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THEORETICAL STUDY OF A TWO PATCH METAPOPULATION SCHISTOSOMIASIS MODEL

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Abstract

In this paper we present a deterministic nonlinear model which provides mathematical and epidemiological insights to the influence of metapopulation paradigm on Schistosomiasis transmission dynamics with variations in available control measures between two communities
The qualitative properties of the model are rigorously analyzed and thresholds for Schistosomiasis control and possible eradication was established. Local Asymptotic Stability (LAS) result of equilibrium is established.

***Keywords*:** Schistosomiasis, Metapopulation, Reproduction Number, Patch, Stability.

1.0 Introduction

Schistosomiasis is one of the most prevalent Neglected Tropical Diseases (NTDs) which are a diverse group of tropical ulcerations (infections) that are most common in very poor populations in emerging nations of Africa, Asia, and some part of the Americas. The NTDs affects over one billion people in the world over and about 90 percent of the entire disease load occurred in Africa.

Based on a 2011 report, it was estimated that about 243 million persons live in high risk locations of Schistosomiasis in about 78 countries of the world [1]. In 2012, the figure of those exposed to *S. Haematobium* were approximately 436 million persons in sub-Sahara Africa and about 112 million infected with the disease [2]. Also, a global report in 2012 revealed that about 393 million humans were at high risk to *S. Mansoni* with about 54 million already infected [1].

According to current world reported, Nigerian ranks number one in terms of incidence and prevalence of Schistosomiasis in the world [3]. Records show that an estimated 29 million Nigerians (with about 16 million being children) were infected with Schistosomiasis [4],[5].

Several Mathematical Models for Schistosomiasis transmission dynamics have been formulated since 1973 as seen in [6] - [25]. Although these models have indeed brought great understanding into schistosome dynamics, none of them has investigated the influence of the metapopulation paradigm on the transmission dynamics of Schistosomiasis to the best of our knowledge.

Introducing metapopulation to Schistosomiasis transmission dynamics and qualitatively analyzing the formulated model is the core of this paper. Typically, a patch may represent a city, a village, or a biological habitat. A full understanding of the effect of movement on the geographical spread of infection between patches can definitely improve disease control and prevention measures. Firstly, a single patch model is introduced and subsequently extended to incorporate another patch and which will have parameters for the short term migration of persons between the patches. The movement in this metapopulation model is restricted to the human subpopulation where the susceptible, latently infected and infected humans move between the patches. This movement follows the Lagrangian approach, as seen in the work of [26].

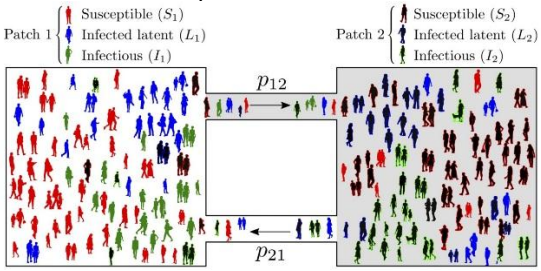

Fig 1.1. Schematic description of movement linking two patches [26].

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where p_{12} and p_{21} respectively denote movement of individuals from patch 1 to 2 and from patch 2 to 1. However, in this work we denote the movement from and to the susceptible, latently infected and infected compartments by η_{ij}, a_{ij} and b_{ij} respectively, where ij denote movement from patch i to j.

In this paper, we focus on formulating a non-linear coupled deterministic mathematical model which will describe the disease dynamics for Schistosomiasis in a 2- patch population setting and mathematically analyze the formulated model. This dynamic is studied in two patches, where movement is only allowed in all the human subpopulations in both patches. The forces of infections for our formulated model (for both human and snail populations in the patches) are modified by introducing control parameters that seeks to prevent the interactions between humans and Cercariae; between Snail and Miracidia on the other hand.

2.0 Formulating the Metapopulation Schistosomiasis model

In formulating this model, we make the assumption 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 [27], [28]. The total population for the metapopulation Schistosomiasis model is partitioned into sixteen non-overlapping compartments comprising of two patches; patches 1 and 2. The compartments for the models are: susceptible humans in patch i (S_{h_i}), latently infected or exposed humans in patch i (E_{h_i}), infected humans in patch i (I_{h_i}), Miracidia concentration in patch i (M_i), population of uninfected Snails in patch i (U_i), latently-infected Snails in patch i (L_i), patent infected Snails (not yet releasing Cercariae) in patch i (I_{s_i}) and free swimming Cercariae ready to enter human skin in patch i (C_i), where $i = 1,2$. From the above, the total human population at any time t, is given by

$$N(t) = \sum_{i=1}^{2} (S_{h_i}(t) + E_{h_i}(t) + I_{h_i}(t))$$

At any time t, influx of persons into the human populations (through birth or immigration) in both patches are assumed to occur only through recruitment into the susceptible human populations in both patches at a rate Λ_{h_i}. The human populations in both patches decreases as susceptible humans interact with Cercariae (larva) by their activities in water bodies where these larvae have been deposited by a specific species of Snails and this results in humans being infected with the parasite; Schistosome which results in Schistosomiasis infection at a rate λ_{h_i}; which is referred to as the force of infection. This is given by:

$$\lambda_{h_i} = \beta_{h_i} \frac{(I - \phi\xi_i)C_i}{C_0 + \varepsilon C_i},$$

and β_{h_i} is the rate at which free swimming Cercariae enters the bodies (where it complete its cycle of developing into full blown Schistosomiasis) of susceptible humans in patch i as a result of the activities of humans in a Cercariae laden water, where we assume that uninfected humans become tainted by coming in contact with Cercariae in infected waters [10]. The parameter ϕ is the efficacy of control measures in the human populations which is assumed to be the equal in the two patches and ξ_i is the availability of control measures in the human populations in patch i. The availability of these control measures in the patches is largely dependent on a number of factors like awareness of individuals about the existence of these measures, government interventions and commitments, accessing these measure and others. The parameter ϕ and ξ_i lies in the interval $0 \leq \phi \leq 1$ and $0 \leq \xi_i \leq 1$, implying that ϕ and ξ_i ranges from 0% to 100%. The parameter C_0 is the saturation constant for Cercariae in both patches and ε is the growth velocity constraint of Cercariae relative to increased infection in both patches. When the parasite is not present in both patches, the practical reaction of the susceptible populations in both patches to the pathogen is given by $\beta_{h_i} \frac{(I-\phi\xi_i)C_i S_{h_i}}{C_0+\varepsilon C_i}$. This reaction refers to the density change of the susceptible populations in both patches per unit time per pathogen as the uninfected populations density is altered [10]. We assume that recovery from Schistosomiasis does not confer any form of immunity; so recovered individuals becomes susceptible at the rate γ_{h_i} in patch i, which is of course an increase to the susceptible populations in both patches. The susceptible human populations in both patches are further depleted as a result of natural mortality rate, μ_h, and for the sake of simplicity and for the fact that epidemiological data are released country by country and does not vary from community to community within the same geographical location, we assume that this rate is the same for all human epidemiological classes in both patches since the patches are assumed to be relatively close (within the same geographical location) and that the migration only take place in the human classes. The susceptible human population in patch 1 is decreased at a rate η_{12} (migration of susceptible humans from patch 1 to 2), which is a plus to the susceptible population in patch 2. Also, the susceptible population in patch 1 is increased at a rate η_{21} (migration of susceptible humans from patch 2 to 1), which is a decrease to the susceptible population in patch 2. Thus we have

$$\frac{dS_{h_1}}{dt} = \Lambda_{h_1} - \lambda_{h_1}S_{h_1} + \gamma_{h_1}I_{h_1} - \mu_h S_{h_1} - \eta_{12}S_{h_1} + \eta_{21}S_{h_2}$$
and
$$\frac{dS_{h_2}}{dt} = \Lambda_{h_2} - \lambda_{h_2}S_{h_2} + \gamma_{h_2}I_{h_2} - \mu_h S_{h_2} - \eta_{21}S_{h_2} + \eta_{12}S_{h_1}$$
respectively for patches 1 and 2.

