

**THE IMPACT OF REDUCED RE-INFECTION ON SCHISTOSOMIASIS TRANSMISSION
DYNAMICS AT POPULATION LEVEL: A THEORETICAL STUDY**

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Abstract

A novel deterministic model for the transmission dynamics of schistosomiasis is designed and deployed to both qualitatively assess the role of the impact of re-infection on the population dynamics for schistosomiasis disease burden in the presence of intermediate stages of development of the pathogen responsible for the disease. The model is shown to undergo the backward bifurcation phenomenon due to the presence of the reduced re-infection parameter. A unique threshold for the reduced rate of re-infection was also obtained. A special case of the model showed that the disease-free equilibrium was locally asymptotic stable in the absence of the reduced rate of re-infection.

Keywords: Backward bifurcation, disease, reduced re-infection, schistosomiasis and stability

1. Introduction

Schistosomiasis is an acute and chronic parasitic epidemic precipitated by *Schistosoma spp* which are blood flukes [1-7]. Global estimates reveal that at least 220.8 million persons required preventive treatment in 2017 [6-7]. Preventive medical care, which should be repeated over a number of years, will decrease and curtail morbidity [3-7]. About 78 countries have reported schistosomiasis outbreak worldwide [29-33]. People are infected during casual agricultural, domestic, occupational, and recreational activities, which bring them in direct contact with infested water [1, 3-7]. The absence of hygiene, coupled with play lifestyles of children of school age such as fishing or swimming in water infested, make them specifically vulnerable to infection [1, 3-7]. Schistosomiasis control focuses on reducing disease through periodic, large-scale population treatment with praziquantel; a more comprehensive approach including potable water, adequate sanitation, and snail control would also reduce transmission [9-10]. However, preventive chemotherapy for schistosomiasis, where people and communities are targeted for large-scale treatment, is only required in 52 endemic countries with moderate-to-high transmission [3-7].

Generally, several authors have developed mathematical models for investigating schistosomiasis disease dynamics with different questions in mind which have enriched the literature [8-31], and in particular, Qi *et al.* [27] investigated the effect of re-infection on schistosomiasis dynamics amongst other issues of interest.

It is evident, from the foregoing, that the several mathematical models have been developed to analyze schistosomiasis infection but none has looked at the possibility of the impact of the intermediate stages of development of the *Schistosoma spp* on the burden of the disease in the population in the presence of re-infection of individuals treated for the disease, to the best of the authors' knowledge.

Hence, we propose a new mathematical model to provide insight into schistosomiasis dynamics with the impact of the reduced re-infection of individuals treated for the disease, incorporating the intermediate stages (cercariae and miracidia, respectively) of development of the pathogen responsible. The paper is organized as follows: Section 2 contains the model formulation. The qualitative mathematical analysis is done in Sections 3 while Section 4 gives the conclusion.

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Table 1: Model variables and their definitions

Variables	Description
S_H	Susceptible human population
E_{HS}	Human population exposed to schistosomiasis
I_{HS}	Human population infected with schistosomiasis
T_{HS}	Human population treated for schistosomiasis
L	Miracidia (parasite larvae just after hatching from the eggs) population
S_S	Susceptible snail population
I_S	Snail population infected with miracidia in the aquatic environment
J	Cercariae (larvae in the water that penetrates the human skin) population

Table 2: Model parameters and their definitions

Parameter	Description
Λ_H	Human recruitment rate
μ_H	Natural death rate of humans
ψ	Reduced rate of infection with schistosomiasis
α_1	Progression rate from latently to actively infected with schistosomiasis
Λ_S	Recruitment rate for snail population
μ_S	Snail mortality rate
ϵ	Limitation of the growth velocity
L_0	Saturation constant for the miracidia
β_L	Miracidial infection rate
N_e	Number of eggs secreted by humans
γ	Rate at which eggs successfully become miracidia
μ_L	Miracidial death rate
ϕ	Cercarial production rate
J_0	Saturation constant for the cercariae
β_J	Cercarial infection rate
μ_J	Cercarial death rate

The purpose of this current study is to mathematically (i.e., theoretically) investigate the impact of re-infection on the population dynamics for schistosomiasis disease burden in the presence of intermediate stages of development of the pathogen responsible for the disease.

In this study, in Section 2, a novel mathematical model is formulated to investigate the effect of re-infection on the dynamics of schistosomiasis at population level; important thresholds governing the disease dynamics are obtained and the local and global asymptotic stabilities of equilibria are established. Section 3 concludes the paper.

2.0 Model Formulation

The total human population at time t , denoted by $N_H(t)$, is split into the mutually exclusive compartments of susceptible to infections ($S_H(t)$), exposed to schistosomiasis ($E_{HS}(t)$), infected with schistosomiasis ($I_{HS}(t)$), treated for schistosomiasis ($T_{HS}(t)$), individuals so that

$$N_H(t) = S_H(t) + E_{HS}(t) + I_{HS}(t) + T_{HS}(t).$$

Similarly, the entire snail population in the freshwater environment at time t , given by $N_S(t)$, is broken down into the mutually exclusive compartments of susceptible snails ($S_S(t)$) and snails penetrated with miracidia ($I_S(t)$), where

$$N_S(t) = S_S(t) + I_S(t).$$

The miracidia and cercariae population at the different stages in the life-cycle of the *Schistosoma spp* are depicted by $L(t)$ and $J(t)$ compartments respectively.

The model is the following deterministic system of eight non-linear ordinary differential equations (the parameters of the model are tabulated in Table 2):

$$\begin{aligned}
 S'_H &= \Lambda_H - \lambda_J S_H - \mu_H S_H, \\
 E'_{HS} &= \lambda_J (S_H + \psi T_{HS}) - (\alpha_1 + \mu_H) E_{HS}, \\
 I'_{HS} &= \alpha_1 E_{HS} - (\zeta_S + \delta_S + \mu_H) I_{HS}, \\
 T'_{HS} &= \zeta_S I_{HS} - \psi \lambda_J T_{HS} - \mu_H T_{HS}, \\
 L' &= N_e \gamma I_{HS} - \mu_L L, \\
 S'_S &= \Lambda_S - \lambda_L S_S - \mu_S S_S, \\
 I'_S &= \lambda_L S_S - \mu_S I_S, \\
 J' &= \phi I_S - \mu_J J.
 \end{aligned}
 \tag{2.1}$$

where the force of infection associated with schistosomiasis (following penetration by cercariae) and snail penetration by miracidia respectively are given below:

$$\lambda_J = \frac{\beta_J J(t)}{J_0 + \epsilon J(t)},
 \tag{2.2}$$

$$\lambda_L = \frac{\beta_L L(t)}{L_0 + \epsilon L(t)}.
 \tag{2.3}$$

2.1 Positivity and Boundedness of Solutions

Theorem 2.1: Let the basic data for the tuberculosis-schistosomiasis co-infection model be given as $S_H(0) > 0, E_{HS}(0) > 0, I_{HS}(0) > 0, T_{HS}(0) > 0, L(0) > 0, S_S(0) > 0, I_S(0) > 0$ and $J(0) > 0$. Then the orbits $(S_H(t), E_{HS}(t), I_{HS}(t), T_{HS}(t), L(t), S_S(t), I_S(t), J(t))$ of the model with positive basic conditions, will continue to be positive for all time $t > 0$.

Proof: Let $t_1 = \sup\{t > 0: S_H(0) > 0, E_{HS}(0) > 0, I_{HS}(0) > 0, T_{HS}(0) > 0, L(0) > 0, S_S(0) > 0, I_S(0) > 0, J(0) > 0\}$. Consider the first equation of model (2.1), given below as

$$\frac{dS_H(t)}{dt} = \Lambda_H - (\lambda_J + \mu_H) S_H(t),
 \tag{2.4}$$

which can be re-expressed as

$$\begin{aligned}
 \frac{d}{dt} [S_H(t) \exp\{\mu_H t + \int_0^t \lambda_J(\tau) d\tau\}] \\
 \geq \Lambda_H \exp\left\{\mu_H t + \int_0^t \lambda_J(\tau) d\tau\right\}.
 \end{aligned}
 \tag{2.5}$$

$$\begin{aligned}
 S_H(t_1) \exp\{\mu t_1 + \int_0^{t_1} \lambda_J(\tau) d\tau\} - S_H(0) \\
 \geq \int_0^{t_1} \Lambda_H \left[\exp\left\{\mu_H y + \int_0^y \lambda_J(\tau) d\tau\right\} \right] dy,
 \end{aligned}
 \tag{2.6}$$

So that,

$$\begin{aligned}
 S_H(t_1) &\geq S_H(0) \exp \left[-\mu_H t_1 - \int_0^{t_1} \lambda_J(\tau) d\tau \right] \\
 &\quad + [\exp\{-\mu_H t_1 - \int_0^{t_1} \lambda_J(\tau) d\tau\}] \\
 &\quad \times \int_0^{t_1} \Lambda_H \left[\exp\left\{\mu_H y + \int_0^y \lambda_J(\tau) d\tau\right\} \right] dy > 0.
 \end{aligned}
 \tag{2.7}$$

Hence, $S_H(t) > 0, \forall t > 0$.

