Mathematical model of schistosomiasis with health education and molluscicide intervention

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Abstract. In this article, a mathematical model is developed to study the impact of health education and molluscicide intervention on the spread of schistosomiasis. The model constructed consists of seven ordinary differential equations that describe susceptible human, latent human, infectious human, susceptible snail, infected snail, miracidia and cercarie. After analyzing non-negativity and boundedness of solutions of the model, we determine the disease free equilibrium point and the endemic equilibrium point as well as their existence conditions. The basic reproduction number is determined using the next generation matrix approach. The local stability condition of the disease-free equilibrium point is proved by using linearization and Descartes’ sign rule. The Center Manifold Theory is used to prove the local stability condition of the endemic equilibrium point and to identify the existence of bifurcation. We constructed Lyapunov function to show that the disease-free equilibrium point is globally asymptotically stable under sufficient condition. We present numerical simulations to support our theoretical study. Numerical simulations show that health education and molluscicide intervention are able to reduce schistosomiasis cases in human and snail populations. Molluscicide intervention is a very effective method to control the spread of schistosomiasis.

1. Introduction
Schistosomiasis is a disease caused by parasitic worms (helminths). Schistosomiasis is also called bilharzia or bilharziasis or snail fever [1]. The parasites that cause this disease are worms of schistosoma genus. The species that usually infect humans are Schistosoma haematobium (\textit{S. haematobium}), \textit{S. japonicum}, \textit{S. mansoni}, \textit{S. intercalatum}, \textit{S. mekongi}, and \textit{S. malayensis} [2]. At least 78 countries are endemic areas. It is estimated that more than 200 million people suffer from schistosomiasis and no less than 750 million people are at risk of schistosoma infection. Approximately, the number of deaths due to schistosomiasis is 280 thousands annually [3]. Berquist [4]; Ross [5] stated that integrated control is needed so that the spread of schistosomiasis can be controlled. Integrated control is a combination of mass drug administration, WASH, education, and snail control. Chiyaka and Garira [6] developed a schistosomiasis model that takes into consideration the miracidia and cercariae compartment. The two compartments are the stages of schistosoma development. Gao et al [7] modified the model by adding parameters that related to treatment and elimination of snail and cercariae. Mathematical model that takes into account the incubation period of schistosomiasis is discussed in [8]. Unlike the models that have been described, the proposed schistosomiasis model takes into consideration health education and
molluscicide intervention.
This paper is organized as follows. Model formulation; non-negativity and boundedness of solutions of the model; equilibrium points and their existence condition are discussed in section 2. We determine the basic reproduction number and prove the stability condition of the equilibrium points in section 3. Numerical simulations to support our theoretical study and to provide a better interpretation are presented in section 4. Some conclusions and a short discussion are given in section 5.

2. Model formulation

We formulate the model with basic assumptions as follows:

- The human population is divided into three distinct compartments: susceptible human \((S_h)\), latent human \((E_h)\), and infectious human \((I_h)\).
- The snail population is divided into two distinct compartments: susceptible snail \((S_v)\) and infectious snail \((I_v)\).
- Parasites are divided into two stages: cercariae \((C)\) and miracidia \((M)\).
- Susceptible human is exposed to schistosomiasis and become latent human due to direct contact with cercariae.
- Susceptible snail can become infectious due to direct contact with miracidia.
- Only infectious human can release parasite eggs.
- There is no latent period in the snail population.
- Latent and infectious humans are able to recover from schistosomiasis.
- Humans who have recovered can get reinfected.
- There is no recovery for infectious snail.

Transitions and interactions between compartments in accordance with the basic assumptions can be seen in Figure 1.

![Figure 1. Transition and interaction diagram of model (1)](image-url)
Based on the basic assumptions and Figure 1, we obtain a system as follows:

\[
\begin{align*}
\frac{dS_h}{dt} &= \Lambda_h - (1 - \omega_v \omega) \beta_{ch} CS_h + \theta_{is} I_h - d_h S_h + \theta_{es} E_h, \\
\frac{dI_h}{dt} &= (1 - \omega_v \omega) \beta_{ch} CS_h - (\theta_{ci} + \theta_{es} + d_h) E_h, \\
\frac{dE_h}{dt} &= \theta_{ci} E_h - (\theta_{is} + d_h) I_h, \\
\frac{dS_v}{dt} &= \Lambda_v - \beta_{mv} MS_v - (d_v + d_e) S_v, \\
\frac{dI_v}{dt} &= \beta_{mv} MS_v - (d_v + d_e) I_v, \\
\frac{dM}{dt} &= \sigma I_v - d_e C, \\
\frac{dC}{dt} &= k_6 I_h - d_m M,
\end{align*}
\]

where \(\omega, \omega_v, d_e \in [0, 1]\), and other parameters are positive. \(\omega\) is the proportion of susceptible humans who receive health education related to schistosomiasis (health education coverage of susceptible human), \(\omega_v\) is effectiveness of implementation of education, \(d_e\) is molluscicide induced snail death rate, \(\Lambda_h\) is recruitment rate for human, \(\Lambda_v\) is recruitment rate for snail, \(d_h\) is human natural death rate, \(d_v\) is snail natural death rate, \(d_e\) is cercariae natural death rate, \(d_m\) is miracidia natural death rate, \(\theta_{ci}\) is transition rate from latent human to infectious human, \(\theta_{es}\) is latent human recovery rate, \(\theta_{is}\) is infectious human recovery rate, \(\alpha\) is parasite egg hatch rate, \(g_h\) is average weight of human stool per day, \(h_h\) is average number of eggs per gram of stool, \(\beta_{ch}\) is effective contact rate between susceptible human and cercariae, \(\beta_{mv}\) is effective contact rate between susceptible snail and miracidia. To make model analysis easier, we simplify system (1) and obtain a new system as follows:

