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Hindawi
Journal of Mathematics
Volume 2022, Article ID 2094979, 18 pages
https://doi.org/10.1155/2022/2094979

Research Article

Modelling the Role of Treatment, Public Health Education, and Chemical Control Strategies on Transmission Dynamics of Schistosomiasis

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Received 5 September 2022; Revised 30 November 2022; Accepted 7 December 2022; Published 19 December 2022

Academic Editor: Kenan Yildirim

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In this study, a mathematical model for the transmission dynamics and control of schistosomiasis is studied using a system of nonlinear ordinary differential equations. The basic reproduction number $\mathcal{R}_0$ for the model is obtained, and its dependence on model parameters is discussed. Analytical results reveal that the disease-free equilibrium point is globally stable if and only if the basic reproduction number $\mathcal{R}_0 < 1$, indicating that the disease would be wiped out of the community, and the endemic equilibrium point is globally stable if $\mathcal{R}_0 > 1$ and the disease would persist at the endemic steady state. Numerical simulations reveal that a combination of treatment, public health education, and chemical control intervention strategies significantly increased the number of susceptible human population and susceptible snail population while significantly decreasing the number of infected humans, miracidia, infected snails, and cercariae. The results further indicate that a combination of treatment, public health education, and chemical control intervention strategies can effectively manage the transmission of schistosomiasis in endemic areas.

1. Introduction

Schistosomiasis, commonly known as bilharzia or snail fever, is a disease caused by parasitic flukes of genus *Schistosoma*. It is listed among the neglected tropical diseases (NTDs) and is endemic in many countries, particularly in tropical countries of Africa, South America, and Asia. Schistosomiasis is the second most devastating tropical disease after malaria [1], and its prevalence has continued to prevail amidst efforts to curb it.

The human is the definitive host of the parasite and the snail is the intermediate host. The snail lives in fresh, shallow, and slow running water sources. The three main schistosomes that infect humans are *Schistosoma japonicum*, *Schistosoma mansoni* which cause intestinal and hepatosplenic disease, and *Schistosoma haematobium* that causes urogenital pathology. Each species of the schistosome is transmitted by a snail of different species; however, the species share the same pattern in the reproductive cycle [1–3].

The infection cycle begins when an infected host eliminates eggs of the parasite through urine (*Schistosoma haematobium*) or feaces (*Schistosoma mansoni* and *Schistosoma japonicum*) into fresh water bodies [2]. The excreted eggs can stay up to seven days and hatch into miracidia that infect fresh water snails of appropriate species [4]. The miricidia then multiply through asexual reproduction within the body of the snail [1], and within 4–6 weeks after infection, the snails release cercariae into water which can then penetrate the skin of a new definitive host (human) and take about 4–6 weeks to mature in the blood vessels of the urinary or the digestive system. The cercariae continue to develop and mature to adult size and pair in sexual reproduction [5]. The period between cercariae penetration of the human skin and release of schistosome eggs in human waste takes about five weeks [6] and subsequent egg laying continues the life

cycle of the parasite. Drinking water or eating food that has been washed with untreated water can also lead to infection.

The disease is characterized by an itchy skin within the first few hours of penetration, fever, cough, headache, difficulty in breathing, loss of appetite and weight, diarrhea and blood in stool. In advanced cases, children develop thin legs, seizures and spinal cord inflammation which lead to physical and mental development problems [5, 7]. However, most of these conditions can be reversed and prevented through early and regular treatment with praziquantel [8]. Globally, 76.9 million people received treatment for schistosomiasis in 2020, and of these, 59.9 million were school aged children (55.2 million in African region) and 17 million adults (14.5 million in African region) [9].

Studies to determine the effectiveness of the control strategies on schistosomiasis have been undertaken before. Gao et al. [10] studied the effect of treatment and snail control on schistosomiasis transmission, Das et al. [11] studied the optimal control of snail population and sanitation and treatment on schistosomiasis transmission, Nur et al. [12] determined the effect of health education and snail control on schistosomiasis transmission, and Kanyi et al. [13] determined the effect of treatment and sanitation on schistosomiasis transmission. Although there have been studies that address the role of reducing the intermediate host population (snail population) in the spread and control dynamics of schistosomiasis, the combined effect of treatment, public health education, and chemical control interventions on the disease transmission dynamics has not been explicitly established. Therefore, there is need to examine the effectiveness of control measures that include environmental modifications that separate humans from contaminated water sources through public health campaigns and snail reduction with chemical control measures to limit schistosomiasis transmission. In this study, a deterministic mathematical model describing the effect of treatment, public health education, and snail chemical control strategies on schistosomiasis transmission is formulated. The model considers control of schistosomiasis through human treatment using praziquantel and chemical control intervention by use of molluscicide (niclosamide) to reduce snail population and public health education which is intended to create change in human behavior, so as to maintain improved sanitation and hence reduce schistosome worm egg disposal in water by infected individuals.

The rest of the study is structured as follows. In the next section, we present the schistosomiasis model. In Section 3, we find and determine sufficient conditions for the stability of the disease-free and endemic equilibria and analyze the reproduction number of the disease, while Section 4 provides numerical results, and Section 5 concludes the study.

2. Model Formulation

2.1. Variables of the Model. The model consists of the human population divided into two epidemiological classes: the susceptible human population $S_H(t)$ and the infected human population $I_H(t)$ such that the total human population is given by $N_H(t) = S_H(t) + I_H(t)$. The snail host population is categorized into two epidemiological classes, namely, the susceptible snail population $S_S(t)$ and infected snail population $I_S(t)$ such that the total snail population is given by $N_S(t) = S_S(t) + I_S(t)$. The model also includes the population of the free-living larva stages, the miracidia $M(t)$ and cercariae $C(t)$.

2.2. Assumptions. The following assumptions are considered in the formulation of the model:

(i) There is no vertical transmission of the disease in humans

(ii) There is no immigration of infectious humans

(iii) Seasonal and weather variations do not affect snail populations

(iv) Susceptible host is infected only by contact with infected water

(v) The public health education awareness campaign covers the entire population

(vi) Treatment provides cure but does not prevent reinfection

(vii) The interaction between the miracidia and the susceptible snails, and the interaction between the cercariae and the susceptible humans has negligible effect on the pathogen population

2.3. Parameters of the Model. The parameters used in the model are described as follows:

Λ_H: Recruitment rate into the human population

Λ_S: Recruitment rate into the snail population

η: Recovery rate due to treatment

λ_H: Force of infection of the susceptible human population

λ_S: Force of infection of the susceptible snail population

ϕ: Public health education efficacy parameter

ρ: Number of schistosome eggs excreted by infected human

γ_1: Rate at which excreted eggs hatch into miracidia

γ_2: Rate at which infected snails release cercariae

β_1: Rate at which susceptible humans get infected

β_2: Rate at which susceptible snails get infected

ϵ: Limitation rate of growth velocity

$\mathscr{C}_0$: Saturation constant for cercariae

$\mathscr{M}_0$: Saturation constant for miracidia

δ_H, δ_S: Disease induced mortality rates of the human and snail population, respectively

$\theta_S, \theta_M, \theta_C$: Elimination rates of snail, miracidia, and cercariae by molluscicide, respectively

μ_H, μ_S: Natural death rates of the human and snail population, respectively

μ_M, μ_C: Natural death rates of the miracidia and cercariae population, respectively

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The parameters λ_H and λ_S are given by $\lambda_H = \beta_1 C/\mathscr{C}_0 + \epsilon C$ and $\lambda_S = \beta_2 M/\mathscr{M}_0 + \epsilon M$, respectively.

2.4. Equations of the Model. From the compartmental diagram in Figure 1 and applying the assumptions, definitions of variables and parameters above, the following system of differential equations that describe the effect of integrated control strategies on schistosomiasis transmission is obtained:

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H + \eta I_H - (1-\phi)\lambda_H S_H - \mu_H S_H,\\
\frac{dI_H}{dt} &= (1-\phi)\lambda_H S_H - (\eta + \mu_H + \delta_H) I_H,\\
\frac{dM}{dt} &= (1-\phi)\rho\gamma_1 I_H - (\mu_M + \theta_M) M,\\
\frac{dS_S}{dt} &= \Lambda_S - \lambda_S S_S - (\mu_S + \theta_S) S_S,\\
\frac{dI_S}{dt} &= \lambda_S S_S - (\mu_S + \delta_S + \theta_S) I_S,\\
\frac{dC}{dt} &= \gamma_2 I_S - (\mu_C + \theta_C) C.
\end{aligned} \tag{1}$$

Let $d_1 = \eta + \mu_H + \delta_H$, $d_2 = \mu_M + \theta_M$, $d_3 = \mu_S + \theta_S$, $d_4 = \mu_S + \delta_S + \theta_S$, and $d_5 = \mu_C + \theta_C$, then system (1) can be written as

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H + \eta I_H - (1-\phi)\lambda_H S_H - \mu_H S_H,\\
\frac{dI_H}{dt} &= (1-\phi)\lambda_H S_H - d_1 I_H,\\
\frac{dM}{dt} &= (1-\phi)\rho\gamma_1 I_H - d_2 M,\\
\frac{dS_S}{dt} &= \Lambda_S - \lambda_S S_S - d_3 S_S,\\
\frac{dI_S}{dt} &= \lambda_S S_S - d_4 I_S,\\
\frac{dC}{dt} &= \gamma_2 I_S - d_5 C.
\end{aligned} \tag{2}$$

3. Model Analysis

3.1. Positivity and Boundedness of the Solutions

Theorem 1. *Let the initial population be*

$$\left\{S_{H_0}, I_{H_0}, M_0, S_{S_0}, I_{S_0}, C_0 \geq 0\right\} \in \Omega. \tag{3}$$

Then, the solution set $\{S_H(t), I_H(t), M(t), S_S(t), I_S(t), C(t)\}$ of (2) is positive for all $t \geq 0$.

