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Original Article

Theoretical Study of the Impact of Availability and Efficacy of Controls on the Transmission Dynamics of Schistosomiasis

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Abstract *- In this paper we present a deterministic nonlinear model which provides mathematical and epidemiological insights to the influence of availability and efficacy of control on the transmission dynamics of Schistosomiasis. Schistosomiasis is second only to Malaria in terms of impact as the most brutal parasitic disease. There is also presently no vaccine in commercial quantity against the parasite, Schistosoma spp. Thus, the need to explore other control measures. The disease free equilibrium of the model was shown to be locally asymptotically stable if* $\mathcal{R}_c < 1$*, and unstable and if* $\mathcal{R}_c > 1$*. Again, the endemic equilibrium as also shown to be globally asymtotically stable for the special case when there is permanent immunity upon recovery.*

Keywords *- Schistosomiasis, Reproduction Number, Availability, Efficacy, Control.*

1. Introduction

Schistosomiasis popularly called snail fever is an acute and chronic disease caused by a parasitic worm (Schistosomatidae) (CDC, 2012; Inobaya et al., 2014). The parasite (Schistosoma hematobium) was identified in Egypt by Theodore Bilharz in 1851 (Inobaya et al., 2014). Other names for Schistosomiasis include Bilharza (named after Theodor Bilharz), Snail fever and Katayama fever (Chitsulo et al., 2000; Mushayabasa and Bhunu, 2011; Zou and Ruan, 2015; CDC, 2012). When Schistosomiasis affects the intestine, it is called Intestinal Schistosomiasis and this is caused by the parasite Schistosoma mansoni (discovered in 1907), and when it affects the urinary system it is referred to as Urinary Schistosomiasis which is caused by the parasitic worm Schistosoma Haematobium (discovered in 1852) (CDC, 2012; Inobaya et al., 2014). Schistosomiasis is second only to Malaria in terms of impact as the most brutal parasitic disease (CDC, 2012; Inobaya et al., 2014). The parasitic worm Schistosoma spp which causes the disease gains access to humans by appending and penetrating the skin, after which, the parasites migrate to the portal veins through the venous system, where the parasites produce eggs (Adenowo et al., 2015; Chiyaka et al., 2010; Yang, 2003). At this stage, symptoms such as abdominal irritation, fever, Hematochezia; blood in stools are experienced (Adenowo et al., 2015; Chiyaka et al., 2010; Yang, 2003). The disease is mostly common in Africa, South America, the Middle East, Asia, and the Caribbean (Hotez and Kamath, 2009; Hotez et al., 2012). It is worthy of note that the various forms of the infection are caused by different snail species serving as their intermediate hosts (Feasey et al., 2010). The snail specie Biomphalaria is responsible for S. Mansoni, Oncomelania is responsible for S. Japonicum, while the snail specie Tricula (Neotricula aperta) is in turn responsible for *S*. Mekongi and Bulinus is responsible for *S*. Haematobium and S. Intercalatum (Feasey et al., 2010). Humans by their activities around water bodies get into freshwater bodies that harbour snails that house Schistosoma sporocysts which evolve into cercariae (Yang, 2003). Cercariae then glue to and pierce the human skin and eventually reach the portal blood or bladder after migrating via the lung and blood capillaries to blood vessels (Adenowo et al., 2015; Yang, 2003). Within a period of about 6 weeks, the cercariae metamorphoses into male and female adult worms from schistosomula (Adenowo et al., 2015; Yang, 2003). Human proteins are absorbed by the worms into their structures, so there is little or no immune response by most infected humans (Chiyaka et al., 2010; Yang, 2003). After the adult worms mate in the portal or bladder, egg are produced (Adenowo et al., 2015; Chiyaka et al., 2010; Yang, 2003).

The eggs however stimulate a strong immune response in most humans (Adenowo et al., 2015; Chiyaka et al., 2010; Yang, 2003). There is so much uncertainty surrounding the lifespan of the adult worms; it ranges from 5-10 years and sometimes longer than 30 years (Yang, 2003). Less than 50% of the eggs produced move via the bladder tissue or bowel and are released into freshwater via feces or urine, while the remaining eggs which were not passed out as feces or urinary become trapped in

neighbouring tissues or are transported by the circulatory or lymphatic structure and can become placed in almost any organ of the body (Adenowo et al., 2015; Chiyaka et al., 2010; Yang, 2003). The eggs shed via urine or feces discard their shells and breed into a fringe of hairlike free-swimming larva (Miracidium), which locates a particular specie of snail, penetrates and infects it and within 4-6 weeks, the snail produces thousands of Cercariae (Adenowo et al., 2015; Chiyaka et al., 2010; Yang, 2003). The life cycle is much more compounded by S. Japonicum species (responsible for Intestinal Schistosomiasis) that sometimes infect domestic and wild animals which now become another host system (by implication having three host) and it is endemic in certain regions in the Philippines and some specific locations in China (Adenowo et al., 2015; Chitsulo et al., 2000; Chiyaka et al., 2010; Hotez et al., 2006; Yang, 2003).

Schistosomiasis can be prevented if humans can avoid any possible contact with cercariae laden fresh water and boiling of fresh water suspected to infected with Schistosoma spp before possible usage for domestic purposes and consumption (CDC, 2012; Inobaya et al., 2014). Exposing fresh water suspected to be infected with Schistosoma spp for at least a day before using it for domestic purposes and using thin netting sifts for isolating cercariae from fresh water can also help in reducing the spread of Schistosomiasis (CDC, 2012; Inobaya et al., 2014). Precautionary chemotherapy can be used as a measure for regulating the condition of being infected and can result in decline in the incidence, range, and seriousness of the effects of Schistosomiasis (WHO, 2013). Mass drug administration programmes can also help to bring down the incidence and burden of the disease (CDC, 2012; Inobaya et al., 2014). Snail elimination from some freshwater sources using a chemical substance (repeated treatment is required) known as molluscicides (snail bait) have been delineated to reduce the incidence of Schistosomiasis (CDC, 2012; Inobaya et al., 2014). Sadly, individuals who have been cured and exhibit no symptoms of Schistosomiasis can easily get tainted again if they come into contact with cercariae laden fresh water; as the human immune response to Schistosomiasis is unable to thwart a reinfection. Meaning that recovery from disease does not confer permanent immunity (CDC, 2012; Inobaya et al., 2014). Presently, no vaccine in commercial quantity is available against the parasite, Schistosoma spp., but scientific experimentation is in progress and hopefully in the near future, a vaccine may become available (CDC, 2012; Inobaya et al., 2014). The drug used in most patient is Praziquantel (which is a derivative of Pyrazinosoquinolone) and it is strictly potent against mature worms and not potent against the eggs or not fully developed worms (CDC, 2012; WHO, 1993; WHO, 2017a). However, Praziquantel causes speedy breakdown of the worms which subsequently empowers the human immune system to fight the parasite (CDC, 2012).