\[
\begin{align*}
\frac{dS_h}{dt} &= \Lambda_h - k_1 \beta_{ch} CS_h + \theta_{is} I_h - d_h S_h + \theta_{es} E_h, \\
\frac{dI_h}{dt} &= k_1 \beta_{ch} CS_h - k_2 E_h, \\
\frac{dE_h}{dt} &= \theta_{ci} E_h - k_3 I_h, \\
\frac{dS_v}{dt} &= \Lambda_v - \beta_{mv} MS_v - k_4 S_v, \\
\frac{dI_v}{dt} &= \beta_{mv} MS_v - k_4 I_v, \\
\frac{dM}{dt} &= k_5 I_v - d_e C, \\
\frac{dC}{dt} &= k_6 I_h - d_m M,
\end{align*}
\]

where

\[
\begin{align*}
k_1 &= 1 - \omega_v \omega, \\
k_2 &= \theta_{ci} + \theta_{es} + d_h, \\
k_3 &= \theta_{is} + d_h, \\
k_4 &= d_v + d_e, \\
k_5 &= \sigma, \\
k_6 &= \alpha h_h g_h.
\end{align*}
\]

\[2.1. \text{Non-negativity and boundedness of solutions}\]

The system (2) represents the dynamic of schistosomiasis spread in human and snail populations. On that account, it is important to verify that \(S_h(t), E_h(t), I_h(t), S_v(t), I_v(t), C(t), M(t) \geq 0\) for \(t \geq 0\).

**Theorem 1.** If initial value \(S_h(0), E_h(0), I_h(0), S_v(0), I_v(0), C(0), M(0) \geq 0\) then the solutions \((S_h(t), E_h(t), I_h(t), S_v(t), I_v(t), C(t), M(t))\) of system (2) are non-negative for \(t \geq 0\).

**Proof.** Theorem 1 can be proved by using reduction to absurdity method. Let \(G(t) = \min\{S_h(t), E_h(t), I_h(t), S_v(t), I_v(t), C(t), M(t)\}\). Suppose that the conclusion of Theorem 1 is wrong, thus there is a \(t^* > 0\) such that \(G(t^*) = 0, G(t) > 0\) for \(t \in [0, t^*]\), and \(G(t^+) < 0\) for
If $G(t^*) = S_h(t^*)$ then $E_h(t^*), I_h(t^*), S_v(t^*), I_v(t^*), C(t^*), M(t^*) > 0$. From the first equation of system (2), we get

$$\frac{dS_h(t^*)}{dt} = \Lambda_h - k_1 \beta_{dh}C(t^*)S_h(t^*) + \theta_{is}I_h(t^*) - d_h S_h(t^*) + \theta_{es}E_h(t^*),$$

$$= \Lambda_h - 0 + \theta_{is}I_h(t^*) - 0 + \theta_{es}E_h(t^*),$$

$$= \Lambda_h + \theta_{is}I_h(t^*) + \theta_{es}E_h(t^*),$$

$$> 0. \tag{4}$$

If $\frac{dS_h(t^*)}{dt} > 0$, then according to monotonic function properties, it is found that $S_h(t^{**}) > 0$ for $t^{**} > t^*$. This result contradicts with the fact that $S_h(t^{**}) < 0$ for $t^{**} > t^*$. Therefore, it is proved that $S_h(t) \geq 0$ for $t \geq 0$. In the same way, we can prove that $E_h(t), I_h(t), S_v(t), I_v(t), C(t), M(t) \geq 0$ for $t \geq 0$.

**Theorem 2.** A closed and bounded set

$$\Omega_+ = \{S_h(t), E_h(t), I_h(t), S_v(t), I_v(t), C(t), M(t) \in \mathbb{R}_+^7 | N_h(t) \leq \frac{\Lambda_h}{d_h}, N_v(t) \leq \frac{\Lambda_v}{d_v}, C(t) \leq \frac{\Lambda_C}{d_C}, M(t) \leq \frac{\Lambda_M}{d_M}, t \geq 0\} \tag{5}$$

is positively invariant set of system (2) where $N_h(t) = S_h(t) + E_h(t) + I_h(t)$ and $N_v(t) = S_v(t) + I_v(t)$. Solutions of system (2) with initial value in $\Omega_+$ remain in $\Omega_+$.