Proof. From the first equation of system (2), we get

$$\Lambda_H + \eta I_H - \left((1-\phi)\lambda_H + \mu_H\right) S_H \geq -\left((1-\phi)\lambda_H + \mu_H\right) S_H. \tag{4}$$

This implies that

$$\frac{dS_H}{dt} \geq -\left((1-\phi)\lambda_H + \mu_H\right) S_H. \tag{5}$$

By separating variables, (5) is integrated to give

$$\ln S_H \geq -\left((1-\phi)\lambda_H + \mu_H\right) t + K, \tag{6}$$

where K is the constant of integration. Applying the initial condition $S_H(0) = S_{H_0}$ to (6) gives $K = \ln S_{H_0}$.

Hence,

$$S_H(t) \geq S_{H_0} e^{-\left((1-\phi)\lambda_H + \mu_H\right) t}. \tag{7}$$

Therefore,

$$S_H(t) \geq 0. \tag{8}$$

Again from system (2), one gets

$$\begin{aligned}
\frac{dI_H}{dt} &\geq -d_1 I_H,\\
\frac{dM}{dt} &\geq -d_2 M,\\
\frac{dS_S}{dt} &\geq -\lambda_S S_S - d_3 S_S,\\
\frac{dI_S}{dt} &\geq -d_4 I_S,\\
\frac{dC}{dt} &\geq -d_5 C.
\end{aligned} \tag{9}$$

Now, separating the variables and integrating with the initial conditions $I_H(0) = I_{H_0}, M(0) = M_0, S_S(0) = S_{S_0}, I_S(0) = I_{S_0}, C(0) = C_0$ gives

$$\begin{aligned}
I_H(t) &\geq I_{H_0} e^{-d_1 t} \geq 0,\\
M(t) &\geq M_0 e^{-d_2 t} \geq 0,\\
S_S(t) &\geq S_{S_0} e^{-\left(\lambda_S + d_3\right) t} \geq 0,\\
I_S(t) &\geq I_{S_0} e^{-d_4 t} \geq 0,\\
C(t) &\geq C_0 e^{-d_5 t}
\end{aligned} \tag{10}$$

Thus, the solution set of model (2) is positive for all values of $t \geq 0$. □

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Theorem 2. *The feasible solution set of the model system (2) is mathematically and epidemiologically well-posed in the domain as*

$$\Omega = \left\{ \left(S_H, I_H, M, S_S, I_S, C\right) \in \mathbb{R}_6^+ \colon N_H \le \frac{\Lambda_H}{\mu_H}, M \le \frac{(1-\phi)\rho\gamma_1\Lambda_H}{\mu_H d_2}, N_S \le \frac{\Lambda_S}{d_4}, C \le \frac{\gamma_2\Lambda_S}{d_3 d_5} \right\}. \tag{11}$$

Proof. From $N_H(t) = S_H(t) + I_H(t)$, it follows that

$$\frac{\mathrm{d}N_H}{\mathrm{d}t} = \Lambda_H - \mu_H N - \delta_H I_H \le \Lambda_H - \mu_H N, \tag{12}$$

and integrating (12) with the initial condition $N_H(0) = N_{H_0}$ gives

$$N_H(t) \le \frac{\Lambda_H}{\mu_H} - \frac{1}{\mu_H}\left(\Lambda_H - \mu_H N_{H_0}\right)e^{-\mu_H t}, \tag{13}$$

and as $t \longrightarrow \infty$ in (13), we get

$$N_H(t) \le \frac{\Lambda_H}{\mu_H}. \tag{14}$$

For the snail host population, we get

$$\begin{aligned} N_S(t) &= S_S(t) + I_S(t), \\ \frac{\mathrm{d}N_S}{\mathrm{d}t} &= \Lambda_S - d_3 N_S - \delta_S I_S \le \Lambda_S - d_3 N_S. \end{aligned} \tag{15}$$

Integrating (15) with initial condition $N_S(0) = N_{S_0}$ gives

$$N_S(t) \le \frac{\Lambda_S}{d_3} - \frac{1}{d_3}\left(\Lambda_S - d_3 N_{S_0}\right)e^{-d_3 t}. \tag{16}$$

As $t \longrightarrow \infty$ in (16),

$$N_S(t) \le \frac{\Lambda_S}{d_3}. \tag{17}$$

For the miracidia free-living larva stage subpopulation,

$$\frac{\mathrm{d}M}{\mathrm{d}t} = (1-\phi)\rho\gamma_1 I_H - d_2 M \le (1-\phi)\rho\gamma_1\frac{\Lambda_H}{\mu_H} - d_2 M, \tag{18}$$

and integrating (18) with the initial condition $M(0) = M_0$ gives

$$M(t) \le \frac{(1-\phi)\rho\gamma_1\Lambda_H}{\mu_H d_2} - \frac{1}{d_2}\left((1-\phi)\rho\gamma_1\frac{\Lambda_H}{\mu_H} - d_2 M_0\right)e^{-d_2 t}. \tag{19}$$

As $t \longrightarrow \infty$ in (19), we get

$$M(t) \le \frac{(1-\phi)\rho\gamma_1\Lambda_H}{\mu_H d_2}. \tag{20}$$

For the cercariae free-living larva stage subpopulation,

$$\frac{\mathrm{d}C}{\mathrm{d}t} = \gamma_2 I_S - d_5 C \le \gamma_2\frac{\Lambda_S}{d_3} - d_5 C. \tag{21}$$

Integrating (21) with initial condition $C(0) = C_0$ gives

$$C(t) \le \frac{\gamma_2\Lambda_S}{d_3 d_5} - \frac{1}{d_5}\left(\gamma_2\frac{\Lambda_S}{d_3} - d_5 C_0\right)e^{-d_5 t}. \tag{22}$$

As $t \longrightarrow \infty$ in (22), we have

$$C(t) \le \frac{\gamma_2\Lambda_S}{d_3 d_5}. \tag{23}$$

Thus, the region Ω is positively invariant, and hence, the model is well posed and biologically meaningful. □

3.2. The Disease-Free Equilibrium Point. The disease-free equilibrium point is obtained by setting the right-hand side of (2) to zero and is defined at the steady state where there is no disease. Therefore, in absence of the disease, we have $I_H = M = I_S = C = 0$. Hence, the disease-free equilibrium point is given as

$$E_0 = \left(S_H, 0, 0, S_S, 0, 0\right) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, \frac{\Lambda_S}{d_3}, 0, 0\right). \tag{24}$$

3.3. Basic Reproduction Number. The basic reproduction number $\mathscr{R}_0$ is the average number of secondary infections caused by an infectious individual in a fully susceptible population. It is a threshold for predicting the disease as well as evaluating the effectiveness of the intervention strategies.

The value of $\mathscr{R}_0$ is obtained using the next generation operator approach described in [14, 15]. Let F_i be the matrix of the rates of the appearance of new infections in compartment i and V_i be matrix of the rates of the transfer of individuals into and out of compartment i. Thus, from (2), we have

$$\begin{aligned} F_i &= \begin{bmatrix} (1-\phi)\lambda_H S_H \\ 0 \\ \lambda_S S_S \\ 0 \end{bmatrix}, \\ V_i &= \begin{bmatrix} d_1 I_H \\ d_2 M - (1-\phi)\rho\gamma_1\lambda_H \\ d_4 I_S \\ d_5 C - \gamma_2 I_S \end{bmatrix}. \end{aligned} \tag{25}$$

Let F and V be the Jacobian matrices of F_i and V_i, respectively, evaluated at the disease-free equilibrium point (24). Then, one gets

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$$F = \begin{bmatrix} 0 & 0 & 0 & \dfrac{(1-\phi)\beta_1\Lambda_H}{\mathscr{C}_0\mu_H} \\ 0 & 0 & 0 & 0 \\ 0 & \dfrac{\beta_2\Lambda_S}{\mathscr{M}_0 d_3} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} d_1 & 0 & 0 & 0 \\ -(1-\phi)\rho\gamma_1 & d_2 & 0 & 0 \\ 0 & 0 & d_4 & 0 \\ 0 & 0 & -\gamma_2 & d_5 \end{bmatrix}. \tag{26}$$

Evaluating FV^{-1}, we get

$$FV^{-1} = \begin{bmatrix} 0 & 0 & \dfrac{\beta_1\gamma_2(1-\phi)\Lambda_H}{\mathscr{C}_0 d_4 d_5 \mu_H} & \dfrac{(1-\phi)\beta_1\Lambda_H}{\mathscr{C}_0 d_5 \mu_H} \\ 0 & 0 & 0 & 0 \\ \dfrac{(1-\phi)\rho\gamma_1\beta_2\Lambda_S}{\mathscr{M}_0 d_1 d_2 d_3} & \dfrac{\beta_2\Lambda_S}{\mathscr{M}_0 d_2 d_3} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}. \tag{27}$$

The eigenvalues of matrix FV^{-1} are obtained from

$$\det\left[FV^{-1} - \lambda I\right] = 0, \tag{28}$$

where I is a 4 by 4 identity matrix. This gives

$$\lambda^2\left(\lambda^2 - \left(\frac{(1-\phi)\beta_1\rho\gamma_1\Lambda_H}{\mathscr{C}_0 d_1 d_2 \mu_H}\right)\left(\frac{(1-\phi)\beta_2\rho\gamma_2\Lambda_S}{\mathscr{M}_0 d_3 d_4 d_5}\right)\right) = 0, \tag{29}$$

and the roots of the characteristic (29) are $\lambda = 0, \sqrt{\beta_1\beta_2\rho\gamma_1\gamma_2\Lambda_H\Lambda_S(1-\phi)^2/\mathscr{C}_0\mathscr{M}_0\mu_H d_1 d_2 d_3 d_4 d_5}$ and $\lambda = -\sqrt{\beta_1\beta_2\rho\gamma_1\gamma_2\Lambda_H\Lambda_S(1-\phi)^2/\mathscr{C}_0\mathscr{M}_0\mu_H d_1 d_2 d_3 d_4 d_5}$.