Newly infected humans with Schistosomiasis in each patch ($\lambda_{h_i}S_{h_i}$) are assumed to advance in their infection and subsequently progress to the class of latently infected humans (E_{h_i}) in their respective patches per unit time. As the infection grows stronger and the symptoms of Schistosomiasis become more glaring, the population of latently infected humans with Schistosomiasis is depleted at a rate κ_{h_i}, which is the progression rate from the latently infected class to infectious class in patch i. Since individuals are free to move within the population (within and between patches), the population of latently infected individuals in patch 1 is further reduced at the rate a_{12} (migration rate of latently infected humans from patch 1 to 2), and the population of latently infected individuals in patch 2 is increased at this rate. Furthermore, the population of latently infected individuals in patch 1 is increased at a rate a_{21} (migration rate of latently infected humans from patch 2 to 1), while the population of latently tainted individuals in patch 2 is reduced at this rate. The population of persons latently infected with Schistosomiasis in both patches is depleted due to natural death, (μ_h). The differential equations for the latently infected individuals for patches 1 and 2 are respectively given by:

$$\frac{dE_{h_1}}{dt} = \lambda_{h_1}S_{h_1} - (\kappa_{h_1} + \mu_h + a_{12})E_{h_1} + a_{21}E_{h_2}$$
and
$$\frac{dE_{h_2}}{dt} = \lambda_{h_2}S_{h_2} - (\kappa_{h_2} + \mu_h + a_{21})E_{h_2} + a_{12}E_{h_1}$$

The population of individuals infected with Schistosomiasis in patch i (I_{h_i}) is increased due to the influx of latently infected individuals in patch i (E_{h_i}) who have progressed in their infection at a rate κ_{h_i}. Slight or moderate movement of infected individuals from one patch to another in search of better medical facilities or better amenities or means of livelihood in that sick state can cause a depletion in one patch and an increase in the other. The parameter b_{12} is the rate at which infected individuals migrate from patch 1 to 2, which is a reduction in the infected class in patch 1 and an increase in the infected population in patch 2 while the infected human population in patch 2 is reduced at a rate b_{21} (movement rate of infected humans from patch 2 to 1), and the infected human population in patch 1 is increased at this rate. The infected humans population in patch i is again reduced for the following reasons: those treated of Schistosomiasis becomes susceptible and move to the susceptible class (since recovery does not guarantee immunity) at a rate γ_{h_i}; individuals die from the infection at a rate of δ_{h_i} (infection induced death) and natural mortality rate μ_h. Thus we have,

$$\frac{dI_{h_1}}{dt} = \kappa_{h_1}E_{h_1} - (\gamma_{h_1} + \delta_{h_1} + \mu_h + b_{12})I_{h_1} + b_{21}I_{h_2}$$
and
$$\frac{\mathrm{dI_{h_2}}}{\mathrm{dt}} = \kappa_{\mathrm{h_2}}\mathrm{E_{h_2}} - (\gamma_{\mathrm{h_2}} + \delta_{\mathrm{h_2}} + \mu_{\mathrm{h}} + \mathrm{b_{21}})\mathrm{I_{h_2}} + \mathrm{b_{12}I_{h_1}}$$
respectively for patches 1 and 2.

The pathogenesis of Schistosomiasis is incomplete without man's interaction with water bodies around the patches. There is no direct interaction between human and the intermediate host; the fresh water Snail. The Snail just serve as a host for the free swimming Miracidia larva to metamorphose into a another larva called Cercariae that can infect humans once humans come in contacted with Cercariae laden water bodies.

By the activities of infected individuals around water bodies in the patches, a fraction of eggs (*Schistosoma spp*) is released from the bodies of infected humans in both patches with the poo or urine and enters fresh water where they immediately hatch into liberated swimming thread-like larva called Miracidium at the rate θ_{M_i} in patch i. These Miracidia are supposed to find a suitable Snail species to infect, otherwise it dies naturally at the rate μ_M in both patches; this is a reduction to the class of Miracidia . Natural mortality for Miracidium is assumed to be same in both patches. Thus we have

$$\frac{dM_1}{dt} = \theta_{M_1}I_{h_1} - \mu_M M_1$$
and
$$\frac{dM_2}{dt} = \theta_{M_2}I_{h_2} - \mu_M M_2$$
respectively for patches 1 and 2.

Now, to model the dynamics of the intermediate host; the fresh water Snail. We assume that Snails infected by Miracidia, by reason of castration, do not reproduce and that periodic and climatic variations have no effect on the population of Snails

and contact arrangements (Chiyaka and Garira, 2009). We also assume that the Snails do not travel between patches and we presume that the entire Snail populace in the freshwater environment at time t is given by $N_s(t)$ and it is broken down into the jointly exclusive classes of Uninfected Snails in patch i ($U_i(t)$), latently-infected Snails in patch i ($L_i(t)$) and Patent infected Snails penetrated with Miracidia (not yet releasing Cercariae) in patch i ($I_{s_i}(t)$), where

$N_s(t) = U_i(t) + L_i(t) + I_{s_i}(t)$.

Fresh water Snails of the specific species are assumed to come into the entire Snail populations in both patches only through recruitment into the susceptible Snail population in patch i at the rate Λ_{s_i}. The force at which susceptible fresh water Snails are penetrated by Miracidia in patch i is given by

$$\lambda_{s_i} = \beta_{s_i} \frac{(I - \pi\nu_i)M_i}{M_0 + \varepsilon M_i},$$

where β_{s_i} is the rate at which Miracidia penetrates uninfected fresh water Snails in patch i. π is the efficacy of control measures in the aquatic environment which is intended to limit the metamorphosis of schistosome eggs into free swimming Miracidia larvae which is capable of infecting the fresh water Snail and the efficacy is assumed to be the same in both patches. The parameter ν_i is the availability of these control measures in the aquatic environment in patch i. π and ν_i lie in the interval $0 \leq \pi \leq 1$ and $0 \leq \nu_i \leq 1$, implying that π and ν_i range from 0% to 100%. The parameter M_0 is the constant of saturation for Miracidia in both patches and ε is the velocity growth limitation of Miracidia with the rise in infection in both patches. The Snail population decreases as susceptible Snails are penetrated by Miracidia (at the rate λ_{s_1}). The susceptible Snail population in patches 1 and 2 is further reduces due to the natural Snail mortality (μ_s). Hence the equations representing the dynamics of the uninfected/susceptible Snail populations in patches 1 and 2 are respectively given as