**Proof.** Let $N_h = S_h + E_h + I_h$, then $\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dI_h}{dt}$. By adding up all equations of system (2), we get

$$\frac{dN_h}{dt} + d_h N_h = \Lambda_h - d_h N_h, \tag{6}$$

with general solution as follows

$$N_h(t) = e^{-\int d_h dt} \left( \int e^{\int d_h dt} \Lambda_h dt \right),$$

$$= e^{-d_h t} \left( \frac{\Lambda_h}{d_h} e^{d_h t} + U \right),$$

$$= \frac{\Lambda_h}{d_h} + U e^{-d_h t}. \tag{7}$$

for any value $U$. Let $N_h(0) = N_{h0}$, we obtain:

$$N_h(t) = \frac{\Lambda_h}{d_h} + \left( N_{h0} - \frac{\Lambda_h}{d_h} \right) e^{-d_h t}. \tag{8}$$

It is obvious that $e^{-d_h t} > 0$ for all $t \in \mathbb{R}^+$. If we choose initial value $N_{h0} \leq \frac{\Lambda_h}{d_h}$ that leads to $N_{h0} - \frac{\Lambda_h}{d_h} \leq 0$, we have

$$N_h(t) = \frac{\Lambda_h}{d_h} + \left( N_{h0} - \frac{\Lambda_h}{d_h} \right) e^{-d_h t},$$

$$\leq \frac{\Lambda_h}{d_h} + 0 \times e^{-d_h t},$$

$$= \frac{\Lambda_h}{d_h}. \tag{9}$$

and

$$\lim_{t \to +\infty} N_h(t) = \lim_{t \to +\infty} \left( \frac{\Lambda_h}{d_h} + \left( N_{h0} - \frac{\Lambda_h}{d_h} \right) e^{-d_h t} \right), \tag{10}$$

$$= \frac{\Lambda_h}{d_h}.$$

Therefore, the solutions of system (2) satisfy $N_h(t) = S_h(t) + E_h(t) + I_h(t) \leq \frac{\Lambda_h}{d_h}$ provided that initial value $N_h(0) \leq \frac{\Lambda_h}{d_h}$. Similarly, we can prove that $N_v(t) = S_v(t) + I_v(t) \leq \frac{\Lambda_v}{d_v}$. These results imply that $I_h$ and $I_v$ are bounded. In line with these facts and by using Gronwall’s inequality, it is easy to show that $M(t) \leq \frac{\Lambda_M}{d_M}$, and $C(t) \leq \frac{\Lambda_C}{d_C}$. It is proved that all solutions of system (2) are bounded. Based on these results and Theorem 1, we conclude that $\Omega_+$ is positively invariant set of system (2).
2.2. Steady states

The System (2) has two equilibrium points, namely disease free equilibrium point (DFE) and endemic equilibrium point (EE).

(i) The disease free equilibrium point is defined by

\[ \Xi_0 = (S^*_h, E^*_h, I^*_h, S^*_v, I^*_v, C^*, M^*) = \left( \frac{\Lambda}{d_h}, 0, 0, \frac{\Lambda}{k_4}, 0, 0 \right). \]  

\[ \Xi_0 \text{ always exists in } R^7_+. \]

(ii) The endemic equilibrium point is defined by

\[ \Xi_1 = (S^{**}_h, E^{**}_h, I^{**}_h, S^{**}_v, I^{**}_v, C^{**}, M^{**}), \]

where

\[ S^{**}_h = \frac{1}{\theta_{ei}} \left( \frac{1}{\theta_{es}} + k_3(\theta_{es} - k_2) \right) I^{**}_h, \]

\[ E^{**}_h = \frac{1}{\theta_{ei}} \left( R_e - 1 \right) d_c d_m k_4 k_2 d_h, \]

\[ I^{**}_h = \frac{1}{\beta_{ch} k_2 k_3 k_6} \left( d_c k_4 d_m k_2 k_3 d_h \right) \]

\[ S^{**}_v = \frac{1}{\beta_{mv} k_2 k_4 d_m}, \]

\[ I^{**}_v = \frac{1}{\beta_{mv} k_2 k_4 d_m}, \]

\[ C^{**} = \frac{1}{d_c k_4 k_6 d_m k_5}, \]

\[ M^{**} = \frac{1}{d_m k_6 k_7}. \]

\[ R_e = \frac{k_1 \beta_{ch} k_2 k_3 k_4 k_6 \theta_{ci} \Lambda_h}{d_c k_4^2 d_m k_2 k_3 d_h}. \]

\[ \Xi_1 \text{ exists in } R^7_+ \text{ provided that } R_e > 1. \]

3. Model analysis

3.1. Basic reproduction number

The basic reproduction number from an epidemiological point of view is defined as average number of new infections in a vulnerable population produced by one infectious individual. The basic reproduction number is computed using the next generation matrix approach developed in [9].

\[ F = D\mathcal{F}(\Xi_0) \]

\[ \mathcal{F} = \begin{pmatrix} \beta_{ch} k_1 C S_h \\
0 \\
\beta_{mv} M S_v \\
0 \\
0 \\
0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} k_2 E_h \\
k_3 I_h - \theta_{ei} E_h \\
k_4 I_v \\
C - k_2 I_v \\
d_m M - k_6 I_h \end{pmatrix}. \]

Substituting \( \Xi_0 \) into the jacobian matrix of \( \mathcal{F} \) and \( \mathcal{V} \), we find that:

\[ F = D\mathcal{F}(\Xi_0) = \begin{pmatrix} 0 & 0 & \beta_{ch} k_1 S^*_h \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \end{pmatrix}, \]
We obtain next generation matrix as follows

\[
FV^{-1} = \begin{pmatrix}
0 & 0 & 0 & \beta_{ch} k_1 S_h^* & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0
\end{pmatrix}
\]

