1 \omega_1 \rho \theta_{h\beta_1 (r_1 {x_5}^* - g_3 K_1) {x_1}^{*2}}} v_7 \\ \mathscr{M} \left(\frac{r_2 {x_5}^*}{K_2} - \frac{g_1 g_3 K_1}{r_1 {x_5}^*} \right) v_7 \end{pmatrix}$$

where $\mathscr{M} = \frac{C_o M_o r_1 v_5^2 (1+ \mathrm{at}_1 {x_5}^*)^2 (v_1+\delta_1)(\omega_1+v_3)(1+ \mathrm{at}_1 {x_5}^*)^2}{K_1 \omega_1 \rho \theta_h \beta_1 \eta_1 t_1 (r_1 {x_5}^* - g_3 K_1) {x_1}^*}$.

$$a = \sum_{k,\ i,\ j=1}^{9} v_k u_i u_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} \left(E_1^d, \beta^{**} \right)$$

$$b = \sum_{k,\ j=1}^{9} v_k u_i \frac{\partial^2 f_k}{\partial x_i \beta^{**}} \left(E_1^d, \beta^{**} \right)$$

where the non-vanishing second-order partial derivatives at the disease-free equilibrium E_0^d, are algebraically given by

$$\begin{cases} \frac{\partial^2 f_1}{\partial x_1 \partial x_7} = \frac{-\beta_1}{C_o}, \ \frac{\partial^2 f_2}{\partial x_1 \partial x_7} = \frac{\beta_1}{C_o}, \ \frac{\partial^2 f_3}{\partial x_2 \partial x_3} = \frac{-\rho \theta_h}{K}, \ \frac{\partial^2 f_5}{\partial x_4 \partial x_5} = \frac{-\beta^{**}}{M_o}, \ \frac{\partial^2 f_5}{\partial {x_5}^2} = \frac{-2r_1}{K_1}, \ \frac{\partial^2 f_5}{\partial x_5 \partial x_6} = \frac{-r_1}{K_1}, \\ \frac{\partial^2 f_5}{\partial x_5 \partial x_9} = \frac{-t_1}{(1+ \mathrm{at}_1 {x_5}^*)^2}, \frac{\partial^2 f_6}{\partial x_4 \partial x_5} = \frac{\beta^{**}}{M_o}, \ \frac{\partial^2 f_6}{\partial x_5 \partial x_6} = \frac{\partial^2 f_6}{\partial x_6 \partial x_8} = -e_2, \ \frac{\partial^2 f_6}{\partial x_6 \partial x_6} = -2e_2, \\ \# \\ \frac{\partial^2 f_6}{\partial x_6 \partial x_9} = \frac{t_2}{\alpha_2}, \ \frac{\partial^2 f_8}{\partial x_5 \partial x_8} = \ \frac{\partial^2 f_8}{\partial x_6 \partial x_8} = \frac{-r_2}{K_2}, \frac{\partial^2 f_6}{\partial {x_8}^2} = \frac{-2r_2}{K_2}, \ \frac{\partial^2 f_5}{\partial x_4 \partial \beta^*} = \frac{-{x_5}^*}{M_o}, \ \frac{\partial^2 f_6}{\partial x_4 \partial \beta^*} = \frac{{x_5}^*}{M_o} \end{cases}$$

We compute the values of the coefficients a and b according to the formulae in Castillo-Chavez and Song [34], where:

Thus, considering the signs and combining similar terms we obtain

$$a = \left(v_5 u_4 u_5 \frac{\beta^{**}}{M_o} + v_5 u_5 u_6 \frac{r_1}{K_1} + v_6 u_5 u_6 e_2 + v_5 u_5 u_6 \frac{r_2}{K_2} \right)$$
$$- \left(v_2 u_1 u_7 \frac{\beta_1}{C_o} + v_3 u_2 u_3 \frac{\rho \theta_h}{K} + 2 v_5 {u_5}^2 \frac{-t_1}{(1+ \mathrm{at}_1 {x_5}^*)^2} + v_6 u_6 u_8 e_2 + 2 v_6 {u_6}^2 e_2 + v_6 u_6 u_9 \frac{t_2}{\alpha_2} + 2 v_6 {u_8}^2 \frac{r_2}{K_2} \right)$$

$$b = \frac{u_4 x_5^*}{M_o}(v_5 + v_6)$$

The positivity of the coefficient b has been established. As pointed out by Castillo-Chavez and Song [34], the behavior of coefficient a determines the specific characteristics of the local dynamics of the equilibrium points. Consequently, if $a < 0$, the model system (Eq. (17)) will exhibit forward bifurcation where the endemic equilibrium is locally asymptotically stable. Conversely, if $a > 0$, it will undergo backward bifurcation. This can lead to the coexistence of stable and endemic equilibria, and reducing the reproduction number $R_{XY} < 1$ at disease free equilibrium does not guarantee disease eradication. This makes disease control more difficult, as both competition and predation measures may still result in persistent low endemicity. To anticipate and manage backward bifurcation, in addition to competition and predation of snail-vector control measures, it is essential to implement more interventions such as increased treatment coverage and public health education. In addition, in order to respond to the dynamic changes in the system and to prevent the disease from re-emerging, monitoring and adaptive management strategies should be put in place.