Numerous mathematical models have been used to study the dynamics of Schistosomiasis at population level. Feng et al. (2002) formulated a mathematical model for Schistosomiasis which depended on density and the age of the infection in snail dynamics. Their model incorporated practical attributes like treatment of human using drugs, infection age in snail host, Schistosomiasis distribution within the human host and infection related death in the human and snail subpopulations to study Schistosomiasis dynamics. They were also able to scrutinize numerous control strategies to ascertain the cost advantage of treatment programs. Allen and Victory (2003) formulated a model for Schistosomiasis which involved the definitive human host, transitional snail hosts, another natural host and a snail species which is a competitor as well as resistant to the infection. Results of the simulations showed that the competitor snail species can affect the Schistosomiasis dynamics. Chiyaka and Garira (2009) developed a unique model that mathematically studied the host-parasite dynamics of Schistosomiasis. They constructed a deterministic mathematical model which incorporated miracidia and cercariae concentrations to studied the dynamics of Schistosomiasis. Their simulation results showed that control strategies aimed at disease transmission from snail to man were more productive than those aimed at transmission from man to snail. Longxing et al.(2014) built a model which mathematically investigated the effect of flooding on the basic reproduction number and the Schistosomiasis transmission dynamics in Anhui Province of China. Their result showed that the system can be destabilize by flooding giving rise to a Hopf bifurcation. Ngarakana-Gwasira et al. (2016) extended the work of Chiyaka and Garira (2009) and investigated the impact of aquatic environment (taken in circumstances of rain fall arrangements) and temperature from 1950 to 2000 on the dynamics of Schistosomiasis in Zimbabwe. Their results suggested that Schistosomiasis can best be transmitted at a temperature of about 23 degrees celsius and that high threshold number which implied high disease incidence was recorded in Zambezi valley and lower downs of the country. Olowu, et al (2021a) investigated a two patch metapopulation Schistosomiasis model with sixteen deterministic ordinary differential equations where the thresholds for effective control of Schistosomiasis were established. Olowu, et al (2021b) investigated the quantitative analysis of a two patch metapopulation model. Their findings showed effective control of Schistosomiasis in one patch can result to a drastic reduction in another patch. Bada et al (2021) developed a mathematical model to examine the effect of case detection on the transmission dynamics of schistosomiasis and established that a significant decrease in the schistosomiasis cases could be achieved in the population if there is an increase in the proportion of the detected human cases of schistosomiasis which are set for treatment immediately. They further established that schistosomiasis can be controlled in a population if the public health control programmes provide and implement strategies for detection as well as the timely treatment of a very large number of persons infected with schistosomiasis. Ako, et al (2021) developed a 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. The model was shown to undergo the backward bifurcation phenomenon due to the presence of the reduced re-infection parameter which implied that as long as there is re-infection of the population with schistosomiasis, the disease will remain endemic in the given population. They also established a unique threshold for the reduced rate of re-infection and a special case of their model showed that the disease-free equilibrium was locally asymptotic stable in the absence of the reduced rate of re-infection.

These models have brought a lot of insight into the transmission dynamics of Schistosomiasis but they have had relatively little or no impact on investigating the impact of control parameters that seeks to curtail the interactions between humans and cercariae and between snails and miracidia. Therefore, we propose a mathematical model for Schistosomiasis which will be used to investigate the impact of the availability and efficacy of control measures on the transmission dynamics of Schistosomiasis. Given that Schistosomiasis is mostly endemic in poor countries. The focus is on how the availability and efficacy of control measures will impact on the dynamics of the infection in such a population.

2. Model Formulation

In formulating the model, we assume that the entire population is homogeneous, well-mixed and all individuals have equal chances of being infected and that the number of effective contacts (resulting in an infection) is assumed to depend on the frequency of contacts between susceptible humans and Cercariae infected water (Hethcote, 2000; Mishra, 2010). The total human population for the Schistosomiasis model is partitioned into Susceptible humans (S_h), Latently infected or Exposed humans (E_h) and Infected humans (I_h). The non-human compartments are divided into Miracidia concentration (M), population of uninfected Snails (U), Latently-infected snails (L), patent infected Snails (not yet releasing cercariae) (I_s) and free swimming Cercariae ready to enter human skin (C), From the above, the total human population and non-human population at any time t, are given by

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) \text{ and } N_n(t) = M(t) + U(t) + L(t) + I_s(t) + C(t).$$

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \lambda_h S_h + \gamma_h I_h - \mu_h S_h \\
\frac{dE_h}{dt} &= \lambda_h S_h - (\kappa_h + \mu_h) E_h \\
\frac{dI_h}{dt} &= \kappa_h E_h - (\gamma_h + \delta_h + \mu_h) I_h \\
\frac{dM}{dt} &= \theta_M I_h - \mu_M M \\
\frac{dU}{dt} &= \Lambda_s - \lambda_s U - \mu_s U \\
\frac{dL}{dt} &= \lambda_s U - (\kappa_s + \mu_s) L \\
\frac{dI_s}{dt} &= \kappa_s L - (\delta_s + \mu_s) I_s \\
\frac{dC}{dt} &= \theta_C I_s - \mu_C C.
\end{aligned}$$

Where

$$\lambda_h = \beta_h \frac{(1 - \phi\xi)C}{C_0 + \varepsilon C} \text{ and } \lambda_s = \beta_s \frac{(1 - \pi\nu)M}{M_0 + \varepsilon M}$$

are the forces of infection. A schematic representation/description (which is a graphic description of the movement of individuals between the various compartments of the system) is given in Figure 1. The state variables as well as the parameters used in the mathematical formulation are given in Tables 1 and 2, respectively.

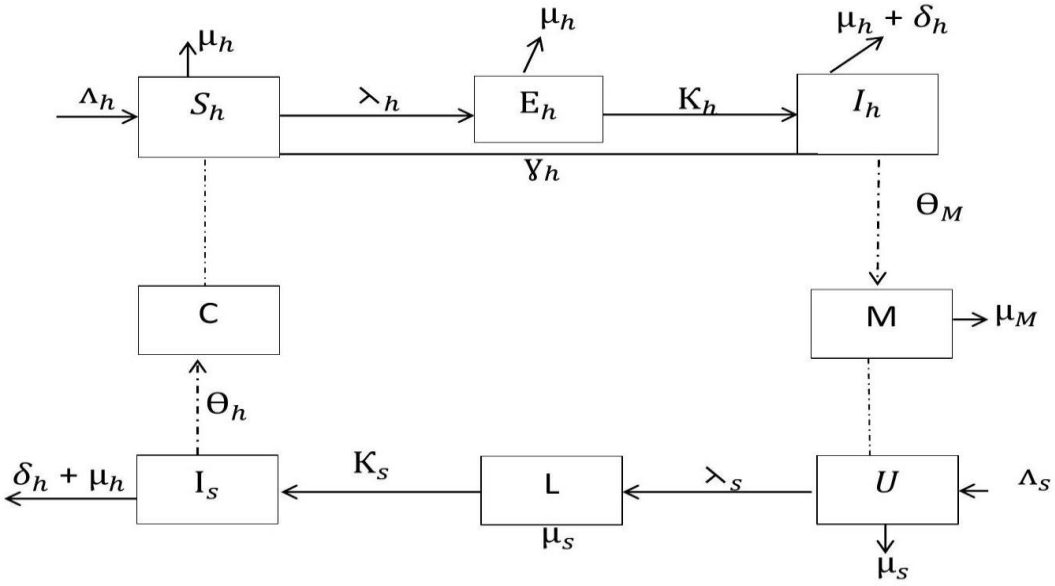

Fig. 1 Schematic representation of the transmission dynamics of the Schistosomiasis model