According to Theorem 2 in [9], the reproduction number is the spectral radius of the next generation matrix. Thus, we get

\[
R_0 = \rho(FV^{-1}) = +\sqrt{\frac{\beta_{ch} k_1 S_h^*}{d_c} \frac{\beta_{mv} S_v^*}{d_m} \frac{\beta_{ch} k_1 S_h^*}{d_c} \frac{\beta_{mv} S_v^*}{d_m}}.
\]

Substituting \( S_h^* \) and \( S_v^* \) into \( R_0 \) yields

\[
R_0 = \sqrt{\frac{\beta_{ch} k_1 \Lambda_h \beta_{mv} \Lambda_v \theta_{ei}}{d_c k_4 d_h} \frac{\beta_{mv} \Lambda_v \theta_{ei}}{k_4 k_5 d_m}}.
\]

We observe that \( R_c = R_0^2 \). Because \( R_0 \) is positive, \( R_c > 1 \) if \( R_0 > 1 \). In accordance with existence condition of the endemic equilibrium point, \( \Xi_1 \) exists if \( R_0 > 1 \).

### 3.2. Local stability of the disease free equilibrium point

**Theorem 3.** The disease free equilibrium point \( \Xi_0 \) of system (2) defined by (11) is locally asymptotically stable if \( R_0 < 1 \) and unstable if \( R_0 > 1 \).

**Proof.** Theorem 3 can be proved by linearization technique. The jacobian matrix of (2) is:

\[
J = \begin{pmatrix}
\beta_{ch} k_1 C - d_h & \theta_{es} & \theta_{is} & 0 & 0 & -\beta_{ch} k_1 S_h & 0 \\
\beta_{ch} k_1 C & -k_2 & 0 & 0 & 0 & -\beta_{ch} k_1 S_h & 0 \\
0 & \theta_{ei} & -k_3 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & -\beta_{mv} M - k_4 & 0 & 0 & -\beta_{mv} S_v \\
0 & 0 & 0 & \beta_{mv} M & -k_4 & 0 & 0 \\
0 & 0 & 0 & k_5 & -d_c & 0 & 0 \\
0 & 0 & k_6 & 0 & 0 & -d_m & 0
\end{pmatrix}
\]
After substituting $\Xi_0$ into (16), we get

$$J(\Xi_0) = \begin{pmatrix}
-d_h & \theta_{es} & \theta_{ts} & 0 & 0 & -\beta_{ch}k_1S_h^* & 0 \\
0 & -k_2 & 0 & 0 & 0 & \beta_{ch}k_1S_h^* & 0 \\
0 & \theta_{ei} & -k_3 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & -k_4 & 0 & 0 & -\beta_{mv}S_v^* \\
0 & 0 & 0 & 0 & k_5 & -d_c & 0 \\
0 & 0 & k_6 & 0 & 0 & 0 & -d_m \\
\end{pmatrix}.$$ (17)

The characteristic polynomial of (17) is

$$\det(\lambda I - J(\Xi_0)) = (\lambda + d_h)(\lambda + k_2)(\lambda + k_3)(\lambda + k_4)(\lambda + d_c)(\lambda + d_m)$$
$$+ (\lambda + d_h)\theta_{ei}(-k_6)(-k_6)(-k_5)(-\beta_{ch}k_1S_h^*)(-\beta_{mv}S_v^*)$$
$$+ (\lambda + d_h)(\lambda + k_4)((\lambda + k_2)(\lambda + k_3)(\lambda + k_4)(\lambda + d_c)(\lambda + d_m)$$
$$- \theta_{ei}k_6k_5k_1\beta_{ch}k_1S_h^*\beta_{mv}S_v^*)$$
$$= (\lambda + d_h)(\lambda + k_4)Q(\lambda)$$
$$= 0.$$

It is easy to see that two eigenvalues are negative. $\lambda_1 = -d_h$ and $\lambda_2 = -k_4$. Next, we determine the eigenvalues in $Q(\lambda)$.

$$Q(\lambda) = (\lambda + k_2)(\lambda + k_3)(\lambda + k_4)(\lambda + d_c)(\lambda + d_m) - \theta_{ei}k_6k_5k_1\beta_{ch}k_1S_h^*\beta_{mv}S_v^*$$
$$= \lambda^5 + q_1\lambda^4 + q_2\lambda^3 + q_3\lambda^2 + q_4\lambda + (1 - R_0)k_2k_3k_4d_c d_m$$ (18)
$$= 0,$$

where

$$q_1 = k_2 + k_3 + k_4 + d_c + d_m,$$
$$q_2 = \sum_{1 \leq z_1 < z_2}^5 (p_{z_1}p_{z_2}),$$
$$q_3 = \sum_{1 \leq z_1 < z_2 < z_3}^5 (p_{z_1}p_{z_2}p_{z_3}),$$
$$q_4 = \sum_{1 \leq z_1 < z_2 < z_3 < z_4}^5 (p_{z_1}p_{z_2}p_{z_3}p_{z_4}),$$

and $p_1 = k_2, p_2 = k_3, p_3 = k_4, p_4 = d_c, p_5 = d_m$. Since $k_2, k_3, k_4, d_c, d_m \in \mathbb{R}_+$, $q_1, q_2, q_3, q_4 \in \mathbb{R}_+$.