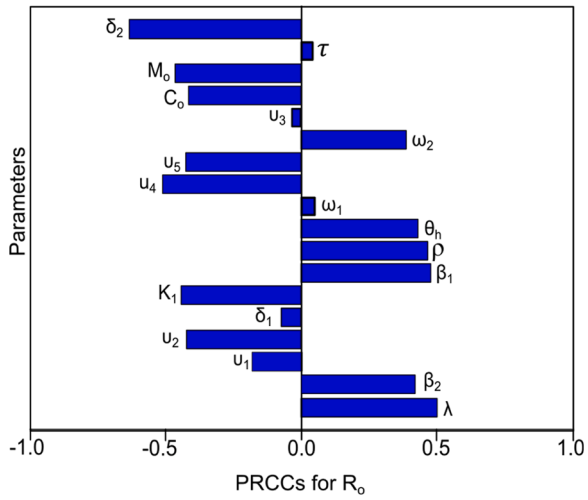

Fig. 2. Partial rank correlation coefficients (PRCC) for sensitivity analysis of R_0 with respect to each model parameter when no control measures are implemented.

4. Numerical simulations

We use the R statistical environment version 4.0.3 [35], and the main R package for executing ordinary differential equations [36]. We use information from relevant literature to determine the parameters of the model (Table 2). Parameters not typically found in the literature are replicated using expert knowledge, taking into account the prevailing understanding of vector and disease dynamics in schistosomiasis. For instance, the documented initial age of infection in children is typically around 2 years (730 days) [37]. However, it is worth noting that the disease can also affect very young children who are under the age of 2 if they come into contact with contaminated freshwater sources through activities such as washing. Furthermore, as reported by Cross and Benke [38], the interaction between species in competitive scenarios can exert