Similarly, considering the second equation of model (2.1), given below as

$$\frac{dE_{HS}(t)}{dt} = \lambda_J (S_H(t) + \psi T_{HS}(t)) - (\alpha_1 + \mu_H) E_{HS}(t),
 \tag{2.8}$$

It follows from (2.8) above that

$$\frac{dE_{HS}(t)}{dt} \geq -(\alpha_1 + \mu_H) E_{HS}(t),
 \tag{2.9}$$

which can be re-expressed as

$$\frac{d}{dt} [E_{HS}(t)\exp\{(\alpha_1 + \mu_H)t\}] \geq 0. \tag{2.10}$$

Thus, integrating (2.10) with respect to $t \in [0, t_1]$, we obtain

$$E_{HS}(t_1)\exp\{(\alpha_1 + \mu_H)t_1\} - E_{HS}(0) \geq 0, \tag{2.11}$$

So that,

$$E_{HS}(t_1) \geq E_{HS}(0)\exp\{-(\alpha_1 + \mu_H)t_1\} > 0. \tag{2.12}$$

Hence, $E_{HS}(t) > 0, \forall t > 0$.

Similarly, considering the third equation of model (2.1), given below as

$$\frac{dI_{HS}(t)}{dt} = \alpha_1 E_{HS}(t) - (\zeta_S + \delta_S + \mu_H)I_{HS}(t), \tag{2.13}$$

It follows from (2.13) above that

$$\frac{dI_{HS}(t)}{dt} \geq -(\zeta_S + \delta_S + \mu_H)I_{HS}(t), \tag{2.14}$$

which can be re-expressed as

$$\frac{d}{dt} [I_{HS}(t)\exp\{(\zeta_S + \delta_S + \mu_H)t\}] \geq 0. \tag{2.15}$$

Thus, integrating (2.15) with respect to $t \in [0, t_1]$, we obtain

$$I_{HS}(t_1)\exp\{(\zeta_S + \delta_S + \mu_H)t_1\} - I_{HS}(0) \geq 0, \tag{2.16}$$

So that,

$$I_{HS}(t_1) \geq I_{HS}(0)\exp\{-(\zeta_S + \delta_S + \mu_H)t_1\} > 0. \tag{2.17}$$

Hence, $I_{HS}(t) > 0, \forall t > 0$.

Similarly, considering the fourth equation of model (2.1), given below as

$$\frac{dT_{HS}(t)}{dt} = \zeta_S I_{HS}(t) - \psi \lambda_J T_{HS}(t) - \mu_H T_{HS}(t), \tag{2.18}$$

It follows from above that

$$\frac{dT_{HS}(t)}{dt} \geq -\psi \lambda_J T_{HS}(t) - \mu_H T_{HS}(t), \tag{2.19}$$

which can be re-expressed as

$$\frac{d}{dt} [T_{HS}(t)\exp\{\mu_H t + \int_0^t \psi \lambda_J(\tau) d\tau\}] \geq 0. \tag{2.20}$$

Thus, integrating (2.20) with respect to $t \in [0, t_1]$, we obtain

$$T_{HS}(t_1)\exp\left\{\mu_H t_1 + \int_0^{t_1} \psi \lambda_J(\tau) d\tau\right\} - T_{HS}(0) \geq 0, \tag{2.21}$$

So that,

$$T_{HS}(t_1) \geq T_{HS}(0)\exp\left\{-\mu_H t_1 - \int_0^{t_1} \psi \lambda_J(\tau) d\tau\right\} > 0. \tag{2.22}$$

Hence, $T_{HS}(t) > 0, \forall t > 0$.

Similarly, considering the fifth equation of model (2.1), given below as

$$\frac{dL(t)}{dt} = N_e \gamma I_{HS} - \mu_L L, \tag{2.23}$$

It follows from above that

$$\frac{dL(t)}{dt} \geq -\mu_L L(t), \tag{2.24}$$

which can be re-expressed as

$$\frac{d}{dt} [L(t)\exp\{\mu_L t\}] \geq 0. \tag{2.25}$$

Thus, integrating (2.25) with respect to $t \in [0, t_1]$, we obtain

$$L(t_1)\exp\{\mu_L t_1\} - L(0) \geq 0, \tag{2.26}$$

So that,

$$L(t_1) \geq L(0)\exp\{-\mu_L t_1\} > 0. \tag{2.27}$$

Hence, $L(t) > 0, \forall t > 0$.

Similarly, considering the sixth equation of model (2.1), given below as

$$\frac{dS_S(t)}{dt} = \Lambda_S - \lambda_L S_S - \mu_S S_S, \tag{2.28}$$

which can be re-expressed as

$$\begin{aligned} \frac{d}{dt} [S_S(t)\exp\{\mu_S t + \int_0^t \lambda_L(\tau) d\tau\}] \\ \geq A_S \exp\left\{\mu_S t + \int_0^t \lambda_L(\tau) d\tau\right\}. \end{aligned} \tag{2.29}$$

Thus, integrating with respect to $t \in [0, t_1]$, we obtain

$$\begin{aligned} S_S(t_1)\exp\{\mu_S t_1 + \int_0^{t_1} \lambda_L(\tau) d\tau\} - S_S(0) \\ \geq \int_0^{t_1} A_S \left[\exp\left\{\mu_S x + \int_0^x \lambda_L(\tau) d\tau\right\} \right] dy, \end{aligned} \tag{2.30}$$

So that,

$$\begin{aligned} S_S(t_1) \geq S_S(0)\exp\left[-\mu_S t_1 - \int_0^{t_1} \lambda_L(\tau) d\tau\right] \\ + [\exp\{-\mu_S t_1 - \int_0^{t_1} \lambda_L(\tau) d\tau\}] \\ \times \int_0^{t_1} A_S \left[\exp\left\{\mu_S x + \int_0^x \lambda_L(\tau) d\tau\right\} \right] dx > 0. \end{aligned} \tag{2.31}$$

Hence, $S_S(t) > 0, \forall t > 0$.

Similarly, considering the seventh equation of model (2.1), given below as

$$\frac{dI_S(t)}{dt} = \lambda_L S_S(t) - \mu_S I_S(t), \tag{2.32}$$

It follows from (2.32) above that

$$\frac{dI_S(t)}{dt} \geq -\mu_S I_S(t), \tag{2.33}$$

which can be re-expressed as

$$\frac{d}{dt} [I_S(t)\exp\{\mu_S t\}] \geq 0. \tag{2.34}$$

Thus, integrating (2.34) with respect to $t \in [0, t_1]$, we obtain

$$I_S(t_1)\exp\{\mu_S t_1\} - I_S(0) \geq 0, \tag{2.35}$$

So that,

$$I_S(t_1) \geq I_S(0)\exp\{-\mu_S t_1\} > 0. \tag{2.36}$$

Hence, $I_S(t) > 0, \forall t > 0$.

Finally, considering the eighth equation of model (2.1), given below as

$$\frac{dJ(t)}{dt} = \phi I_S - \mu_J J, \tag{2.37}$$

It follows from (2.37) above that

$$\frac{dJ(t)}{dt} \geq -\mu_J J(t), \tag{2.38}$$

which can be re-expressed as

$$\frac{d}{dt} [J(t)\exp\{\mu_J t\}] \geq 0. \tag{2.39}$$

Thus, integrating (2.39) with respect to $t \in [0, t_1]$, we obtain

$$J(t_1)\exp\{\mu_J t_1\} - J(0) \geq 0, \tag{2.40}$$

So that,

$$J(t_1) \geq J(0)\exp\{-\mu_J t_1\} > 0. \tag{2.41}$$

Hence, $J(t) > 0, \forall t > 0$.

Thus, we have established positivity for all the state variables in model for all time.

We proceed to establish the boundedness of solutions to the model (2.1).