$$\frac{dU_1}{dt} = \Lambda_{s_1} - \lambda_{s_1}U_1 - \mu_s U_1$$

and

$$\frac{dU_2}{dt} = \Lambda_{s_2} - \lambda_{s_2}U_2 - \mu_s U_2$$

The population of latently infected Snails in patch i (L_i) is assumed to increase due to the inflow of the population of susceptible Snails penetrated by Miracidia at the rate λ_{s_i}. The population of latently infected Snail is reduced at a rate κ_{s_i}; the rate at which latently tainted Snails penetrated with Miracidia become patently infected Snails (not yet releasing Cercariae) and natural snail mortality (μ_s). Thus, we have

$$\frac{\mathrm{dL_1}}{\mathrm{dt}} = \lambda_{s_1}\mathrm{U_1} - (\kappa_{s_1} + \mu_s)\mathrm{L_1}$$

and

$$\frac{dL_2}{dt} = \lambda_{s_2}U_2 - (\kappa_{s_2} + \mu_s)L_2$$

respectively for patches 1 and 2.

The population of infected Snails in patch i (I_{s_i}) is assumed to increase due to the progression of latently infected Snails at a rate κ_{s_i}. The population of infected Snail in patch i is reduced at a rate δ_{s_i} ; which is the parasite induced death and natural Snail mortality (μ_s). Hence for patches 1 and 2 respectively, we have

$$\frac{dI_{s_1}}{dt} = \kappa_{s_1}L_1 - (\delta_{s_1} + \mu_s)I_{s_1}$$

and

$$\frac{dI_{s_2}}{dt} = \kappa_{s_2}L_2 - (\delta_{s_2} + \mu_s)I_{s_2}.$$

The concentration of free swimming Cercariae ($C_i(t)$) in patch i is increased by the released of free swimming Cercariae by infected Snails (ready to enter human skin) at the rate θ_{C_i} into the water bodies. We assume that the population decreases due to the natural Cercariae mortality (μ_C). Hence,

$$\frac{dC_1}{dt} = \theta_{C_1}I_{s_1} - \mu_C C_1$$

and

$$\frac{\mathrm{dC_2}}{\mathrm{dt}} = \theta_{\mathrm{C_2}}\mathrm{I_{s_2}} - \mu_{\mathrm{C}}\mathrm{C_2}$$

Based on the assumptions above, the formulated metapopulation model for Schistosomiasis for two patches, which is a system of sixteen non-linear ordinary differential equations is given in system (2.1). A schematic representation/description (which is a graphic description of the movement of individuals between different compartments, the snail dynamics as well as the different stages in the pathogenesis of Schistosomiasis) of the system is given in Figure 2.1. The state variables as well as the parameters in the mathematical formulation are given in Tables 2.1 and 2.2, respectively.

$$
\begin{aligned}
\frac{dS_{h_1}}{dt} &= \Lambda_{h_1} - \lambda_{h_1} S_{h_1} + \gamma_{h_1} I_{h_1} - \mu_h S_{h_1} - \eta_{12} S_{h_1} + \eta_{21} S_{h_2},\\
\frac{dE_{h_1}}{dt} &= \lambda_{h_1} S_{h_1} - (\kappa_{h_1} + \mu_h + a_{12}) E_{h_1} + a_{21} E_{h_2},\\
\frac{dI_{h_1}}{dt} &= \kappa_{h_1} E_{h_1} - (\gamma_{h_1} + \delta_{h_1} + \mu_h + b_{12}) I_{h_1} + b_{21} I_{h_2},\\
\frac{dM_1}{dt} &= \theta_{M_1} I_{h_1} - \mu_M M_1,\\
\frac{dU_1}{dt} &= \Lambda_{s_1} - \lambda_{s_1} U_1 - \mu_s U_1,\\
\frac{dL_1}{dt} &= \lambda_{s_1} U_1 - (\kappa_{s_1} + \mu_s) L_1,\\
\frac{dI_{s_1}}{dt} &= \kappa_{s_1} L_1 - (\delta_{s_1} + \mu_s) I_{s_1},\\
\frac{dC_1}{dt} &= \theta_{C_1} I_{s_1} - \mu_C C_1,\\
\frac{dS_{h_2}}{dt} &= \Lambda_{h_2} - \lambda_{h_2} S_{h_2} + \gamma_{h_2} I_{h_2} - \mu_h S_{h_2} - \eta_{21} S_{h_2} + \eta_{12} S_{h_1},\\
\frac{dE_{h_2}}{dt} &= \lambda_{h_2} S_{h_2} - (\kappa_{h_2} + \mu_h + a_{21}) E_{h_2} + a_{12} E_{h_1},\\
\frac{dI_{h_2}}{dt} &= \kappa_{h_2} E_{h_2} - (\gamma_{h_2} + \delta_{h_2} + \mu_h + b_{21}) I_{h_2} + b_{12} I_{h_1},\\
\frac{dM_2}{dt} &= \theta_{M_2} I_{h_2} - \mu_M M_2,\\
\frac{dU_2}{dt} &= \Lambda_{s_2} - \lambda_{s_2} U_2 - \mu_s U_2,\\
\frac{dL_2}{dt} &= \lambda_{s_2} U_2 - (\kappa_{s_2} + \mu_s) L_2,\\
\frac{dI_{s_2}}{dt} &= \kappa_{s_2} L_2 - (\delta_{s_2} + \mu_s) I_{s_2},\\
\frac{dC_2}{dt} &= \theta_{C_2} I_{s_2} - \mu_C \qquad (2.1)
\end{aligned}
$$

Figure 2.1. Schematic representation of a two patch metapopulation Schistosomiasis model.

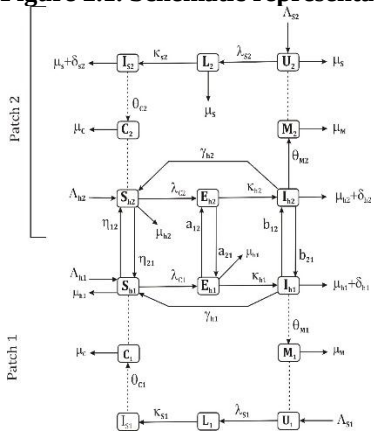

Table 2.1: Model Variables and Description

Variables	Description
$S_{h_i}(t)$	Susceptible individuals in patch i
$E_{h_i}(t)$	Latently tainted individuals in patch i
$I_{h_i}(t)$	Infected individuals in patch i
$M_i(t)$	Miracidia concentration in patch i
$U_i(t)$	Uninfected snails in patch i
$L_i(t)$	Latently-infected snails in patch i
$I_{s_i}(t)$	Tainted snails not yet releasing cercariae in patch i
$C_i(t)$	Free swimming Cercariae ready to enter human skin patch i.