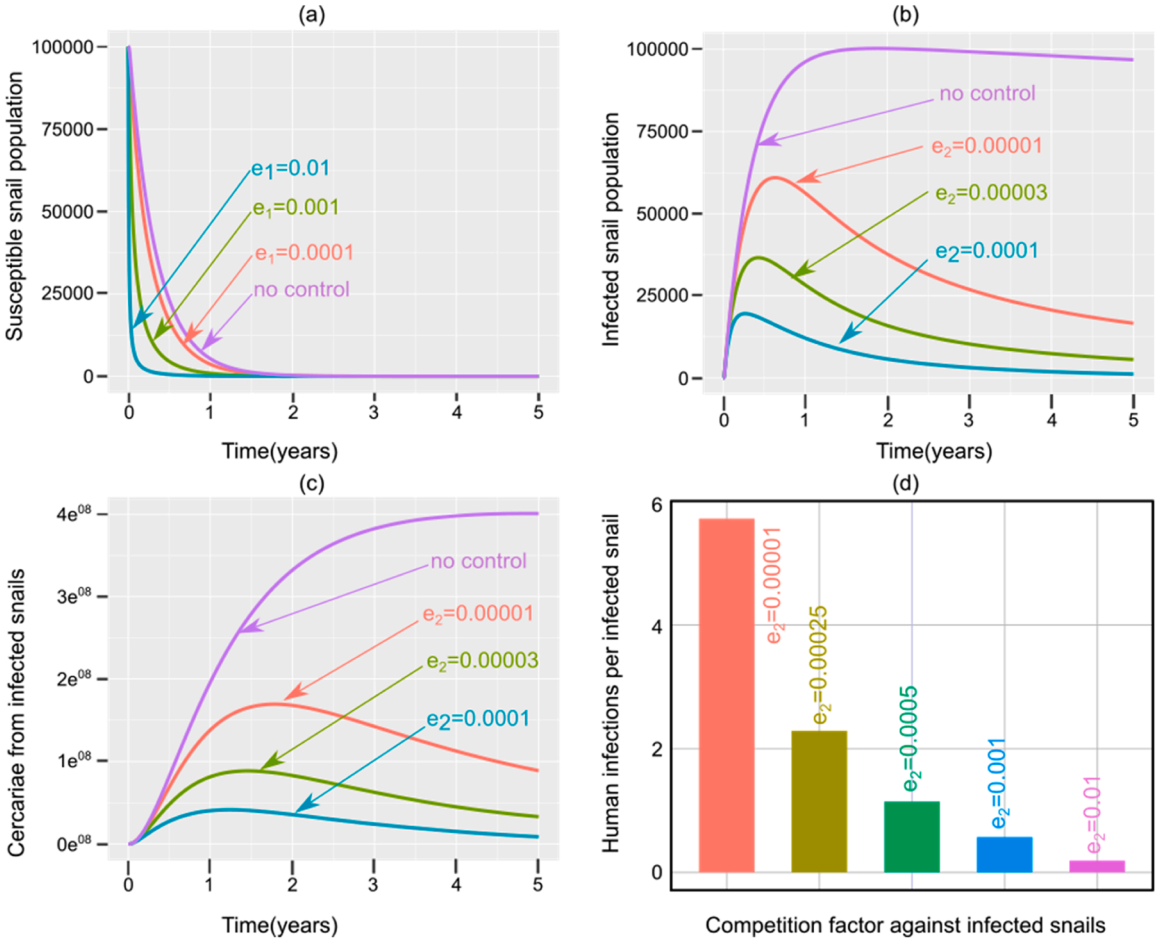

Fig. 3. Impact of varying competition against (a) susceptible snail population, and (b) infected snail population. The subsequent influence on (c) infectious cercariae emerging from infected snails, and (d) human infections per infected snail.

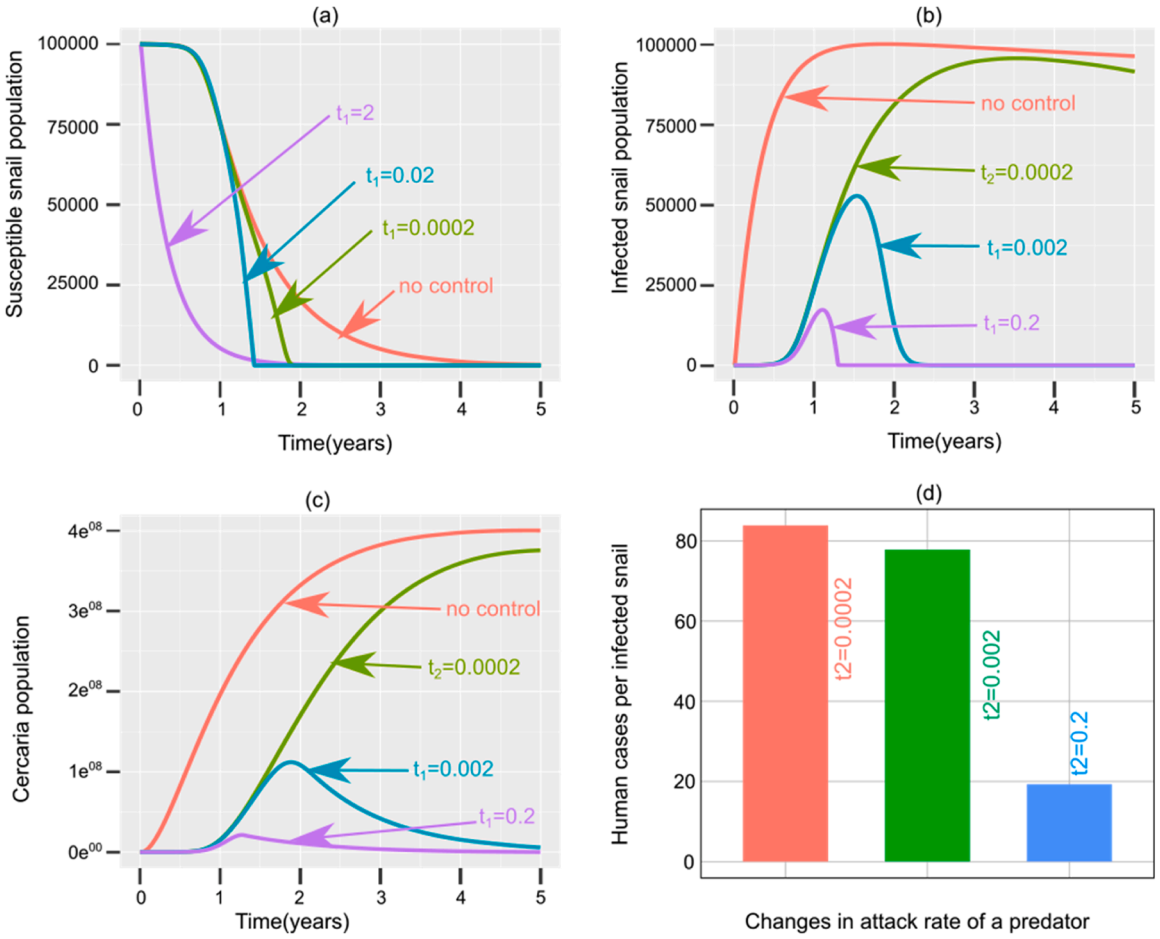

Fig. 4. Impact of varying the attack rate (t_1, t_2) of a predator on (a) susceptible snail population, and (b) infected snail population. The subsequent influence on (c) infectious cercaria emerging from infected snail population, and (d) human infections per infected snail.