The basic reproduction number is the dominant eigenvalue of the matrix FV^{-1} and is thus given as

$$\begin{aligned} \mathscr{R}_0 &= \sqrt{\frac{\beta_1\beta_2\rho\gamma_1\gamma_2\Lambda_H\Lambda_S(1-\phi)^2}{\mathscr{C}_0\mathscr{M}_0\mu_H d_1 d_2 d_3 d_4 d_5}} \\ &= \sqrt{\left(\frac{(1-\phi)\beta_1\rho\gamma_1\Lambda_H}{\mathscr{C}_0 d_1 d_2 \mu_H}\right)\left(\frac{(1-\phi)\beta_2\rho\gamma_2\Lambda_S}{\mathscr{M}_0 d_3 d_4 d_5}\right)} \\ &= \sqrt{\mathscr{R}_0^H \mathscr{R}_0^S}, \end{aligned} \tag{30}$$

where

$$\begin{aligned} \mathscr{R}_0^H &= \frac{(1-\phi)\beta_1\rho\gamma_1\Lambda_H}{\mathscr{C}_0 d_1 d_2 \mu_H}, \\ \mathscr{R}_0^S &= \frac{(1-\phi)\beta_2\rho\gamma_2\Lambda_S}{\mathscr{M}_0 d_3 d_4 d_5}. \end{aligned} \tag{31}$$

Thus, the basic reproduction number, $\mathscr{R}_0$, can be written in terms of $\mathscr{R}_0^H$ and R_0^S, where $\mathscr{R}_0^H$ describes the average number of secondary infections by an infected snail in a completely susceptible population (the average number of susceptible humans infected by the infectious snail) and $\mathscr{R}_0^S$ describes the average number of secondary infections by an infected human in a completely susceptible population (the average number of susceptible snails infected by the infectious human).

3.4. The Endemic Equilibrium Point. The endemic equilibrium point determines the persistence of the disease in the community and this is evaluated when $\mathscr{R}_0 > 1$.

Theorem 3. *System (2) has a unique endemic equilibrium point when* $R_0 > 1$.

Proof. Setting the right-hand side of system (2) to zero, we have

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$$\begin{aligned}
\Lambda_H + \eta I_H - (1-\phi)\lambda_H S_H - \mu_H S_H &= 0,\\
(1-\phi)\lambda_H S_H - d_1 I_H &= 0,\\
(1-\phi)\rho\gamma_1 I_H - d_2 M &= 0,\\
\Lambda_S - \lambda_S S_S - d_3 S_S &= 0,\\
\lambda_S S_S - d_4 I_S &= 0,\\
\gamma_2 I_S - d_5 C &= 0.
\end{aligned} \tag{32}$$

Adding the first two equations of (15) and rearranging gives

$$S_H = \frac{\Lambda_H - (\mu_H + \delta_H) I_H}{\mu_H}. \tag{33}$$

From the third equation of (15), $M = (1-\phi)\rho\gamma_1 / d_2 I_H$. Substituting this into the fourth equation gives

$$S_S = \frac{\Lambda_S\left(\mathscr{M}_0 d_2 + \epsilon(1-\phi)\rho\gamma_1 I_H\right)}{\mathscr{M}_0 d_2 d_3 + (\beta_2 + \epsilon d_3)(1-\phi)\rho\gamma_1 I_H}. \tag{34}$$

Putting (33) and (34) into the fifth equation of (32) gives

$$I_S = \frac{\Lambda_S \beta_2 (1-\phi)\rho\gamma_1 I_H}{\mathscr{M}_0 d_2 d_3 d_4 + d_4(\beta_2 + \epsilon d_3)(1-\phi)\rho\gamma_1 I_H}. \tag{35}$$

From the sixth equation of (32), we have

$$C = \frac{\gamma_2}{d_6} I_S. \tag{36}$$

Consequently, (35) and (36) yield

$$C = \frac{\Lambda_S \gamma_2 \beta_2 (1-\phi)\rho\gamma_1 I_H}{d_5\left(\mathscr{M}_0 d_2 d_3 d_4 + d_4(\beta_2 + \epsilon d_3)(1-\phi)\rho\gamma_1 I_H\right)}. \tag{37}$$

Now, using (34) and (36) in the second equation of (32) and then simplifying gives

$$I_H\left(A I_H - B\left(\mathscr{R}_O^2 - 1\right)\right) = 0, \tag{38}$$

where

$$\begin{aligned}
A &= \rho\gamma_1(1-\phi)\left[\mathscr{C}_0\mu_H d_1 d_4 d_5(\epsilon d_3 + \beta_2) + \epsilon\mu_H\Lambda_S\gamma_2\beta_2 + \Lambda_S\beta_1\beta_2\gamma_2(1-\phi)(\mu_H + \delta_H)\right],\\
B &= \mathscr{C}_0\mathscr{M}_0 d_1 d_2 d_3 d_4 d_5.
\end{aligned} \tag{39}$$

From (38), $I_H = 0$ corresponds to the disease-free equilibrium and $I_H = B/A(\mathscr{R}_O^2 - 1)$ for $A \neq 0$ indicates endemicity of the disease. Thus, system (2) has a unique endemic equilibrium point $E_1(S_H^*, I_H^*, M^*, S_S^*, I_S^*, C^*)$ when $\mathscr{R}_0 > 1$, where

$$\begin{aligned}
S_H^* &= \frac{\Lambda_H - (\mu_H + \delta_H) I_H^*}{\mu_H},\\
I_H^* &= \frac{\mathscr{C}_0\mathscr{M}_0 d_1 d_2 d_3 d_4 d_5}{\rho\gamma_1(1-\phi)\left[\mathscr{C}_0\mu_H d_1 d_4 d_5(\epsilon d_3 + \beta_2) + \epsilon\mu_H\Lambda_S\gamma_2\beta_2 + \Lambda_S\beta_1\beta_2\gamma_2(1-\phi)(\mu_H + \delta_H)\right]}\left(R_O^2 - 1\right),\\
M^* &= \frac{(1-\phi)\rho\gamma_1}{d_2} I_H^*,\\
S_S^* &= \frac{\Lambda_S\left(\mathscr{M}_0 d_2 + \epsilon(1-\phi)\rho\gamma_1 I_H^*\right)}{\mathscr{M}_0 d_2 d_3 + (\beta_2 + \epsilon d_3)(1-\phi)\rho\gamma_1 I_H^*},\\
I_S^* &= \frac{\Lambda_S\beta_2(1-\phi)\rho\gamma_1 I_H^*}{\mathscr{M}_0 d_2 d_3 d_4 + d_4(\beta_2 + \epsilon d_3)(1-\phi)\rho\gamma_1 I_H^*},\\
C^* &= \frac{\Lambda_S\gamma_2\beta_2(1-\phi)\rho\gamma_1 I_H^*}{d_5\left(\mathscr{M}_0 d_2 d_3 d_4 + d_4(\beta_2 + \epsilon d_3)(1-\phi)\rho\gamma_1 I_H^*\right)}.
\end{aligned} \tag{40}$$

3.5. Local and Global Stability of the Disease-Free Equilibrium Point

Theorem 4. *The disease-free equilibrium point E_0 of system (2) is locally asymptotically stable whenever $R_0 < 1$ and unstable whenever $R_0 > 1$.*

□

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Proof. The Jacobian matrix of system (2) evaluated at the disease-free equilibrium point E_0 is given by

$$J_{E_0} = \begin{bmatrix} -\mu_H & 0 & 0 & 0 & 0 & \frac{-(1-\phi)\beta_1\Lambda_H}{\mathscr{C}_0\mu_H} \\ 0 & -d_1 & 0 & 0 & 0 & \frac{(1-\phi)\beta_1\Lambda_H}{\mathscr{C}_0\mu_H} \\ 0 & (1-\phi)\rho\gamma_1 & -d_2 & 0 & 0 & 0 \\ 0 & 0 & \frac{-\beta_2\Lambda_S}{\mathscr{M}_0 d_3} & -d_3 & 0 & 0 \\ 0 & 0 & \frac{\beta_2\Lambda_S}{\mathscr{M}_0 d_3} & 0 & -d_4 & 0 \\ 0 & 0 & 0 & 0 & \gamma_2 & -d_5 \end{bmatrix}. \tag{41}$$

The disease-free equilibrium point E_0 is locally asymptotically stable if the real parts of the eigenvalues of J_{E_0} are negative; otherwise, it is unstable.

The eigenvalues of J_{E_0} are obtained by setting the characteristic polynomial.

