Multi-Host Transmission Dynamics of Schistosomiasis and Effective Control

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Abstract—**This paper presents a dynamic model for the transmission of schistosomiasis, which involves two definitive hostshumans and bovinesand an intermediate host, snails. The study demonstrates the positive invariance and non-negativity of the system. It outlines the conditions necessary for the existence of both disease-free and endemic equilibrium points. Additionally, it provides criteria for the local and global stability of the disease-free equilibrium point. The local stability of the endemic equilibrium point is analyzed using central manifold theory, while global stability is established through the construction of a Lyapunov function, simultaneously proving the existence of forward bifurcation in the system. A sensitivity analysis of the basic reproduction number concerning various parameters reveals that the effective contact rate between hosts and cercariae, along with the hatching rate of cercariae, are critical factors influencing the extinction of schistosomiasis. Consequently, strategies such as minimizing contact between humans and livestock in freshwater contaminated with cercariae, as well as effectively eliminating schistosomiasis eggs, can be implemented to control the disease's spread.**

Index Terms*—*Schistosoma, equilibrium point, local stability, global stability, forward bifurcation.

1 Introduction

SCHISTOSOMIASIS is a parasitic disease first described by Theodor Bilharz in 1851, and the disease was originally named bilharzia. This disease is caused by parasitic trematode worms called schistosomes and is currently the second most widespread endemic parasitic disease globally [1], [2]. Infection occurs when individuals come into contact with freshwater that contains larvae (cercariae) of these parasites. An estimated 264.4 million people required treatment for schistosomiasis in 2022 [3]. Of the 23 known Schistosoma species, Schistosoma haematobium, Schistosoma mansoni and Schistosoma japonicum mainly infect humans [4]. Schistosoma japonicum is particularly common in China, the Philippines, and Indonesia [5].

Schistosoma japonicum has a complex life cycle consisting of two free-living stages, miracidia and cercariae. And two host populations, the intermediate host snails and the definitive host mammals. In addition to humans, over 40 mammals, including bovine, sheep, and dogs, can act as final hosts for Schistosoma japonicum [6]. Its distribution closely aligns with that of the snails, making schistosomiasis typically endemic. The disease generally develops when cercariae penetrate the skin of the definitive host. After about five weeks, paired male and female adults in the final host begin to lay eggs, some of which are excreted into the environment. In freshwater, these eggs hatch and release ciliated miracidia that seek out and infect intermediate hosts (snails) to promote sporangium development. After about four weeks, the sporozoites begin to reproduce asexually, releasing thousands of swimming infective cercariae into the water. These cercariae actively search for and penetrate definitive hosts, maturing into adults within the host's body [7]. The interaction between human and animal schistosomiasis exacerbates the prevalence of the disease and complicates efforts for its prevention and control.

Mathematical modeling of schistosomiasis transmission dynamics serves as an effective method for comprehending disease transmission patterns, identifying critical factors in epidemic spread, and aiding in the development of disease control strategies. The initial mathematical model for schistosomiasis transmission was introduced by Macdonald [8] in 1965. Since then, various mathematical models have been proposed to investigate the transmission dynamics of schistosomiasis. Barbour enhanced Macdonald's model [9] in 1996, which monitored the dy-

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namics of both infected humans and snails within a community. Chiyaka et al. [7] constructed a deterministic mathematical model considering miracidia and cercariae in 2009. Gao et al. [10] modified it in 2011, adding parameters for infected human population treatment. In 2015, Zou and Ruan [11] categorized schistosomiasis epidemic areas in China into three types: plain areas with water networks, mountainous and hilly regions, and swamp and lake areas, proposing tailored control strategies for each. Gao et al. [12] modify the Barbour's two-host model with seasonal fluctuations in 2017. In 2021, Ronoh et al. [13] developed a six-dimensional differential equation model that examined schistosomiasis transmission among humans, non-human mammals, and cercariae, incorporating a saturation rate for transmission between humans and non-human mammals, while also assessing the influence of immune and environmental factors. Nur et al. [14] established a seven-dimensional differential equation model that analyzed transmission among humans, snails, miracidia, and cercariae, evaluating the effects of health education and molluscicides based on model parameters. In 2023, Zhang and zhao [15] explored a multi-host schistosomiasis model that included seasonality and temperature-dependent delays. Building on these studies, we propose a mathematical model for schistosomiasis transmission that considers intermediate host snails and final host mammals (bovines and humans), followed by an analysis of its dynamic properties.

The organization of this paper is as follows. In section 2, we propose a schistosomiasis model incorporates an intermediate host and two definitive hosts, and study the non-negative properties of solutions and the positive invariance of feasible regions. In section 3, we determine the basic reproduction numbers for the model and equilibrium points and their existence condition. and then we give some sufficient conditions for global stability of the disease-free equilibrium point. By using central manifold theory, we discuss the local stability of endemic equilibria. By constructing the Lyapunov function, we prove the global stability of endemic equilibrium point. In Section 4, numerical simulations are presented for supporting the analytic results. Finally, section 5 concludes the paper with a brief discussion.

2 Model formulation

In this section, we develop a mathematical model to primarily illustrate the transmission dynamics of Schistosomiasis japonicum in China. Numerous studies indicate that bovines are the primary source of infection for the spread of Schistosoma japonicum in the country [16], while other mammals like dogs, pigs, mice, and goats contribute minimally to the overall transmission [17]. Therefore, this article presents a mathematical model that describes the transmission dynamics of schistosomiasis among humans, bovines, and snails, while also including a contaminated environment that harbors cercaria as a transmission vector.

Let $S_h(t)$ and $I_h(t)$ denote the number of susceptible and infected humans at time t, respectively. $S_b(t)$ and $I_b(t)$ denote the number of susceptible and infected bovines at time t, respectively. $S_v(t)$ and $I_v(t)$ denote the density of susceptible and infected snail population, and $C_e(t)$ denote the density of cercaria population at time t, respectively. The schistosomiasis model is described by the following system of 7 ordinary differential equations

$$\begin{cases} \dfrac{dS_h}{dt} = \Lambda_h + \gamma I_h - \beta_h C_e S_h - \mu_h S_h, \\ \dfrac{dI_h}{dt} = \beta_h C_e S_h - (\mu_h + \delta + \gamma) I_h, \\ \dfrac{dS_b}{dt} = \lambda_b - \beta_b C_e S_b - \mu_b S_b, \\ \dfrac{dI_b}{dt} = \beta_b C_e S_b - \mu_b I_b, \\ \dfrac{dS_v}{dt} = \Lambda_v - \beta_v C_e S_v - (\mu_v + d_v) S_v, \\ \dfrac{dI_v}{dt} = \beta_v C_e S_v - (\mu_v + d_v) I_v, \\ \dfrac{dC_e}{dt} = \alpha(I_h + I_b) - \mu_c C_e, \end{cases} \tag{2.1}$$

where all parameters are positive, and $\beta_h, \beta_b, \beta_v, \gamma, \delta, \alpha, d_v, \mu_h, \mu_b, \mu_v, \mu_c \in (0, 1)$. The parameters of system (2.1) are described as in Table 1:

Table 1: Description of parameters in system (2.1)

Parameter	Description	value
β_h	effective contact rate between susceptible human and cercariae	[18]
β_b	effective contact rate between susceptible bovine and cercariae	[18]
β_v	effective contact rate between susceptible snail and cercariae	[7]
γ	recovery rate of infected humans	[19]
δ	human death rate due to infection	[20]
d_v	the killing rate of using drugs to kill snails	[7]
α	parasite egg hatch rate	estimated
Λ_h, λ_b,Λ_v	recruitment rate for humans, bovines, snails, respectively	estimated
μ_b	natural death rate of bovines	[18]
μ_h, μ_v, μ_c	natural death rate of humans, snails, cercariae, respectively	estimated

The model system (2.1) will be analyzed in a biologically feasible region Ω, given by

$$\Omega := \{(S_h, I_h, S_b, I_b, S_v, I_v, C_e) \in \mathbb{R}^7_+ : N_h \le \frac{\Lambda_h}{\mu_h}, N_b \le \frac{\lambda_b}{\mu_b}, N_v \le \frac{\Lambda_v}{\mu_v + d_v}, C_e \le \frac{\alpha(\Lambda_h \mu_b + \lambda_b \mu_h)}{\mu_h \mu_b \mu_c}\},$$

where $N_h = S_h + I_h$, $N_b = S_b + I_b$, $N_v = S_v + I_v$ represent the total number of humans, the total number of bovines, and the total number of snails, respectively.