Table 1. State variables of the model

Variable	Description
$S_h(t)$	Susceptible individuals
$E_h(t)$	Latently tainted individuals
$I_h(t)$	Infected individuals
$M(t)$	Miracidia concentration
$U(t)$	Uninfected snails
$L(t)$	Latently-infected snails
$I_s(t)$	Tainted snails not yet releasing cercariae
$C(t)$	Free swimming Cercariae ready to enter human skin

Table 2. Parameters of the model

Parameter	**Description**
μ_k (k = h, s)	Natural death rate for the kth sub population.
Λ_k (k=h, s)	Recruitment rate for the kth sub population.
β_k (k=h, s)	Cercariae and Miracidia infectious rate respectively for the kth sub population.
C_0	Saturation constant for Cercariae.
M_0	Saturation constant for Miracidia.
ε	Limitation of growth velocity of Cercariae and Miracidia.
K_k ($k = h, s$)	progression rate from latent class to infectious classes in the kth sub population.
δ_k ($k = h, s$)	Disease and parasite induced death respectively for humans and snails the kth sub population.
γ_h	Recovery rate for humans.
θ_M	Rate at which egg produced by adult Schistosome hatch and develop to free swimming Miracidia.
θ_C	Rate at which patent infected snails release cercariae.
ϕ	Efficacy of control in the human population.
ξ	Availability of control in the human population.
π	Efficacy of control in the aquatic (Snail) environment.
v	Availability of control in the aquatic environment
μ_M	Natural death rate for miracidia
μ_C	Natural death rate for cercariae

3. Analysis of the Model

The qualitative properties of the model will be explored in this section.

3.1. Basic Properties of the Model

In this section, we show that the state variables of the model are always non-negative and bounded for all time, t, since the model describes Human and Snail populations, Miracidia and Cercariae concentrations which cannot be non-positive. We also showed that the orbits generated by the model are positively invariant for all time, t.

Theorem 3.1: Let the initial data of the availability and efficacy of control Schistosomiasis model be given as $X(0) \geq 0$,

where: $X(t) = (S_h(t), E_h(t), I_h(t), M(t), U(t), L(t), I_s(t), C(t))$. Then the orbits $X(t)$ of the availability and efficacy of control Schistosomiasis model with non-negative initial conditions will always be non-negative for all time $t > 0$.

Proof:

Let $t_1 = \sup\{t > 0 : X(t) \geq 0 \in [0, t]\}$. Thus for $t_1 > 0$, from the first equation of model, it follows

$$\frac{dS_h(t)}{dt} = \Lambda_h - (\lambda_h + \mu_h)S_h + \gamma_h I_h(t)$$

which can be rewritten as

$$\left[\frac{d}{dt} + (\lambda_h + \mu_h)\right] S_h(t) \geq \Lambda_h$$

which implies

$$\frac{d}{dt}\left[S_h(t)\exp\left\{(\mu_h)t + \int_0^t \lambda_h(\tau)d\tau\right\}\right] \geq \Lambda_h \exp\left\{(\mu_h)t + \int_0^t \lambda_h(\tau)d\tau\right\}$$

as a result,

$$S_h(t_1)\exp\left\{(\mu_h)t_1 + \int_0^{t_1} \lambda_h(\tau)d\tau\right\} - S_h(0) \geq \int_0^{t_1} \Lambda_h[\text{e x p}\{(\mu_h)y + \int_0^y \lambda_h(\tau)d\tau\}]dy,$$

hence,

$$S_h(t_1) \geq S_h(0)\exp\left[-(\mu_h)t_1 - \int_0^{t_1} \lambda_h(\tau)d\tau\right] + [\text{e x p}\{-(\mu_h)t_1 - \int_0^{t_1} \lambda_h(\tau)d\tau\}]\int_0^{t_1} \Lambda_h\left[\exp\left\{(\mu_h)y + \int_0^y \lambda_h(\tau)d\tau\right\}\right]dy \geq 0.$$

Hence $S_h(t) \geq 0, \forall t > 0$.

Considering equation 2 of model:

$$\frac{dE_h}{dt} = \lambda_h S_h - (\kappa_h + \mu_h)E_h$$

$$\frac{dE_h}{dt} \geq -(\kappa_h + \mu_h)E_{h_1}$$

Integrating with respect to t in $[0, t_1]$, yields

$$E_h(t_1) \geq E_h(0)\exp\{-(\kappa_h + \mu_h)t_1\} > 0.$$

Hence $E_h(t) > 0$ for all $t > 0$.

Following the procedure above for equation (3) – (8) of the model, it can be shown that $I_h(t) > 0$, $M(t) > 0$, $U(t) > 0$, $L(t) > 0$, $I_s(t) > 0$ and $C_1(t) > 0$ for all $t > 0$.

Hence the trajectories X(t), where $X(t) = (S_h(t), E_h(t), I_h(t), M(t), U(t), L(t), I_s(t), C(t)$ generated by the availability and efficacy of control Schistosomiasis model with non-negative initial data/conditions will always be non-negative for all time $t > 0$.

Next, we need to prove that each of the subpopulations: Humans, Miracidia, Snails and Cercariae (since we cannot lump all the subpopulations in one invariant set) are bounded and also determine the bound and finally show that the domains of these subpopulations are positively-invariant and attracts all the positive trajectories (there exist a unique solution to the initial value problem, and solution exists for all time) of the model

Lemma 3.1: Let $S_h(t), E_h(t), I_h(t), M(t), U(t), L(t), I_s(t), C(t)$ be trajectories of the model with initial conditions and the biological feasible region given by the set $\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_M \times \mathcal{D}_S \times \mathcal{D}_C \subset \mathbb{R}^3_+ \times \mathbb{R}^1_+ \times \mathbb{R}^3_+ \times \mathbb{R}^1_+ \subset \mathbb{R}^8_+$ where:

$$\mathcal{D}_h = \left\{(S_h, E_h, I_h) \in \mathbb{R}^3_+ : N_h \leq \frac{\Lambda_h}{\mu_h}\right\}$$
$$\mathcal{D}_M = \left\{(M) \in \mathbb{R}^1_+ : M \leq \frac{\theta_M \Lambda_h}{\mu_M \mu_h}\right\}$$
$$\mathcal{D}_S = \left\{(U, L, I_s) \in \mathbb{R}^3_+ : N_S \leq \frac{\Lambda_s}{\mu_s}\right\}$$
$$\mathcal{D}_C = \left\{(C) \in \mathbb{R}^1_+ : C \leq \frac{\theta_C \Lambda_s}{\mu_C \mu_s}\right\}$$

is positively-invariant and attracts all the non-negative trajectories of model.