a substantial influence on the disparities observed in their respective growth rates. Therefore, for comparison purposes, we assume that inherent natural increase (r_2) of the competitor snail will exhibit a 15 % increment over the inherent natural increase (r_1) of the IH snail. This is closer to the 10 % assumed in a similar study conducted by Allen and Victory [29]. In addition, we assume that $r_1, r_2 \in [0, 0.5]$ because values outside this range would indicate a doubling of the population within less than two time units, and this may not be realistic given reproduction and survival characteristics of snails. In addition, we assume that both the competitor and the IH snail have the same carrying capacities. Furthermore, we assume that infected snails are more impacted by competition and that the competition coefficients are such that $e_3 < e_2 < e_1 \in [0, 1]$ when the non-host competing snail outcompetes, otherwise the control agent is outcompeted. In addition, due to the limited availability of data regarding IH snail predation, the corresponding parameter values were adjusted over a range such that a, $b \in [0, 3]$, and $t_1, t_2 \in [0, 4]$ to account for different potential predator behaviors. For example, a predator's interest in its next prey may decrease as the number of prey items consumed increases, and some may typically require a longer period of handling before they resume hunting their next prey.

4.1. Sensitivity analysis of the model parameters

We performed a Partial Rank Correlation Coefficient (PRCC) test to measure the robustness of the model to parameter values and support the qualitative results of our analyses. PRCC helps identify which input parameters have the most significant impact on the output (R_0) of the model. This information can be crucial in understanding the behavior of the model, identifying influential factors, and making informed decisions, such as determining which parameters should be targeted for intervention strategies. Hence, we used the range of parameter values in Table 2 and the expression for the basic reproduction number, R_0, in equation Eq. (12) to compute the PRCC (Fig. 2).

Parameters with positive PRCC values increase R_0 when increased and therefore lead to higher risk of infection and vice versa (Fig. 2). However, an increase in parameters with negative PRCC values including δ_2, υ_2, K_1, υ_4, υ_5, C_o, and M_o (Fig. 2) results in R_0 reduction, critical for disease extinction. Notably, snail mortality parameter, δ_2 has a greater influence on reducing R_0, suggesting that increasing δ_2 could potentially decrease the endemicity of the disease. In addition, R_0 is a decreasing function of δ_2 and υ_2 therefore it makes sense that control intervention targeting δ_2 and υ_2 would be an accepted strategy. This study underscores the incorporation of biological control measures for IH snails, with a particular focus on assessing how snail competitors and predators influence the IH snail populations. Additionally, it examines the subsequent impact on the cercaria population originating from infected IH snails, which, in turn, poses a risk of disease transmission to humans.

4.2. Impact of a snail competitor on the population dynamics of IH snails and cercariae

We use a pure competition model with a snail competitor as a control strategy to mitigate IH snail and the subsequent cercaria populations. We fix $e_3 = 0.001$ and vary the competition factors e_1 and e_2 to account