$P_6(\lambda) = \det(J_{E_0} - \lambda I)$ equals to zero. Thus, we have

$$P_6(\lambda) = (-\mu_H - \lambda)(-d_3 - \lambda)\begin{vmatrix} -d_1-\lambda & 0 & 0 & \frac{-(1-\phi)\beta_1\Lambda_H}{\mathscr{C}_0\mu_H} \\ (1-\phi)\rho\gamma_1 & -d_2-\lambda & 0 & 0 \\ 0 & \frac{-\beta_2\Lambda_S}{\mathscr{M}_0 d_3} & -d_4-\lambda & 0 \\ 0 & 0 & \gamma_2 & -d_5-\lambda \end{vmatrix} = 0. \tag{42}$$

From (42), it is clear that $\lambda_1 = -\mu_H, \lambda_2 = -d_3$. The other remaining eigenvalues are obtained by considering the matrix

$$\begin{bmatrix} -d_1-\lambda & 0 & 0 & \frac{-(1-\phi)\beta_1\Lambda_H}{\mathscr{C}_0\mu_H} \\ (1-\phi)\rho\gamma_1 & -d_2-\lambda & 0 & 0 \\ 0 & \frac{-\beta_2\Lambda_S}{\mathscr{M}_0 d_3} & -d_4-\lambda & 0 \\ 0 & 0 & \gamma_2 & -d_5-\lambda \end{bmatrix}, \tag{43}$$

whose characteristic equation is

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0, \tag{44}$$

where $a_1 = d_1 + d_2 + d_4 + d_5, a_2 = d_1d_2 + d_1d_4 + d_1d_5 + d_2d_4 + d_2d_5 + d_4d_5, a_3 = d_1d_2d_4 + d_1d_2d_5 + d_1d_4d_5 + d_2d_4d_5$, $a_4 = d_1d_2d_4d_5\ (1 - \mathscr{R}_0^2)$.

According to [16], the Routh–Hurwitz conditions for the stability of the polynomial (44) are as follows:

(i) $a_1 > 0$

(ii) $a_1a_2 - a_3 > 0$

(iii) $a_3(a_1a_2 - a_3) - a_1^2a_4 > 0$

(iv) $a_4 > 0$

Clearly (i) is satisfied and (iv) holds true whenever $\mathscr{R}_0 < 1$. It remains to check conditions (ii) and (iii):

and

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$$a_1a_2 - a_3 = d_1^2d_2 + d_1^2d_4 + d_1^2d_5 + d_1d_2^2 + 2d_1d_2d_4 + 2d_1d_2d_5 + d_2^2d_4 + d_2^2d_5 + d_1d_4^2 + 2d_1d_4d_5 + d_2d_4^2 + 2d_2d_4d_5 + d_4^2d_5 + d_2d_5^2 + d_4d_5^2 > 0, \tag{45}$$

$$\begin{aligned} a_3(a_1a_2 - a_3) - a_1^2a_4 &= (d_1d_2d_4 + d_1d_2d_5 + d_1d_4d_5 + d_2d_4d_5) \\ &[(d_1 + d_2 + d_4 + d_5)(d_1d_2 + d_1d_4 + d_1d_5 + d_2d_4 + d_2d_5 + d_4d_5) - (d_1d_2d_4 + d_1d_2d_5 + d_1d_4d_5 + d_2d_4d_5)] \\ &-(d_1 + d_2 + d_4 + d_5)^2d_1d_2d_4d_5\left(1 - R_0^2\right) > 0, \text{ if } \mathscr{R}_0 < 1. \end{aligned} \tag{46}$$

Thus, the Routh–Hurwitz conditions hold when all the eigenvalues have negative real parts making E_0 locally asymptotically stable whenever $\mathscr{R}_0 < 1$.

To study the global asymptotic stability of the disease-free equilibrium, the method described by Castillo-Chavez et al. [17] and in [18] has been employed. The model system (2) is expressed in the form:

$$\begin{aligned} \frac{dX_1}{dt} &= F(X_1, X_2), \\ \frac{dX_2}{dt} &= G(X_1, X_2), \quad G(X_1, 0) = 0, \end{aligned} \tag{47}$$

where $X_1 = (S_H, S_S)$ comprises of the uninfected subpopulation and $X_2 = (I_H, M, I_S, C)$ comprises of the infected subpopulation. Let $U_0 = (X_1^*, 0)$ be the disease-free equilibrium point of system (47). Then, U_0 is globally asymptotically stable provided that $\mathscr{R}_0 < 1$ and that the following conditions hold true:

(**H**1): For $dX_1/dt = F(X_1, 0)$, $X_1^* = (S_H^0, S_S^0)$ is globally asymptotically stable

(**H**2): $G(X_1, X_2) = AX_2 - \widehat{G}(X_1, X_2), \widehat{G}(X_1, X_2) \geq 0$ for $(X_1, X_2) \in \Omega$, where $A = D_{X_2}G(X_1^*, 0)$ is an M-matrix (the off-diagonal elements are non-negative) and Ω is the region where the model makes biological sense □

Theorem 5. *The disease-free equilibrium point E_0 for system (2) is globally asymptotically stable provided $\mathscr{R}_0 < 1$ and conditions* (**H**1) *and* (**H**2) *hold true.*

Proof. For system (2), we have

$$\begin{aligned} \frac{dX_1}{dt} &= F(X_1, X_2) \\ &= \begin{bmatrix} \Lambda_H + \eta I_H - (1-\phi)\lambda_H S_H - \mu_H S_H \\ \Lambda_S - \lambda_S S_S - d_3 S_S \end{bmatrix}, \\ \frac{dX_2}{dt} &= G(X_1, X_2) \\ &= \begin{bmatrix} (1-\phi)\lambda_H S_H - d_1 I_H \\ (1-\phi)\rho\gamma_1 I_H - d_2 M \\ \lambda_S S_S - d_4 I_S \\ \gamma_2 I_S - d_5 C \end{bmatrix}, \end{aligned} \tag{48}$$

$$F(X_1, 0) = \begin{bmatrix} \Lambda_H - \mu_H S_H \\ \Lambda_S - d_3 S_S \end{bmatrix}.$$

As $t \longrightarrow \infty$, we observe that $(X_1, 0) \longrightarrow E_0 \in \Omega$, that is, $S_H \longrightarrow \Lambda_H/\mu_H$ and $S_S \longrightarrow \Lambda_S/d_3$. Thus, $(H1)$ holds true:

$$A = \frac{\partial G(X_1, 0)}{\partial X_2}$$

$$|_{U_0} = \begin{bmatrix} -d_1 & 0 & 0 & \dfrac{(1-\phi)\beta_1 S_H^0}{\mathscr{C}_0} \\ (1-\phi)\rho\gamma_2 & -d_2 & 0 & 0 \\ 0 & \dfrac{\beta_2 S_H^0}{\mathscr{M}_0} & -d_4 & 0 \\ 0 & 0 & \gamma_2 & -d_5 \end{bmatrix}, \tag{49}$$

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$$\widehat{G}(X_1, X_2) = AX_2 - G(X_1, X_2)$$
$$= \begin{bmatrix} \beta_1(1-\phi)C\left(\dfrac{S_H^0}{\mathscr{C}_0} - \dfrac{S_H}{\mathscr{C}_0 + \epsilon C}\right) \\ 0 \\ \beta_2 M\left(\dfrac{S_S^0}{\mathscr{M}_0} - \dfrac{S_S}{\mathscr{M}_0 + \epsilon M}\right) \\ 0 \end{bmatrix}. \tag{50}$$

From (49), clearly A is an M-matrix (all the off-diagonal elements are non-negative), and from (50), $\widehat{G}(X_1, X_2) \geq 0$ since $S_H^0/\mathscr{C}_0 \geq S_H/\mathscr{C}_0 + \epsilon C$ and $S_S^0/\mathscr{M}_0 \geq S_S/\mathscr{M}_0 + \epsilon M$. Thus, (H2) holds true, and the proof is complete. □

3.6. Local Stability of the Endemic Equilibrium. The center manifold theory by Castillo-Chavez [19] is used to establish the local asymptotic stability of the endemic equilibrium point.

Theorem 6. *Consider the following general system of ordinary differential equations with a parameter ϑ:*

$$\frac{\mathrm{d}x}{\mathrm{d}t} = f(x, \vartheta), \quad f: \mathbb{R}^n \times \mathbb{R} \longrightarrow \mathbb{R}^n,\ f \in \mathbb{C}^2(\mathbb{R}^n \times \mathbb{R}), \tag{51}$$

and without loss of generality, it is assumed that 0 is an equilibrium point of the system (51) for all values of the parameter ϑ, that is,

$$f(0, \vartheta) \equiv 0 \text{ for all } \vartheta. \tag{52}$$

Assume the following points:

A1. $A = D_x f(0,0) = (\partial f_i/\partial x_j(0,0))$ is the linearization matrix of system (51) around the equilibrium 0 with ϑ evaluated at 0. Zero is a simple eigenvalue of A, and other eigenvalues have negative real parts.

A2. The matrix A has a non-negative right eigenvector w and a left eigenvector v each corresponding to the zero eigenvalue.

Let f_k be the k^{th} component of f, and then, we get

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$
$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \vartheta}(0,0). \tag{53}$$

Then, the local dynamics of the system around 0 are completely determined by the signs of a and b that can be given as follows:

(i) $a > 0, b > 0$. When $\vartheta < 0$ with $|\vartheta| \ll 1$, 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 < \vartheta \ll 1$, 0 is unstable, and there exists a negative and locally asymptotically stable equilibrium.

(ii) $a < 0, b < 0$. When $\vartheta < 0$ with $|\vartheta| \ll 1$, 0 is unstable; when $0 < \vartheta \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium.

(iii) $a > 0, b < 0$. When $\vartheta < 0$ with $|\vartheta| \ll 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \vartheta \ll 1$, 0 is stable, and a positive unstable equilibrium appears.

(iv) $a < 0, b > 0$. When ϑ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.