Proof:

(a) To determine the bound for the human subpopulation, we add up the right hand side of the vector field for the human population in the model, which is the rate of change of the total population described by the model and it is given by:

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_h I_h$$

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_h I_h$$

$$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h$$

which is a linear first order ODE with integrating factor given as $e^{\mu_h t}$. Thus, we obtain

$$\frac{dN_h}{dt} e^{\mu_h t} + \mu_h N_h e^{\mu_h t} \leq \Lambda_h e^{\mu_h t}$$

which can be rewritten as

$$\int_0^t \frac{dN_h}{d\tau} e^{\mu_h \tau} d\tau \leq \Lambda_h \int_0^t e^{\mu_h \tau} d\tau$$

Integrating and using the starting condition $N_h(t) = N_h(0)$, we obtain

$$N_h(t)e^{\mu_h t} - N_h(0) \leq \frac{\Lambda_h}{\mu_h}(e^{\mu_h t} - 1).$$

Solving for $N_h(t)$ gives

$$N_h(t) \leq N_h(0)e^{\mu_h t} + \frac{\Lambda_h}{\mu_h}(1 - e^{\mu_h t}).$$

If $N_h(0) \leq \frac{\Lambda_h}{\mu_h}$, then $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$. Hence, the domain $\mathcal{D}_h$ is positively invariant under the flows of the model. 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 approaches $\frac{\Lambda_h}{\mu_h}$ as $t \to \infty$. Thus, the domain $\mathcal{D}_h$ attracts all trajectories and no trajectory goes out of any boundary of $\mathcal{D}_h$ in $\mathbb{R}^3_+$.

(b) To determine the bound for the concentration of the Miracidia in the model

$$\frac{dM}{dt} = \theta_M I_h - \mu_M M$$

$\frac{dM}{dt} \leq \theta_M \frac{\Lambda_h}{\mu_h} - \mu_M M$, since $N_h = S_h + E_h + I_h \leq \frac{\Lambda_h}{\mu_h} \Longrightarrow I_h \leq \frac{\Lambda_h}{\mu_h}$.

which is a linear equation with integrating factor given as $e^{\mu_M t}$. Thus, we obtain

$$\frac{dM}{dt} e^{\mu_M t} + \mu_M M e^{\mu_M t} \leq \theta_M \frac{\Lambda_h}{\mu_h} e^{\mu_M t}.$$

Which can be re-written as

$$\int_0^t \frac{dM}{d\tau} e^{\mu_M \tau} d\tau \leq \theta_M \frac{\Lambda_h}{\mu_h} \int_0^t e^{\mu_M \tau} d\tau$$

Integrating and using the initial condition, $M(t) = M(0)$, we obtain

$$M(t)e^{\mu_M t} - M(0) \leq \frac{\Lambda_h \theta_M}{\mu_h \mu_M}(e^{\mu_M t} - 1).$$

Solving for $M(t)$ gives

$$M(t) \leq M(0)e^{\mu_M t} + \frac{\Lambda_h \theta_M}{\mu_h \mu_M}(1 - e^{\mu_M t}).$$

If $M(0) \leq \frac{\Lambda_h \theta_M}{\mu_h \mu_M}$, then $M(t) \leq \frac{\Lambda_h \theta_M}{\mu_h \mu_M}$. Hence, the domain $\mathcal{D}_M$ is positively invariant under the flow of the model. Moreover, if $M(0) > \frac{\Lambda_h \theta_M}{\mu_h \mu_M}$, then either the orbits enters the domain $\mathcal{D}_M$ in finite time or $M(t)$ asymptotically approaches $\frac{\Lambda_h \theta_M}{\mu_h \mu_M}$ as $t \to \infty$. Thus, the domain $\mathcal{D}_M$ attracts all trajectories and no trajectory goes out of any boundary of $\mathcal{D}_M$ in $\mathbb{R}^1_+$.

(c) For the bound of the Snail population, we add up the right hand side of the vector field of the Snail population in the model and this yields

$$\frac{dN_s}{dt} = \Lambda_s + (U + L + I_s)\mu_s - \delta_s I_s$$

$$\frac{dN_s}{dt} = \Lambda_s - \mu_h N_s - \delta_s I_s$$

it follows that

$$\frac{dN_s}{dt} \leq \Lambda_s - \mu_s N_s$$

which is a linear equation with integrating factor given as $e^{\mu_s t}$. Thus, we obtain

$$\frac{dN_s}{dt} e^{\mu_s t} + \mu_s N_s e^{\mu_s t} \leq \Lambda_s e^{\mu_s t}$$

which can be rewritten as

$$\int_0^t \frac{dN_s}{d\tau} e^{\mu_s \tau} d\tau \leq \Lambda_s \int_0^t e^{\mu_s \tau} d\tau$$

Integrating and using the initial condition $N_s(t) = N_s(0)$, we obtain

$$N_s(t)e^{\mu_s t} - N_s(0) \leq \frac{\Lambda_s}{\mu_s}(e^{\mu_s t} - 1)$$

Solving for $N_s(t)$ gives

$$N_s(t) \leq N_s(0)e^{-\mu_s t} + \frac{\Lambda_s}{\mu_s}(1 - e^{-\mu_s t})$$

If $N_s(0) \leq \frac{\Lambda_s}{\mu_s}$, then $N_s(t) \leq \frac{\Lambda_s}{\mu_s}$. Hence, the domain $\mathcal{D}_s$ is positively invariant under the flow of the model. 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 $t \to \infty$. Thus, the domain $\mathcal{D}_s$ attracts all trajectories and no trajectory goes out of any boundary of $\mathcal{D}_s$ in $\mathbb{R}^3_+$.