Table 2.2: Model Parameters and Description

Parameter	Description
$\mu_k(k = h, M, s, C)$	Natural death rate for the kth sub population.
$\Lambda_{k_i}(k = h, s)$	Recruitment rate for the kth sub population in patch i.
$\beta_{k_i}(k = h, s)$	Cercariae and Miracidia infectious rate respectively for the kth sub population in patch i.
C_0	Saturation constant for Cercariae.
M_0	Saturation constant for Miracidia.
ε	Limitation of growth velocity of Cercariae and Miracidia.
$\kappa_{k_i}(k = h, s)$	Progression rate from latent class to infectious classes in the kth sub population in patch i.
$\delta_{k_i}(k = h, s)$	Disease and parasite induced death respectively for humans and snails the kth sub population in patch i.
γ_{h_i}	Recovery rate for humans in patch i
θ_{M_i}	Rate at which egg produced by adult Schistosome hatch and develop to free swimming Miracidia in patch i.
θ_{C_i}	Rate at which patent infected snails release cercariae in patch i.
η_{ij}	Movement rate of susceptible individuals from patch i to j.
a_{ij}	Rate at which latently infected individuals move from patch i to j.
b_{ij}	Movement rate of infected individuals from patch i to j.
ϕ	Efficacy of control in the human population.
ξ_i	Availability of control in the human population in patch i.
π	Efficacy of control in the aquatic (Snail) environment.
ν_i	Availability of control in the aquatic environment in patch i.

3.0 Fundamental properties of the Metapopulation Schistosomiasis 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 (2.1) are positively invariant for all time, t.

Theorem 3.1

Let the initial data of the metapopulation Schistosomiasis model be given as $X(0) \geq 0$, where: X(t)= (S_{h_1}(t), S_{h_2}(t), E_{h_1}(t), E_{h_2}(t), I_{h_1}(t), I_{h_2}(t), M_1(t), M_2(t), U_1(t), U_2(t), L_1(t), L_2(t), I_{s_1}(t), I_{s_2}(t), C_1(t), C_2(t)). Then the orbits X(t) of the metapopulation 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 (2,1), it follows

$$\frac{dS_{h_1}(t)}{dt} = \Lambda_{h_1} - (\lambda_{h_1} + \mu_h + \eta_{12})S_{h_1}(t) + \gamma_{h_1}I_{h_1}(t) + \eta_{21}S_{h_2}(t), \qquad (3.1)$$

Equation (3.1) can be rewritten as

$$[\frac{d}{dt} + (\lambda_{h_1} + \mu_h + \eta_{12})]S_{h_1}(t) \geq \Lambda_{h_1}$$

which implies

$$\frac{d}{dt}[S_{h_1}(t)\exp\{(\mu_h + \eta_{12})t + \int_0^t \lambda_{h_1}(\tau)d\tau\}] \geq \Lambda_{h_1}\exp\{(\mu_h + \eta_{12})t + \int_0^t \lambda_{h_1}(\tau)d\tau\}.$$

as a result,

$$S_{h_1}(t_1)\exp\{(\mu_h + \eta_{12})t_1 + \int_0^{t_1} \lambda_{h_1}(\tau)d\tau\} - S_{h_1}(0) \geq \int_0^{t_1} \Lambda_{h_1}[\exp\{(\mu_h + \eta_{12})y + \int_0^y \lambda_{h_1}(\tau)d\tau\}]dy,$$

hence,

$$S_{h_1}(t_1) \geq S_{h_1}(0)\exp[-(\mu_h + \eta_{12})t_1 - \int_0^{t_1} \lambda_{h_1}(\tau)d\tau] + [\exp\{-(\mu_h + \eta_{12})t_1 - \int_0^{t_1} \lambda_{h_1}(\tau)d\tau\}] \int_0^{t_1} \Lambda_{h_1}[\exp\{(\mu_h + \eta_{12})y + \int_0^y \lambda_{h_1}(\tau)d\tau\}]dy \geq 0.$$

Hence $S_{h_1}(t) \geq 0, \ \forall \ t > 0$.
Considering equation 2 of model (2.1)
$\frac{dE_{h_1}}{dt} = \lambda_{h_1} S_{h_1} - (\kappa_{h_1} + \mu_h + a_{12})E_{h_1} + a_{21}E_{h_2}$.
It follows from that
$\frac{dE_{h_1}}{dt} \geq -(\kappa_{h_1} + \mu_h + a_{12})E_{h_1}$
Integrating with respect to t in $[0, t_1]$, yields
$E_{h_1}(t_1) \geq E_{h_1}(0)\exp\{-(\kappa_{h_1} + \mu_h + a_{12})t_1\} > 0$.
Hence $E_{h_1}(t) > 0$ for all $t > 0$.
Consider the third equation of model (2.1) which is given as
$\frac{dI_{h_1}}{dt} = \kappa_{h_1} E_{h_1} - (\gamma_{h_1} + \delta_{h_1} + \mu_h + b_{12})I_{h_1} + b_{21}I_{h_2}$.
It follows from that
$\frac{dI_{h_1}}{dt} \geq -(\gamma_{h_1} + \delta_{h_1} + \mu_h + b_{12})I_{h_1}$,
Integrating with respect to t in $[0, t_1]$, yields
$I_{h_1}(t_1) \geq I_{h_1}(0)\exp\{-(\gamma_{h_1} + \delta_{h_1} + \mu_h + b_{12})t_1\} > 0$.
Hence $I_{h_1}(t) > 0$ for all $t > 0$.
Consider equation 4 of model (2.1) given as
$\frac{dM_1}{dt} = \theta_{M_1} I_{h_1} - \mu_M M_1$.
It follows from that
$\frac{dM_1}{dt} \geq -\mu_M M_1$,
Integrating with respect to t in $[0, t_1]$, yields
$M_1(t_1) \geq M_1(0)\exp\{-\mu_M t_1\} > 0$.
Hence $M_1(t) > 0$ for all $t > 0$.
Consider equation 5 of model (2.1) given as
$\frac{dU_1}{dt} = \Lambda_{s_1} - (\lambda_{s_1} + \mu_s)U_1$.
It follows from that
$\frac{dU_1}{dt} \geq -(\lambda_{s_1} + \mu_s)U_1$.
Integrating with respect to t in $[0, t_1]$, yields
$U_1(t_1) \geq U_1(0)\exp\{-(\lambda_{s_1} + \mu_s)t_1\} > 0$.
Hence $U_1(t) > 0$ for all $t > 0$.
Consider equation 6 of model (2.1) given as
$\frac{dL_1}{dt} = \lambda_{s_1} U_1 - (\kappa_{s_1} + \mu_s)L_1$.
It follows from that
$\frac{dL_1}{dt} \geq -(\kappa_{s_1} + \mu_s)L_1$.
Integrating with respect to t in $[0, t_1]$, yields
$L_1(t_1) \geq L_1(0)\exp\{-(\kappa_{s_1} + \mu_s)t_1\} > 0$.
Hence $L_1(t) > 0$ for all $t > 0$.
Consider equation 7 of model (2.1) given as
$\frac{dI_{s_1}}{dt} = \kappa_{s_1} L_1 - (\delta_{s_1} + \mu_s)I_{s_1}$.
It follows from that
$\frac{dI_{s_1}}{dt} \geq -(\delta_{s_1} + \mu_s)I_{s_1}$.
Integrating with respect to t in $[0, t_1]$, yields
$I_{s_1}(t_1) \geq I_{h_1}(0)\exp\{-(\delta_{s_1} + \mu_s)t_1\} > 0$.
Hence $I_{s_1}(t) > 0$ for all $t > 0$.