Theorem 2.2: Let $(S_H(t), E_{HS}(t), I_{HS}(t), T_{HS}(t), L(t), S_S(t), I_S(t), J(t))$ be trajectories of the system with initial conditions and the biological feasible region given by the set $\mathcal{D}_1 = \mathcal{D}_H \times \mathcal{D}_L \times \mathcal{D}_S \times \mathcal{D}_J \subset \mathbb{R}_+^4 \times \mathbb{R}_+^1 \times \mathbb{R}_+^2 \times \mathbb{R}_+^1 \subset \mathbb{R}_+^8$, where:

$$\mathcal{D}_H = \{(S_H, E_{HS}, I_{HS}, T_{HS}) \in \mathbb{R}_+^4 : N_H \leq \frac{\Lambda_H}{\mu_H}\}$$

$$\mathcal{D}_L = \{L \in \mathbb{R}_+^1 : L \leq \frac{N_e \gamma \Lambda_H}{\mu_L \mu_H}\}$$

$$\mathcal{D}_S = \{(S_S, I_S) \in \mathbb{R}_+^2 : N_S \leq \frac{\Lambda_S}{\mu_S}\}$$

$$\mathcal{D}_J = \{J \in \mathbb{R}_+^1 : J \leq \frac{\phi \Lambda_S}{\mu_J \mu_S}\}$$

is positively-invariant and attracts the entire positive trajectories of the model .

Proof: Adding up the right flank of the vector field for the human population in (2.1), yields

$$\frac{dN_H}{dt} = \Lambda_H - \mu_H N - \delta_S I_{HS}. \tag{2.42}$$

From (2.42), it ensues that $\frac{dN_H}{dt} \leq \Lambda_H - \mu_H N_H$. Hence, $\frac{dN_H}{dt} \leq 0$ if $N_H(t) \geq \frac{\Lambda_H}{\mu_H}$. Employing a standard comparison theorem [32], we prove that $N_H(t) \leq N_H(0)e^{-\mu_H t} + \frac{\Lambda_H}{\mu_H}(1 - e^{-\mu_H t})$. In particular, if $N_H(0) \leq \frac{\Lambda_H}{\mu_H}$, thus $N_H(t) \leq \frac{\Lambda_H}{\mu_H}$ for every $t > 0$. Hence, the set \mathcal{D}_H is positively invariant. Moreover, if $N_H(0) > \frac{\Lambda_H}{\mu_H}$, then either the orbits enters the domain \mathcal{D}_H in finite time or $N_H(t)$ asymptotically advances towards $\frac{\Lambda_H}{\mu_H}$ as $t \rightarrow \infty$. Thus, the domain \mathcal{D}_H attracts every trajectory in \mathbb{R}_+^4 .

$$\frac{dL}{dt} = N_e \gamma I_{HS} - \mu_L L. \tag{2.43}$$

From (2.43), which follows that $\frac{dL}{dt} \leq \frac{N_e \gamma \Lambda_H}{\mu_H} - \mu_L L$ since $N_H = S_H + E_{HS} + I_{HS} + T_{HS} \leq \frac{\Lambda_H}{\mu_H} \Rightarrow I_{HS} \leq \frac{\Lambda_H}{\mu_H}$. Hence, $\frac{dL}{dt} \leq 0$ if $L(t) \geq \frac{N_e \gamma \Lambda_H}{\mu_L \mu_H}$. Employing a standard comparison theorem [32], we prove that $L(t) \leq L(0)e^{-\mu_L t} + \frac{N_e \gamma \Lambda_H}{\mu_L \mu_H}(1 - e^{-\mu_L t})$. In particular, if $L(0) \leq \frac{N_e \gamma \Lambda_H}{\mu_L \mu_H}$, then $L(t) \leq \frac{N_e \gamma \Lambda_H}{\mu_L \mu_H}$ for all $t > 0$. Hence, the set \mathcal{D}_L is positively invariant. Moreover, if $L(0) > \frac{N_e \gamma \Lambda_H}{\mu_L \mu_H}$, then either the orbits enters the domain \mathcal{D}_L in finite time or $L(t)$ asymptotically approaches $\frac{N_e \gamma \Lambda_H}{\mu_L \mu_H}$ as $t \rightarrow \infty$. Thus, the domain \mathcal{D}_L attracts every trajectory in \mathbb{R}_+^1 .

For the snail population, we add up the right flank of the vector field of the snail population in (2.1), which gives

$$\frac{dN_S}{dt} = \Lambda_S - \mu_S N_S. \tag{2.44}$$

From (2.44), it ensues that $\frac{dN_S}{dt} \leq 0$ if $N_S(t) \geq \frac{\Lambda_S}{\mu_S}$. Consequently, $N_S(t) = N_S(0)e^{-\mu_S t} + \frac{\Lambda_S}{\mu_S}(1 - e^{-\mu_S t})$. Then the $\limsup_{t \rightarrow \infty} N_S(t) = \frac{\Lambda_S}{\mu_S}$. In particular, if $N_S(0) \leq \frac{\Lambda_S}{\mu_S}$, then $N_S(t) \leq \frac{\Lambda_S}{\mu_S}$ for every $t > 0$. Hence, the set \mathcal{D}_S is positively invariant. Moreover, if $N_S(0) > \frac{\Lambda_S}{\mu_S}$, then either the orbits enters the domain \mathcal{D}_S in finite time or $N_S(t)$ asymptotically approaches $\frac{\Lambda_S}{\mu_S}$ as $t \rightarrow \infty$. Thus, the domain \mathcal{D}_S attracts every trajectory in \mathbb{R}_+^2 .

For the concentration of the cercariae, we consider the right flank of the vector field J in (2.1), yields

$$\frac{dJ}{dt} = \phi I_S - \mu_J J. \tag{2.45}$$

From (2.45), $\frac{dJ}{dt} = \phi I_S - \mu_J J$ which follows that $\frac{dJ}{dt} \leq \frac{\phi \Lambda_S}{\mu_J \mu_S} - \mu_J J$ since $N_S = S_S + I_S \leq \frac{\Lambda_S}{\mu_S} \Rightarrow I_S \leq \frac{\Lambda_S}{\mu_S}$. Hence, $\frac{dJ}{dt} \leq 0$ if $J(t) \geq \frac{\phi \Lambda_S}{\mu_J \mu_S}$. Employing a standard comparison theorem [32], we prove that $J(t) \leq J(0)e^{-\mu_J t} + \frac{\phi \Lambda_S}{\mu_J \mu_S}(1 - e^{-\mu_J t})$. In particular, if $J(0) \leq \frac{\phi \Lambda_S}{\mu_J \mu_S}$, then $J(t) \leq \frac{\phi \Lambda_S}{\mu_J \mu_S}$ for all $t > 0$. Hence, the set \mathcal{D}_J is positively invariant. Moreover, if $J(0) > \frac{\phi \Lambda_S}{\mu_J \mu_S}$, then either the orbits enters the domain \mathcal{D}_J in finite time or $J(t)$ asymptotically approaches $\frac{\phi \Lambda_S}{\mu_J \mu_S}$ as $t \rightarrow \infty$. Thus, the domain \mathcal{D}_J attracts every trajectory in \mathbb{R}_+^1 .

Therefore, it is sufficient to study the dynamics of the flows engendered by the model system in \mathcal{D} . We conclude, therefore, that the model is together mathematically and epidemiologically well-posed.

3.0 Mathematical Analysis of the Model

We proceed to qualitatively analyze the model (2.1).

3.1 Local Asymptotic Stability (LAS) of the Disease-free Equilibrium (DFE)

The model system has a disease-free equilibrium (DFE) given by

$$\begin{aligned} \mathcal{E}_0 &= (S_H^*, E_{HS}^*, I_{HS}^*, T_{HS}^*, L^*, S_S^*, I_S^*, J^*) \\ &= \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, \frac{\Lambda_S}{\mu_S}, 0, 0 \right) \end{aligned} \tag{3.1}$$

The linear stability of \mathcal{E}_0 is established by deploying the next-generation operator method on the model system [26]. Deploying specific notations as espoused by [33], it follows that matrices F and V, respectively, for the fresh infection terms and the other transition terms, are given by

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\beta_I \Lambda_H}{J_0 \mu_H} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta_L \Lambda_S}{L_0 \mu_S} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} (\alpha_1 + \mu_H) & 0 & 0 & 0 & 0 \\ -\alpha_1 & (\zeta_S + \delta_S + \mu_H) & 0 & 0 & 0 \\ 0 & 0 & \mu_S & 0 & 0 \\ 0 & -N_e \gamma & 0 & -\mu_L & 0 \\ 0 & 0 & -\phi & 0 & \mu_J \end{bmatrix}$$

It ensues that *effective reproduction number*, denoted by $\mathcal{R}_{HS} = \rho(FV^{-1})$, is denoted by

$$\mathcal{R}_{HS} = \sqrt{\frac{\alpha_1 \beta_J \beta_L \Lambda_H \Lambda_S \phi N_e \gamma}{J_0 L_0 \mu_H \mu_J \mu_L \mu_S^2 (\alpha_1 + \mu_H) (\zeta_S + \delta_S + \mu_H)}} \tag{3.2}$$

where $\rho(FV^{-1})$ is the spectral radius belonging to the matrix FV^{-1} . Consequently, the result below stems from the conclusion of Theorem 2 in [33].