(d) To determine the bound for the concentration of the Cercariae in the model and it yields

$$\frac{dC}{dt} = \theta_C I_s - \mu_C C$$

$$\frac{dC}{dt} = \theta_C I_s - \mu_C C$$

it follows that,

$\frac{dC}{dt} \leq \theta_C \frac{\Lambda_s}{\mu_s} - \mu_C C$, since $N_s = U + L + I_s \leq \frac{\Lambda_s}{\mu_s} \Longrightarrow I_s \leq \frac{\Lambda_s}{\mu_s}$.

which is a linear equation with integrating factor given as $e^{\mu_C t}$. Thus, we obtain

$$\frac{dC}{dt} e^{\mu_C t} + \mu_C C e^{\mu_C t} \leq \theta_C \frac{\Lambda_s}{\mu_C} e^{\mu_C t}$$

which can be rewritten as

$$\int_0^t \frac{dC}{d\tau} e^{\mu_C \tau} d\tau \leq \theta_C \frac{\Lambda_s}{\mu_s} \int_0^t e^{\mu_C \tau} d\tau$$

Integrating and using the initial condition $C(t) = C(0)$, we obtain

$$C(t)e^{\mu_C t} - C(0) \leq \frac{\Lambda_s \theta_C}{\mu_s \mu_C}(e^{\mu_C t} - 1).$$

Solving for $C(t)$ gives

$$C(t) \leq C(0)e^{-\mu_C t} + \frac{\Lambda_s \theta_C}{\mu_s \mu_C}(1 - e^{-\mu_C t})$$

If $C(0) \leq \frac{\Lambda_s \theta_C}{\mu_s \mu_C}$, then $C(t) \leq \frac{\Lambda_s \theta_C}{\mu_s \mu_C}$. Hence, the domain $\mathcal{D}_C$ is positively invariant under the flow of the model. Moreover, if $C(0) > \frac{\Lambda_s \theta_C}{\mu_s \mu_C}$, then either the orbits enters the domain $\mathcal{D}_C$ in finite time or $C(t)$ asymptotically approaches $\frac{\Lambda_s \theta_C}{\mu_s \mu_C}$ as $t \to \infty$. Thus, the domain $\mathcal{D}_C$ attracts all trajectories and no trajectory goes out of any boundary of $\mathcal{D}_C$ in $\mathbb{R}_+^1$.

From the above, we have shown that $\mathcal{D}_h, \mathcal{D}_M, \mathcal{D}_s$ and $\mathcal{D}_C$ are positively invariant and since $\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_M \times \mathcal{D}_s \times \mathcal{D}_C$, it implies that the domain $\mathcal{D}$ is positively-invariant and an attractor, so that no trajectory leaves via any boundary of $\mathcal{D}$.

$$\mathcal{D} = \begin{cases} (S_h, E_h, I_h,) \in \mathbb{R}_+^3 : N_h \leq \dfrac{\Lambda_h}{\mu_h} \\ (M) \in \mathbb{R}_+^1 : M \leq \dfrac{\theta_M \Lambda_h}{\mu_M \mu_h} \\ (U, L, I_s) \in \mathbb{R}_+^3 : N_S \leq \dfrac{\Lambda_s}{\mu_s} \\ (C) \in \mathbb{R}_+^1 : C \leq \dfrac{\theta_C \Lambda_s}{\mu_C \mu_s} \end{cases}$$

It implies that the right hand side of the model is smooth, hence there exist a unique solution to the initial value problem, and solution exists for all time. Hence the model is well posed when considered from both mathematical and epidemiological point of views and it is therefore sufficient to study the dynamics of the flows generated by the model in $\mathcal{D}$.

3.2. Local Asymptotic Stability of the Disease Free Equilibrium

The Disease Free Equilibrium (DFE) of the model is obtained by Setting the right-hand side of the equations in the model as well as the infected compartments (i.e., state variables of the infected classes) to zero and solving the resulting system. The DFE for the model is given by:

$$\mathcal{E}_0^p = (S_h^0, E_h^0, I_h^0, M^0, U^0, L^0, I_s^0, C^0) = \left(\frac{\Lambda_h}{\mu_h}, 0,0,0, \frac{\Lambda_s}{\mu_s}, 0,0,0\right).$$

The method of next generation matrix operator proposed by van den Driessche and Watmough (2002) is used to investigate whether the DFE of the system is Local Asymptotic Stability (LAS). Using notations similar to the ones used in van den Driessche and Watmough (2002), the matrices F and V, of new infection terms as well as the remaining transfer terms, are respectively, given by:

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & \frac{A_1\beta_s\Lambda_s}{M_0\mu_s} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{A_2\beta_h\Lambda_h}{C_0\mu_h} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} T_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & T_1 & 0 & 0 & 0 & 0 \\ -\kappa_s & 0 & T_4 & 0 & 0 & 0 \\ 0 & -\kappa_h & 0 & T_2 & 0 & 0 \\ 0 & 0 & 0 & -\theta_M & \mu_M & 0 \\ 0 & 0 & -\theta_C & 0 & 0 & \mu_C \end{pmatrix}$$

The reproduction number, $\mathcal{R}_c = \rho(FV^{-1})$, with $\rho(\cdot)$ being the largest eigenvalue associated with matrix FV^{-1}, is given by

$$\mathcal{R}_c = \sqrt{\frac{A_1A_2\beta_h\beta_s\Lambda_h\Lambda_s\kappa_h\kappa_s\theta_M\theta_C}{T_1T_2T_3T_4C_0M_0\mu_h\mu_M\mu_s\mu_C}},$$

where: $A_1 = (1-\pi\nu), A_2 = (1-\phi\xi), T_1 = (\kappa_h + \mu_h), T_2 = (\gamma_h + \delta_h + \mu_h), T_3 = (\kappa_s + \mu_s), T_4 = (\delta_s + \mu_s)$.

The result in Lemma 3.2 is deduce from Theorem 2 of van den Driessche and Watmough (2002):

Lemma 3.2: The DFE of the availability and efficacy of control model is LAS in $\mathcal{D}$ if $\mathcal{R}_c < 1$, and unstable if $\mathcal{R}_c > 1$.

In Epidemiological sense, Lemma 3.2 implies that Schistosomiasis can be eradicated from the population with the availability and efficacy of control when $\mathcal{R}_c < 1$, if the starting sizes of the sub-populations of the model lie in the basin of attraction of the DFE and that a little inflow of sick humans with Schistosomiasis into the population where control is available would not result to large outbreaks and Schistosomiasis will become endemic in the population where control is available if $\mathcal{R}_c > 1$.

3.3. Analysis of the Reproduction Number $(\mathcal{R}_c)$

The sensitivity of $\mathcal{R}_c$ to some key parameters $(\phi, \xi, \pi, \nu, \gamma_h)$ is investigated by considering the partial derivatives of $\mathcal{R}_c$ with respect to these parameters.