Theorem 7. *The unique endemic equilibrium point, E_1 of system (2) is locally asymptotically stable if $R_0 > 1$ but close to 1.*

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Proof. We apply the change of variable on system (2) as follows. Let $S_H = x_1, I_H = x_2, M = x_3, S_S = x_4, I_S = x_5, C = x_6$. The model system (2) can be written as $\mathrm{d}X/\mathrm{d}t = F$, where $X = (x_1, x_2, x_3, x_4, x_5, x_6)^T$ and $F = (f_1, f_2, f_3, f_4, f_5, f_6)$:

$$\begin{aligned}
\frac{\mathrm{d}x_1}{\mathrm{d}t} &= f_1 \\
&= \Lambda_H + \eta x_2 - (1-\phi)\frac{\beta_1 x_6}{\mathscr{C}_0 + \epsilon x_6}x_1 - \mu_H x_1, \\
\frac{\mathrm{d}x_2}{\mathrm{d}t} &= f_2 \\
&= (1-\phi)\frac{\beta_1 x_6}{\mathscr{C}_0 + \epsilon x_6}x_1 - d_1 x_2, \\
\frac{\mathrm{d}x_3}{\mathrm{d}t} &= f_3 \\
&= (1-\phi)\rho\gamma_1 x_2 - d_2 x_3, \\
\frac{\mathrm{d}x_4}{\mathrm{d}t} &= f_4 \\
&= \Lambda_S - \frac{\beta_2 x_3}{\mathscr{M}_0 + \epsilon x_3}x_4 - d_3 x_4, \\
\frac{\mathrm{d}x_5}{\mathrm{d}t} &= f_5 \\
&= \frac{\beta_2 x_3}{\mathscr{M}_0 + \epsilon x_3}x_4 - d_4 x_5, \\
\frac{\mathrm{d}x_6}{\mathrm{d}t} &= f_6 \\
&= \gamma_2 x_5 - d_5 x_6.
\end{aligned} \tag{54}$$

Taking β_1 as the bifurcation parameter while considering the case $\mathscr{R}_0 = 1$ gives

$$\beta_1 = \beta_1^* = \frac{\mathscr{C}_0 \mathscr{M}_0 \mu_H d_1 d_2 d_3 d_4 d_5}{\beta_2 \rho \gamma_1 \gamma_2 \Lambda_H \Lambda_S (1-\phi)^2}. \tag{55}$$

The linearization around the disease-free equilibrium point evaluated at β_1^* gives

$$A = \begin{bmatrix}
-\mu_H & 0 & 0 & 0 & 0 & \dfrac{-(1-\phi)\beta_1^* \Lambda_H}{\mathscr{C}_0 \mu_H} \\
0 & -d_1 & 0 & 0 & 0 & \dfrac{(1-\phi)\beta_1^* \Lambda_H}{\mathscr{C}_0 \mu_H} \\
0 & (1-\phi)\rho\gamma_1 & -d_2 & 0 & 0 & 0 \\
0 & 0 & \dfrac{-\beta_2 \Lambda_S}{\mathscr{M}_0 d_3} & -d_3 & 0 & 0 \\
0 & 0 & \dfrac{\beta_2 \Lambda_S}{\mathscr{M}_0 d_3} & 0 & -d_4 & 0 \\
0 & 0 & 0 & 0 & \gamma_2 & -d_5
\end{bmatrix}. \tag{56}$$

Matrix A has zero as an eigenvalue, and this can be deduced from (21)–(23) with $\mathscr{R}_0 = 1$, which yields

$$\lambda\left(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3\right) = 0. \tag{57}$$

The right eigenvector $w = (w_1, w_2, w_3, w_4, w_5, w_6)^T$ corresponding to the zero eigenvalue is computed from $Aw = 0$, which yields

$$\begin{aligned}
w_1 &= \frac{-\beta_1^*(1-\phi)\Lambda_H}{\mu_H^2 \mathscr{C}_0} w_6, \\
w_2 &= \frac{\beta_1^*(1-\phi)\Lambda_H}{\mathscr{C}_0 \mu_H d_1} w_6, \\
w_3 &= \frac{\rho\gamma_1 \beta_1^* \Lambda_H (1-\phi)^2}{\mathscr{C}_0 \mu_H d_1 d_2} w_6, \\
w_4 &= \frac{-\rho\gamma_1 \beta_1^* \beta_2 \Lambda_S \Lambda_H (1-\phi)^2}{\mathscr{M}_0 \mathscr{C}_0 \mu_H d_1 d_2 d_3^2} w_6, \\
w_5 &= \frac{d_5}{\gamma_2} w_6, \\
w_6 &> 0.
\end{aligned} \tag{58}$$

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The left eigenvector $v = (v_1, v_2, v_3, v_4, v_5, v_6)^T$ is computed from $vA = 0$ which yields

$$\begin{aligned} v_1 &= v_4 = 0, \\ v_2 &= \frac{\mathscr{C}_0 \mu_H d_5}{\beta_1^* (1-\phi)\Lambda_H} v_6, \\ v_3 &= \frac{\mathscr{C}_0 \mu_H d_1 d_5}{\rho \gamma_1 \beta_1^* \Lambda_H (1-\phi)^2} v_6, \\ v_5 &= \frac{\gamma_2}{d_4} v_6, \\ v_6 &> 0. \end{aligned} \tag{59}$$

Letting $w_6 = 1$, it can be seen from (58) that any other right eigenvector of matrix A corresponding to the zero eigenvalue is a constant multiple of w (the same is true for the left eigenvector in (59) by letting $v_6 = 1$). Hence, zero is a simple eigenvalue.

In the computation of a and b, only nonzero partial derivatives of system (54) are used. Since $v_1 = v_4 = 0$, the nonzero partial derivatives computed at the disease-free equilibrium are from f_2, f_3, f_5, and f_6. These are obtained as

$$\begin{aligned} \frac{\partial^2 f_2}{\partial x_1 \partial x_6} &= \frac{\beta_1^*}{\mathscr{C}_0}(1-\phi), \\ \frac{\partial^2 f_2}{\partial^2 x_6} &= -\frac{2\epsilon \beta_1^*}{\mathscr{C}_0^2}(1-\phi)x_1^*, \\ \frac{\partial^2 f_5}{\partial x_3 \partial x_4} &= \frac{\beta_2}{\mathscr{M}_0}, \end{aligned} \tag{60}$$

$\partial^2 f_5/\partial^2 x_3 = -2\epsilon\beta_2 x_5^*/M_0^2$ and $\partial^2 f_2/\partial x_6 \partial \beta_1^* = (1-\phi)/\mathscr{C}_0 x_1^*$. Thus, it follows that

$$\begin{aligned} a &= v_2 w_1 w_6 \frac{\partial^2 f_2}{\partial x_1 \partial x_6} + v_2 w_6^2 \frac{\partial^2 f_2}{\partial^2 x_6} + v_5 w_3 w_4 \frac{\partial^2 f_5}{\partial x_3 \partial x_4} + v_5 w_3^2 \frac{\partial^2 f_5}{\partial^2 x_3}, \\ a &= -v_6 w_6^2 \left(\frac{\beta_1^*(1-\phi)d_5}{\mathscr{C}_0 \mu_H} + \frac{2\epsilon d_5}{\mathscr{C}_0} + \frac{d_4 d_5^2}{\Lambda_S \gamma_2} + \frac{2\epsilon d_3 d_4 d_5^2}{\Lambda_S \beta_2 \gamma_2^2} \right) < 0, \\ b &= \frac{d_6}{\beta_1^*} v_7 w_7 > 0. \end{aligned} \tag{61}$$

Since $a < 0$ and $b > 0$, then by condition (iv) of Theorem 5, system (54) exhibits a forward bifurcation at $\mathscr{R}_0 = 1$, implying that the unique endemic equilibrium is locally asymptotically stable when $\mathscr{R}_0 > 1$ and close to 1. □

3.7. Global Stability of the Endemic Equilibrium Point

Theorem 8. *If $\mathscr{R}_0 \geq 1$, the endemic equilibrium E_1 of system (2) is globally asymptotically stable.*

Proof. To analyze the global stability of endemic equilibrium, we consider the candidate Lyapunov function $L(S_H, I_H, M, S_S, I_S, C)$ given by

$$\begin{aligned} L = &\left(S_H - S_H^* + S_H^* \ln \frac{S_H^*}{S_H} \right) + \left(I_H - I_H^* + I_H^* \ln \frac{I_H^*}{I_H} \right) + \left(M - M^* + M^* \ln \frac{M^*}{M} \right) \\ &+ \left(S_S - S_S^* + S_S^* \ln \frac{S_S^*}{S_S} \right) + \left(I_S - I_S^* + I_S^* \ln \frac{I_S^*}{I_S} \right) + \left(C - C^* + C^* \ln \frac{C^*}{C} \right). \end{aligned} \tag{62}$$

Clearly, $L(S_H^*, I_H^*, M^*.S_S^*, I_S^*, C^*) = 0$, and $L > 0$, otherwise. Moreover, L is radially unbounded in R_+^6. So, it remains to determine the sign of $L\prime$. Differentiating (62) with respect to t gives

$$\begin{aligned} \frac{\mathrm{d}L}{\mathrm{d}t} = &\left(\frac{S_H - S_H^*}{S_H} \right) \frac{\mathrm{d}S_H}{\mathrm{d}t} + \left(\frac{I_H - I_H^*}{I_H} \right) \frac{\mathrm{d}I_H}{\mathrm{d}t} + \left(\frac{M - M^*}{M} \right) \frac{\mathrm{d}M}{\mathrm{d}t} + \left(\frac{S_S - S_S^*}{S_S} \right) \frac{\mathrm{d}S_S}{\mathrm{d}t} \\ &+ \left(\frac{I_S - I_S^*}{I_S} \right) \frac{\mathrm{d}I_S}{\mathrm{d}t} + \left(\frac{C - C^*}{C} \right) \frac{\mathrm{d}C}{\mathrm{d}t}. \end{aligned} \tag{63}$$