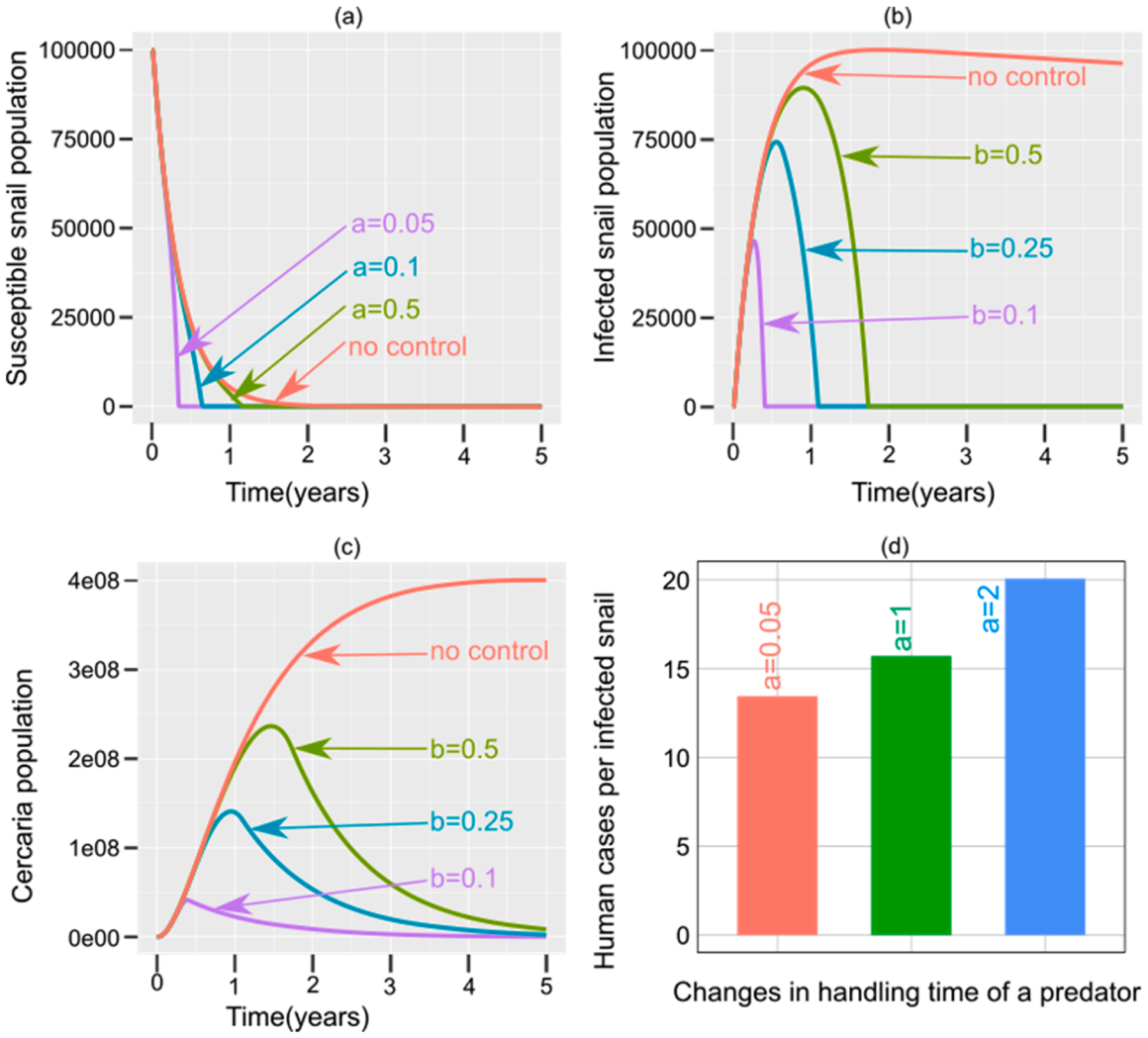

Fig. 5. Impact of varying the handling time (a, b) of a predator on (a) susceptible snail population, and (b) infected snail population. The subsequent influence on (c) infectious cercaria emerging from infected snail population, and (d) human infections per infected snail.

Table 3
Various parameter sets representing distinct combinations of both snail competitors and snail predators.

	e_1	e_2	t_1	t_2	a	b
Set 1	0.001	0.00001	0.002	2	3	0.5
Set 2	0.0001	0.00003	0.02	1	0.5	0.1
Set 3	0.00003	0.0001	0.2	0.04	0.05	0.5
Set 4	0.000001	0.001	2	0.004	0.005	1

for the variability in competition against the IH snail populations. This accounts for different potential competitive snails that may behave more specifically and competitively than the intermediate host in an ecosystem. Our findings demonstrate a substantial reduction in the number of susceptible IH snail population with increasing values of e_1 (Fig. 3a) and a reduction in infected snail population, as well as the subsequent emergence of cercaria population from infected snails, with increasing values of e_2 (Fig. 3b, 3c). As a result, there is a corresponding decrease in the number of infected individuals per infected snail (Fig. 3d). Based on our observations, snail competitors with higher competitive abilities, such as $e_1, e_2 > 0.001$, are promising as effective biological control agents. In particular, these competitors have the potential to suppress especially the population of infected IH snails. Consequently, the subsequent infective cercaria population from infected snails is reduced leading to a potential reduction in human infections and the transmission of schistosomiasis. This reduction could potentially drive the reproduction number below 1, indicating a potential for disease extinction. However, it is worth noting that some snail competitors with lower competitive abilities ($e_1, e_2 < 0.00025$) are ineffective control agents. Their coexistence can only bring about a partial reduction in the IH snail population. The number of subsequent infectious cercaria population and the corresponding human infection per infected snail are still high. Thus, the reproduction number remains higher than one, allowing the disease to persist.

4.3. Impact of a predator on the population dynamics of IH snails and cercariae

We use the predator-only model with the predator as the only control strategy. To account for variation in predation caused by several potential predators of IH snails with specific predation abilities, we vary the predation parameters. First, we investigate the effects of varying the attack rates t_1 and t_2 of susceptible and infected snails, respectively. This is done by fixing the handing times $a = 0.25$ and $b = 0.175$. The results show that increasing the predator attack rate has a negative effect on the IH susceptible snail population (Fig. 4a) and the infected snail population as well as the subsequent cercaria population (Fig. 4b, 4c). Consequently, there is a concurrent reduction in the number of infected