For the model system (2.1) to be biologically meaningful, it is important to indicate that all solutions with non-negative initial values will remain non-negative when $t \geq 0$, hence we give the following Theorem.

Theorem 2.1. *If initial value* $S_h(0), I_h(0), S_b(0), I_b(0), S_v(0), I_v(0), C_e(0) \geq 0$, *then the solutions* $(S_h(t), I_h(t), S_b(t), I_b(t), S_v(t), I_v(t), C_e(t))$ *of system* (2.1) *are non-negative when* $t \geq 0$, *and* Ω *is a positive invariant set of the system* (2.1).

Proof. Let $F(t) = min\{S_h(t), I_h(t), S_b(t), I_b(t), S_v(t), I_v(t), C_e(t)\}$. Assuming exists $t_* > 0$, such that $F(t_*) = 0$, $F(t) > 0$ for $t \in [0, t_*)$, and when $t_*^+ > t_*$, $F(t_*^+) < 0$. If $F(t_*) = S_h(t_*)$, then $I_h(t_*), S_b(t_*), I_b(t_*), S_v(t_*), I_v(t_*), C_e(t_*) > 0$. From the first equation of system (2.1), we have

$$\begin{aligned}\frac{dS_h(t_*)}{dt} &= \Lambda_h + \gamma I_h(t_*) - \beta_h C_e(t_*) S_h(t_*) - \mu_h S_h(t_*),\\ &= \Lambda_h + \gamma I_h(t_*) > 0.\end{aligned}$$

If $(dS_h(t_*)/dt) > 0$, by the monotonic function property, when $t_*^+ > t_*$, we have $F(t_*^+) > 0$. This is contradict with the previous assumption. Thus, when $t \geq 0$, we have $S_h(t) \geq 0$. Similarly, when $t \geq 0$, we can prove that $I_h(t), S_b(t), I_b(t), S_v(t), I_v(t), C_e(t) \geq 0$.

Next, we prove the positively invariance of Ω for system (2.1). From system (2.1), we can get that the total number of humans satisfies differential equation

$$\begin{aligned}\frac{dN_h}{dt} &= \Lambda_h - \mu_h N_h - \delta I_h,\\ &\leq \Lambda_h - \mu_h N_h.\end{aligned}$$

This means that, $N_h(t) \to \Lambda_h/\mu_h$ as $t \to +\infty$. Similarly, $N_b(t) \to \lambda_b/\mu_m$, $N_v \to \Lambda_v/(\mu_v + d_v)$ as $t \to +\infty$.

From the 7th equation of system (2.1), we get

$$\begin{aligned}\frac{dC_e}{dt} &= \alpha(I_h + I_b) - \mu_c C_e,\\ &\leq \alpha\left(\frac{\Lambda_h}{\mu_h} + \frac{\lambda_b}{\mu_b}\right) - \mu_c C_e.\end{aligned}$$

This means that, $C_e \leq (\Lambda_h\mu_b + \lambda_b\mu_h)\alpha/(\mu_h\mu_b\mu_c)$ as $t \to +\infty$. Therefore, it is proven that Ω is a positive invariant set of System (2.1). □

3 Model analysis

3.1 Basic reproduction number

The basic reproduction number, denoted by R_0, defined as the expected number of secondary cases produced by a typical infected individual in a completely susceptible population during its entire period of infectiousness (see [21]). R_0 is often used to predict trends in disease transmission and to evaluate the effectiveness of control measures. We compute the basic reproduction number using the next generation matrix approach as described by Castillo-Chavez et al [22]. This is achieved by rewriting the model system (2.1) in the form

$$\begin{cases}\dfrac{dX}{dt} = f(X, Y, Z),\\ \dfrac{dY}{dt} = g(X, Y, Z),\\ \dfrac{dZ}{dt} = h(X, Y, Z).\end{cases} \tag{3.1}$$

where $X = (S_h, S_b, S_v), Y = (I_h, I_b, I_v), Z = C_e$. Components of X, Y and Z represent the number of susceptible, infected individuals that cannot transmit the disease and infected individuals capable of transmitting the disease, respectively. Let $U_0 = (\Lambda_h/\mu_h, \lambda_b/\mu_b, \Lambda_v/(\mu_v + d_v), 0, 0, 0, 0)$ denote the disease free equilibrium of system(3.1). Denote $\widetilde{g}(x^*, Z) = (\widetilde{g_1}(x^*, Z), \widetilde{g_2}(x^*, Z), \widetilde{g_3}(x^*, Z))$, where

$$\begin{aligned}\widetilde{g_1}(x^*, Z) &= \frac{\Lambda_h\beta_h C_e}{\mu_h(\mu_h + \delta + \gamma)},\\ \widetilde{g_2}(x^*, Z) &= \frac{\lambda_b\beta_b C_e}{\mu_b^2},\\ \widetilde{g_3}(x^*, Z) &= \frac{\Lambda_v\beta_v C_e}{(\mu_v + d_v)^2}.\end{aligned}$$

Let $A = D_Z h(X^*, \widetilde{g}(x^*, 0), 0)$, by calculation, we can get that

$$A = \alpha\left(\frac{\Lambda_h\beta_h}{\mu_h(\mu_h + \delta + \gamma)} + \frac{\lambda_b\beta_b}{\mu_b^2}\right) C_e - \mu_c C_e.$$

Then A can be written as $A = M - D$, here

$$\begin{aligned}M &= \frac{\Lambda_h\beta_h}{\mu_h(\mu_h + \delta + \gamma)} + \frac{\lambda_b\beta_b}{\mu_b^2} \geq 0,\\ D &= \mu_c > 0.\end{aligned}$$

The basic reproductive number is the spectral radius (dominant eigenvalue) of the matrix MD^{-1}, that is,

$$R_0 = \rho(MD^{-1}) = \frac{\alpha}{\mu_c}\left(\frac{\Lambda_h\beta_h}{\mu_h(\mu_h + \delta + \gamma)} + \frac{\lambda_b\beta_b}{\mu_b^2}\right). \tag{3.2}$$

3.2 Equilibrium points

Theorem 3.1. *In Ω, the disease-free equilibrium point $E_0 : (\Lambda_h/\mu_h, 0, \lambda_b/\mu_b, 0, \Lambda_v/(\mu_v + d_v), 0, 0)$ is the unique equilibrium point of system (2.1) when $R_0 \leq 1$; System (2.1) has exactly two equilibria points E_0 and $E_c : (S_h^*, I_h^*, S_b^*, I_b^*, S_v^*, I_v^*, C_e^*)$ when $R_0 > 1$, where*

$$S_h^* = \frac{\Lambda_h + \gamma I_h^*}{\mu_h + \beta_h C_e^*}, \qquad I_h^* = \frac{\beta_h \Lambda_h C_e^*}{\mu_h \gamma + (\delta + \mu_h)(\mu_h + \beta_h C_e^*)},$$
$$S_b^* = \frac{\lambda_b}{\mu_b + \beta_b C_e^*}, \qquad I_b^* = \frac{\beta_b \lambda_b C_e^*}{\mu_b(\mu_b + \beta_b C_e^*)},$$
$$S_v^* = \frac{\Lambda_v}{\mu_v + d_v + \beta_v C_e^*}, \quad I_v^* = \frac{\beta_v \Lambda_v C_e^*}{(\mu_v + d_v)(\mu_v + d_v + \beta_v C_e^*)},$$
$$C_e^* = \frac{-B + \sqrt{B^2 - 4AC}}{2A},$$

and

$$\begin{aligned} A &= \left(\beta_h \beta_b \mu_b \mu_c (\delta + \mu_h)\right)/\alpha, \\ B &= \mu_b \mu_c (\beta_b \mu_h (\mu_h + \delta + \gamma) + \beta_h \mu_b (\mu_h + \delta))/\alpha \\ &\quad - \beta_h \beta_b \Lambda_h \mu_b - \beta_h \beta_b \lambda_b (\mu_h + \delta) \\ C &= \mu_h \mu_c \mu_b^2 (\mu_h + \delta + \gamma)(1 - R_0). \end{aligned}$$

Proof. To get equilibria points of system (2.1), we need to solve

$$\begin{cases} \Lambda_h + \gamma I_h - \beta_h C_e S_h - \mu_h S_h = 0, \\ \beta_h C_e S_h - (\mu_h + \delta + \gamma) I_h = 0, \\ \lambda_b - \beta_b C_e S_b - \mu_b S_b = 0, \\ \beta_b C_e S_b - \mu_b I_b = 0, \\ \Lambda_v - \beta_v C_e S_v - (\mu_v + d_v) S_v = 0, \\ \beta_v C_e S_v - (\mu_v + d_v) I_v = 0, \\ \alpha(I_h + I_b) - \mu_c C_e = 0. \end{cases} \tag{3.3}$$