(a) If we consider how $\mathcal{R}_c$ changes with respect to the product of availability (ξ) and the efficacy (ϕ) of the control measures in the human subpopulation, we obtain:

$$\frac{\partial\mathcal{R}_c^2}{\partial\phi\xi} = -\frac{(1-\pi\nu)\beta_h\beta_s\theta_C\theta_M\kappa_h\kappa_s\Lambda_h\Lambda_s}{C_0M_0\mu_h\mu_M\mu_s\mu_CT_1T_2T_3T_4}$$

Clearly, it follows that $\frac{\partial\mathcal{R}_c^2}{\partial\phi\xi} < 0$ unconditionally. This implies that increasing the availability of the control measures (ξ) and sustaining the efficacy (ϕ) in the human subpopulation such that their combination will be close to a $\mathbf{100}\%$ will always result to a decrease in the incidence of Schistosomiasis in the population irrespective of other parameter values in $\mathcal{R}_c$.

(b) Considering how $\mathcal{R}_c$ changes with respect to the product of availability of the control measures (ν) and the efficacy (π) of the control measures in the aquatic environment, we obtain:

$$\frac{\partial \mathcal{R}_c^2}{\partial \pi \nu} = -\frac{(1-\phi\xi)\beta_h\beta_s\theta_C\theta_M\kappa_h\kappa_s\Lambda_h\Lambda_s}{C_0M_0\mu_h\mu_M\mu_s\mu_C T_1T_2T_3T_4}$$

Obviously, it follows that $\frac{\partial \mathcal{R}_c^2}{\partial \pi \nu} < 0$ unconditionally. This implies that increasing the availability of the control measures (ν) and sustaining the efficacy (π) in the aquatic environment such that their combination is close to a $\mathbf{100}\%$ will always result to a decrease in the incidence of Schistosomiasis in the population irrespective of other parameter values in $\mathcal{R}_c$.

(c) Considering how $\mathcal{R}_c$ changes with respect to the treatment/recovery rate (γ_h) in the human subpopulation, we obtain:

$$\frac{\partial \mathcal{R}_c^2}{\partial \gamma_h} = -\frac{(1-\phi\xi)(1-\pi\nu)\beta_h\beta_s\theta_C\theta_M\kappa_h\kappa_s\Lambda_h\Lambda_s}{C_0M_0\mu_h\mu_M\mu_s\mu_C T_1T_2^2T_3T_4}$$

Clearly, it follows that $\frac{\partial \mathcal{R}_c^2}{\partial \gamma_h} < 0$ unconditionally. This implies that increasing the treatment rate (γ_h) in the human population will always result to a decrease in the incidence of Schistosomiasis in the population irrespective of other parameter values in $\mathcal{R}_c$.

We further investigate the sensitivity of $\mathcal{R}_c$ with respect to its sensitivity to some key parameters $(\phi, \xi, \pi, \nu, \gamma)$ that describes the effect of the control parameters and recovery on the availability and efficacy of control Schistosomiasis dynamics by considering the limiting values of $\mathcal{R}_c$ as extremely large values are assigned to these parameters.

(a) Considering the limiting value of $\mathcal{R}_c$ as $\phi\xi \to 1$, we have

$$\lim_{\phi\xi\to 1} \mathcal{R}_c = \lim_{\phi\xi\to 1} \sqrt{\frac{A_1A_2\beta_h\beta_s\Lambda_h\Lambda_s\kappa_h\kappa_s\theta_M\theta_C}{T_1T_2T_3T_4C_0M_0\mu_h\mu_M\mu_s\mu_C}} = 0$$

The limiting value suggests that increasing the availability of the control measures (ξ) and sustaining the efficacy (ϕ) in the human subpopulation such that their combination is a $\mathbf{100}\%$ can help in effective Schistosomiasis control in the population and significant decrease in the reproduction number to a value below unity.

(b) Considering the limiting value of $\mathcal{R}_c$ as $\pi\nu \to 1$, we obtain

$$\lim_{\pi\nu\to 1} \mathcal{R}_c = \lim_{\pi\nu\to 1} \sqrt{\frac{A_1A_2\beta_h\beta_s\Lambda_h\Lambda_s\kappa_h\kappa_s\theta_M\theta_C}{T_1T_2T_3T_4C_0M_0\mu_h\mu_M\mu_s\mu_C}} = 0$$

The limiting value suggests that increasing the availability of the control measures (ν) and sustaining the efficacy (π) in the aquatic environment such that their combination is a $\mathbf{100}\%$ can help in effective control and eradication of Schistosomiasis in the population and significant decrease in the value of the reproduction number to a value below unity.

(c) Considering the limiting value of $\mathcal{R}_c$ as $\gamma_h \to \infty$, we obtain

$$\lim_{\gamma_h\to\infty} \mathcal{R}_c = \lim_{\gamma_h\to\infty} \sqrt{\frac{A_1A_2\beta_h\beta_s\Lambda_h\Lambda_s\kappa_h\kappa_s\theta_M\theta_C}{T_1T_2T_3T_4C_0M_0\mu_h\mu_M\mu_s\mu_C}} = 0$$

The limiting value indicates that at a very high treatment/ recovery rate $(\gamma_h \to \infty)$, Schistosomiasis can be be completely eradicated from the population and bringing the value of the corresponding reproduction number to a value below unity.

In conclusion, these analysis showed that both the availability and efficacy of control measures in the human population and the aquatic environment will go a long way in controlling the spread of human Schistosomiasis.

3.4. Existence of Endemic Equilibrium Point

The existence of Endemic Equilibrium Point (EEP) of the model. This is the equilibrium for which the disease persist in the population.

Let $\mathcal{E}_1^p = (S_h^{**}, E_h^{**}, I_h^{**}, M^{**}, U^{**}, L^{**}, I_s^{**}, C^{**})$ be an EEP for the model.

The EEP of the model is obtained by solving the right hand side of the equations in the model in terms of the forces of infection at the EEP.

$$\begin{aligned} S_h^{**} &= \frac{\Lambda_h T_1 T_2}{T_5\lambda_C^{**} + \mu_h T_1 T_2},\ E_h^{**} = \frac{\Lambda_h \lambda_C^{**} T_2}{T_5\lambda_C^{**} + \mu_h T_1 T_2},\ I_h^{**} = \frac{\Lambda_h \lambda_C^{**} \kappa_h}{T_5\lambda_C^{**} + \mu_h T_1 T_2} \\ M^{**} &= \frac{\theta_M \kappa_h + \lambda_C^{**}\Lambda_h}{\mu_M(T_5\lambda_C^{**} + \mu_h T_1 T_2)},\ U^{**} = \frac{\Lambda_s}{\lambda_s^{**} + \mu_s},\ L^{**} = \frac{\Lambda_s \lambda_s}{T_3(\lambda_s^{**} + \mu_s)} \\ I_s^{**} &= \frac{\Lambda_s \lambda_s \kappa_s}{T_3 T_4(\lambda_s^{**} + \mu_s)},\ C^{**} = \frac{\Lambda_s \lambda_s \kappa_s \theta_C}{T_3 T_4 \mu_s(\lambda_s^{**} + \mu_s)} \end{aligned}$$

where: $T_1 = (\kappa_h + \mu_h), T_2 = (\gamma_h + \delta_h + \mu_h), T_3 = (\kappa_s + \mu_s), T_4 = (\delta_s + \mu_s),$ $T_5 = T_1 T_2 - \gamma_h \kappa_h > 0, T_6 = C_0 \mu_C T_3 T_4 + \varepsilon \Lambda_s \kappa_s \theta_s, T_7 = M_0 \mu_M T_5 + \varepsilon \theta_M \kappa_h \Lambda_h.$