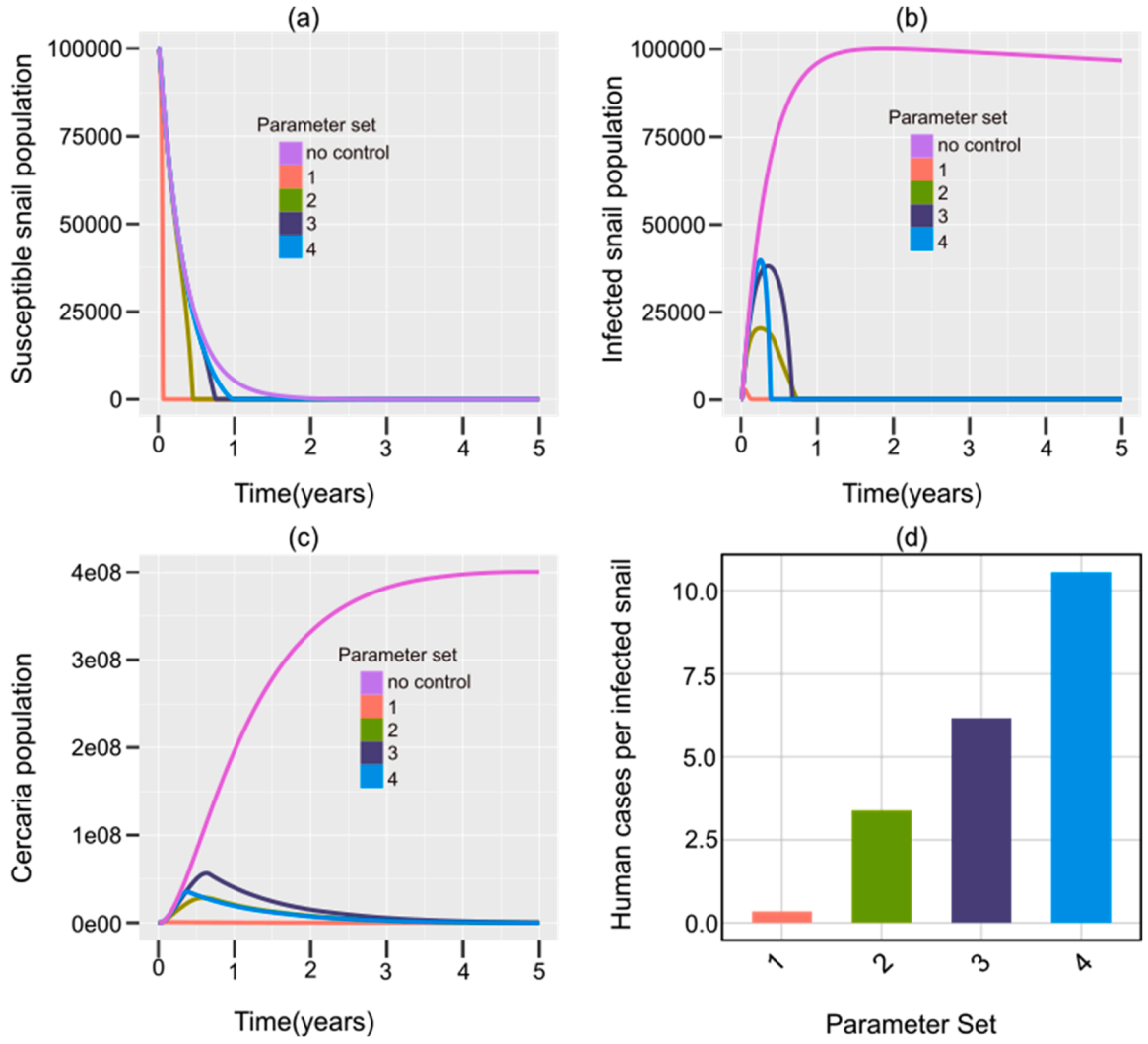

Fig. 6. Impact of varying competition factors (e_1, e_2), attack rates (t_1, t_2), and handling times (a, b) on (a) susceptible snail population, and (b) infected snail population. The subsequent influence on (c) infectious cercaria emerging from infected snail population, and (d) human infections per infected snail.

individuals per infected snail (Fig. 4d) and the disease is less prevalent. Thus, predators with higher attack rates tend to be more effective as biological control agents for IH snails (Fig. 4d). On the contrary, predators characterized by a low attack rate ($t_2 < 0.0002$ hour/day) exhibit reduced efficiency in regulating snail populations and achieving a reduction of human infections to less than one individual (Fig. 4d) and schistosomiasis may persist in the population.

Second, the effects of changing the handling times a and b of susceptible and infected snails, respectively, are examined. This is done by fixing the attack rate $t_1 = 0.02$ and $t_2 = 0.4$.