Consider equation 8 of model (2.1) given as

$\frac{dC_1}{dt} = \theta_{C_1} I_{s_1} - \mu_C C_1.$

It follows from that

$\frac{dC_1}{dt} \geq -\mu_C C_1.$

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

$C_1(t_1) \geq C_1(0)\exp\{-\mu_C t_1\} > 0.$

Hence $C_1(t) > 0$ for all $t > 0$.

Consider equation 9 of model (2.1) given as

$\frac{dS_{h_2}}{dt} = \Lambda_{h_2} - (\lambda_{h_2} + \mu_h + \eta_{21})S_{h_2} + \gamma_{h_2} I_{h_2} + \eta_{12} S_{h_1}.$ (3.2)

Equation (3.2) can be rewritten as

$[\frac{d}{dt} + (\lambda_{h_2} + \mu_h + \eta_{21})]S_{h_2}(t) \geq \Lambda_{h_2}$

Which follows that

$\frac{d}{dt}[S_{h_2}(t)\exp\{(\mu_h + \eta_{21})t + \int_0^t \lambda_{h_2}(\tau)d\tau\}] \geq \Lambda_{h_2}\exp\{(\mu_h + \eta_{21})t + \int_0^t \lambda_{h_2}(\tau)d\tau\}$. (3.3)

Equation (3.3) results to

$S_{h_2}(t_1)\exp\{(\mu_h + \eta_{21})t_1 + \int_0^{t_1} \lambda_{h_2}(\tau)d\tau\} - S_{h_2}(0) \geq \int_0^{t_1} \Lambda_{h_2}[\exp\{(\mu_h + \eta_{21})y + \int_0^y \lambda_{h_2}(\tau)d\tau\}]dy,$ (3.4)

Simplifying equation (3.4) gives

$$S_{h_2}(t_1) \geq S_{h_2}(0)\exp[-(\mu_h + \eta_{21})t_1 - \int_0^{t_1} \lambda_{h_2}(\tau)d\tau] + [\exp\{-(\mu_h + \eta_{21})t_1 - \int_0^{t_1} \lambda_{h_2}(\tau)d\tau\}] \int_0^{t_1} \Lambda_{h_2}[\exp\{(\mu_h + \eta_{21})y + \int_0^y \lambda_{h_2}(\tau)d\tau\}]dy \geq 0.$$

Hence $S_{h_2}(t) > 0$ for all $t > 0$.

Consider the tenth equation of model (2.1) , given below as

$\frac{dE_{h_2}}{dt} = \lambda_{h_2} S_{h_2} - (\kappa_{h_2} + \mu_h + a_{21})E_{h_2} + a_{12} E_{h_1}.$

It follows from that

$\frac{dE_{h_2}}{dt} \geq -(\kappa_{h_2} + \mu_h + a_{21})E_{h_2}.$

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

$E_{h_2}(t_1) \geq E_{h_2}(0)\exp\{-(\kappa_{h_2} + \mu_h + a_{21})t_1\} > 0.$

Hence $E_{h_2}(t) > 0$ for all $t > 0$.

Consider the eleventh equation of model (2.1), given below as

$\frac{dI_{h_2}}{dt} = \kappa_{h_2} E_{h_2} - (\gamma_{h_2} + \delta_{h_2} + \mu_h + b_{21})I_{h_2} + b_{12} I_{h_1}.$

It follows from that

$\frac{dI_{h_2}}{dt} \geq -(\gamma_{h_2} + \delta_{h_2} + \mu_h + b_{21})I_{h_2}.$

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

$I_{h_2}(t_1) \geq I_{h_2}(0)\exp\{-(\gamma_{h_2} + \delta_{h_2} + \mu_h + b_{21})t_1\} > 0.$

Hence $I_{h_2}(t) > 0$ for all $t > 0$.

Consider the twelfth equation of model (2.1), given below as

$\frac{dM_2}{dt} = \theta_{M_2} I_{h_2} - \mu_M M_2.$

It follows from that

$\frac{dM_2}{dt} \geq -\mu_M M_2.$

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

$M_2(t_1) \geq M_2(0)\exp\{-\mu_M t_1\} > 0.$

Hence $M_2(t) > 0$ for all $t > 0$.

Consider the thirteenth equation of model (2.1), given below as

$\frac{dU_2}{dt} = \Lambda_{s_2} - (\lambda_{s_2} + \mu_s)U_2.$

It follows from that
$\frac{dU_2}{dt} \geq -(\lambda_{s_2} + \mu_s)U_2.$
Integrating with respect to t in $[0, t_1]$, yields
$U_2(t_1) \geq U_2(0)\exp\{-(\lambda_{s_2} + \mu_s)t_1\} > 0.$
Hence $U_2(t) > 0$ for all $t > 0$.
Consider the fourteenth equation of model (2.1), given below as
$\frac{dL_2}{dt} = \lambda_{s_2}U_2 - (\kappa_{s_2} + \mu_s)L_2.$
It follows from that
$\frac{dL_2}{dt} \geq -(\kappa_{s_2} + \mu_s)L_2.$
Integrating with respect to t in $[0, t_1]$, yields
$L_2(t_1) \geq L_2(0)\exp\{-(\kappa_{s_2} + \mu_s)t_1\} > 0.$
Hence $L_2(t) > 0$ for all $t > 0$.
Consider the fifteenth equation of model (2.1), given below as
$\frac{dI_{s_2}}{dt} = \kappa_{s_2}L_2 - (\delta_{s_2} + \mu_s)I_{s_2}.$
It follows from that
$\frac{\mathrm{dI}_{s_2}}{\mathrm{dt}} \geq -(\delta_{s_2} + \mu_s)\mathrm{I}_{s_2}.$
Integrating with respect to t in $[0, t_1]$, yields
$I_{s_2}(t_1) \geq I_{s_2}(0)\exp\{-(\delta_{s_2} + \mu_s)t_1\} > 0.$
Hence $I_{s_2}(t) > 0$ for all $t > 0$.
And finally last equation of model (2.1), given as
$\frac{dC_2}{dt} = \theta_{C_2}I_{s_2} - \mu_C C_2.$
It follows from that
$\frac{dC_2}{dt} \geq -\mu_C C_2.$
Integrating with respect to t in $[0, t_1]$, yields
$C_2(t_1) \geq C_2(0)\exp\{-\mu_C t_1\} > 0.$
Hence $C_2(t) > 0$ for all $t > 0$.
From the above, we have thus shown that for the metapopulation Schistosomiasis model (2.1), $X(t) \geq 0$, where X(t)= (S_{h_1}(t), S_{h_2}(t), E_{h_1}(t),\\ E_{h_2}(t), I_{h_1}(t), I_{h_2}(t), M_1(t), M_2(t), U_1(t), U_2(t), L_1(t), L_2(t), I_{s_1}(t), I_{s_2}(t), C_1(t), C_2(t)). Hence the trajectories X(t) generated by the metapopulation 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 (2.1)