Substituting these equations into the force of infection and after several algebraic simplifications, the EEP of the model satisfies the following polynomial at the steady state:

$$\lambda_s^{**} K_1 + K_0 = 0$$

Where $K_1 = A_1 \beta_h \Lambda_s \kappa_s \theta_C T_7 + M_0 \mu_M \mu_h T_1 T_2$ *and* $K_0 = C_0 M_0 \mu_M \mu_C \mu_s \mu_h T_1 T_2 T_3 T_4 (1 - \mathcal{R}_c^2)$

From the above analysis, we have thus established the results in the Lemma 3.3

Lemma 3.3: The model, has a unique EEP given by $\mathcal{E}_1^p$, whenever $\mathcal{R}_c^2 > 1$ and no EEP when $\mathcal{R}_c^2 < 1$.

The epidemiological interpretation of the unique EEP is that, in a population, the long established necessity of having the reproduction number below one is both necessary and sufficient for the effective control of Schistosomiasis in the population.

3.5. Global Asymptotic Stability of the DFE

In this subsection, we prove that the Disease Free equilibrium (DFE) of the model is Global Asymptotic Stability (GAS).

Theorem 3.2: The DFE of the model is GAS in $\mathcal{D}$ whenever $\mathcal{R}_c \leq 1$ and not stable if $\mathcal{R}_c > 1$.

Proof: Consider the following Lyapunov function given by

$$Q = B_1 E_h + B_2 I_h + B_3 M + B_4 L + B_5 I_s + B_6 C,$$

where

$$\begin{aligned} B_1 &= \frac{A_1 \beta_s^* \kappa_s \theta_C \kappa_h \theta_M U^*}{M_0 \mu_C \mu_M T_1 T_2 T_3 T_4},\ B_2 = \frac{A_1 \beta_s \kappa_s \theta_C \theta_M U^*}{M_0 \mu_C \mu_M T_2 T_3 T_4},\ B_3 = \frac{A_1 \beta_s \kappa_s \theta_C U^*}{M_0 T_3 T_4 \mu_C \mu_M)}, \\ B_4 &= \frac{\kappa_s \theta_C \mathcal{R}_c}{\mu_C T_3 T_4},\ B_5 = \frac{\theta_C \mathcal{R}_c}{\mu_C T_4},\ B_6 = \frac{\mathcal{R}_c}{\mu_C}. \end{aligned}$$

The Lyapunov function has time derivative given by

$$\dot{Q} = B_1 \dot{E}_h + B_2 \dot{I}_h + B_3 \dot{M} + B_4 \dot{L} + B_5 \dot{I}_s + B_6 \dot{C}$$

Substituting the right hand side of model gives

$$\begin{aligned}\dot{Q} = \;& B_1(\lambda_s^* S_h^* - T_1 E_h^*) B_2(\kappa_h E_h^* - T_2 I_h^*) + B_3(\theta_M I_h^* - \mu_M M^*) \\ & -B_4(\lambda_s^* U * - T_3 L^*) + B_5(\kappa_s L^* - T_4 I_s^*) + B_6(\theta_C I_s^* - \mu_C C^*), \\ = \;& \frac{A_1 A_2 \beta_h \beta_s \kappa_s \theta_C \kappa_h \theta_M C^* U^* S_h^*}{M_0 T_1 T_2 T_3 T_4 \mu_c \mu_M (C_0 + \varepsilon C^*)} + \frac{A_1 \kappa_s \theta_C \mathcal{R}_c \beta_s M^* U^*}{T_3 T_4 \mu_C (M_0 + \varepsilon M^*)} - \frac{A_1 \beta_s \kappa_s \theta_C U^* M^*}{M_0 T_3 T_4 \mu_C} - \\ & \mathcal{R}_c C^*, \\ = \;& \mathcal{R}_c C^* \left[\frac{C_0 S_h^*}{S_h^*(C_0 + \varepsilon C^*} \mathcal{R}_c - 1\right] + \frac{A_1 \beta_s M^* \kappa_s \theta_C U^*}{T_3 T_4 \mu_C M_0} \left[\frac{M_0 U^*}{U^*(M_0 + \varepsilon M^*)} \mathcal{R}_c - 1\right], \\ \leq \;& \mathcal{R}_c C^* [\mathcal{R}_c - 1] + \frac{A_1 \beta_s M^* \kappa_s \theta_C U^*}{T_3 T_4 \mu_C M_0} [\mathcal{R}_c - 1]\end{aligned}$$

Hence, $\dot{Q} \leq 0$ whenever $\mathcal{R}_c \leq 1$ with $\dot{Q} = 0$ if and only if $I_h = I_s = M = C = 0$. Since $\dot{Q} \leq 0$ then Q is a Lyapunov function in the domain $\mathcal{D}$. From LaSalle's Invariance Principle (LaSalle and Lefschetz, 1976), it follows that:

$$(E_h(t), I_h(t), M(t), L_s(t), I_s(t), C(t)) \to (0,0,0,0,0,0) \text{ as } t \to \infty.$$

Consequently, every orbits of the equations of the model tends to the DFE of model, as $t \to \infty$ for $\mathcal{R}_c \leq 1$.

The result from the analysis showed that in the population, the DFE of the model is GAS whenever $\mathcal{R}_c \leq 1$ since a unique EEP exist for all time. Hence, Schistosomiasis will be eliminated from the population whenever $\mathcal{R}_c \leq 1$. It is worthy to note that, in this case, having $\mathcal{R}_c \leq 1$ is both a necessary and sufficient condition for effective Schistosomiasis control, irrespective of the starting sizes of the sub-populations.

3.6. Global Asymptotic Stability of the Endemic Equilibrium Point

In establishing the Global Asymptotic Stability (GAS) of the Endemic Equilibrium Point (EEP), we consider a particular case of the model when recovery for infected humans is negligible (i.e., $\gamma_h = 0$).

Let $\mathcal{D}_0$ be the stable manifold of the DFE of the model and be given as $\mathcal{D}_0 = \{(S_h, E_h, I_h, M, U, L, I_s, C) \in \mathcal{D}: E_h = I_h = M = L = I_s = C = 0\}$. The following result established the GAS of the EEP.

Theorem 3.3: The unique EEP, $\mathcal{E}_{1p}$ corresponding to the special case of the model (when $\gamma_h = 0$) is GAS in the domain $\mathcal{D} \setminus \mathcal{D}_0$ whenever $\mathcal{R}_c > 1$.