Lemma 3.1: The DFE \mathcal{E}_0 of is locally asymptotically stable on the condition that $\mathcal{R}_{HS} < 1$ and unstable on the condition that $\mathcal{R}_{HS} > 1$.

The threshold quantity, \mathcal{R}_{HS} , is the effective reproduction number of the disease [33-34]. It is a measure of the mean number of secondary schistosomiasis infections engendered by a typical infected human in a completely exposed population or at the DFE [33-34]. The epidemiological connotation of Lemma 3.1 implies that whenever \mathcal{R}_{HS} is less than one, schistosomiasis can be annihilated from the populace if the basic (initial) sizes of the classes of the model system (2.1) are in the basin of attraction of the infection-free equilibrium \mathcal{E}_0 . Thus, a small arrival of schistosomiasis-infected humans into the populace will not engender enormous schistosomiasis outbreaks, with the resultant effect of the disease dying out over time.

3.2 Analysis of the Effective Reproduction Number, \mathcal{R}_{HS}

Utilizing the threshold parameter, \mathcal{R}_{HS} , we wish to determine the effect of the medical care rate (ζ_S) of humans occupying the infectious class on the control of schistosomiasis in the population.

Calculating the partial derivatives of \mathcal{R}_{HS} with respect to the parameter under scrutiny (ζ_S) further exposes the consequence of this parameter on schistosomiasis regulation among the populace. This implies

$$\frac{\partial \mathcal{R}_{HS}^2}{\partial \zeta_S} = - \frac{\alpha_1 \beta_J \beta_L \Lambda_H \Lambda_S \phi N_e \gamma}{J_0 L_0 \mu_H \mu_J \mu_L \mu_S^2 (\alpha_1 + \mu_H) (\zeta_S + \delta_S + \mu_H)^2} < 0 \tag{3.3}$$

Apparently, it ensues from (3.3) that the partial derivative is negative, unconditionally. Thus, effective medical care rate of schistosomiasis at the phase of infection will exert a positive consequence in decreasing the burden of schistosomiasis among the populace, regardless of the rates of the other parameters in the expression on the right flank of (3.3). It is obvious from (3.2) that

$$\lim_{\zeta_S \rightarrow \infty} \mathcal{R}_{HS} = 0 \tag{3.4}$$

From (3.4), a near complete annihilation of schistosomiasis is feasible. In this situation, an effective strategy will be to pay close attention to medical care programmes for infected humans.

Lemma 3.2: Effective treatment rate (ζ_S) for the infectious phase of infection will exert a positive influence in decreasing the schistosomiasis hardship in a populace, regardless of the rates of the other parameters that constitute the effective reproduction number.

3.3 Endemic Equilibrium Point (EEP)

Let the endemic equilibrium point, \mathcal{E}_S^* , of the system is defined by

$$\mathcal{E}_S^* = (S_H^{**}, E_{HS}^{**}, I_{HS}^{**}, T_{HS}^{**}, L^{**}, S_S^{**}, I_S^{**}, J^{**}) \tag{3.5}$$

where

$$\begin{aligned}
 S_H^{**} &= \frac{\Lambda_H}{\lambda_j^{**} + \mu_H}, \\
 E_{HS}^{**} &= \frac{\Lambda_H(\zeta_S + \delta_S + \mu_H)\lambda_j^{**}(\psi\lambda_j^{**} + \mu_H)}{(\lambda_j^{**} + \mu_H)[(\alpha_1 + \mu_H)(\zeta_S + \delta_S + \mu_H)(\psi\lambda_j^{**} + \mu_H) - \alpha_1\zeta_S\psi\lambda_j^{**}]}, \\
 I_{HS}^{**} &= \frac{\alpha_1\Lambda_H\lambda_j^{**}(\psi\lambda_j^{**} + \mu_H)}{(\lambda_j^{**} + \mu_H)[(\alpha_1 + \mu_H)(\zeta_S + \delta_S + \mu_H)(\psi\lambda_j^{**} + \mu_H) - \alpha_1\zeta_S\psi\lambda_j^{**}]}, \\
 T_{HS}^{**} &= \frac{\alpha_1\Lambda_H\zeta_S\lambda_j^{**}(\psi\lambda_j^{**} + \mu_H)}{(\psi\lambda_j^{**} + \mu_H)(\lambda_j^{**} + \mu_H)[(\alpha_1 + \mu_H)(\zeta_S + \delta_S + \mu_H)(\psi\lambda_j^{**} + \mu_H) - \alpha_1\zeta_S\psi\lambda_j^{**}]}, \\
 L^{**} &= \frac{\alpha_1\Lambda_H N_e \gamma \lambda_j^{**}(\psi\lambda_j^{**} + \mu_H)}{\mu_L(\lambda_j^{**} + \mu_H)[(\alpha_1 + \mu_H)(\zeta_S + \delta_S + \mu_H)(\psi\lambda_j^{**} + \mu_H) - \alpha_1\zeta_S\psi\lambda_j^{**}]}, \\
 S_S^{**} &= \frac{\Lambda_S}{\lambda_L^{**} + \mu_S}, \\
 I_S^{**} &= \frac{\Lambda_S\lambda_L^{**}}{\mu_S(\lambda_L^{**} + \mu_S)}, \\
 J^{**} &= \frac{\Lambda_S\phi\lambda_L^{**}}{\mu_J\mu_S(\lambda_L^{**} + \mu_S)}.
 \end{aligned} \tag{3.6}$$

The forces of infection, respectively, are:

$$\lambda_j^{**} = \frac{\beta_J J^{**}}{J_0 + \epsilon J^{**}}, \tag{3.7}$$

and

$$\lambda_L^{**} = \frac{\beta_L L^{**}}{L_0 + \epsilon L^{**}}. \tag{3.8}$$

Substituting the value for J^{**} in (3.6) into (3.7), the force of infection for cercarial penetration becomes:

$$\lambda_j^{**} = \frac{\beta_J \Lambda_S \phi \lambda_L^{**}}{J_0 \mu_J \mu_S (\lambda_L^{**} + \mu_S) + \epsilon \Lambda_S \phi \lambda_L^{**}}, \tag{3.9}$$

while substituting the value for L^{**} into (3.8), the force of infection for miracidial penetration becomes:

$$\lambda_L^{**} = \frac{\alpha_1 \beta_L \Lambda_H N_e \gamma \lambda_j^{**} (\psi \lambda_j^{**} + \mu_H)}{L_0 \mu_L (\lambda_j^{**} + \mu_H) [(\alpha_1 + \mu_H)(\zeta_S + \delta_S + \mu_H)(\psi \lambda_j^{**} + \mu_H) - \alpha_1 \zeta_S \psi \lambda_j^{**}] + \epsilon \alpha_1 \Lambda_H N_e \gamma \lambda_j^{**}}. \tag{3.10}$$

Substituting (3.10) into (3.9) and after several algebraic manipulations and simplifications, it is shown that the EEP associated with the system (2.1) satisfies the polynomial (expressed as a function of λ_j^{**})

$$\lambda_j^{**} (A_{22}(\lambda_j^{**})^2 + A_{11}\lambda_j^{**} + A_{00}) = 0. \tag{3.11}$$

Now,

$$\lambda_j^{**} = 0 \tag{3.12}$$

or

$$A_{22}(\lambda_j^{**})^2 + A_{11}\lambda_j^{**} + A_{00} = 0. \tag{3.13}$$

where

$$\begin{aligned}
 A_{00} &= J_0 L_0 \mu_H^2 \mu_J \mu_L \mu_S^2 (\alpha_1 + \mu_H) (\zeta_S + \delta_S + \mu_H) (1 - \mathcal{R}_{HS}^2), \\
 A_{11} &= \epsilon \alpha_1 \beta_L \Lambda_H \Lambda_S N_e \gamma \mu_H + J_0 \mu_H \mu_J \mu_S \alpha_1 N_e \gamma (\beta_L + \epsilon \Lambda_H \mu_S) \\
 &\quad + J_0 L_0 \mu_H \mu_J \mu_L \mu_S^2 (\alpha_1 + \mu_H) (\zeta_S + \delta_S + \mu_H) \\
 &\quad + \psi J_0 L_0 \mu_H \mu_J \mu_L \mu_S^2 \alpha_1 (\delta_S + \mu_H) \\
 &\quad + \psi J_0 L_0 \mu_H^2 \mu_J \mu_L \mu_S^2 (\zeta_S + \delta_S + \mu_H) - \psi \alpha_1 \beta_J \beta_L \Lambda_H \Lambda_S N_e \gamma \phi \\
 A_{22} &= \psi (\epsilon \alpha_1 \beta_L \Lambda_H \Lambda_S N_e \gamma \phi + J_0 \mu_J \mu_S \alpha_1 N_e \gamma (\beta_L + \epsilon \Lambda_H \mu_S) \\
 &\quad + J_0 L_0 \alpha_1 \mu_J \mu_L \mu_S^2 (\delta_S + \mu_H) + J_0 L_0 \mu_H \mu_J \mu_L \mu_S^2 (\zeta_S + \delta_S + \mu_H))
 \end{aligned} \tag{3.14}$$

The components of the EEP are obtained when we solve for λ_j^{**} from the polynomial given in (3.13). Thus, we substitute the values obtained for λ_j^{**} into (3.6). The above result is captured in the theorem below.