From (3.3), we obtain that

$$S_h = \frac{\Lambda_h + \gamma I_h}{\mu_h + \beta_h C_e}, \qquad I_h = \frac{\beta_h \Lambda_h C_e}{\mu_h \gamma + (\delta + \mu_h)(\mu_h + \beta_h C_e)},$$
$$S_b = \frac{\lambda_b}{\mu_b + \beta_b C_e}, \qquad I_b = \frac{\beta_b \lambda_b C_e}{\mu_b(\mu_b + \beta_b C_e)},$$
$$S_v = \frac{\Lambda_v}{\mu_v + d_v + \beta_v C_e}, I_v = \frac{\beta_v \Lambda_v C_e}{(\mu_v + d_v)(\mu_v + d_v + \beta_v C_e)},$$

and C_e satisfies $\left(AC_e^2 + BC_e + C\right) C_e = 0$, where A, B, C are expressed in the statement of the Theorem 3.1. We observe that system (2.1) always has the disease free equilibrium E_0.

Obviously, $A > 0$ and it is easy to check that

$$B > (\mu_h \mu_b \mu_c \beta_b (\mu_h + \delta + \gamma) + \beta_h \mu_c \mu_b^2 (\mu_h + \delta))(1 - R_0)/\alpha.$$

When $R_0 < 1$, we get $B > 0$ and $C > 0$. Thus, $AC_e^2 + BC_e + C = 0$ has no positive roots. When $R_0 = 1$, we get $B > 0$ and $C = 0$. Equation $AC_e^2 + BC_e + C = 0$ has nonzero negative root $C_e = -B/A$. Therefore, when $R_0 \leq 1$, E_0 is the unique equilibrium point of system (2.1). When $R_0 > 1$, we have $C < 0$, equation $AC_e^2 + BC_e + C = 0$ has a unique positive root C_e^*, given in Theorem 3.1. Thus, system (2.1) has exactly two equilibria E_0 and E_c, given in the statement of this theorem. □

3.3 Stability of equilibrium points

3.3.1 Local stability analysis for disease free equilibrium point E_0

In this subsection, we study the stability of the disease free equilibrium E_0.

Theorem 3.2. *The disease free equilibrium point E_0 of system (2.1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. We compute the Jacobian matrix at E_0 and get

$$J(E_0) = \begin{pmatrix} -\mu_h & \gamma & 0 & 0 & 0 & 0 & -\beta_h S_h^* \\ 0 & -(\mu_h + \delta + \gamma) & 0 & 0 & 0 & 0 & \beta_h S_h^* \\ 0 & 0 & -\mu_m & 0 & 0 & 0 & -\beta_m S_m^* \\ 0 & 0 & 0 & -\mu_m & 0 & 0 & \beta_m S_m^* \\ 0 & 0 & 0 & 0 & 0 & -(\mu_n + d_n) & -\beta_n S_n^* \\ 0 & 0 & 0 & 0 & -(\mu_n + d_n) & 0 & \beta_n S_n^* \\ 0 & \alpha & 0 & \alpha & 0 & 0 & -\mu_c \end{pmatrix}. \tag{3.4}$$

From the characteristic equation $|(\lambda I - J(E_0))| = 0$ of system (2.1) at E_0, it can obtain that

$$(\lambda + \mu_h)(\lambda + \mu_h + \delta + \gamma)(\lambda + \mu_m)^2(\lambda + \mu_n + d_n)^2 \left(\lambda + \mu_c - \frac{\alpha \beta_h S_h^*}{\mu_h + \delta + \gamma} - \frac{\alpha \beta_m S_m^*}{\mu_m}\right) = 0,$$

further

$$\begin{aligned} &\lambda_1 = -\mu_h < 0, \qquad \lambda_2 = -(\mu_h + \delta + \gamma) < 0, \\ &\lambda_{3,4} = -\mu_m < 0, \qquad \lambda_{5,6} = -(\mu_n + d_n) < 0, \\ &\lambda_7 = \frac{\alpha \beta_h S_h^*}{\mu_h + \delta + \gamma} + \frac{\alpha \beta_m S_m^*}{\mu_m} - \mu_c \end{aligned}$$

$$= \alpha \left(\frac{\beta_h \Lambda_h}{\mu_h(\mu_h + \delta + \gamma)} + \frac{\beta_m \lambda_m}{\mu_m^2} - \frac{\mu_c}{\alpha} \right).$$

When $R_0 < 1$, $\lambda_7 < 0$, all eigenvalues of (3.4) are negative. Therefore, the disease-free equilibrium point E_0 is locally asymptotically stable. When $R_0 > 1$, $\lambda_7 > 0$, (3.4) has a positive eigenvalue. Therefore, the disease free equilibrium point E_0 is unstable in this case. □

3.3.2 Local stability analysis for endemic equilibrium point E_c

Center manifold theory is often used to analyze the stability of non-hyperbolic equilibrium points. A non-hyperbolic equilibrium point is one in which the linearized matrix has at least one eigenvalue with zero real part. Therefore, we use the center manifold theory [23] to analyze the local asymptotic stability of endemic equilibrium point. This theory is applied to analyze the existence of forward bifurcations and backward bifurcations. When forward bifurcation occurs, it implies that the endemic equilibrium point is locally asymptotically stable for $R_0 > 1$, but in the neighborhood of 1. The theorem is as follows.

Theorem 3.3. *The system* (2.1) *exhibits a forward bifurcation at* $R_0 = 1$. *Therefore, the endemic equilibrium point* E_c *is locally asymptotically stable for* $R_0 > 1$ *but close to 1.*

Proof. In the proof of Theorem 3.2, it is shown that if $R_0 = 1$ then one eigenvalue of (3.4) is zero and the other eigenvalue is negative. Therefore, the bifurcation point will be evaluated when $R_0 = 1$. Assuming that R_0 is a function of the bifurcation parameter, we chose β_h as bifurcation parameter. By $R_0 = 1$, and solve for β_h, we get bifurcation point

$$\beta_h^* = \frac{\mu_h(\mu_h + \delta + \gamma)(\mu_c \mu_b^2 - \alpha \lambda_b \beta_b)}{\alpha \Lambda_h \mu_b^2}.$$

When $R_0 = 1$, $\beta_h = \beta_h^*$, the Jacobian matrix $J(E_0, \beta_h^*)$ of system (2.1) at point E_0 is the same as the Jacobian matrix $J(E_0)$ in (3.4).

From the previous analysis, we can get that $J(E_0, \beta_h^*)$ has an eigenvalue $\lambda = 0$, and the other eigenvalues are negative. Therefore, we can apply the Center manifold theory [23] to analyze the dynamic behavior of system (2.1) near $\beta_h = \beta_h^*$. By Theorem 4.1 of the paper [23], it can be shown that the Jacobian matrix at $\beta_h = \beta_h^*$ has a right eigenvector and a left eigenvector associated with the zero eigenvalue, which are given by

$$\vec{u} = \begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \\ u_5 \\ u_6 \\ u_7 \end{pmatrix} = \begin{pmatrix} -\frac{\beta_h \Lambda_h (u_h + \delta)}{\mu_h^2} u_7 \\ \frac{\beta_h \Lambda_h}{\mu_h(u_h + \delta + \gamma)} u_7 \\ -\frac{\beta_b \lambda_b}{\mu_b^2} u_7 \\ \frac{\beta_b \lambda_b}{\mu_b^2} u_7 \\ -\frac{\beta_n \Lambda_v}{\mu_v(\mu_v + d_v)} u_7 \\ \frac{\beta_n \Lambda_v}{\mu_v(\mu_v + d_v)} u_7 \\ u_7 \end{pmatrix},$$

and

$$\vec{v} = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)$$
$$= \left(0, \frac{\alpha}{u_h + \delta + \gamma} v_7, 0, \frac{\alpha}{u_b} v_7, 0, 0, v_7\right),$$

respectively, where $u_7 > 0$, $v_7 > 0$. Before applying center manifold theory [23], we make small change as follows, let

$$S_h = x_1, I_h = x_2, S_b = x_3, I_b = x_4,$$
$$S_v = x_5, I_v = x_6, C_e = x_7,$$

and

$$X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7)^T,$$

then the system (2.1) can be written in the following form:

$$dX/dt = f(X) = (f_1, f_2, f_3, f_4, f_5, f_6, f_7). \tag{3.5}$$

where

$$\begin{aligned} f_1 &= \Lambda_h + \gamma x_2 - \beta_h x_1 x_7 - \mu_h x_1, \\ f_2 &= \beta_h x_1 x_7 - (\mu_h + \delta + \gamma) x_2, \\ f_3 &= \lambda_m - \beta_m x_3 x_7 - \mu_m x_3, \\ f_4 &= \beta_m x_3 x_7 - \mu_m x_4, \\ f_5 &= \Lambda_n - \beta_n x_5 x_7 - (\mu_n + d_n) x_5, \\ f_6 &= \beta_n x_5 x_7 - (\mu_n + d_n) x_6, \\ f_7 &= \alpha(x_2 + x_4) - \mu_c x_7. \end{aligned}$$