Theorem 3.2

Let S_{h_1}(t), S_{h_2}(t), E_{h_1}(t), E_{h_2}(t), I_{h_1}(t), I_{h_2}(t), M_1(t), M_2(t), U_1(t) ,U_2(t), L_1(t), L_2(t), I_{s_1}(t), I_{s_2}(t), C_1(t), C_2(t) be trajectories of the system with initial conditions and the biological feasible region given by the set $\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_M \times \mathcal{D}_S \times \mathcal{D}_C \subset \mathbb{R}_+^6 \times \mathbb{R}_+^2 \times \mathbb{R}_+^6 \times \mathbb{R}_+^2 \subset \mathbb{R}_+^{16}$ where:

$\mathcal{D}_h = \{(S_{h_1}, S_{h_2}, E_{h_1}, E_{h_2}, I_{h_1}, I_{h_2}) \in \mathbb{R}_+^6 : N_h \leq \frac{\Lambda_h}{\mu_h}\}$

$\mathcal{D}_M = \{(M_1, M_2) \in \mathbb{R}_+^2 : M \leq \frac{\theta_M \Lambda_h}{\mu_M \mu_h}\}$

$\mathcal{D}_S = \{(U_1, U_2, L_1, L_2, I_{s_1}, I_{s_2}) \in \mathbb{R}_+^6 : N_S \leq \frac{\Lambda_s}{\mu_s}\}$

$\mathcal{D}_C = \{(C_1, C_2) \in \mathbb{R}_+^2 : C \leq \frac{\theta_C \Lambda_s}{\mu_C \mu_s}\}$

is positively-invariant and attracts all the non-negative trajectories of model (2.1).

Proof

To determine the bound for the human subpopulation, we add up the right hand side of the vector field for the human population in both patches in model (2.1), which is the rate of change of the total population described by the system and it is given by:

$\frac{dN_h}{dt} = \Lambda_{h_1} + \Lambda_{h_2} - \mu_h N - (\delta_{h_1} I_{h_1} + \delta_{h_2} I_{h_2})$. (3.5)

From (3.5), we have

$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_h I_h$. (3.6)

From (3.6), it follows that

$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h$. (3.7)

Equation (3.7) is a linear first order ODE with integrating factor given as $e^{\mu_h t}$. Thus, we obtain

$\frac{dN_h}{dt} e^{\mu_h t} + \mu_h N_h e^{\mu_h t} \leq \Lambda_h e^{\mu_h t}$. (3.8)

Equation (3.8) 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 $N_h(t)$ from , 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 system . 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 $\mathbb{R}^6_+$.

To determine the bound for the concentration of the Miracidia in both patches, we add up the right hand side of the vector field M_1 and M_2 in and it yields

$\frac{dM}{dt} = \theta_{M_1} I_{h_1} + \theta_{M_2} I_{h_2} - (M_1 + M_2)\mu_M$. (3.9)

From (3.9) , we have

$\frac{dM}{dt} = \theta_M I_h - \mu_M M$. (3.10)

From (3.10), it follows that,

$\frac{dM}{dt} \leq \theta_M \frac{\Lambda_h}{\mu_h} - \mu_M M$. (3.11)

since $N_h = S_h + E_h + I_h \leq \frac{\Lambda_h}{\mu_h} \Rightarrow I_h \leq \frac{\Lambda_h}{\mu_h}$.

Equation (3.11) is a linear equation with integrating factor given as $e^{\mu_M t}$. Thus, we obtain

$\frac{dM}{dt} e^{\mu_M t} + \mu_M M e^{\mu_M t} \leq \theta_M \frac{\Lambda_h}{\mu_h} e^{\mu_M t}$. (3.12)

We can rewrite equation (3.12) 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 $M(t)$ from , 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 system . 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 $\mathbb{R}^2_+$.

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

$\frac{dN_s}{dt} = \Lambda_{s_1} + \Lambda_{s_2} + (U + L + I_s)\mu_s - \delta_{s_1} I_{s_1} - \delta_{s_2} I_{s_2}$. (3.13)

From (3.13), we have

$$\frac{dN_s}{dt} = \Lambda_s - \mu_h N_s - \delta_s I_s. \quad (3.14)$$

From (3.14), it follows that

$$\frac{dN_s}{dt} \leq \Lambda_s - \mu_s N_s. \quad (3.15)$$

Equation (3.15) is a linear equation with integrating factor given as $e^{\mu_s t}$. Thus, we obtain

$$\frac{dN_s}{dt} e^{\mu_s t} + \mu_s N_s e^{\mu_s t} \leq \Lambda_s e^{\mu_s t}. \quad (3.16)$$

Equation (3.16) 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 $N_s(t)$ from , 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 system . 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 $\mathbb{R}^6_+$.

To determine the bound for the concentration of the Cercariae in both patches, we add up the right hand side of the vector field C_1 and C_2 in and it yields

$$\frac{dC}{dt} = \theta_{C_1} I_{s_1} + \theta_{C_2} I_{s_2} - (C_1 + C_2)\mu_C. \quad (3.17)$$

From (3.17) , we have

$$\frac{dC}{dt} = \theta_C I_s - \mu_C C. \quad (3.18)$$

From (3.18), it follows that,

$$\frac{dC}{dt} \leq \theta_C \frac{\Lambda_s}{\mu_s} - \mu_C C. \quad (3.19)$$

since $N_s = U + L + I_s \leq \frac{\Lambda_s}{\mu_s} \Rightarrow I_s \leq \frac{\Lambda_s}{\mu_s}$.

Equation (3.19) is a linear equation with integrating factor given as $e^{\mu_C t}$. Thus, we obtain

$$\frac{dC}{dt} e^{\mu_C t} + \mu_C C e^{\mu_C t} \leq \theta_C \frac{\Lambda_s}{\mu_C} e^{\mu_C t}. \quad (3.20)$$

Equation (3.20) 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 $C(t)$ from , 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 system . 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 $\mathbb{R}^2_+$.

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 $\mathcal{D}$.

$$\mathcal{D} = \begin{cases} (S_{h_1}, S_{h_2}, E_{h_1}, E_{h_2}, I_{h_1}, I_{h_2}) \in \mathbb{R}^6_+ : N_h \leq \dfrac{\Lambda_h}{\mu_h} \\ (M_1, M_2) \in \mathbb{R}^2_+ : M \leq \dfrac{\theta_M \Lambda_h}{\mu_M \mu_h} \\ (U_1, U_2, L_1, L_2, I_{s_1}, I_{s_2}) \in \mathbb{R}^6_+ : N_S \leq \dfrac{\Lambda_s}{\mu_s} \\ (C_1, C_2) \in \mathbb{R}^2_+ : C \leq \dfrac{\theta_C \Lambda_s}{\mu_C \mu_s} \end{cases}$$

It implies that the right hand side of the system is smooth, hence there exist a unique solution to the initial value problem, and solution exists for all time. Hence the system 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 system in $\mathcal{D}$.