The number of negative and positive roots of (18) is determined by using Descartes’ sign rule.

| $R_0 < 1$ | $R_0 > 1$ |
|-----------|-----------|
| Number of positive roots | 0 | 1 |
| Number of negative roots | 5 or 3 or 1 | 4 or 2 or 0 |
Based on Table (1), we conclude:

(i) If $R_0 < 1$, then all eigenvalues of (17) are negative. Hence, the disease free equilibrium point $(\Xi_0)$ is locally asymptotically stable;

(ii) If $R_0 > 1$, then one eigenvalue of (17) is positive. Hence, the disease free equilibrium point $(\Xi_0)$ is unstable;

It is clear that if $R_0 = 1$ then constant term of (18) is zero. Thus, one eigenvalue is zero.

3.3. Local stability of the endemic equilibrium point

**Theorem 4.** The endemic equilibrium point $(\Xi_1)$ of system (2) defined by (12) is locally asymptotically stable if $R_0 > 1$ $(\beta_{ch} > \beta_{ch}^*)$ where

$$
\beta_{ch}^* = \frac{k_2^2 k_3 d_m d_c d_h}{\beta_{me} \Lambda_c \theta_c \kappa_1 \Lambda_h \kappa_5}.
$$

**Proof.** Let $\beta_{ch}$ be bifurcation parameter and suppose $R_0$ is a function of bifurcation parameter. Center manifold theory [10] will be used to analyze local stability of endemic equilibrium point. In the previous subsection, it is shown that if $R_0 = 1$ then one eigenvalue of (17) is zero and the other eigenvalues are negative. Therefore, bifurcation point $(\beta_{ch}^*)$ will be evaluated when $R_0 = 1$.

$$
R_0 = \sqrt{\frac{\beta_{ch} \Lambda_h \kappa_1 \kappa_5}{d_c k_4 d_h} \cdot \frac{\beta_{me} \Lambda_c \theta_c \kappa_1 \Lambda_h}{d_c k_2 d_m}} = 1
$$

$$
\beta_{ch}^* = \frac{k_2^2 k_3 d_m d_c d_h}{\beta_{me} \Lambda_c \theta_c \kappa_1 \Lambda_h \kappa_5}.
$$

It is straightforward to show that the right eigenvector of $J(\Xi_0, \beta_{ch}^*)$ corresponding to zero eigenvalue is

$$
\tilde{w} = \begin{pmatrix}
w_1 \\
w_2 \\
w_3 \\
w_4 \\
w_5 \\
w_6 \\
w_7 \\
\end{pmatrix} = \begin{pmatrix}
-d_m (\theta_c + \theta_c + \beta_{ch}^*) k_1 S_h^* w_6 \\
\beta_{ch}^* k_2 k_3 \\
\theta_c \beta_{ch}^* k_1 S_h^* w_6 \\
-\beta_{me} S_c^* k_2 \beta_{ch}^* k_1 S_h^* w_6 \\
\beta_{me} S_c^* k_4 d_m k_3 k_2 \\
k_2 \theta_c \beta_{ch}^* k_1 S_h^* w_6 \\
k_4 d_m k_3 k_2 \\
\end{pmatrix},
$$

with $w_6 > 0$. The left eigenvector of $J(\Xi_0, \beta_{ch}^*)$ corresponding to zero eigenvalue is

$$
\tilde{v}^T = \begin{pmatrix}
v_1 \\
v_2 \\
v_3 \\
v_4 \\
v_5 \\
v_6 \\
v_7 \\
\end{pmatrix} = \begin{pmatrix}
0 \\
k_6 \beta_{me} S_c^* k_2 \beta_{ch}^* k_1 S_h^* v_2 \\
k_3 d_m k_4 d_c \\
k_5 \beta_{ch}^* k_1 S_h^* v_2 \\
k_4 d_m k_3 k_2 \\
k_2 \beta_{ch}^* S_c^* k_5 \beta_{ch}^* k_1 S_h^* v_2 \\
k_6 \beta_{me} S_c^* k_5 \beta_{ch}^* k_1 S_h^* v_2 \\
\end{pmatrix}.
$$
with \( v_2 > 0 \). Before applying center manifold theory \([10]\), we make small change as follows

\[
\begin{align*}
x_1 &= S_h, \\
x_2 &= E_h, \\
x_3 &= I_h, \\
x_4 &= S_v, \\
x_5 &= I_v, \\
x_6 &= C, \\
x_7 &= M,
\end{align*}
\]

and

\[
\begin{align*}
f_1 &= \Lambda_h - k_1 \beta_{ch} x_6 x_1 + \theta_t x_3 - d_h x_1 + \theta_e x_2, \\
f_2 &= k_1 \beta_{ch} x_6 x_1 - k_2 x_2, \\
f_3 &= \theta_e x_2 - k_3 x_3, \\
f_4 &= \Lambda_v - \beta_{mv} x_7 x_4 - k_4 x_4, \\
f_5 &= \beta_{mv} x_7 x_4 - k_4 x_5, \\
f_6 &= k_5 x_5 - d_c x_6, \\
f_7 &= k_6 x_3 - d_m x_7.
\end{align*}
\]