For system (3.5), the associated non-zero partial derivatives of $f(X)$ at E_0 for the model are given by

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_7}(E_0; \beta_h^*) = \beta_h^*, \quad \frac{\partial^2 f_2}{\partial x_7 \partial x_1}(E_0; \beta_h^*) = \beta_h^*,$$
$$\frac{\partial^2 f_4}{\partial x_3 \partial x_7}(E_0; \beta_h^*) = \beta_b, \quad \frac{\partial^2 f_4}{\partial x_7 \partial x_3}(E_0; \beta_h^*) = \beta_b,$$
$$\frac{\partial^2 f_2}{\partial x_7 \partial \beta_h}(E_0; \beta_h^*) = \frac{\Lambda_h}{\mu_h}.$$

From this, we obtain

$$a = \Sigma_{k,i,j=1}^{n} v_k u_i u_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0; \beta_h^*)$$

$$= v_2u_1u_7\left(\frac{\partial^2 f_2}{\partial x_1\partial x_7}(E_0;\beta_h^*)+\frac{\partial^2 f_2}{\partial x_7\partial x_1}(E_0;\beta_h^*)\right)$$
$$+ v_4u_3u_7\left(\frac{\partial^2 f_4}{\partial x_3\partial x_7}(E_0;\beta_h^*)+\frac{\partial^2 f_4}{\partial x_7\partial x_3}(E_0;\beta_h^*)\right)$$
$$= -2\alpha v_7u_7^2\left(\frac{\beta_h\Lambda_h(\mu_h+\delta)}{\mu_h^2(\mu_h+\delta+\gamma)}+\frac{\beta_b\lambda_b}{\mu_b^3}\right)<0,$$

and

$$\begin{aligned} b =&\Sigma_{k,i=1}^n v_ku_i\frac{\partial^2 f_k}{\partial x_i\partial\beta_h}(E_0;\beta_h^*)\\ &= v_2u_7\frac{\partial^2 f_2}{\partial x_7\partial\beta_h}(E_0;\beta_h^*)\\ &= \frac{\Lambda_h}{\mu_h}v_2u_7>0. \end{aligned}$$

Because $a<0$, $b>0$, according to Theorem 4.1 in [23], when $\beta_h>\beta_h^*$, i.e. $R_0>1$, the system (2.1) will experience forward bifurcation and a positive locally asymptotically stable equilibrium point will appear. In Theorem (3.1), it has been proven that the system has a unique positive equilibrium point, which is the endemic equilibrium point E_c. Therefore, when $R_0>1$ and approaches 1, E_c is locally asymptotically stable. □

3.3.3 Global stability of the disease free equilibrium point E_0

Next, we prove the global asymptotic stability of the disease free equilibrium point. In order to prove the global asymptotic stability of E_0, we adopt the method in [22].

Rewrite the system (2.1) as follows:

$$\begin{cases}\dfrac{dX}{dt}=F(X,Z),\\ \dfrac{dZ}{dt}=G(X,Z), \qquad G(X,\mathbf{0})=0,\end{cases} \tag{3.6}$$

where, $X=(S_h,S_b,S_v)$ represents the number of uninfected individuals, $Z=(I_h,I_b,I_v,C_e)$ represents the number of infected and infectious individuals. $U_0=(X_*,\mathbf{0})=(\Lambda_h/\mu_h,\lambda_h/\mu_b,\Lambda_v/(\mu_v+d_v),0,0,0,0)$ represents the disease free equilibrium point of system (3.6), here $X_*=(\Lambda_h/\mu_h,\lambda_b/\mu_b,\Lambda_v/(\mu_v+d_v))$.

According to [22], when system (3.6) satisfies the following conditions (H1) and (H2), the global stability of the disease free equilibrium point U_0 can be obtained.

(H1) $dX/dt=F(X,0)$, X_* is globally asymptotically stable.

(H2) $G(X,Z)=AZ-\hat{G}(X,Z)$, $\hat{G}(X,Z)\geq 0$ for $(X,Z)\in\Omega$, where $A=D_ZG(X_*,0)$ is an M matrix (the off-diagonal elements A are nonnegative) and Ω is the region where the model makes biological sense.

Theorem 3.4. *The disease free equilibrium point U_0 of system (3.6) is globally asymptotically stable if $R_0<1$. That is, the disease free equilibrium point E_0 of the system (2.1) is globally asymptotically stable.*

Proof. In our case, from the system $dX/dt=F(X,0)$, we obtain that

$$\begin{cases}\dfrac{dS_h}{dt}=\Lambda_h-\mu_hS_h,\\ \dfrac{dS_b}{dt}=\lambda_b-\mu_bS_b,\\ \dfrac{dS_v}{dt}=\Lambda_v-(\mu_v+d_v)S_v\end{cases} \tag{3.7}$$

The coefficient matrix of the system (3.7) at the $X_*=(\Lambda_h/\mu_h,\lambda_h/\mu_b,\Lambda_v/(\mu_v+d_v))$ is

$$J(x_*)=\begin{pmatrix}-\mu_h & 0 & 0\\ 0 & -\mu_b & 0\\ 0 & 0 & -(\mu_v+d_v)\end{pmatrix}.$$

It eigenvalues are all negative. According to the Routh-Hurwitz criterion [24], x_* is globally asymptotically stable.

From the system (3.6), we obtained that

$$A=\begin{pmatrix}-(\mu_h+\delta+\gamma) & 0 & 0 & \frac{\beta_h\Lambda_h}{\mu_h}\\ 0 & -\mu_b & 0 & \frac{\beta_b\lambda_b}{\mu_b}\\ 0 & 0 & -(\mu_v+d_v) & \frac{\beta_v\Lambda_v}{\mu_v+d_v}\\ \alpha & \alpha & 0 & -\mu_c\end{pmatrix}.$$

$$\hat{G}(X,Z)=\begin{pmatrix}\left(\frac{\Lambda_h}{\mu_h}-S_h\right)\beta_hC_e\\ \left(\frac{\lambda_b}{\mu_b}-S_b\right)\beta_mC_e\\ \left(\frac{\Lambda_v}{\mu_v+d_v}-S_v\right)\beta_vC_e\\ 0\end{pmatrix}.$$

Obviously, the non-diagonal elements of A are nonnegative, so A is a M-matrix. For $S_h\leq\Lambda_h/\mu_h$, $S_b\leq\lambda_b/\mu_b$, $S_v\leq\Lambda_v/(\mu_v+d_v)$ in Ω, so the $\hat{G}(X,Z)\geq 0$. Therefore, the system (3.6) satisfies conditions (H1) and (H2), and Theorem 3.5 is proved. □

3.3.4 Global stability of the endemic equilibrium point E_c

In this subsection, we prove the global asymptotic stability of the endemic equilibrium point E_c. In order to prove the global asymptotic stability of E_c, we constructing the Lyapunov function use the approach in [25].