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Substituting for values of dS_H/dt, dI_H/dt, dM/dt, dS_S/dt, dI_S/dt, and dC/dt in (63) yields

$$\begin{aligned}\frac{dL}{dt} &= \left(\frac{S_H - S_H^*}{S_H}\right)\left(\Lambda_H + \eta I_H - (1-\phi)\lambda_H S_H - \mu_H S_H\right) + \left(\frac{I_H - I_H^*}{I_H}\right)\left((1-\phi)\lambda_H S_H - d_1 I_H\right)\\ &+ \left(\frac{M - M^*}{M}\right)\left((1-\phi)\rho\gamma_1 I_H - d_2 M\right) + \left(\frac{S_S - S_S^*}{S_S}\right)\left(\Lambda_S - \lambda_S S_S - d_3 S_S\right)\\ &+ \left(\frac{I_S - I_S^*}{I_S}\right)\left(\lambda_S S_S - d_4 I_S\right) + \left(\frac{C - C^*}{C}\right)\left(\gamma_2 I_S - d_5 C\right).\end{aligned} \tag{64}$$

Rearranging positive and negative terms in (64) gives

$$\frac{dL}{dt} = P - Q, \tag{65}$$

where

$$\begin{aligned}P &= \Lambda_H + \eta I_H + \left((1-\phi)\lambda_H + \mu_H + \lambda_S + d_3\right)S_H^* + \left((1-\phi)\lambda_H S_H + \lambda_S\right)S_S + d_1 I_H^*\\ &+ \left(\eta + (1-\phi)\rho\gamma_1\right)I_H + d_2 M^* + \Lambda_S + \lambda_S S_S + d_4 I_S^* + \gamma_2 I_S + d_5 C^*,\\ Q &= \left((1-\phi)\lambda_H + \mu_H\right)S_H + \left(\Lambda_H + \eta I_H\right)\frac{S_H^*}{S_H} + d_1 I_H + (1-\phi)\lambda_H S_H \frac{I_H^*}{I_H} + d_2 M\\ &+ (1-\phi)\rho\gamma_1 I_H \frac{M^*}{M} + \left(\lambda_S + d_3\right)S_S + \Lambda_S \frac{S_S^*}{S_S} + d_4 I_S + \lambda_S S_S \frac{I_S^*}{I_S} + d_5 C + \gamma_2 I_S \frac{C^*}{C}.\end{aligned} \tag{66}$$

If $P \le Q$, then $dL/dt \le 0$ noting that $dL/dt = 0$ if and only if $S_H = S_H^*$, $I_H = I_H^*$, $M = M^*$, $S_S = S_S^*$, $I_S = I_S^*$, and $C = C^*$. Therefore, the largest compact invariant set in $\{(S_H, I_H, M, S_S, I_S, C) \in \Omega: dL/dt = 0\}$ is the singleton $\{E_1\}$, where E_1 is the endemic equilibrium point of system (2). Thus, by Lasalle's invariance principle [20], it implies that E_1 is globally asymptotically stable in Ω if $P \le Q$; which holds if and only if $\mathcal{R}_0 \ge 1$. □

3.8. Sensitivity Analysis. In this subsection, sensitivity analysis of the reproduction number $\mathcal{R}_0$ on different parameters is carried out in order to determine their relative importance to the disease transmission in the community. The approach by Chitnis et al. [21] has been employed to obtain the relative change in a state variable when the parameter changes. An expression for the sensitivity of $\mathcal{R}_0$ with respect to p is given by

$$\gamma_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p}\frac{p}{\mathcal{R}_0}, \tag{67}$$

where p is any sensitive parameter to be considered in relation to $\mathcal{R}_0$.

From (67), expressions for the sensitivity indices are as follows:

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$$
\begin{aligned}
\gamma_{\beta_1}^{\mathscr{R}_0} &= \gamma_{\beta_2}^{\mathscr{R}_0} \\
&= \gamma_{\rho}^{\mathscr{R}_0} = \gamma_{\beta_1}^{\mathscr{R}_0} \\
&= \gamma_{\gamma_1}^{\mathscr{R}_0} = \gamma_{\gamma_2}^{\mathscr{R}_0} \\
&= \gamma_{\lambda_H}^{\mathscr{R}_0} = \gamma_{\lambda_S}^{\mathscr{R}_0} \\
&= \frac{1}{2}, \\
\gamma_{\mu_C}^{\mathscr{R}_0} &= -\frac{1}{2}\frac{\mu_C}{d_5}, \\
\gamma_{\theta_M}^{\mathscr{R}_0} &= -\frac{1}{2}\frac{\theta_M}{d_2}, \\
\gamma_{\mu_M}^{\mathscr{R}_0} &= -\frac{1}{2}\frac{\mu_M}{d_1}, \\
\gamma_{\delta_H}^{\mathscr{R}_0} &= -\frac{1}{2}\frac{\delta_H}{d_1} \\
&= \gamma_{\delta_S}^{\mathscr{R}_0} = -\frac{1}{2}\frac{\delta_S}{d_4}, \\
\gamma_{\theta_C}^{\mathscr{R}_0} &= -\frac{1}{2}\frac{\theta_C}{d_5}, \\
\gamma_{\theta_S}^{\mathscr{R}_0} &= -\frac{1}{2}\frac{\theta_S(d_3+d_4)}{d_3 d_4}, \\
\gamma_{\mu_S}^{\mathscr{R}_0} &= -\frac{1}{2}\frac{\mu_S(d_3+d_4)}{d_3 d_4}.
\end{aligned}
\tag{68}
$$

The parameter values used are $\mu_C = 0.004, \mu_S = 0.000569, \delta_S = 0.0004012, \mu_M = 0.008, \theta_S = 0.1, \eta = 0.0181$, and $\mu_H = 0.0000384$.

Table 1 shows the sensitivity indices of $\mathscr{R}_0$ with respect to different parameters from the most sensitive to the least sensitive. It is noted that the value of $\mathscr{R}_0$ increases when parameter values $\beta_1, \beta_2, \rho, \gamma_1, \gamma_2, \Lambda_H$, and Λ_S increase, while the other parameters values are kept constant. Since these parameters have positive indices, the endemicity of the disease is increased. When parameter $\mu_H, \theta_S, \theta_C, \mu_C, \delta_H, \mu_S, \theta_M$, and δ_S are increased while the other parameters values are kept constant, the value of $\mathscr{R}_0$ decreases implying that the endemicity of the disease is decreased.

4. Numerical Simulations

In this section, we numerically simulate the combined effect of treatment with praziquantel, public health education and chemical control strategies on schistosomiasis transmission. The model was simulated using ODE solvers coded in MATLAB computer software. The parameter values used are shown in Table 2 with the initial conditions such as $S_H(0) = 120000$, $I_H(0) = 15000$, $M(0) = 200$, $S_S(0) = 400$, $I_S(0) = 200$, and $C(0) = 0$.

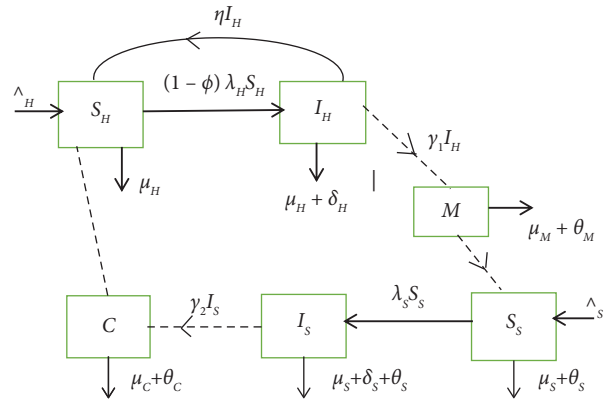


Figure 1: Compartmental diagram for schistosomiasis transmission.

Table 1: Numerical values of sensitivity indices of $\mathscr{R}_0$.

Parameter symbol	Sensitivity index
μ_H	−0.5009
β_1	+0.5000
β_2	+0.5000
ρ	+0.5000
γ_1	+0.5000
γ_2	+0.5000
Λ_H	+0.5000
Λ_S	+0.5000
μ_M	−0.4956
θ_S	−0.4952
θ_C	−0.3333
μ_C	−0.1667
δ_H	−0.0885
μ_S	−0.0056
θ_M	−0.0044
δ_S	−0.0020

Figures 2 and 3 show numerical simulation for a mathematical model of schistosomiasis with the treatment strategy only, where $\eta = 0.0181$, $\theta_S = 0$, $\theta_M = 0$, $\theta_C = 0$, and $\phi = 0$.

The results indicate that with the treatment strategy only, the subpopulations of infected humans, I_H, and miracidia, M, decrease in the early stages of treatment and begin to increase again. The subpopulations of susceptible humans and susceptible snails decrease, while the infected snails and cercariae increase. It is noted that with treatment intervention only, schistosomiasis persists within the population.

Figure 4 shows the numerical simulation of schistosomiasis model with treatment and public health education control strategy when $\phi = 0.2, 0.5$, and 0.9. The results reveal that the susceptible humans and snail subpopulations increase with increase in public health awareness from $\phi = 0.2$ to $\phi = 0.9$. The subpopulations of infected humans, infected snails, miracidia, and cercariae decrease as ϕ increases. This is because of increased awareness among the human population to maintain proper sanitation as well as reduced contact with contaminated water.

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Table 2: Parameter values used in numerical simulations of the schistosomiasis model.