The findings indicate that reducing the handling time of the IH snail by the predator has adverse effects on both the IH susceptible snail population (Fig. 5a) and the infected snail population, together with the subsequent cercaria population (5b, 5c). In our findings, predators with shorter handling times, exemplified by values where $a = b < 0.05$, consistently prove to be more effective biological control agents in reducing the IH snail population (Fig. 5a, b), consequently leading to a reduction in the subsequent infectious cercaria population (Fig. 5c). Consequently, the occurrence of human infections per infected snail is notably lower (Fig. 5d), thereby alleviating the burden of schistosomiasis. Conversely, predators characterized by longer handling times ($b >$ 1 hour/day) have limited efficacy as control agents. They are less efficient in reducing the number of snail hosts, which, in turn, results in the persistence of high human infection rates even in the presence of such control measures (Fig. 5d). This persistence suggests that schistosomiasis may endure within the population. Thus, the dynamics ofschistosomiasis can be greatly determined by the handling time (a, b) and attack rate (t_1,t_2) of the IH snails by the potential predator.

4.4. Impact of both a snail competitor and a predator on the population dynamics of IH snails and cercariae

We investigate various scenarios employing both biological control agents. We use randomized sets of competition and predation parameters to simulate situations in which competition and/or predation can exert either a strong or weak influence, or achieve intermediate impact on IH snail populations. We use four sets of parameters as defined in Table 3.

The findings reveal that the presence of potential snail competitors and snail predators alongside IH snails can have a substantial impact on their populations, leading to variations in emerging cercariae populations and a reduction in the subsequent number of human infections per infected snail, depending on the specific parameter sets (Fig. 6). In general, decreasing e_1, t_2, a, and increasing e_2, t_1, b were found to be optimal for effective management of IH snail populations and controlling schistosomiasis. For instance, in Set 4, the combination of a snail competitor with high competitive abilities and a snail predator with strong predation capabilities, especially against infected snail

populations, results in a lower incidence of human infections compared to unity (Fig. 6d). This suggests that schistosomiasis transmission is significantly less likely to occur, potentially creating disease-free environments in areas where both biological agents are introduced or coexist naturally. However, it is important to note that certain combinations of snail competitors and snail predators can lead to high numbers of IH snails, subsequent infectious cercariae, and human infections per infected snail (Fig. 6d), which may contribute to disease persistence. In such cases, these agents are less effective in controlling the disease.

5. Discussion

We formulated and analyzed a deterministic model for the control of schistosomiasis by competitive and predatory control of the IH snails. In the analysis, we explored the feasible region, and computed the stability and characteristics of disease-free and endemic equilibrium points. Additionally, bifurcation analyses were conducted, revealing instances of backward bifurcation for specific parameter values. Sensitivity analysis of the basic reproduction number (R_0) highlighted δ_2 as the most negatively influential sensitivity parameter, thus identifying it as a potential intervention target. Therefore, the primary intervention strategy was to introducenon-host competiting snails and/or snail predators to reduce the population of IH snails and the infectious cercariae emerging from the infected snail population.

Our study reveals that the distinct competitive advantage demonstrated by competitor snails over IH snails significantly diminishes the population size of IH snails, particularly among the infected subgroup, along with a subsequent reduction in the emergence of cercariae. This finding is consistent with empirical observations from field and laboratory investigations, indicating that within a competitive ecological context, characterized by resource scarcity, outcompeted populations remain constrained in size. s As a result, some species within the population die off, while the survivors have reduced diminished reproductive rates and produce fewer infectious cercariae [45–48]. In some cases of species shift, displacement, and invasion are most likely. In particular, Pointier and McCullough [47] specifically documented a decline in the population of IH snails *Biomphalaria glabrata*, coinciding with the proliferation of competitors such as *Melanoides tuberculata* and *Thiara granifera* in certain habitats. This phenomenon results in limited interaction of the schistosomes with humans and reduced disease prevalence. Our findings are consistent with mathematical models suggesting that the use of a suitable snail competitor as a biological control can strongly influence infection dynamics, stop local transmission, and potentially eradicate schistosomiasis [10,29]. However, the reverse case is also proven, certain potential competitors are only less/average competitors and that competition has less impact on susceptible IH snails, As a result, cercariae-human interaction is high and disease can potentially spread. This result complements a field study in which *Lanistes carinatus* species were introduced into water canals and only a small decrease in the density of *B. pfeifferi* was subsequently observed [49].

Predator attack and handling times are also important factors affecting the control of schistosomiasis because they determine the extent of predation on IH snails. According to our results, schistosomiasis prevalence correlates with handling time and inversely with attack time. According to our results, predators making quick handling decisions and attacking quickly may prey more on infected IH snails than on healthy or susceptible IH snails, thus reducing their population and shedding infectious cercariae. The findings of our study are supported by studies showing that infected prey (snails) tend to be weaker, more immobile, live in more accessible areas, and change their appearances or behaviors, leaving them more vulnerable to predators [50,51], whereas healthy snails may seek refuge beneath submerged aquatic plants near transmission sites [29,15,49]. The presence of predators therefore plays a significant role in both regulating and contributing to the success of schistosomiasis control at transmission sites. A higher abundance of potential snail-eating predators increases the effectiveness of schistosomiasis control. Conversely, the low presence or absence disease control predators explains the persistence of schistosomiasis in certain areas [44,19]. For example, Madsen and Stauffer [44] observed that the decline in fish density due to overfishing in Lake Malawi was linked to increased schistosomiasis transmission in that region. On the other hand, certain predators tend to opt for alternative food sources, failing to effectively impact and reduce IH snail populations, thus limiting their potential as effective biological control agents [49]. For example, *Marisa cornuarietis* consumes not only juvenile and potentially adult vector snails but also other snails [52].