Theorem 3.5. *When $R_0 > 1$, the endemic equilibrium point E_c of the system (2.1) is globally asymptotically stable if $r = 0$ and $C_e/C_e^* \leq 1$.*

Proof. Constructing the Lyapunov function as follow

$$\begin{aligned} V = \; & \frac{I_h^* + I_b^*}{I_b^*}\Big(S_h - S_h^* - S_h^* ln\frac{S_h}{S_h^*} + I_h - I_h^* - I_h^* ln\frac{I_h}{I_h^*}\Big) \\ & + \frac{\beta_h S_h^*}{\beta_b S_b^*}\Big(S_b - S_b^* - S_b^* ln\frac{S_b}{S_b^*} + I_b - I_b^* - I_b^* ln\frac{I_b}{I_b^*}\Big) \\ & + \Big(S_v - S_v^* - S_v^* ln\frac{S_v}{S_v^*} + I_v - I_v^* - I_v^* ln\frac{I_v}{I_v^*}\Big) \\ & + \frac{\beta_h C_e^* S_h^*}{\alpha I_b^*}\Big(C_e - C_e^* - C_e^* ln\frac{C_e}{C_e^*}\Big). \end{aligned}$$

Differentiating V along system (2.1) gives

$$\begin{aligned} V' = \; & \frac{I_h^* + I_b^*}{I_b^*}\Big(\Big(1 - \frac{S_h^*}{S_h}\Big)S_h' + \Big(1 - \frac{I_h^*}{I_h}\Big)I_h'\Big) \\ & + \frac{\beta_h S_h^*}{\beta_b S_b^*}\Big(\Big(1 - \frac{S_b^*}{S_b}\Big)S_b' + \Big(1 - \frac{I_b^*}{I_b}\Big)I_b'\Big) \\ & + \Big(\Big(1 - \frac{S_v^*}{S_v}\Big)S_v' + \Big(1 - \frac{I_v^*}{I_v}\Big)I_v'\Big) \\ & + \frac{\beta_h C_e^* S_h^*}{\alpha I_b^*}\Big(\Big(1 - \frac{C_e^*}{C_e}\Big)C_e'\Big). \end{aligned} \tag{3.8}$$

At endemic equilibrium state, we have

$$\begin{aligned} & \Lambda_h = \beta_h C_e^* S_h^* + \mu_h S_h^* - \gamma I_h^*, \mu_h + \delta + \gamma = \frac{\beta_h C_e^* S_h^*}{I_h^*}, \\ & \lambda_b = \beta_b C_e^* S_b^* + \mu_b S_b^*, \mu_b = \frac{\beta_b C_e^* S_b *}{I_b^*}, \\ & \Lambda_v = \beta_v C_e^* S_v^* + (\mu_v + d_v) S_v^*, \\ & \mu_c = \frac{\alpha(I_h^* + I_b^*)}{C_e^*}, \mu_v + d_v = \frac{\beta_v C_e^* S_v^*}{I_v^*}. \end{aligned} \tag{3.9}$$

Substitute equation (3.9) into equation (3.8) and expand to simplify it, we get

$$\begin{aligned} V' = & -\frac{\mu_h(I_h^* + I_b^*)}{I_b^*}\frac{(S_h - S_h^*)^2}{S_h} - \frac{\mu_b \beta_h S_h^*}{\beta_b S_b^*}\frac{(S_b - S_b^*)^2}{S_b} \\ & - \frac{(\mu_v + d_v)(S_v - S_v^*)^2}{S_v} + r I_h^*\Big(\frac{S_h^*}{S_h} + \frac{I_h}{I_h^*} - \frac{I_h S_h^*}{I_h^* S_h} - 1\Big) \\ & + \frac{I_h^* \beta_h C_e^* S_h^*}{I_b^*}\Big(3 - \frac{S_h^*}{S_h} - \frac{C_e^* I_h}{C_e I_h^*} - \frac{I_h^* C_e S_h}{I_h C_e^* S_h^*}\Big) \\ & + \beta_h C_e^* S_h^*\Big(5 + \frac{C_e}{C_e^*} - \frac{S_h^*}{S_h} - \frac{I_h}{I_h^*} - \frac{I_h^* C_e S_h}{I_h C_e^* S_b^*} \\ & - \frac{S_b^*}{S_b} - \frac{I_b C_e^*}{I_b^* C_e} - \frac{I_b^* C_e S_b}{I_b C_e^* S_b^*}\Big) \\ & + \beta_v C_e^* S_v^*\Big(2 + \frac{C_e}{C_e^*} - \frac{S_v^*}{S_v} - \frac{I_v}{I_v^*} - \frac{I_v^* C_e S_v}{I_v C_e^* S_v^*}\Big). \end{aligned}$$

With the assumption that $r = 0$ and $C_e/C_e^* \leq 1$, by the arithmetic-geometric mean inequality, it follows that

$$V' \leq 0.$$

Moreover, the equality $V' = 0$ holds if and only if $S_h = S_h^*$, $I_h = I_h^*$, $S_b = S_b^*$, $I_b = I_b^*$, $S_v = S_v^*$, $I_v = I_v^*$ and $C_e = C_e^*$. Therefore, E_c of the system (2.1) is globally asymptotically stable when $R_0 > 1$ and $r = 0, C_e/C_e^* \leq 1$. □

4 Numerical simulations

In this section, some numerical simulations are used to verify the correctness of the above theory. Meanwhile, through the sensitivity analysis of the model parameters, the control strategy of schistosoma was proposed. The initial value of the numerical simulation in this section is the same, which is $(S_h(0), I_h(0), S_b(0), I_b(0), S_v(0), I_v(0), C_e(0)) = (200, 20, 200, 20, 500, 50, 100)$.

4.1 Parameter sensitivity analysis

Uncertainty and sensitivity analysis techniques help us to evaluate and control the uncertainty in the model output generated by the uncertainty in the parameter input. When the output reaches the set value, the corresponding maximum and minimum values of the parameters are the variation range of the parameters, which is the basic time sensitivity measurement method [26]. Temporal sensitivity measurement is also known as a local analysis method because it aims to obtain an address point estimate rather than the entire distribution. In sensitivity analyses, we used Latin Hypercube Sampling followed by transformation on a logarithmic scale.

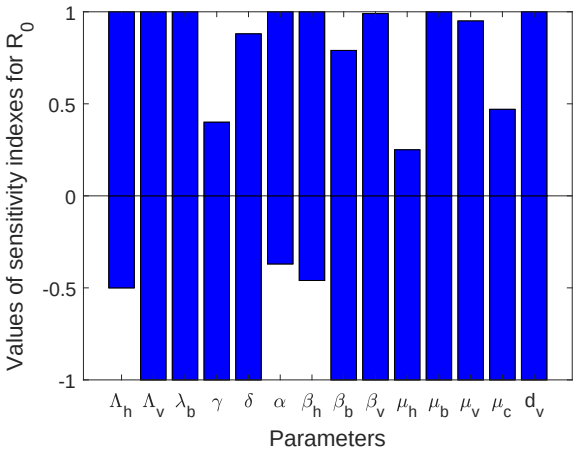


Fig. 1: Parameter sensitivity analysis for $R_0 > 1$.

In Fig.1 and 2, we present the analysis of the sensitivity and uncertainty of the parameters, with the length of the

blue bands indicating the sensitivity of the parameters. The shorter the band, the higher the sensitivity of the corresponding parameter. A positive part indicates an increase in the basal value and a negative part indicates a decrease in the basal value. In Fig.1, the sensitivity analysis of the parameters to changes in the magnitude of R_0 is depicted when $R_0 > 1$. In Fig.2, the sensitivity analysis of the amount of presence of I_h, I_b, I_v and C_e on the relevant parameters is indicated. In Fig.2(i-iv), we see that I_h, I_b, I_v and C_e are all most sensitive to the recruitment rate Λ_h of definitive host humans, the hatching rate α of parasite eggs, the effective contact rate β_h between susceptible and cercariae, and the natural mortality rate μ_h of humans.

4.2 Time series and bifurcation

In Fig.3 (i), taking $(\Lambda_h, \Lambda_b, \lambda_v, \gamma, \delta, \alpha, \beta_h, \beta_b, \beta_v, \mu_h, \mu_b, \mu_v, \mu_c, d_v) = (400, 20, 200, 1/43, 0.000027, 0.0092, 0.001, 0.00186, 0.00615, 0.384, 0.35, 0.000569, 1/30, 0.3)$. In this case, the basic reproduction number $R_0 < 1$, the disease-free equilibrium point E_0 of the system (2.1) is globally asymptotically stable, which is consistent with Theorem 3.5. In Fig.3 (ii), taking $(\Lambda_h, \Lambda_b, \lambda_v, \gamma, \delta, \alpha, \beta_h, \beta_b, \beta_v, \mu_h, \mu_b, \mu_v, \mu_c, d_v) = (400, 20, 200, 1/43, 0.000027, 0.0092, 0.00406, 0.00186, 0.00615, 0.384, 0.35, 0.000569, 1/30, 0.3)$. In this case, the basic reproduction number $R_0 > 1$, the endemic equilibrium point E_c of the system (2.1) is locally asymptotically stable, which is consistent with Theorem 3.3.