Theorem 3.3: The model system has:

1. two endemic equilibria on the assumption that $A_{11} < 0, A_{00} > 0$ and $\mathcal{R}_{HS} < 1$,
2. one unique endemic equilibrium on the assumption that $A_{11} > 0, A_{00} < 0$ or $A_{11} < 0, A_{00} < 0$ and $\mathcal{R}_{HS} > 1$,
3. nil endemic steady state otherwise, whenever $\mathcal{R}_{HS} < 1$.

It is significant to mention, at this juncture, that item (1) of Theorem 3.3 (above) is indicative of the presence of backward bifurcation in the model. The backward bifurcation phenomenon is pronounced as a consequence of the co-existence of an infection-free state as well as an endemic steady state that are both stable at whatever time the corresponding reproduction number is less than one. This, therefore, implies that the standard condition required for disease control ($\mathcal{R}_{HS} < 1$) is not any more sufficient for effectively regulating schistosomiasis among the populace, although it remains a necessary condition. In such a scenario, effective strategies for schistosomiasis control will now have to be based on the basic conditions of different compartments of the model system under consideration [2]. We observe that the EEP of the model (2.1) possesses a unique endemic equilibrium point when $\mathcal{R}_{HS} > 1$ (and does not have an EEP whenever $\mathcal{R}_{HS} < 1$, and hence no possibility of a backward bifurcation when $\mathcal{R}_{HS} < 1$).

3.4 Backward Bifurcation Analysis

Theorem 3.4: The model (2.1) experiences backward bifurcation at $\mathcal{R}_{HS} = 1$ whenever $\psi > \psi^c$, with ψ^c expressed as

$$\psi^c = \frac{v_2 W_{44} + v_7 W_{55}}{v_2 W_{66}} > 0, \tag{3.15}$$

and

$$\begin{aligned} W_{44} &= \beta_J^* \phi (J_0 L_0 \mu_J \mu_L \mu_S^2 (\alpha_1 + \mu_H) (\zeta_S + \delta_S + \mu_H) + \epsilon \alpha_1 \beta_L \Lambda_H \Lambda_S \phi N_e \gamma), \\ W_{55} &= J_0^2 \alpha_1 \mu_H \mu_J^2 \mu_S^2 N_e \gamma (\beta_L + \epsilon \mu_S), \\ W_{66} &= J_0 L_0 \alpha_1 \beta_J^* \zeta_S \phi \mu_J \mu_L \mu_S^2. \end{aligned} \tag{3.16}$$

Proof: We employ the ensuing alteration of variables. Let $S_H = x_1, E_{HS} = x_2, I_{HS} = x_3, T_{HS} = x_4, L = x_5, S_S = x_6, I_S = x_7$ and $J = x_8$, so that $N_H = x_1 + x_2 + x_3 + x_4$; hence the model (2.1) is re-written as

$$\begin{aligned} \dot{x}_1 &\equiv f_1 = \Lambda_H - \frac{\beta_J x_1 x_8}{J_0 + \epsilon x_8} - \mu_H x_1, \\ \dot{x}_2 &\equiv f_2 = \frac{\beta_J x_1 x_8}{J_0 + \epsilon x_8} (x_1 + \psi x_4) - (\alpha_1 + \mu_H) x_2, \\ \dot{x}_3 &\equiv f_3 = \alpha_1 x_2 - (\zeta_S + \delta_S + \mu_H) x_3, \\ \dot{x}_4 &\equiv f_4 = \zeta_S x_3 - \psi \frac{\beta_J x_4 x_8}{J_0 + \epsilon x_8} - \mu_H x_4, \\ \dot{x}_5 &\equiv f_5 = N_e \gamma x_3 - \mu_L x_5, \\ \dot{x}_6 &\equiv f_6 = \Lambda_S - \frac{\beta_L x_5 x_6}{L_0 + \epsilon x_5} - \mu_S x_6, \\ \dot{x}_7 &\equiv f_7 = \frac{\beta_L x_5 x_6}{L_0 + \epsilon x_5} - \mu_S x_7, \\ \dot{x}_8 &\equiv f_8 = \phi x_7 - \mu_J x_8. \end{aligned} \tag{3.17}$$

The Jacobian for the system (3.16) at the DFE is given by

$$J_{\beta_J^*} = \begin{bmatrix} -\mu_H & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_J \Lambda_H}{J_0 \mu_H} \\ 0 & -(\alpha_1 + \mu_H) & 0 & 0 & 0 & 0 & 0 & \frac{\beta_J \Lambda_H}{J_0 \mu_H} \\ 0 & \alpha_1 & -(\zeta_S + \delta_S + \mu_H) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \zeta_S & -\mu_H & 0 & 0 & 0 & 0 \\ 0 & 0 & N_e \gamma & 0 & -\mu_L & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\frac{\beta_L \Lambda_S}{L_0 \mu_S} & -\mu_S & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_L \Lambda_S}{L_0 \mu_S} & 0 & -\mu_S & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \phi & -\mu_J \end{bmatrix} \tag{3.18}$$

Consider the case when $\mathcal{R}_{HS} = 1$. Working out the value for $\beta_J = \beta_J^*$ from $\mathcal{R}_{HS} = 1$ gives

$$\beta_J = \beta_J^* = \frac{J_0 L_0 \mu_H \mu_J \mu_L \mu_S^2 (\alpha_1 + \mu_H) (\zeta_S + \delta_S + \mu_H)}{\alpha_1 \beta_L \Lambda_H \Lambda_S \phi N_e \gamma} \tag{3.18}$$

Matrix $J_{\beta_J^*}$ possesses a right eigenvector given by $\mathbf{w} = (\omega_1, \omega_2, \dots, \omega_8)^T$, such that

$$\begin{aligned} \omega_1 &= -\frac{(\alpha_1 + \mu_H)(\zeta_S + \delta_S + \mu_H)\omega_3}{\alpha_1\mu_H}, \omega_2 = \frac{(\zeta_S + \delta_S + \mu_H)\omega_3}{\alpha_1}, \\ \omega_3 &= \omega_3 > 0, \omega_4 = \frac{\zeta_S\omega_3}{\mu_H}, \omega_5 = \frac{N_e\gamma\omega_3}{\mu_L}, \omega_6 = -\frac{\beta_L\Lambda_S N_e\gamma\omega_3}{L_0\mu_L\mu_S^2}, \\ \omega_7 &= \frac{\beta_L\Lambda_S N_e\gamma\omega_3}{L_0\mu_L\mu_S^2}, \omega_8 = \frac{\beta_L\Lambda_S\phi N_e\gamma\omega_3}{L_0\mu_J\mu_L\mu_S^2}, \end{aligned} \tag{3.19}$$

In addition, $J_{\beta_j^*}$ possesses a left eigenvector $\mathbf{v} = (v_1, v_2, \dots, v_8)$ satisfying $\mathbf{v} \cdot \mathbf{w} = \mathbf{1}$, with

$$\begin{aligned} v_1 &= 0, v_2 = \frac{\alpha_1 v_3}{\alpha_1 + \mu_H}, v_3 = v_3 > 0, v_4 = 0, \\ v_5 &= \frac{(\zeta_S + \delta_S + \mu_H)v_3}{N_e\gamma}, v_6 = 0, \\ v_7 &= \frac{L_0\mu_L\mu_S(\zeta_S + \delta_S + \mu_H)v_3}{\beta_L\Lambda_S N_e\gamma}, \\ v_8 &= \frac{L_0\mu_L\mu_S^2(\zeta_S + \delta_S + \mu_H)v_3}{\beta_L\Lambda_S\phi N_e\gamma}. \end{aligned} \tag{3.20}$$