Non-zero derivatives of (19) are

\[
\begin{align*}
\frac{\partial^2 f_2}{\partial x_6 \partial x_1} (\Xi_0; \beta_{ch}^*) &= k_1 \beta_{ch}^*, \\
\frac{\partial^2 f_2}{\partial x_6 \partial x_1} (\Xi_0; \beta_{ch}^*) &= k_1 \beta_{ch}^*, \\
\frac{\partial^2 f_2}{\partial x_6 \partial x_1} (\Xi_0; \beta_{ch}^*) &= \beta_{mv}, \\
\frac{\partial^2 f_2}{\partial x_7 \partial x_4} (\Xi_0; \beta_{ch}^*) &= \beta_{mv}, \\
\frac{\partial^2 f_2}{\partial x_6 \partial x_1} (\Xi_0; \beta_{ch}^*) &= k_1 \Lambda_h. \\
\end{align*}
\]

Hence, we get

\[
\begin{align*}
a &= \sum_{k, i, j=1}^n \left( v_k w_i w_j \left( \frac{\partial^2 f_2}{\partial x_6 \partial x_1} (\Xi_0; \beta_{ch}^*) \right) \right) \\
&= v_2 w_1 w_6 \frac{\partial f_2}{\partial x_6 \partial x_1} (\Xi_0; \beta_{ch}^*) \biggr|_{\beta_{ch}^* = \beta_{ch}} + v_2 w_6 w_1 \frac{\partial f_2}{\partial x_6 \partial x_1} (\Xi_0; \beta_{ch}^*) \biggr|_{\beta_{ch}^* = \beta_{ch}} + v_3 w_4 w_7 \frac{\partial f_2}{\partial x_6 \partial x_1} (\Xi_0; \beta_{ch}^*) \biggr|_{\beta_{ch}^* = \beta_{ch}} \biggr|_{\beta_{ch}^* = \beta_{ch}} \\
&= -2 \left( \frac{d_k (\theta_t + \theta_e + d_h) \beta_{mv}^* k_1 S_i^* h}{d_h k_6 k_2 k_5} \right) v_2 w_6 k_1 \beta_{ch}^* \\
&< 0,
\end{align*}
\]

and

\[
\begin{align*}
b &= \sum_{k, i=1}^n \left( v_k w_i \left( \frac{\partial^2 f_2}{\partial x_6 \partial x_1} (\Xi_0; \beta_{ch}^*) \right) \right) \\
&= v_2 w_6 \frac{k_1 \Lambda_h}{d_h} \biggr|_{\beta_{ch}^* = \beta_{ch}} \biggr|_{\beta_{ch}^* = \beta_{ch}} > 0.
\end{align*}
\]

Since \( a < 0 \) and \( b > 0 \), based on Theorem 4.1 in \([10]\), forward bifurcation occurs and there is a positive and locally asymptotically stable equilibrium point if \( \beta_{ch} > \beta_{ch}^* (R_0 > 1) \). It is proved that the system (2) has only one positive equilibrium point, that is, the endemic equilibrium point \((\Xi_1)\), if \( R_0 > 1 \). Hence, \((\Xi_1)\) is locally asymptotically stable if \( R_0 > 1 \).
3.4. Global stability of the disease free equilibrium point

**Theorem 5.** The disease free equilibrium point \( (\Xi_0) \) of system (2) defined by (11) is globally asymptotically stable if \( R_0 < 1 \).

**Proof.** Consider Lyapunov function candidate as follows

\[
L(\vec{x}) = \frac{k_5 \theta_{ci} d_m}{d_m k_2 k_3} E_h + \frac{k_5}{d_m k_3} I_h + \frac{k_6 k_5 k_1 \theta_{ci} \lambda_0}{d_m k_4 d_m k_2 k_3 R_0} I_v + \frac{k_5 k_1 \theta_{ci} \lambda_0}{d_m k_2 k_3 d_c R_0} C + \frac{1}{d_m} M.
\]

with \( \vec{x} = (S_h, E_h, I_h, S_v, I_v, C, M) \in \Omega_+ \). \( \Omega_+ \) is closed and bounded set. It is clear that \( L(\vec{x}) > 0 \) for \( \vec{x} \in \Omega_+ \setminus \{ \Xi_0 \} \) and \( L(\vec{x}) = 0 \) for \( \vec{x} = \Xi_0 \in \Omega_+ \).

Calculating the derivatives of \( L(\vec{x}) \), we find that

\[
L'(\vec{x}) = \frac{k_6 \theta_{ci} d_m}{d_m k_2 k_3} \frac{dE_h}{dt} + \frac{k_6}{d_m k_3} \frac{dI_h}{dt} + \frac{k_6 k_5 k_1 \theta_{ci} \lambda_0}{d_m k_4 d_m k_2 k_3 R_0} \frac{dI_v}{dt} + \frac{k_5 k_1 \theta_{ci} \lambda_0}{d_m k_2 k_3 d_c R_0} \frac{dC}{dt} + \frac{1}{d_m} \frac{dM}{dt}
\]