Parameter	Value	Source
μ_H	0.0000384 day^{-1}	[22]
β_1	0.40 $6 \times 10^{-8} day^{-1}$	[10]
β_2	0.615 $L^{-1} day^{-1}$	[23]
ρ	300	[6]
γ_1	0.00232 day^{-1}	[23]
γ_2	2.6 day^{-1}	[23]
Λ_H	300	Estimated
Λ_S	200	[24]
μ_M	0.9 day^{-1}	[23]
θ_S	0.01 day^{-1}	[11]
θ_C	0.008 day^{-1}	[11]
C_0	9×10^7	[24]
M_0	1×10^6	[10]
ϕ	$0 < \phi < 1$	Estimated
ϵ	0.3	[9]
μ_C	0.004 day^{-1}	[15]
η	0.0181 day^{-1}	[26]
δ_S	0.0004012 day^{-1}	[24]
θ_M	0.008 day^{-1}	[11]
μ_S	0.000569 day^{-1}	[23]
δ_H	0.0039 day^{-1}	[27]

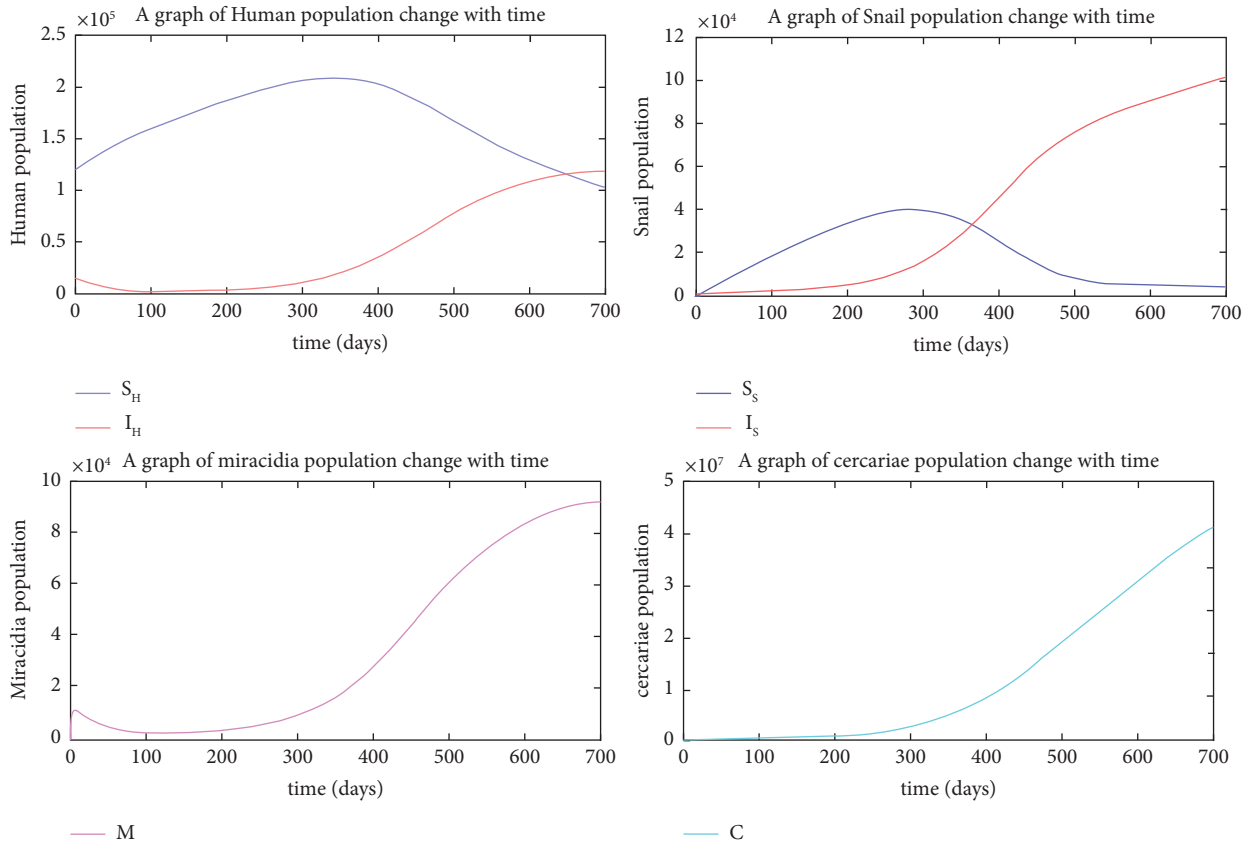


Figure 2: Graph of population against time with treatment intervention only.

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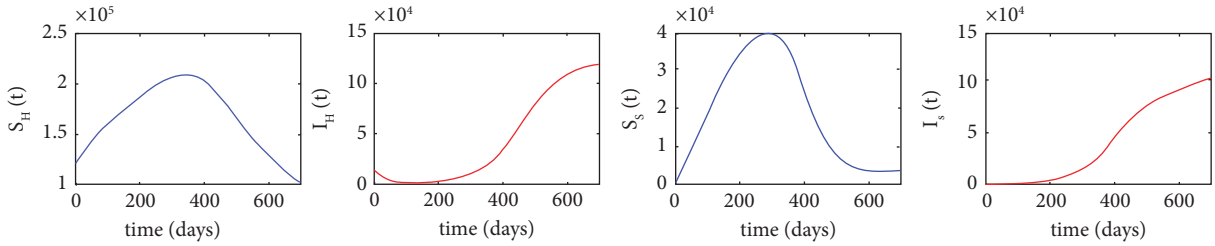


FIGURE 3: Graph of human and snail subpopulations with treatment intervention only.

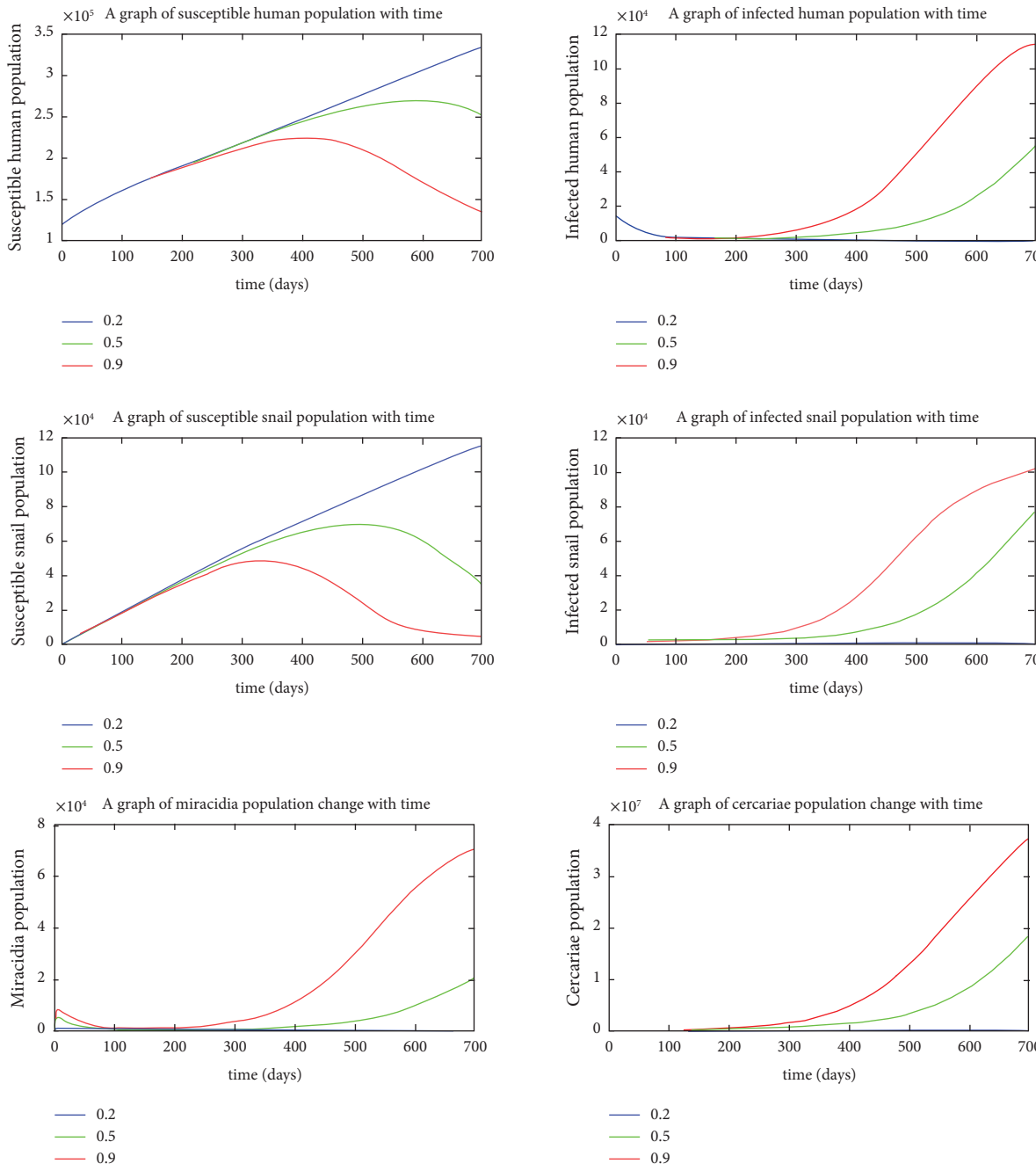


FIGURE 4: Graph of population against time with treatment intervention and public health education control strategy when $\phi = 0.2$, 0.5, and 0.9.