Proof: Consider the following nonlinear Lyapunov function

$$\begin{aligned}Q \quad = \;& S_h - S_h^{**} \ln\left(\frac{S_h}{S_h^{**}}\right) + E_h - E_h^{**} \ln\left(\frac{E_h}{E_h^{**}}\right) + R_1\left(I_h - I_h^{**} \ln\frac{I_h}{I_h^{**}}\right) \\ & + R_2\left(M - M^{**} \ln\frac{M}{M^{**}}\right) + U - U^{**} \ln\left(\frac{U}{U^{**}}\right) + L - L^{**} \ln\left(\frac{L}{L^{**}}\right) \\ & + R_3\left(I_s - I_s^{**} \ln\frac{I_s}{I_s^{**}}\right) + R_4\left(C - C^{**} \ln\frac{C}{C^{**}}\right),\end{aligned}$$

where: $R_1 = \frac{\bar{T}_1}{\kappa_h}$, $R_2 = \frac{T_1 T_2}{\kappa_h \theta_M}$, $R_3 = \frac{T_3}{\kappa_s}$, $R_4 = \frac{T_3 T_4}{\kappa_s \theta_C}$. and time derivatives of the Lyapunov functional Q given by

$$\begin{aligned}\dot{Q} = & \left(1 - \frac{S_h^{**}}{S_h}\right)\dot{S}_h + \left(1 - \frac{E_h^{**}}{E_h}\right)\dot{E}_h + R_1\left(1 - \frac{I_h^{**}}{I_h}\right)\dot{I}_h + R_2\left(1 - \frac{M^{**}}{M}\right)\dot{M} \\ & \left(1 - \frac{U^{**}}{U}\right)\dot{U} + \left(1 - \frac{L^{**}}{L}\right)\dot{L} + R_3\left(1 - \frac{I_s^{**}}{I_s}\right)\dot{I}_s + R_4\left(1 - \frac{C}{C^{**}}\right)\dot{C}.\end{aligned}$$

Substituting the right hand sides of the equations in the model corresponding to $\dot{S}_h, \dot{E}_h, \dot{I}_h, \dot{M}, \dot{U}, \dot{L}, \dot{I}_s, \dot{C}$ into the above and after several algebraic calculations gives:

$$\begin{aligned}\dot{Q} \quad &\leq \mu_h S_h^{**}\left(2-\frac{S_h^{**}}{S_h}-\frac{S_h}{S_h^{**}}\right)\\ &+\lambda^{**}S_h^{**}\left(5-\frac{S_h^{**}}{S_h}-\frac{S_h E_h\ **}{S_h^{**}E_h}-\frac{I_h^{**}E_h}{I_h E_h^{**}}-\frac{I_h M^{**}}{I_h^{**}M}-\frac{M}{M^{**}}\right)\\ &+\mu_s U^{**}\left(2-\frac{U^{**}}{U}-\frac{U}{U^{**}}\right)\\ &+\lambda_s^{**}U^{**}\left(5-\frac{U^{**}}{U}-\frac{LI_s^{**}}{L^{**}I_s}-\frac{C^{**}I_s}{I_s^{**}C^{**}}-\frac{UL^{**}}{U^{**}L}-\frac{C}{C^{**}}\right).\end{aligned}$$

Because the arithmetic mean is greater than the geometric mean, the following inequalities holds

$$\begin{aligned}&2-\frac{S_h^{**}}{S_h}-\frac{S_h}{S_h^{**}}\leq 0,\ 2-\frac{U^{**}}{U}-\frac{U}{U^{**}}\leq 0,\\ &5-\frac{S_h^{**}}{S_h}-\frac{S_h E_h\ **}{S_h^{**}E_h}-\frac{I_h^{**}E_h}{I_h E_h^{**}}-\frac{I_h M^{**}}{I_h^{**}M}-\frac{M}{M^{**}}\leq 0,\\ &5-\frac{U^{**}}{U}-\frac{LI_s^{**}}{L^{**}I_s}-\frac{C^{**}I_s}{I_s^{**}C^{**}}-\frac{UL^{**}}{U^{**}L}-\frac{C}{C^{**}}\leq 0\end{aligned}$$

Thus, $\dot{Q}\leq 0$ whenever $\mathcal{R}_c>1$. Since the relevant state variables in the equation of I_h is at the endemic steady state, these can be substituted into the equations for I_h in the model so that

$$I_h(t)\to I_h^{**}\ \text{ as }\ t\to\infty.$$

Since $\dot{Q}\leq 0$ then Q is a Lyapunov function in $\mathcal{D}\setminus\mathcal{D}_0$.

The result showed that in the Schistosomiasis population, whenever $\gamma_h=0$, the endemic steady state will be GAS whenever $\mathcal{R}_c>1$. Hence, Schistosomiasis will persist in the population regardless of the initial sizes of the subpopulation whenever $\mathcal{R}_c>1$.

4. Conclusion

In this paper, we formulated a novel deterministic mathematical model which investigated the impact of availability and efficacy of control on the transmission dynamics of Schistosomiasis in a population. The eight state variables of the model were shown to be non-negative and bounded for all time, t and that the trajectories generated by the availability and efficacy of control Schistosomiasis model with non-negative starting conditions will always be non-negative for all time, $t>0$. We also showed that the trajectories generated by the model are non-negatively invariant for all time, t. The DFE of the formulated availability and efficacy of control Schistosomiasis model was derived and shown to be LAS whenever the corresponding reproduction number is below one ($\mathcal{R}_c<1$), which suggested that Schistosomiasis can be eradicated from the entire population in the availability and efficacy of control model if the initial sizes of the sub-populations of the model lie in the basin of attraction of the DFE and that a little influx of infected humans with Schistosomiasis into a population where control measures are available would not generate large outbreaks, and unstable and if $\mathcal{R}_c>1$, which implied that Schistosomiasis will become endemic in the population. Analyzing the reproduction number ($\mathcal{R}_c$), by taking the partial derivative of $\mathcal{R}_c$ with respect to some parameters such as the efficacy and availability of controls that prevents transmission from humans to snails and snails to humans show that increasing the availability and efficacy of these controls will significantly reduce the burden of schistosomiasis in the population.The Endemic Equilibrium Point (EEP) was derived and shown to be unique whenever $\mathcal{R}_c^2>1$ and no EEP when $\mathcal{R}_c^2<1$. The DFE of the model was shown to be GAS in $\mathcal{D}$ whenever $\mathcal{R}_c\leq 1$ and not stable if $\mathcal{R}_c>1$. The Global Asymptotic Stability for the Endemic Equilibrium Point of the model was establish for a special case of the model whenever $\gamma_h=0$. The endemic steady state was shown to be GAS whenever $\mathcal{R}_c>1$. Hence, Schistosomiasis will persist in the population regardless of the initial sizes of the subpopulation whenever $\mathcal{R}_c>1$.

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