Our study shows that specific combinations involving the competitive abilities of a snail competitor and the predatory effectiveness of certain species lead to a significant reduction in the population of IH snails, with the reproductive numbers falling below unity, indicating effective disease control measures. However, a notable observation is that heightened levels of competition and predation, especially when IH snails face intense competitive pressure and exploitative predation, could potentially lead to the complete elimination of IH snail populations. This outcome is characterized by instances of species displacement and local extinction, underscoring the delicate balance between competitive and predatory interactions. Furthermore, specific scenarios emerge where the interaction disproportionately impacts infected snails rather than susceptible IH snails. This unique dynamic sustains the diversity of susceptible IH snails without triggering species loss, potentially contributing to a more resilient ecosystem. However, it is essential to acknowledge that certain predators exert a more restricted influence on IH snails, allowing infected individuals to persist. Consequently, this facilitation of infected snails' survival maintains the presence of the disease in their vicinity, emphasizing the complexity of predator-prey relationships within this context. Our findings are substantiated by several studies that demonstrated aggressive predation (e.g., by some crustaceans) and competition between potential competitor snails (e.g., *Thiara granifera* and *Melanoides tuberculata*). This led to ecosystem invasions, which resulted in extinctions and the loss of species, especially in the genera *Biomphalaria* and *Bulinus* [10,15,16,53]. Furthermore, ecologists and parasitologists have criticized some snail predators for to their consumption of non-target gastropod species and aquatic plant communities (water lilies). Consequently, the presence of non-specific predators in large natural water bodies (lakes), poses a threat to freshwater biodiversity, as seen in Lake Malawi and East African lakes [17,43]. Nevertheless, some predatory fish, such as the cichlid *Astatoreochromis alluaudi*, demonstrated only initial success and proved ineffective in sustaining long-term snail control efforts, which is likely due to their slow reproduction rate, which makes them less suitable for large-scale biocontrol initiatives [54]. This can be associated with the fact that fish reproduce too slowly to be useful in large-scale biocontrol efforts. However, it has also been reported that the reintroduction of native predators, e.g., river prawns or crayfish, has further reduced the density of IH snails [10]. Consequently, if biological agents, especially native ones, are experimentally evaluated for their competitive and predatory capabilities, integrated control measures may prove to be both cost-effective and long-lasting. This outcome aligns with the discovery that certain natural predators of disease vectors offer viable ecological approaches to disease control [55]. For instance, an optimal approach could involve using snails as competitors only when their competitive abilities match those of susceptible IH snails and to use predators with average attack rates and quicker handling times. This approach offers a naturally cost-effective in implementation, environmentally friendly, and self-sustaining strategy for managing IH snails and eliminating schistosomiasis. Thus, population growth models such as the one presented here can be helpful in hypothesis testing, landscape planning, or scenario analysis prior to the management of predator and competitor snail populations. We conclude that selective competitor-predator intervention is a way forward to control schistosomiasis in style of a nature-based and sustainable manner.

6. Future work

We propose three important tasks for the future. First, despite the benefits of using a population growth model for *a priori* testing of schistosomiasis dynamics, field and laboratory research is needed to gain a better understanding of the mechanisms responsible for generating these dynamics. Native snail competitors and snail predators need to be tested under controlled and in situ conditions to determine whether their effects on IH snails are minor and ineffective, or whether they exploit them, resulting in invasive, colonizing, or extinction behaviors.

Secondly, the structure and setup of the population growth model and its parameter values can influence the results. For example, adding a climate component to the model could be useful, as climatic variables may affect the schistosomiasis life cycle over time. Furthermore, the lack of experimental parameter values in the literature is a challenge for model parameterization.

Thirdly, there is a need to explore alternative vector control methods, including molluscicides, physical removal, and environmental modifications.

Therefore, our model with its competition rates, attack and handling times should be seen as a tool to test the effects and hypotheses of biological control methods in relation to the spread of schistosomiasis.

Supporting information

All the data used in the study are available in the paper.

CRediT authorship contribution statement

Zadoki Tabo: Writing – original draft, Validation, Software, Methodology, Formal analysis, Data curation, Conceptualization. **Livingstone Luboobi:** Writing – review & editing, Methodology, Investigation, Conceptualization. **Philipp Kraft:** Writing – review & editing, Visualization, Data curation. **Lutz Breuer:** Writing – review & editing, Supervision, Funding acquisition. **Christian Albrecht:** Writing – review & editing, Supervision, Project administration, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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