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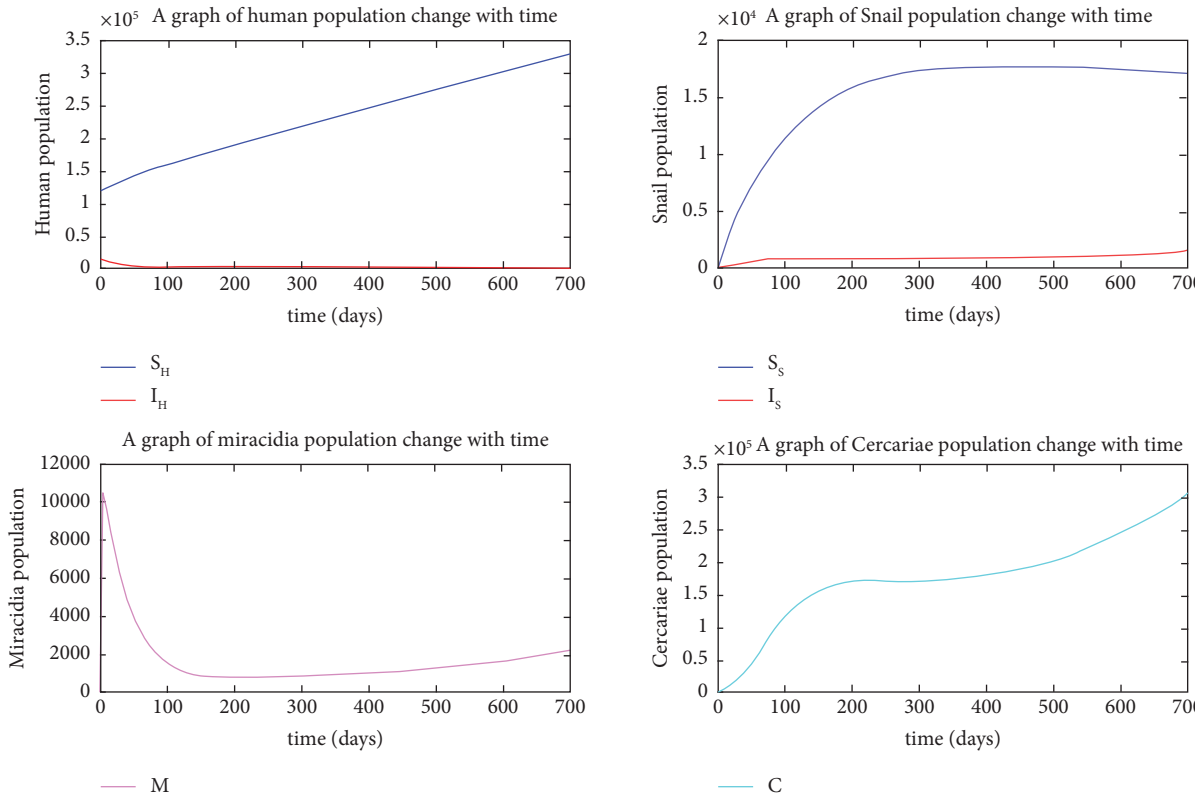


Figure 5: Graph of population against time with treatment intervention and chemical control intervention strategy.

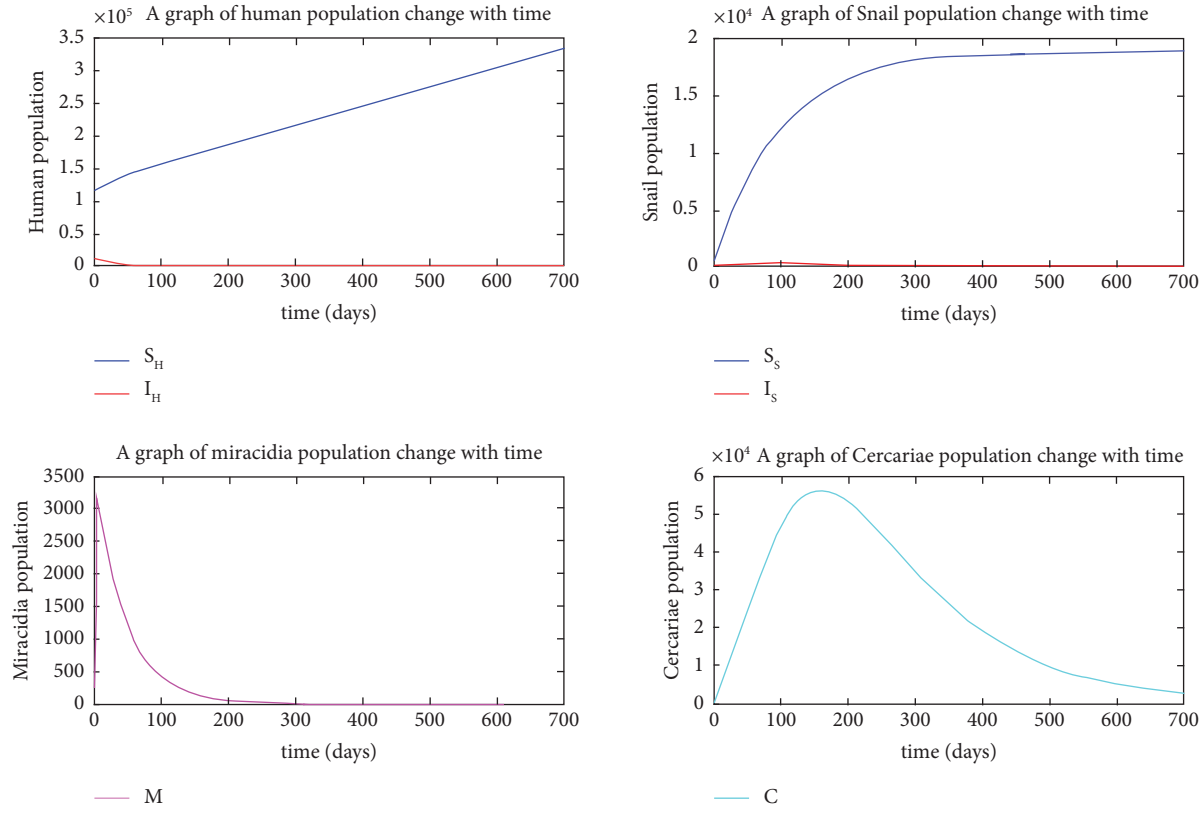


Figure 6: Graph of population against time with treatment intervention, public health education, and chemical control intervention strategy.

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Figure 5 shows numerical simulation of schistosomiasis model with the treatment and chemical control intervention strategy only with parameter values $\eta = 0.0181$, $\theta_S = 0.01$, $\theta_M = 0.008$, $\theta_C = 0.008$, and $\phi = 0$. From the results, it is noted that the subpopulations of susceptible humans and susceptible snails increase relatively higher than that illustrated in Figures 2 and 3 which considers treatment only. The subpopulations of infected humans, infected snails, miracidia, and cercariae also decrease further as compared to those in Figures 2 and 3.

Figure 6 shows numerical simulation of schistosomiasis model with treatment, public health education, and chemical control intervention strategies with parameter values $\eta = 0.0181$, $\theta_S = 0.01$, $\theta_M = 0.008$, $\theta_C = 0.008$, and $\phi = 0.7$. The results indicate that the subpopulations of infected humans, miracidia, infected snails, and cercariae decrease very significantly compared to other strategies illustrated in Figures 2–5, whereas the subpopulations of susceptible humans and susceptible snail increase significantly.

5. Discussion and Conclusion

In this study, a deterministic mathematical model describing the effect of treatment, public health education, and chemical control intervention strategies on schistosomiasis transmission was formulated and analyzed. The model was found to be well posed and biologically meaningful.

Analysis of the model showed existence of two equilibria, namely, the disease-free equilibrium and the endemic equilibrium. The basic reproductive number $\mathscr{R}_0$ of the model was determined, and its biological interpretation explained. $\mathscr{R}_0$ was further decomposed into individual reproductive numbers of the transmission agents of schistosomiasis; that is, $\mathscr{R}_0^H$ is the average number of secondary infections by an infected snail in a completely susceptible population and $\mathscr{R}_0^S$ is the average number of secondary infections by an infected human in a completely susceptible population. The disease-free steady state was found to be locally stable whenever $\mathscr{R}_0 < 1$. The disease-free equilibrium point was also globally stable implying that all initial conditions would converge to it over time; hence, treatment can decrease the disease prevalence. The endemic equilibrium point of the model was found to be locally stable if and only if $\mathscr{R}_0 > 1$. This meant that interventions by policy makers are necessary to eradicate schistosomiasis over time. In order to make endemic equilibrium point unstable, control measures such as mass drug administration in endemic areas are necessary.

Sensitivity analysis identifies the rate at which susceptible humans get infected, β_1 and rate at which susceptible snails get infected, β_2 as the main parameters fueling the spread, while the θ_M and θ_S are the elimination rates of miracidia and snails by molluscicide (chemical control) as inhibitors of the disease. Thus, from the results, it is noted that a combination of treatment, public health education, and chemical control intervention strategies can effectively manage the transmission of schistosomiasis in endemic areas.

Numerical simulations reveal that a combination of treatment, public health education, and chemical control intervention strategies significantly increase the number of susceptible human and susceptible snail populations while significantly decreasing the number of infected humans, miracidia, infected snails, and cercariae as shown in Figure 6. The study shows that treatment only in Figures 2 and 3 is less effective and the disease persists within the population, which agrees with the results of Gurarie et al. The study also reveals that a combination of both treatment and chemical control intervention, illustrated in Figure 5 has more impact compared to that of treatment only in Figures 2 and 3 which agrees with the results of Gao et al. [10].

In conclusion, in order to effectively manage the prevalence of schistosomiasis transmission, there should be a combined control intervention of treatment, public health education, and chemical control intervention strategies.

Data Availability

The data to support this study were obtained from the literature.

Conflicts of Interest

The authors declare that there are no conflicts of interest.

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