Taking $\beta_h \in (0.0001, 0.004)$, the other parameters are the same as in Fig.3 (i). In Fig.4, we give the bifurcation diagram for the system (2.1) which shows an exchange of stability between disease-free and endemic equilibria at $R_0 = 1$. $(S_h(0), I_h(0), S_b(0), I_b(0), S_v(0), I_v(0), C_e(0)) = (200, 20, 200, 20, 500, 50, 100)$.

Next, we analyze the influence of contact rates β_h on I_h, I_b, I_v, and the influence of contact rates β_b on I_h, I_b, I_v, respectively. Changing β_h from 0.005 to 0.008, the other parameters are the same as in Fig.3(i), as shown in Fig.5(i),(ii),(iii), the number of infected individual I_h, I_b, I_v decreases as infection ratios β_h decrease. Changing β_b from 0.001 to 0.004, the other parameters are the same as in Fig.3 (ii), in Fig.5 (iv),(v),(vi), the number of infected individual I_h, I_b, I_v decreases as infection ratios β_b decrease. Further, From Fig.5(iv) we can see that the infected bovines play an important role in the human-to-human transmission of schistosomiasis, and killing infected bovines will help suppress human-to-human transmission of schistosomiasis. However, it is not enough to reduce the effective contact rate between susceptible bovine and cercariae alone, and it needs to be combined with other intervention measures.

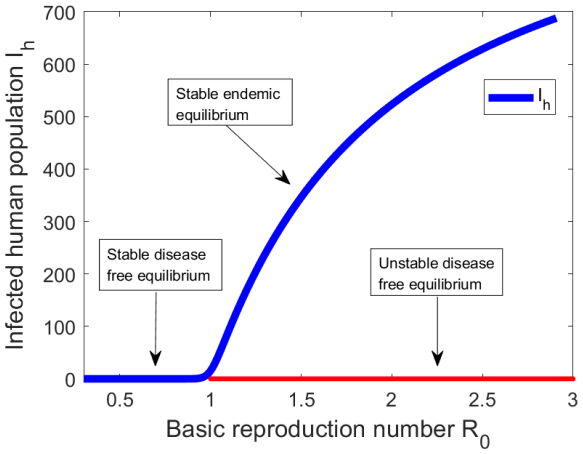


Fig. 4: forward bifurcation diagram

In Fig.7, we show the influence of α on the C_e and I_h. Changing α from 0.007 to 0.01, the other parameters are the same as in Fig.3(i), as shown in Fig.7(i),(ii), the number of infected human populations I_h and the number of cercaria populations C_e both decreased with the decrease of α. It can be seen that the value of α has a significant impact on the extinction of cercariae and the reduction of the number of infected individuals.

As can be seen from the expression in (3.2), R_0 is a strictly increasing function for the parameters β_h, β_b and α, and a strictly decreasing function with respect to parameter γ. In Fig.6, we present some figures to show how the basic reproduction number R_0 changes in terms of various values of some parameters. In Fig.6(i), we take $\beta_h \in (0, 1)$, the other parameters are the same as in Fig.3(i), we observe that the value of R_0 rapidly exceeds 1 as the parameter β_h increases. But in Fig.6(ii) we observe that R_0 never falls below 1 no matter how β_b varies between 0 and 1 when the other parameters are the same as in Fig.3(ii). Thus, the single decreasing of contact rate is insufficient for the complete control of schistosomiasis transmission. In Fig.6(iii), we observe that as the value of the parameter α increases, the value of R_0 rapidly exceeds 1, and in Fig.6(iv) we observe that R_0 decreases as γ increase, whereas it is only when γ is very close to 1 that R_0 is less than 1. This means that simply improving the recovery rate of the human population is also not enough to control of schistosomiasis transmission. As shown in Fig.6(v) and (vi), when we simultaneously reduce the contact rate and improve the recovery rate γ or decrease the hatch rate α, R_0 can quickly decrease to less than 1.

From the above numerical analysis, it can be seen that the effective contact rate β_h, β_b, hatching rate α and recovery

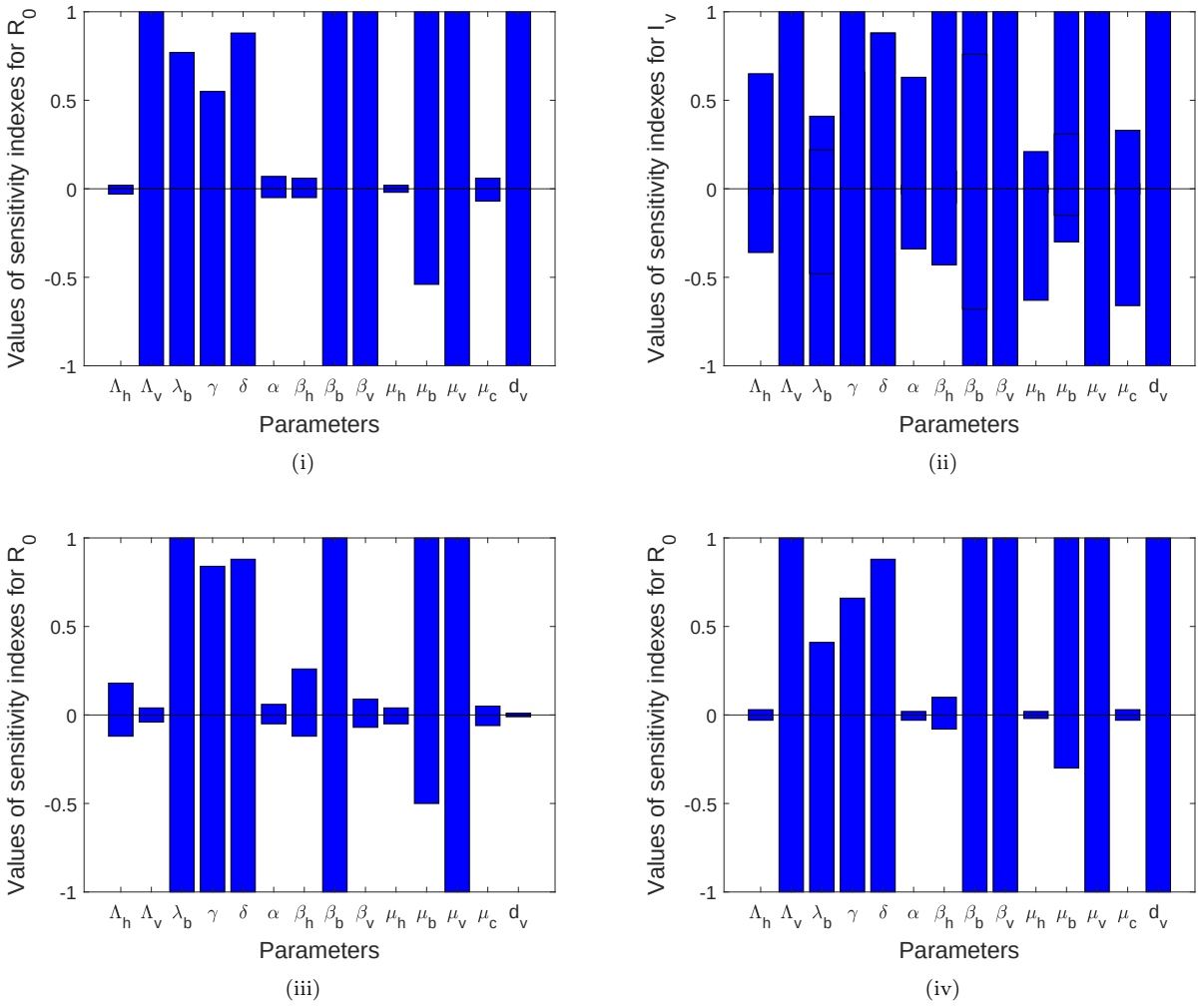


Fig. 2: Sensitivity analysis of the parameters associated with the existence of I_h, I_b, I_v, C_e for $R_0 > 1$.