Applying the *Center Manifold Theory* as espoused by [35], we calculate the related non-zero partial derivatives of the right flanks of the transformed system (3.16), (*appraised in the absence of infection with $\beta_j = \beta_j^*$*) that the related bifurcation coefficients, a and b , are given by

$$a = \sum_{k,l,j=1}^n v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \text{ and } b = \sum_{k,l=1}^n v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0), \tag{3.21}$$

The related non-zero partial derivatives for bifurcation coefficient a for the model system (3.16) (or (2.1)) are:

$$\begin{aligned} \frac{\partial^2 f_2}{\partial x_1 \partial x_8} &= \frac{\beta_j^*}{J_0} = \frac{\partial^2 f_2}{\partial x_8 \partial x_1}, \\ \frac{\partial^2 f_2}{\partial x_4 \partial x_8} &= \frac{\psi\beta_j^*}{J_0} = \frac{\partial^2 f_2}{\partial x_8 \partial x_4}, \\ \frac{\partial^2 f_2}{\partial x_8^2} &= -\frac{2\epsilon\beta_j^*\Lambda_H}{J_0^2\mu_H}, \\ \frac{\partial^2 f_7}{\partial x_5^2} &= -\frac{2\epsilon\beta_L\Lambda_S}{L_0^2\mu_S}, \\ \frac{\partial^2 f_7}{\partial x_5 \partial x_6} &= \frac{\beta_L}{L_0} = \frac{\partial^2 f_7}{\partial x_6 \partial x_5}. \end{aligned} \tag{3.22}$$

It ensues from the above expressions, (after several algebraic calculations), that

$$\begin{aligned} a &= v_2 \sum_{i,j=1}^8 w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j} + v_7 \sum_{i,j=1}^8 w_i w_j \frac{\partial^2 f_7}{\partial x_i \partial x_j} \\ &= \frac{2\beta_L\Lambda_S N_e\gamma v_2 \omega_3^2}{L_0^2\mu_L^2\mu_S^2} [\psi W_{11}] - \frac{2\beta_L\Lambda_S N_e\gamma \omega_3^2}{L_0^2\mu_L^2\mu_S^2} [v_2 W_{22} + v_7 W_{33}]. \end{aligned} \tag{3.23}$$

where

$$\begin{aligned} W_{11} &= \frac{L_0\beta_j^*\zeta_S\phi\mu_L}{J_0\mu_H\mu_J}, \\ W_{22} &= \frac{\phi(J_0 L_0 \beta_j^* \mu_J \mu_L \mu_S^2 (\alpha_1 + \mu_H) (\zeta_S + \delta_S + \mu_H) + \epsilon \alpha_1 \beta_j^* \beta_L \Lambda_H \Lambda_S \phi N_e \gamma)}{J_0^2 \alpha_1 \mu_H \mu_J^2 \mu_S^2}, \\ W_{33} &= N_e \gamma (\beta_L + \epsilon \mu_S). \end{aligned} \tag{3.24}$$

Hence, $a > 0$ implies that

$$\frac{2\beta_L\Lambda_S N_e\gamma v_2 \omega_3^2}{L_0^2\mu_L^2\mu_S^2} [\psi W_{11}] > \frac{2\beta_L\Lambda_S N_e\gamma \omega_3^2}{L_0^2\mu_L^2\mu_S^2} [v_2 W_{22} + v_7 W_{33}]. \tag{3.25}$$

That is, $\psi > \psi^c > 0$, where

$$\psi^c = \frac{v_2 W_{44} + v_7 W_{55}}{v_2 W_{66}} \tag{3.26}$$

where

$$\begin{aligned} W_{44} &= \beta_j^* \phi (J_0 L_0 \mu_j \mu_L \mu_S^2 (\alpha_1 + \mu_H) (\zeta_S + \delta_S + \mu_H) + \epsilon \alpha_1 \beta_L \Lambda_H \Lambda_S \phi N_e \gamma), \\ W_{55} &= J_0^2 \alpha_1 \mu_H \mu_j^2 \mu_S^2 N_e \gamma (\beta_L + \epsilon \mu_S), \\ W_{66} &= J_0 L_0 \alpha_1 \beta_j^* \zeta_S \phi \mu_j \mu_L \mu_S^2. \end{aligned} \tag{3.27}$$

The related non-zero partial derivative for bifurcation coefficient b for the model system (3.16) (or (2.1)) is:

$$\frac{\partial^2 f_2}{\partial x_8 \partial \beta_j^*} = \frac{\Lambda_H}{J_0 \mu_H}. \tag{3.28}$$

It ensues also from that

$$\begin{aligned} b &= v_2 \sum_{i=1}^8 w_i \frac{\partial^2 f_2}{\partial x_i \partial \beta_j^*}, \\ &= v_2 \omega_8 \left[\frac{\Lambda_H}{J_0 \mu_H} \right] \end{aligned} \tag{3.29}$$

Obviously $b > 0$ for all biologically reasonable parameter values. Thus, backward bifurcation appears if and only if the rate of reduced re-infection (ψ), is large enough such that $a > 0$. This, therefore, implies that if the reduced re-infection rate is less than the quantity, ψ^c , the effective reproduction number then becomes a necessary and sufficient tool for promoting control measures that will lead to disease eradication.

Consequent upon the results obtained in Theorem 3.4 above, we claim the following result.

Theorem 3.5: (Non-existence of backward bifurcation) The model (2.1) (or (3.16)) does not experience backward bifurcation in the direction $\mathcal{R}_{HS} = 1$, whenever $\psi = 0$.

Proof: Consider the distinctive case of the model (2.1) with negligible reduced re-infection (i.e., $\psi = 0$). Then the backward bifurcation coefficient, a , in (3.23) reduces to:

$$a = - \frac{2\beta_L \Lambda_S N_e \gamma \omega_3^2}{L_0^2 \mu_L^2 \mu_S^2} [v_2 W_{22} + v_7 W_{33}] < 0. \tag{3.30}$$

Thus, this study has confirmed that the existence of reduced re-infection activates backward bifurcation in the epidemic dynamics of schistosomiasis.

3.5 Global Asymptotic Stability (GAS) of DFE

Consider the special case of the model (2.1) with $\psi = 0$ (i.e., removing the parameter that causes backward bifurcation as discussed above). In this case, the model reduces to

$$\begin{aligned} S'_H &= \Lambda_H - \lambda_j S_H - \mu_H S_H, \\ E'_{HS} &= \lambda_j S_H - (\alpha_1 + \mu_H) E_{HS}, \\ I'_{HS} &= \alpha_1 E_{HS} - (\zeta_S + \delta_S + \mu_H) I_{HS}, \\ T'_{HS} &= \zeta_S I_{HS} - \mu_H T_{HS}, \\ L' &= N_e \gamma I_{HS} - \mu_L L, \\ S'_S &= \Lambda_S - \lambda_L S_S - \mu_S S_S, \\ I'_S &= \lambda_L S_S - \mu_S I_S, \\ J' &= \phi I_S - \mu_j J. \end{aligned} \tag{3.31}$$

We claim the following result.

The DFE of the model (3.31), without re-infection (i.e., $\psi = 0$) is GAS in \mathcal{D}_1 if $\mathcal{R}_{HS} \leq 1$ and unstable on the condition that $\mathcal{R}_{HS} > 1$.