\[
= \frac{k_6 \theta_{ci} d_m}{d_m k_2 k_3} (k_1 \beta_h C S_h - k_2 E_h) + \frac{k_6}{d_m k_3} (\theta_{ci} E_h - k_2 I_h) + \frac{k_6 k_5 k_1 \theta_{ci} \lambda_0}{d_m k_4 d_m k_2 k_3 R_0} \frac{dI_v}{dt} + \frac{k_5 k_1 \theta_{ci} \lambda_0}{d_m k_2 k_3 d_c R_0} \frac{dC}{dt} + \frac{1}{d_m} \frac{dM}{dt}
\]

\[
= \frac{k_6 \theta_{ci} k_1 \beta_h CS_h}{d_m k_2 k_3 (1 - R_0)} + \frac{k_6 k_5 k_1 \theta_{ci} \lambda_0}{d_m k_4 d_m k_2 k_3 R_0} (\frac{\lambda_h}{d_h} \beta_{mu} S_v - \frac{\Lambda_h}{d_h}) - \frac{k_5 k_1 \theta_{ci} \lambda_0}{d_m k_2 k_3 d_c R_0} (\frac{\Lambda_h}{d_h} C)
\]

\[
\leq \frac{k_6 \theta_{ci} k_1 \beta_h CS_h}{d_m k_2 k_3 (1 - R_0)} + \frac{k_6 k_5 k_1 \theta_{ci} \lambda_0}{d_m k_4 d_m k_2 k_3 R_0} (\frac{\Lambda_h}{d_h} \beta_{mu} S_v - \frac{\Lambda_h}{d_h}) - \frac{k_5 k_1 \theta_{ci} \lambda_0}{d_m k_2 k_3 d_c R_0} (\frac{\Lambda_h}{d_h} C)
\]

\[
= \frac{k_6 \theta_{ci} k_1 \beta_h (S_h - \frac{\lambda_h}{d_h})}{d_m k_2 k_3 (1 - R_0)} C + \left( \frac{R_0^2}{R_0^2 - 1} \right) M
\]

\[
= \frac{k_6 \theta_{ci} k_1 \beta_h (\frac{\Lambda_h}{d_h} - S_h)}{d_m k_2 k_3 (1 - R_0)} C + (R_0 - 1) M.
\]

Since \( \frac{\lambda_h}{d_h} \geq S_h \), \( L'(\vec{x}) \leq 0 \) in \( \Omega_+ \) if \( R_0 < 1 \). Furthermore, \( L'(\vec{x}) = 0 \) for \( S_h = \frac{\lambda_h}{d_h} \) and \( M = 0 \) or \( C = 0 \) and \( M = 0 \). These conditions are only satisfied by the disease free equilibrium point \( \Xi_0 = (S_h, E_h, I_h, S_v, I_v, C, M) = (\frac{\lambda_h}{d_h}, 0, 0, \frac{\Lambda_h}{d_h}, 0, 0, 0) \). Thus, the largest compact invariant set contained in \( Z = \{(S_h, E_h, I_h, S_v, I_v, C, M) | L' = 0 \} \) is singleton set, that is, \( \Xi_0 \). Based on LaSalle invariance principle, we conclude that \( \Xi_0 \) is globally asymptotically stable if \( R_0 < 1 \).
4. Numerical simulation

In this section, we present a number of numerical simulations of system (2) to support our theoretical study and to explore the impact of changing parameter $\omega$, $\omega_e$, and $d_r$. We use parameter values that are given in Table (2) while $\omega$, $\omega_e$ and $d_r$ are varied. The initial value that we use is $S_h(0) = 17822$, $E_h(0) = 0$, $I_h(0) = 139$, $S_v(0) = 1000$, $I_v(0) = 10$, $C = 10$, $M = 10$.

| Par. | Value | Dimension | Source |
|------|-------|-----------|--------|
| $d_h$ | $\frac{1}{65 \times 365}$ | day$^{-1}$ | [11] |
| $\theta_{hs}$ | $\frac{1}{10 \times 7}$ | day$^{-1}$ | [12] |
| $\theta_{es}$ | $\frac{1}{6 \times 7}$ | day$^{-1}$ | [13] |
| $\theta_{cs}$ | $\frac{1}{2 \times 7}$ | day$^{-1}$ | [12] |
| $\Lambda_h$ | $22682 \times 1\%$ | day$^{-1}$ | Assumed |
| $\Lambda_v$ | 2.25 | day$^{-1}$ | [8] |
| $d_v$ | 0.000569 | day$^{-1}$ | [6] |
| $d_m$ | 6 | day$^{-1}$ | [11] |
| $d_c$ | 3 | day$^{-1}$ | [11] |
| $\alpha$ | 0.00232 | | [6] |
| $g_h$ | 160 | gram $\times$ day$^{-1}$ | [8] |
| $h_h$ | 10 | gram$^{-1}$ | [8] |
| $\sigma$ | 160 | day$^{-1}$ | [14] |
| $\beta_{hv}$ | $1.914 \times 10^{-9}$ | | [8] |
| $\beta_{mv}$ | $\frac{0.0004}{365}$ | | [15] |
| $\omega$ | (0, 1) | | Assumed |
| $\omega_e$ | (0, 1) | | Assumed |
| $d_r$ | (0, 1) | | Assumed |

4.1. Impact of the proportion of susceptible humans who receive health education (health education coverage of susceptible human)

The first simulation is carried out to compare the dynamics of schistosomiasis spread when $\omega$ is varied. We have fixed $\omega_e = 0.5$ and $d_r = 0$. We used $\omega = 0.1; 0.5; 0.9$, giving $R_0 = 34.2771; 30.4560; 26.0809$, respectively. Based on Theorem 4, the endemic equilibrium point is locally asymptotically stable for three cases. Hence, the disease will persist.
Figure 2. Dynamics of latent human, infectious human, infectious snail, and cercariae with varying $\omega$. The number of latent human and infectious human increase significantly in the first year and then decrease until it reaches an equilibrium point. The number of infectious snail and cercariae increase significantly and the peak have not yet seen. If the time span is extended, the solution curves, $E_h, I_h, I_v, C$, tend to the endemic equilibrium point.