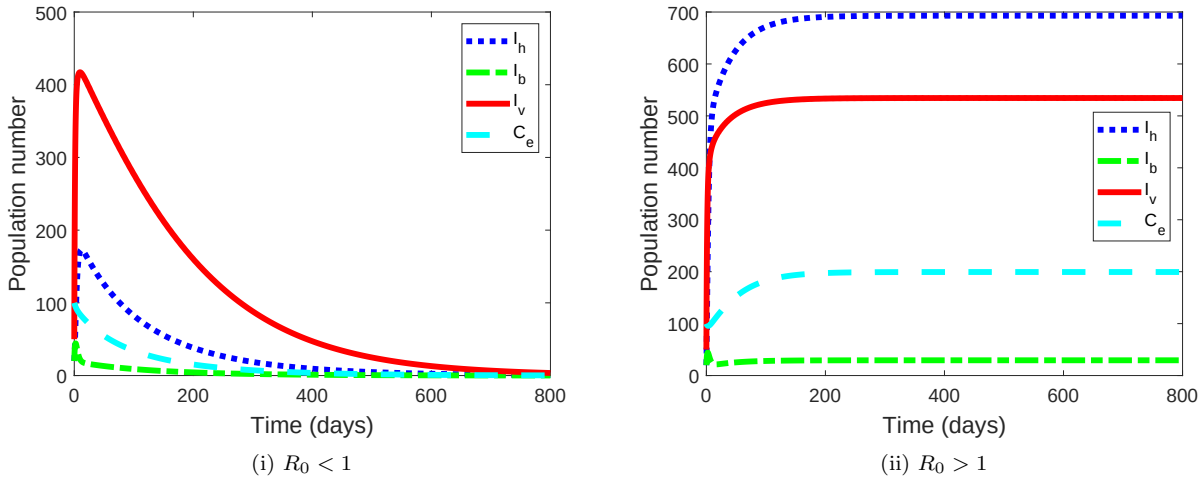
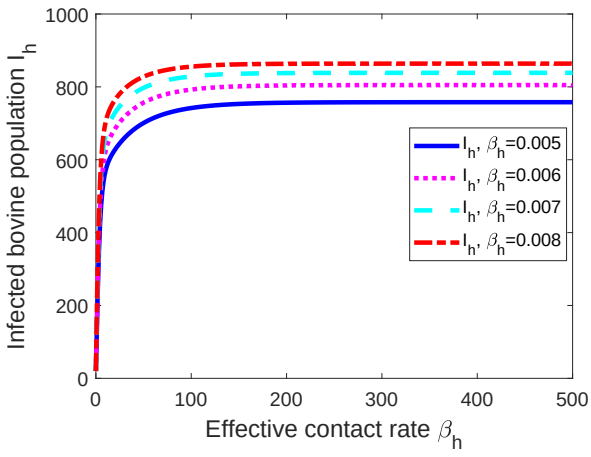
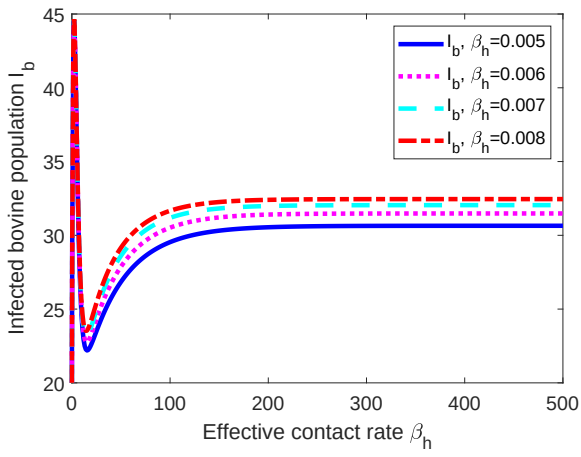


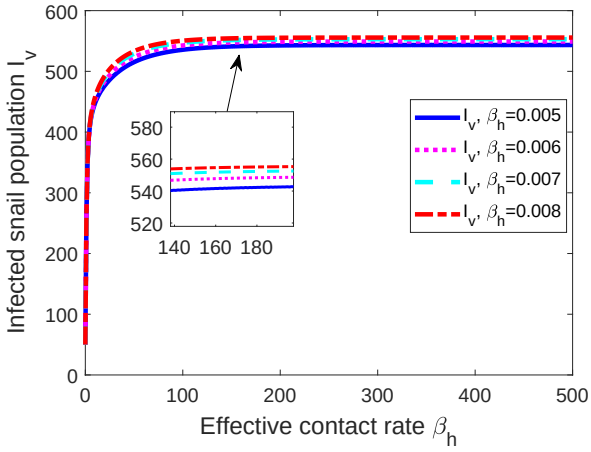
Fig. 3: I_h, I_b, I_v, C_e extinction when $R_0 < 1$ and existence when $R_0 > 1$, respectively.