Proof: Consider the following Lyapunov function

$$\mathcal{U} = K_1 E_{HS} + K_2 I_{HS} + K_3 I_S + K_4 L + K_5 J, \tag{3.32}$$

where

$$\begin{aligned} K_1 &= \frac{\alpha_1 \beta_L \Lambda_S \phi N_e \gamma}{L_0 \mu_j \mu_L \mu_S^2 (\alpha_1 + \mu_H) (\zeta_S + \delta_S + \mu_H)}, & K_2 &= \frac{\beta_L \Lambda_S \phi N_e \gamma}{L_0 \mu_j \mu_L \mu_S^2 (\zeta_S + \delta_S + \mu_H)}, \\ K_3 &= \frac{\mathcal{R}_{HS} \phi}{\mu_j \mu_S}, & K_4 &= \frac{\beta_L \Lambda_S \phi}{L_0 \mu_j \mu_L \mu_S^2}, & \text{and } K_5 &= \frac{\mathcal{R}_{HS}}{\mu_j}, \end{aligned} \tag{3.33}$$

with Lyapunov derivatives (where a dot represents a time derivative)

$$\dot{U} = K_1 \dot{E}_{HS} + K_2 \dot{I}_{HS} + K_3 \dot{I}_S + K_4 \dot{L} + K_5 \dot{J}. \tag{3.34}$$

Substituting the right hand side of model (3.31) into (3.34) gives

$$\begin{aligned} \dot{U} &= K_1 \lambda_J S_H + [\alpha_1 K_2 - (\alpha_1 + \mu_H) K_1] E_{HS} \\ &+ [N_e \gamma K_4 - (\zeta_S + \delta_S + \mu_H) K_2] I_{HS} \\ &+ [\phi K_5 - \mu_S K_3] I_S \\ &- \mu_L K_4 L \\ &- \mu_J K_5 J, \\ &= \frac{\alpha_1 \beta_L \Lambda_S \phi N_e \gamma}{L_0 \mu_J \mu_L \mu_S^2 (\alpha_1 + \mu_H) (\zeta_S + \delta_S + \mu_H)} \left[\left(\frac{\beta_J J}{J_0 + \epsilon J} \right) S_H \right] - \mathcal{R}_{HS} J \\ &+ \frac{\mathcal{R}_{HS} \phi}{\mu_J \mu_S} \left[\left(\frac{\beta_L L}{L_0 + \epsilon L} \right) S_S \right] - \left(\frac{\beta_L \Lambda_S \phi}{L_0 \mu_J \mu_S^2} \right) L. \end{aligned} \tag{3.35}$$

At DFE, $S_H \leq \Lambda_H / \mu_H$, $S_S \leq \Lambda_S / \mu_S$ and $\epsilon = 0$. Hence

$$\therefore \dot{U} \leq \left(\left(\frac{\beta_L \Lambda_S \phi}{L_0 \mu_J \mu_S^2} \right) L + \mathcal{R}_{HS} J \right) [\mathcal{R}_{HS} - 1]. \tag{3.36}$$

Hence, $\dot{U} \leq 0$ whenever $\mathcal{R}_{HS} \leq 1$ with $\dot{U} = 0$ if and only if $L = J = 0$. Hence, U represents a Lyapunov function in \mathcal{D}_1 .

Therefore, it ensues from LaSalle's Invariance Principle [36] that:

$$(E_{HS}(t), I_{HS}(t), I_S(t), L(t), J(t)) \rightarrow (0, 0, 0, 0, 0) \text{ as } t \rightarrow \infty. \tag{3.37}$$

Consequently, every orbit of the equations of the model (3.31), with $\psi = 0$, approaches the DFE of the model (3.31), as $t \rightarrow \infty$ for $\mathcal{R}_{HS} \leq 1$.

This result shows that in a population where there is treatment for active schistosomiasis cases, on the condition that there is negligible re-infection, that is, $\psi = 0$, the DFE will be GAS whenever $\mathcal{R}_{HS} \leq 1$. Hence, schistosomiasis can be annihilated from the populace whenever $\mathcal{R}_{HS} \leq 1$, irrespective of the basic sizes of the sub-populations.

3.6 Global Asymptotic Stability (GAS) of EEP

Assume that the stable manifold of the DFE of the model system (3.31) is

$$\mathcal{D}_0 = \{(S_H, E_{HS}, I_{HS}, T_{HS}, L, S_S, I_S, J) \in \mathcal{D}_1 : E_{HS} = I_{HS} = T_{HS} = L = I_S = J = 0\}.$$

We claim the following result.

Theorem 3.6: The unique EEP, \mathcal{E}_S^* , of model (3.31) with $\psi = 0$ is globally asymptotically stable in $\mathcal{D}_1 \setminus \mathcal{D}_0$ at any time $\mathcal{R}_{HS} > 1$.

Proof: Consider also, the ensuing non-linear Lyapunov function

$$\begin{aligned} Q &= S_H - S_H^{**} \ln \left(\frac{S_H}{S_H^{**}} \right) + E_{HS} - E_{HS}^{**} \ln \left(\frac{E_{HS}}{E_{HS}^{**}} \right) + R_1 \left(I_{HS} - I_{HS}^{**} \ln \frac{I_{HS}}{I_{HS}^{**}} \right) \\ &+ R_2 \left(T_{HS} - T_{HS}^{**} \ln \frac{T_{HS}}{T_{HS}^{**}} \right) + R_3 \left(L - L^{**} \ln \frac{L}{L^{**}} \right) + S_S - S_S^{**} \ln \left(\frac{S_S}{S_S^{**}} \right) \\ &+ I_S - I_S^{**} \ln \left(\frac{I_S}{I_S^{**}} \right) + R_4 \left(J - J^{**} \ln \frac{J}{J^{**}} \right), \end{aligned} \tag{3.38}$$

Where

$$R_1 = \frac{\alpha_1 + \mu_H}{\alpha_1}, \quad R_2 = 0, \quad R_3 = \frac{(\alpha_1 + \mu_H)(\zeta_S + \delta_S + \mu_H)}{\alpha_1 N_e \gamma}, \quad R_4 = \frac{\mu_S}{\phi}. \tag{3.39}$$

Q has Lyapunov derivatives, given as

$$\begin{aligned} \dot{Q} &= \left(1 - \frac{S_H^{**}}{S_H} \right) \dot{S}_H + \left(1 - \frac{E_{HS}^{**}}{E_{HS}} \right) \dot{E}_{HS} + R_1 \left(1 - \frac{I_{HS}^{**}}{I_{HS}} \right) \dot{I}_{HS} + R_2 \left(1 - \frac{T_{HS}^{**}}{T_{HS}} \right) \dot{T}_{HS} \\ &+ R_3 \left(1 - \frac{L^{**}}{L} \right) \dot{L} + \left(1 - \frac{S_S^{**}}{S_S} \right) \dot{S}_S + \left(1 - \frac{I_S^{**}}{I_S} \right) \dot{I}_S + R_4 \left(1 - \frac{J^{**}}{J} \right) \dot{J}. \end{aligned} \tag{3.40}$$

Substituting the right flanks of the equations in model (3.31) corresponding to $\dot{S}_H, \dot{E}_{HS}, \dot{I}_{HS}, \dot{T}_{HS}, \dot{L}, \dot{S}_S, \dot{I}_S, \dot{J}$ into (3.40), after several algebraic calculations gives:

$$\begin{aligned}
 \dot{Q} &= \mu_H S_H^{**} \left(2 - \frac{S_H^{**}}{S_H} - \frac{S_H}{S_H^{**}} \right) \\
 &+ \lambda_j^{**} S_H^{**} \left(4 - \frac{S_H^{**}}{S_H} - \frac{E_{HS} I_{HS}^{**}}{E_{HS}^{**} I_{HS}} - \frac{I_{HS} L^{**}}{I_{HS}^{**} L} - \frac{S_S I_S^{**} L}{S_S^{**} I_S L^{**}} \right) \\
 &+ \mu_S S_S^{**} \left(2 - \frac{S_S^{**}}{S_S} - \frac{S_S}{S_S^{**}} \right) \\
 &+ \mu_S I_S^{**} \left(2 - \frac{I_S J^{**}}{I_S^{**} J} - \frac{S_H E_{HS} J}{S_H^{**} E_{HS} J^{**}} \right) \\
 &+ \lambda_L^{**} S_S^{**} \left(1 - \frac{S_S}{S_S^{**}} \right). \tag{3.41}
 \end{aligned}$$

For as much as the arithmetic mean exceeds the geometric mean, the ensuing inequalities hold

$$\begin{aligned}
 2 - \frac{S_H^{**}}{S_H} - \frac{S_H}{S_H^{**}} \leq 0, \quad 2 - \frac{S_S^{**}}{S_S} - \frac{S_S}{S_S^{**}} \leq 0, \quad 2 - \frac{I_S J^{**}}{I_S^{**} J} - \frac{S_H E_{HS} J}{S_H^{**} E_{HS} J^{**}}, \\
 4 - \frac{S_H^{**}}{S_H} - \frac{E_{HS} I_{HS}^{**}}{E_{HS}^{**} I_{HS}} - \frac{I_{HS} L^{**}}{I_{HS}^{**} L} - \frac{S_S I_S^{**} L}{S_S^{**} I_S L^{**}} \leq 0, \quad 1 - \frac{S_S}{S_S^{**}} \leq 0. \tag{3.42}
 \end{aligned}$$

Thus, $\dot{Q} \leq 0$ whenever $\mathcal{R}_{HS} > 1$.

Since the relevant variables in the equation of I_{HS} is at the endemic equilibrium, they can be supplanted into the equations representing I_{HS} in the model (3.31) so that

$I_{HS}(t) \rightarrow I_{HS}^{**}$ as $t \rightarrow \infty$.