Figure 2 shows the number of latent human, infectious human, infectious snail and cercariae decrease as $\omega$ increases. Nevertheless, schistosomiasis remains persistent although 90% of susceptible human receive health education. this situation may occur because the effectiveness of implementation of education is 50%. To verify this matter, we perform the second simulation in the next subsection.

4.2. Impact of effectiveness of implementation of education

The second simulation is carried out to compare the dynamics of schistosomiasis spread when $\omega_e$ is varied. We have fixed $\omega = 0.9$ and $d_r = 0$. We used $\omega_e = 0.3; 0.6; 0.9$, giving $R_0 = 30.0471; 23.8518; 15.3292$, respectively. We observe that the $R_0$ decreases as $\omega_e$ increases. Furthermore, the last two $R_0$ are less than the last $R_0$ from the previous simulation. Even so, based on Theorem 4, the endemic equilibrium point is locally asymptotically stable for three cases. Thus, the disease will persist.
Figure 3. Dynamics of latent human, infectious human, infectious snail, and cercariae with varying $\omega_e$. The number of latent human and infectious human increase significantly in the first two years and then decrease until it reaches an equilibrium point. The number of infectious snail and cercariae increase significantly and the peak have not yet seen. If the time span is extended, the solution curves, $E_h, I_h, I_v, C$, tend to the endemic equilibrium point.

Figure 3 shows the number of latent human, infectious human, infectious snail and cercariae decrease as $\omega$ increases. However, schistosomiasis remains persistent even though 90% of susceptible humans receive health education with effectiveness of implementation of education is 90%. We recognize the peak for the last two cases from the second simulation are less than the peak from the first simulation. Therefore, an increase in the proportion of susceptible humans who receive health education must be accompanied by an increase in the effectiveness of implementation of education so that the results obtained are maximized. Additionally, these results illustrate that schistosomiasis cannot be eradicated if the only intervention is health education.

4.3. Impact of molluscicide intervention

The third simulation is carried out to compare the dynamics of schistosomiasis spread when $d_r$ is varied. We have fixed $\omega = 0.8$ and $\omega_e = 0.8$. We used $d_r = 0.005; 0.01; 0.015$, giving $R_0 = 2.1559; 1.1360; 0.7712$, respectively. The last $R_0$ is less than unity. Based on Theorem 3, 4 and 5, the endemic equilibrium point is locally asymptotically stable for the first two cases.
and the disease free equilibrium point is asymptotically stable (locally and globally) for the last case. Therefore, the disease will persist for the first two cases and the disease will be eradicated for the last case.

Figure 4 shows the number of latent human, infectious human, infectious snail and cercariae decrease as $d_r$ increases. Yet, schistosomiasis remains persistent if $d_r = 0.005$ and $d_r = 0.01$ despite 80% of susceptible humans receive health education with $\omega_e = 0.8$. Whereas, schistosomiasis is successfully eradicated if $d_r = 0.015$. These results tell us that schistosomiasis can be eradicated by doing integrated control.

5. Evaluation of control measures
Evaluation of intervention parameters, $\omega, \omega_e, d_r$, is discussed in this section. We investigate the effect of changing intervention parameter value on the basic reproduction number.
Figure 5. The first graph shows the effect of changing $\omega$ and $\omega_e$ on $R_0$ while $d_r = 0.005$. We recognize that $R_0$ always greater than one. On top of that, $\omega$ and $\omega_e$ have similar effect on $R_0$. The second and third graph show the effect of changing $d_r, \omega$ and $d_r, \omega_e$ on $R_0$, respectively. Both graphs have the same pattern. We observe that there is a curve that divides the $\omega - d_r$ plane and $\omega_e - d_r$ plane into two areas, respectively. One area indicates $R_0 < 1$ and the other area indicates $R_0 > 1$. Furthermore, it is clear that $R_0$ is strongly influenced by $d_r$.

Figure 2-5 show the prevalence and $R_0$ is more sensitive to variation of $d_r$ than $\omega$ and $\omega_e$. Hence, snail population control is effective method to control schistosomiasis spread.

6. Conclusion
In this article, we have discussed the schistosomiasis model with health education and molluscicide intervention. The numerical simulation results are the same as the results of the theoretical study. Schistosomiasis spread can supressed through health education and molluscicide intervention. However, eradication of the disease cannot be achieved if the intervention is carried out only through health education. Health education intervention must be combined with other interventions. The prevalence and $R_0$ is totally sensitive to $d_r$. Thus, molluscicide intervention is a very effective method to control schistosomiasis spread.
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