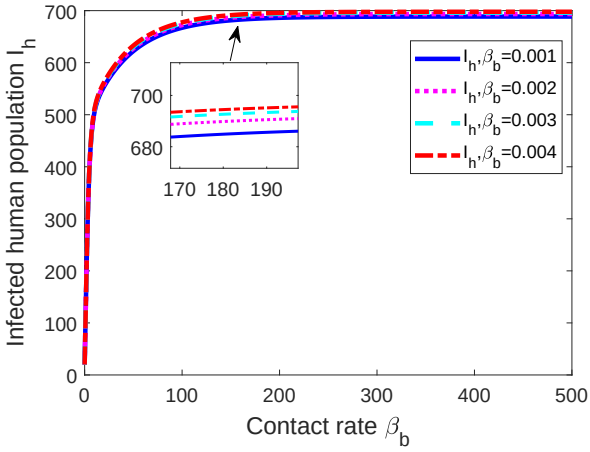
(i) β_h influence on I_h



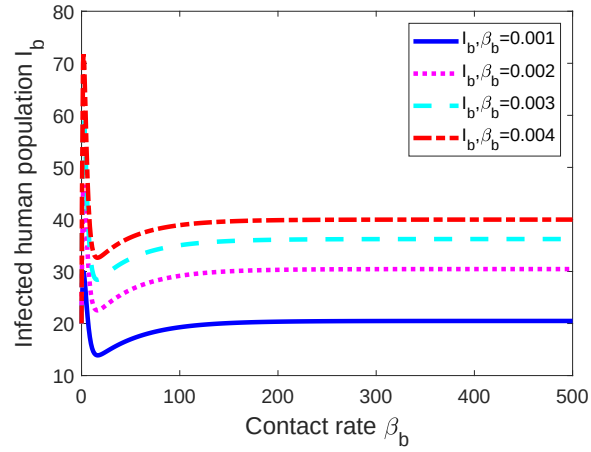
(ii) β_h influence on I_b



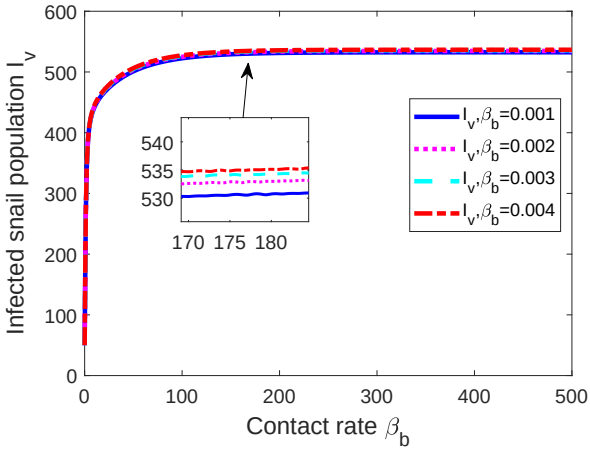
(iii) β_h influence on I_v



(iv) β_b influence on I_h

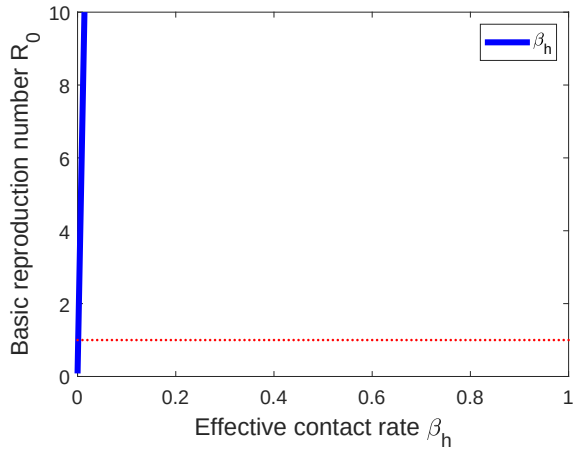


(v) β_b influence on I_b

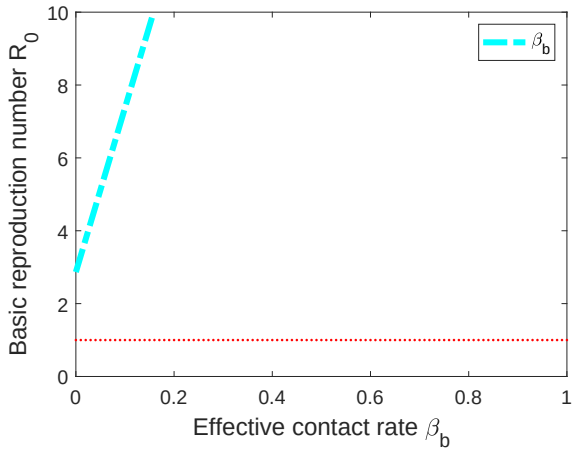


(vi) β_b influence on I_v

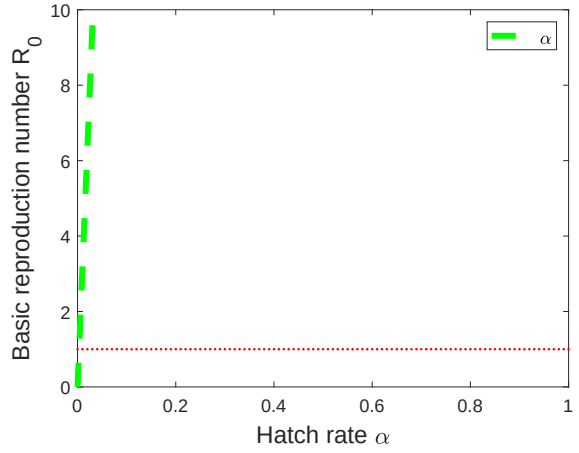
Fig. 5: The impact of contact rate on disease transmission



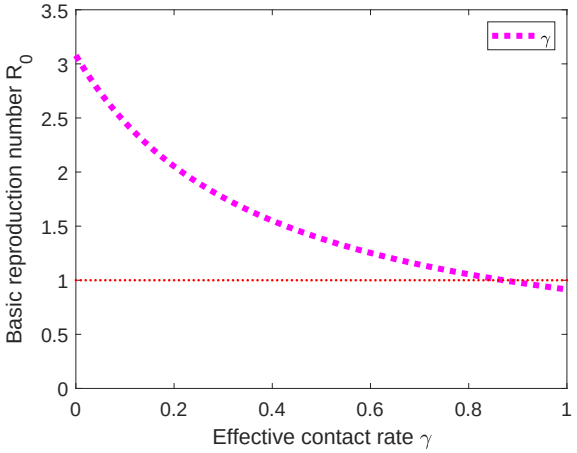
(i) The dependence of R_0 on β_h



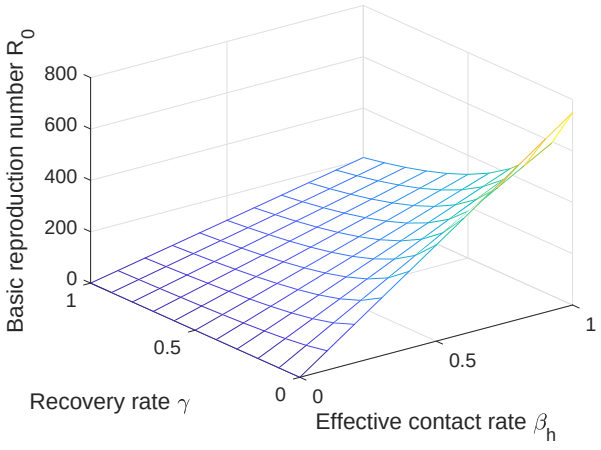
(ii) The dependence of R_0 on β_b



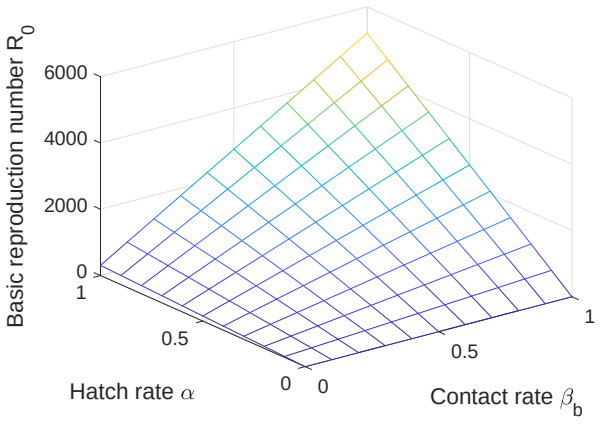
(iii) The dependence of R_0 on α



(iv) The dependence of R_0 on γ

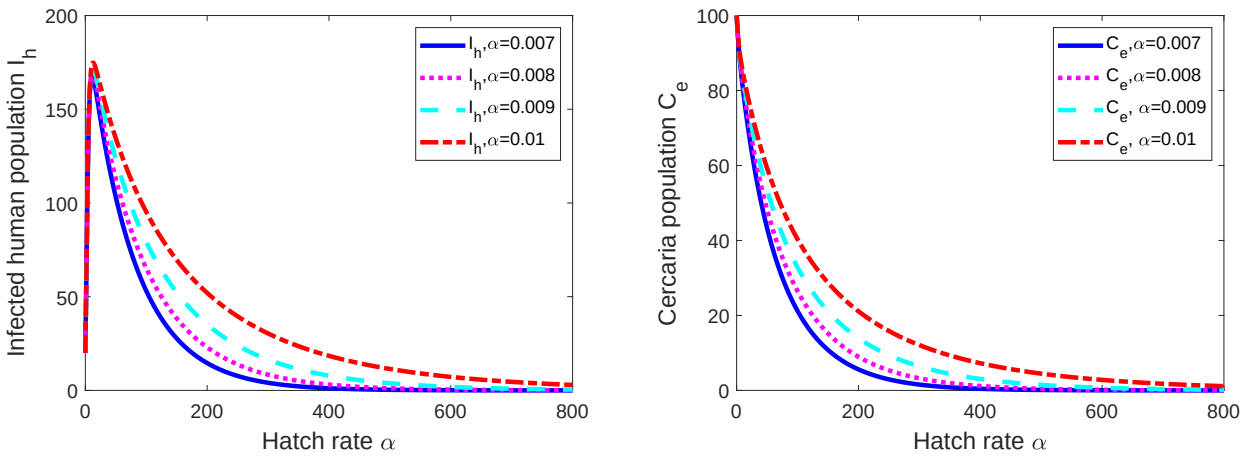


(v) Simultaneously changing β_h and γ



(vi) Simultaneously changing β_b and α

Fig. 6: Changes of R_0



(i) Influence of α on I_h

(ii) Influence of α on C_e

Fig. 7: Influence of α on the C_e and I_h

rate of infected humans γ are key parameters for controlling the spread of schistosomiasis. Therefore, reducing the hatching rate of cercariae, reducing the effective contact rate between humans and cattle with cercariae, and improving the cure rate of humans are effective measures to control the spread of schistosomiasis. However, simply improving the cure rate of humans is not enough, and it needs to be combined with other intervention measures. In fact, due to the fact that the only drug currently available for treating schistosomiasis infection is praziquantel, which has its limitations as it cannot prevent reinfection and continuous use may lead to parasite resistance [27], we also need to combine other measures to control the spread of schistosomiasis. Therefore, in epidemic areas, measures such as increasing hygiene and health education on schistosomiasis, establishing grazing prohibition areas, effectively avoiding contact between humans and livestock with freshwater infected with cercariae, concentrating the treatment of human and animal feces, effectively eliminating the eggs produced by schistosomiasis, and preventing the hatching and reproduction of eggs through medication can be taken to control the spread of schistosomiasis.

5 Conclusions and Future Work

In this paper, we formulate a dynamical model to study the transmission dynamics of schistosomiasis with multi-host. The results from our numerical simulations align with those from our theoretical analysis. The transmission of schistosomiasis can be controlled by avoiding contact with infected water and decreasing the hatching rate of cercaria. By the sensitivity analysis of the model parameters, we obtain that the infected bovines play an important role in the spread of schistosomiasis among humans, and killing the infected bovines will be useful to suppress transmission of schistosomiasis among humans. In addition, improving the cure rate of infected individuals is also very helpful in controlling the spread of infected cases. However, relying solely on one of these two measures is not enough to completely eradicate schistosomiasis, and it needs to be combined with other intervention measures. Because schistosomiasis has a certain latency, the incubation period is very important to control the prevalence of schistosomiasis, and a longer incubation period is more conducive to the prevention and control of schistosomiasis. Thus, it is important to consider whether to incorporate the latency of schistosomiasis into a time-delay model, as this could introduce new dynamic behaviors that worthy further investigation.

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