Therefore, Q represents a Lyapunov function in $\mathcal{D}_1 \setminus \mathcal{D}_0$.

This result shows that in a population where schistosomiasis is endemic, if $\psi = 0$, the EEP will be globally asymptotically stable (GAS) whenever $\mathcal{R}_{HS} > 1$. Hence, schistosomiasis will persist in the population regardless of the initial magnitudes of the sub-populations whenever $\mathcal{R}_{HS} > 1$.

4.0 Conclusion

A new mathematical model to theoretically investigate the role of the impact of reduced re-infection on the population dynamics for schistosomiasis disease burden in the presence of intermediate stages of development of the pathogen responsible for the disease in a given population was developed in this work. The model was shown to undergo the backward bifurcation phenomenon due to the presence of the reduced re-infection parameter. This implies that as long as there is re-infection of the population with schistosomiasis, the disease will remain endemic in the given population. A unique threshold for the reduced rate of re-infection was also obtained. A special case of the model showed that the disease-free equilibrium was locally asymptotic stable in the absence of the reduced rate of re-infection.

References

- [1] Chitsulo, L., Engels, D., Montresor, A. and Savioli, L. (2000). The global status of schistosomiasis and its control, *Acta Tropica* 77, 41-51.
- [2] Gumel, A. B. (2012). Causes of backward bifurcations in some epidemiological models, *J. Math. Anal. Appl.*, 395, pp. 355-365 DOI:10.1016/j.maa.2012.04.077
- [3] World Health Organization (WHO) (2013). Schistosomiasis Progress Report (20012011) and Strategic Plan (20122020). *World Health Organization Press; Geneva, Switzerland*. Available from: <http://www.who.int/schistosomiasis/resources/en/>. (Accessed on November 1, 2016).
- [4] World Health Organization (WHO) (2014). Schistosomiasis, Fact sheet No. 115. Updated February 2014. <http://www.who.int/mediacentre/factsheets/fs115/en/> (accessed on July 9, 2016).
- [5] World Health Organization (WHO) (2017). Schistosomiasis Factsheet 2017. *World Health Organization Press; Geneva, Switzerland*.
- [6] World Health Organization (WHO) (2019). Schistosomiasis Factsheet 2019. *World Health Organization Press; Geneva, Switzerland*.
- [7] Woolhouse, M.E.J. (1991). On the application of mathematical models of schistosome transmission dynamics I: natural transmission, *Acta Trop.*, 49 241.
- [8] Yang, H. M. (2003). Comparison between schistosomiasis transmission modeling considering acquired immunity and age-structured contact pattern with infested water, *Mathematical Biosciences* 184, 126.
- [9] Chiyaka, E. and Garira, W. (2009). Mathematical analysis of the transmission dynamics of schistosomiasis in the human-snail hosts. *J Biol Syst*;17:397423.
- [10] Diaby, M. (2015). Stability Analysis of a Schistosomiasis Transmission Model with Control Strategies, *Biomath* 1, 1504161, <http://dx.doi.org/10.11145/j.biomath.2015.04.161>
- [11] Barbour, A.D. (1982). Schistosomiasis. In: R.M. Anderson (Ed.), *Population Dynamics of Infectious Diseases*, Chapman and Hall, London, pp. 180-208.
- [12] Castillo-Chavez, C., Feng Z. and Xu, D. (2008). A schistosomiasis model with mating structure and time delay. *Mathematical Biosciences* 211, 333–341.

- [13] Chen Z., Zou L., Shen D., Zhang W. and Ruan S. (2010). Mathematical modelling and control of schistosomiasis in Hubei Province, China, *Acta Tropica* 115, 119-125.
- [14] Chiyaka, E. T., Magombedze, G. and Mutimbu, L. (2010). Modelling within host parasite dynamics of schistosomiasis. *Computational and Mathematical Methods in Medicine* Vol. 11, No. 3, 255280.
- [15] Cohen, J.E. (1977). Mathematical models of Schistosomiasis, *Ann. Rev. Eco. Syst.*, 8, pp. 209233.
- [16] Diaby, M., A. and Iggidr, A. (2016). A mathematical analysis of a model with mating structure. *Proceedings of CARI*, 246, pp.402 - 411.
- [17] Diaby, M., A., Iggidr, A., Sy, M., and Sene, A. (2014). Global analysis of a schistosomiasis infection model with biological control. *Applied Mathematics and Computation*, 246, pp.731 - 742.
- [18] Feng, Z., Curtis, J. and Minchella, D . J. (2001). The influence of drug treatment on the maintenance of Schistosome genetic diversity. *J. Math. Bio.* 43, 52-68.
- [19] Feng, Z., Li, C.-C. and Milner, F. A. (2002). Schistosomiasis models with density dependence and age of infection in snail dynamics. *Math. Biosci* 177-178, 271–286.
- [20] Feng, Z., Eppert, A., Milner, F. A. and Minchella, D . J. (2004). Estimation of Parameters Governing the Transmission Dynamics of Schistosomes. *Applied Mathematics Letters* 17, 1105-1112
- [21] Liu, Y. J., Lv, H. M. and Gao, S. J. (2016). A Schistosomiasis Model with Diffusion Effects. *Applied Mathematics* 7, 587-598. <http://dx.doi.org/10.4236/am.2016.77054>
- [22] Macdonald, G. (1965). The Dynamics of Helminth Infections with Special Reference to Schistosomes. *Transaction of the Royal Society of Tropical Medicine and Hygiene*. Vol. 59, No. 5, 489-506.
- [23] Milner, F. A. and Zhao, R. (2008). A Deterministic Model of Schistosomiasis with Spatial Structure. *Mathematical Biosciences and Engineering* Vol. 5, No. 3, pp. 505522, <http://www.mbejournal.org/>
- [24] Ngarakana-Gwasira, E. T., Bhunu, C. P., Masocha, M. and Mashonjowa, E. (2016). Transmission dynamics of schistosomiasis in Zimbabwe: A mathematical and GIS Approach, *Commun Nonlinear Sci Numer Simulat* 35, 137-147.
- [25] Qi, L. and Cui, J. (2013). A Schistosomiasis Model with Mating Structure, *Abstract and Applied Analysis*, Vol. 2013, Article ID 741386, 9 pages, <http://dx.doi.org/10.1155/2013/41386>
- [26] Qi, L., Cui, J., Huang, T., Ye, F. and Jiang, L. (2014). Mathematical Model of Schistosomiasis under Flood in Anhui Province. *Abstract and Applied Analysis* Volume 2014, Article ID 972189, 7 pages, <http://dx.doi.org/10.1155/2014/972189>
- [27] Qi, L., Xue, M., Cui, J., Wang, Q. and Wang, T. (2018). Schistosomiasis Model and its Control in Anhui Province. *Bulletin of Mathematical Biology* Volume 2014, 80: 2435–2451, <http://doi.org/10.1007/s11538-018-0474-7>
- [28] Wang, W., Liang, Y., Hong, Q. and Dai, J. (2013). African schistosomiasis in mainland China: risk of transmission and countermeasures to tackle the risk. *Parasites Vectors* 6, 249.
- [29] Yang, H. M. and Yang, A. C. (1998). The Stabilizing Effects of the Acquired Immunity on the Schistosomiasis Transmission Modeling - The Sensitivity Analysis, *Inst Oswaldo Cruz, Rio de Janeiro*, Vol. 93, Suppl. I: 63-73, 63
- [30] Zhao, R. and Milner, F. A. (2008). A mathematical model of *Schistosoma mansoni* in *Biomphalaria glabrata* with control strategies, *Bulletin of Mathematical Biology*, vol. 70, no. 7, pp. 18861905.
- [31] Zou, L. and Ruan, S. (2015). Schistosomiasis transmission and control in China. *Acta Tropica* 143, 5157.
- [32] Lakshmikantham, V., Leela, S., and Martynyuk, A.A. (1991). Stability analysis of nonlinear systems, *SIAM Review* 33(1): 152154.
- [33] van den Driessche, P. and Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Mathematical Biosciences* 180: 2948.
- [34] Hethcote, H. W. (2000). The Mathematics of Infectious Diseases. *SIAM REVIEW* Vol. 42, No. 4, pp. 599-653.
- [35] Castillo-Chavez, C. and Song, B. (2004). Dynamical Models of Tuberculosis and their Applications, *Mathematical Biosciences and Engineering*, Volume 1, Number 2, pp. 361-404.
- [36] LaSalle, J. P. and Lefschetz, S. (1976). The stability of dynamical systems, *SIAM, Philadelphia*.