4.0. Local Asymptotic Stability of the Disease Free Equilibrium

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

$$\mathcal{E}_0 = (S_{h_1}^0, S_{h_2}^0, E_{h_1}^0, E_{h_2}^0, I_{h_1}^0, I_{h_2}^0, M_1^0, M_2^0, U_1^0, U_2^0, L_1^0, L_2^0, I_{s_1}, I_{s_2}, C_1^0, C_2^0) =$$
$$\left(\frac{P_6\Lambda_{h_1} + \eta_{21}\Lambda_{h_2}}{P_1P_6 - \eta_{12}\eta_{21}}, \frac{\eta_{12}\Lambda_{h_1} + P_1\Lambda_{h_2}}{P_1P_6 - \eta_{12}\eta_{21}}, 0,0,0,0,0,0, \frac{\Lambda_{s_1}}{\mu_s}, \frac{\Lambda_{s_2}}{\mu_s}, 0,0,0,0,0,0\right).$$

where:

$$P_1 = (\eta_{12} + \mu_h), \qquad P_2 = (\kappa_{h_1} + \mu_h + a_{12}), \qquad P_3 = (\gamma_{h_1} + \delta_{h_1} + \mu_h + b_{12}),$$

$$P_4 = (\kappa_{s_1} + \mu_s), \qquad P_5 = (\delta_{s_1} + \mu_s), \qquad P_6 = (\eta_{21} + \mu_h).$$

The method of next generation matrix operator proposed by [29] is used to investigate whether the DFE of the system is Locally Asymptotically Stable (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

$$\mathbf{F} = \begin{pmatrix}
0 & 0 & 0 & 0 & \frac{A_1\beta_{s_1}\Lambda_{s_1}}{M_0\mu_s} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & \frac{A_2\beta_{h_1}P_{11}}{C_0P_{13}} & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{A_3\beta_{s_2}\Lambda_{s_2}}{M_0\mu_s} & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{A_4\beta_{h_2}P_{12}}{C_0P_{13}} & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0
\end{pmatrix}$$

and

$$\mathbf{V} = \begin{pmatrix}
P_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & P_2 & 0 & 0 & 0 & 0 & -a_{21} & 0 & 0 & 0 & 0 & 0 \\
-\kappa_{s_1} & 0 & P_5 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & -\kappa_{h_1} & 0 & P_3 & 0 & 0 & 0 & 0 & 0 & -b_{21} & 0 & 0 \\
0 & 0 & 0 & -\theta_{M_1} & \mu_M & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & -\theta_{C_1} & 0 & 0 & \mu_s & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & P_9 & 0 & 0 & 0 & 0 & 0 \\
0 & -a_{12} & 0 & 0 & 0 & 0 & 0 & P_7 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & -\kappa_{s_2} & 0 & P_{10} & 0 & 0 & 0 \\
0 & 0 & 0 & -b_{12} & 0 & 0 & 0 & -\kappa_{h_2} & 0 & P_8 & 0 & -b_{12} \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\theta_{M_2} & \mu_M & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\theta_{C_2} & 0 & 0 & \mu_s
\end{pmatrix}$$

The reproduction number, $\mathcal{R}_0 = \rho(FV^{-1})$, with $\rho(\cdot)$ being the spectral radius (largest eigenvalue) associated with matrix FV^{-1}, is given by

$$\mathcal{R}_0^2 = \frac{1}{2}[h_1 + \sqrt{h_1^2 - 4h_2}],$$

where:

$$h_1 = \frac{H_1 + H_2}{H_3}$$
$$H_1 = A_1A_2(P_7P_8\kappa_{h_1} + a_{12}b_{21}\kappa_{h_2})P_9P_{10}P_{11}\beta_{h_1}\beta_{s_1}\theta_{M_1}\theta_{C_1}\Lambda_{s_1}\kappa_{s_1}$$
$$H_2 = A_3A_4P_4P_5P_{12}(a_{21}b_{12}\kappa_{h_1} + P_2P_3\kappa_{h_2})\beta_{h_2}\beta_{s_2}\theta_{M_2}\theta_{C_2}\Lambda_{s_2}\kappa_{s_2}$$

$H_3 = (P_2P_7 - a_{12}a_{21})(P_3P_8 - b12b_{21})P_4P_5P_9P_{10}P_{13}^2C_0^2M_0^2\mu_C\mu_M\mu_s,$
$h_2 = \frac{H_4}{H_5}$
$H_4 = A_1A_2A_3A_4\beta_{h_1}\beta_{s_1}\beta_{h_2}\beta_{s_2}\theta_{M_1}\theta_{C_1}\theta_{M_2}\theta_{C_2}\Lambda_{s_1}\Lambda_{s_2}\kappa_{h_1}\kappa_{h_2}\kappa_{s_1}\kappa_{s_2}$
$H_5 = (P_2P_7 - a_{12}a_{21})(P_3P_8 - b12b_{21})P_4P_5P_9P_{10}P_{13}^2C_0^2M_0^2\mu_C^2\mu_M^2\mu_s^2$ $A_1 = (1-\pi\nu_1)$, $A_2 = (1-\phi\xi_1)$, $A_3 = (1-\pi\nu_2)$, $A_4 = (1-\phi\xi_2)$,
$P_7 = (\kappa_{h_2} + \mu_h + a_{21}),$
$P_8 = (\gamma_{h_2} + \delta_{h_2} + \mu_h + b_{21}),$ $P_9 = (\kappa_{s_2} + \mu_s),$ $P_{10} = (\delta_{s_2} + \mu_s),$
$P_{13} = (P_1P_6 - \eta_{12}\eta_{21}) = (\eta_{12}\mu_h + \eta_{21}\mu_h + \mu_h^2) > 0,$
$P_{14} = (P_2P_7 - a_{12}a_{21}) > 0$

The result in Lemma (4.1) follows from Theorem 2 of [29].
Lemma 4.1
The DFE of the model , $\mathcal{E}_0$ is LAS in $\mathcal{D}$ whenever $\mathcal{R}_0^2 < 1$, and it is not stable when $\mathcal{R}_0^2 > 1$.
The threshold number, $\mathcal{R}_0^2$, is a very important epidemiological concept, which is a measure of the mean number of secondary cases engendered by one infected individual in a totally exposed populace [27]. Epidemiologically, Lemma (4.1) implies that whenever $\mathcal{R}_0^2 < 1$, then the initial sizes of the subpopulations of the model lie in the basin of attraction of the DFE and that a little influx of infected humans with Schistosomiasis into the population in both patches where control is available and humans are free to move without restriction between patches would not generate large outbreaks and Schistosomiasis will become endemic in the population in both patches if $\mathcal{R}_0^2 > 1$.

5.0. Existence of endemic steady states

We examine the existence of Endemic Equilibrium Points (EEP) for a special case of the model when $\gamma_{h_1} = \gamma_{h_2} = 0$.
Consider the existence of an EEP associated with a special case of the system (with negligible recovery rate in patches 1 and 2, i.e., $\gamma_{h_1} = \gamma_{h_2} = 0$). Hence, substituting $\gamma_{h_1} = \gamma_{h_2} = 0$ into the model yields:

$\frac{dS_{h_1}}{dt} = \Lambda_{h_1} - \lambda_{h_1}S_{h_1} + \gamma_{h_1}I_{h_1} - \mu_hS_{h_1} - \eta_{12}S_{h_1} + \eta_{21}S_{h_2},$
$\frac{dE_{h_1}}{dt} = \lambda_{h_1}S_{h_1} - (\kappa_{h_1} + \mu_h + a_{12})E_{h_1} + a_{21}E_{h_2},$
$\frac{dI_{h_1}}{dt} = \kappa_{h_1}E_{h_1} - (\delta_{h_1} + \mu_h + b_{12})I_{h_1} + b_{21}I_{h_2},$
$\frac{dM_1}{dt} = \theta_{M_1}I_{h_1} - \mu_MM_1,$
$\frac{dU_1}{dt} = \Lambda_{s_1} - \lambda_{s_1}U_1 - \mu_sU_1,$
$\frac{dL_1}{dt} = \lambda_{s_1}U_1 - (\kappa_{s_1} + \mu_s)L_1,$
$\frac{dI_{s_1}}{dt} = \kappa_{s_1}L_1 - (\delta_{s_1} + \mu_s)I_{s_1},$
$\frac{dC_1}{dt} = \theta_{C_1}I_{s_1} - \mu_CC_1,$
$\frac{dS_{h_2}}{dt} = \Lambda_{h_2} - \lambda_{h_2}S_{h_2} + \gamma_{h_2}I_{h_2} - \mu_hS_{h_2} - \eta_{21}S_{h_2} + \eta_{12}S_{h_1},$
$\frac{dE_{h_2}}{dt} = \lambda_{h_2}S_{h_2} - (\kappa_{h_2} + \mu_h + a_{21})E_{h_2} + a_{12}E_{h_1},$
$\frac{dI_{h_2}}{dt} = \kappa_{h_2}E_{h_2} - (\delta_{h_2} + \mu_h + b_{21})I_{h_2} + b_{12}I_{h_1},$
$\frac{dM_2}{dt} = \theta_{M_2}I_{h_2} - \mu_MM_2,$
$\frac{dU_2}{dt} = \Lambda_{s_2} - \lambda_{s_2}U_2 - \mu_sU_2,$
$\frac{dL_2}{dt} = \lambda_{s_2}U_2 - (\kappa_{s_2} + \mu_s)L_2,$
$\frac{dI_{s_2}}{dt} = \kappa_{s_2}L_2 - (\delta_{s_2} + \mu_s)I_{s_2},$
$\frac{dC_2}{dt} = \theta_{C_2}I_{s_2} - \mu_CC_2.$

with $\lambda_{h_i} = \beta_{h_i}\frac{(I-\phi\xi_i)C_i}{C_0+\varepsilon C_i}$ and $\lambda_{s_i} = \beta_{s_i}\frac{(I-\pi\nu_i)M_i}{M_0+\varepsilon M_i}$ being the forces of infection for human and snail subpopulations.

Let
$\mathcal{E}_1 = (S_{h_1}^{**}, S_{h_2}^{**}, E_{h_1}^{**}, E_{h_2}^{**}, I_{h_1}^{**}, I_{h_2}^{**}, M_1^{**}, M_2^{**}, U_1^{**}, U_2^{**}, L_1^{**}, L_2^{**}, I_{s_1}^{**}, I_{s_2}^{**}, C_1^{**}, C_2^{**}$.
The state variables in the system are solved for, as functions of the forces of infection for patch i (where i =1, 2), i.e., $\lambda_{h_i}^{**} = \beta_{h_i} \frac{(I-\phi\xi_i)C_i^{**}}{C_0+\varepsilon C_i^{**}}$ and $\lambda_{s_i}^{**} = \beta_{s_i} \frac{(I-\pi\nu_i)M_i^{**}}{M_0+\varepsilon M_i^{**}}$ at the endemic steady state, by equating the right-hand side of the equations in system to zero. This yields

$$S_{h_1}^{**} = \frac{\Lambda_{h_1}(\lambda_{C_2}^{**} + P_6) + \eta_{21}\Lambda_{h_2}}{(\lambda_{C_1}^{**}\lambda_{C_2}^{**} + P_6\lambda_{C_1}^{**} + P_1\lambda_{C_2}^{**} + G_1)},$$
$$S_{h_2}^{**} = \frac{\Lambda_{h_2}(\lambda_{C_1}^{**} + P_1) + \eta_{12}\Lambda_{h_1}^{**}}{(\lambda_{C_1}^{**}\lambda_{C_2}^{**} + P_6\lambda_{C_1}^{**} + P_1\lambda_{C_2}^{**} + G_1)},$$
$$E_{h_1}^{**} = \frac{G_3\lambda_{C_1}^{**}\lambda_{C_2}^{**} + G_4\lambda_{C_1}^{**} + G_5\lambda_{C_2}^{**}}{G_2(\lambda_{C_1}^{**}\lambda_{C_2}^{**} + P_6\lambda_{C_1}^{**} + P_1\lambda_{C_2}^{**} + G_1)},$$
$$E_{h_2}^{**} = \frac{G_6\lambda_{C_1}^{**}\lambda_{C_2}^{**} + G_7\lambda_{C_1)^{**}} + G_8\lambda_{C_2}^{**}}{G_2(\lambda_{C_1}^{**}\lambda_{C_2}^{**} + P_6\lambda_{C_1}^{**} + P_1\lambda_{C_2}^{**} + G_1)},$$
$$I_{h_1}^{**} = \frac{G_{10}\lambda_{C_1}^{**}\lambda_{C_2}^{**} + G_{11}\lambda_{C_1}^{**} + G_{12}\lambda_{C_2}^{**}}{G_2G_9(\lambda_{C_1}^{**}\lambda_{C_2}^{**} + P_6\lambda_{C_1}^{**} + P_1\lambda_{C_2}^{**} + G_1)},$$
$$I_{h_2}^{**} = \frac{G_{13}\lambda_{C_1}^{**}\lambda_{C_2}^{**} + G_{14}\lambda_{C_1}^{**} + G_{15}\lambda_{C_2}^{**}}{G_2G_9(\lambda_{C_1}^{**}\lambda_{C_2}^{**} + P_6\lambda_{C_1}^{**} + P_1\lambda_{C_2}^{**} + G_1)}.$$
$$M_1^{**} = \frac{\theta_{M_1}(G_{10}\lambda_{C_1}^{**}\lambda_{C_2}^{**} + G_{11}\lambda_{C_1}^{**} + G_{12}\lambda_{C_2}^{**})}{\mu_M G_2G_9(\lambda_{C_1}^{**}\lambda_{C_2}^{**} + P_6\lambda_{C_1}^{**} + P_1\lambda_{C_2}^{**} + G_1)},$$
$$M_2^{**} = \frac{\theta_{M_2}(G_{13}\lambda_{C_1}^{**}\lambda_{C_2}^{**} + G_{14}\lambda_{C_1}^{**} + G_{15}\lambda_{C_2}^{**})}{\mu_M G_2G_9(\lambda_{C_1}^{**}\lambda_{C_2}^{**} + P_6\lambda_{C_1}^{**} + P_1\lambda_{C_2}^{**} + G_1)},$$
$$U_1^{**} = \frac{\Lambda_{s_1}}{\lambda_{s_1}^{**} + \mu_s},$$
$$U_2^{**} = \frac{\Lambda_{s_2}}{\lambda_{s_2}^{**} + \mu_s},$$
$$L_1^{**} = \frac{\Lambda_{s_1}\lambda_{s_1}^{**}}{P_4(\lambda_{s_1}^{**} + \mu_s)},$$
$$L_2^{**} = \frac{\Lambda_{s_2}\lambda_{s_2}^{**}}{P_9(\lambda_{s_2}^{**} + \mu_s)},$$
$$I_{s_1}^{**} = \frac{\Lambda_{s_1}\lambda_{s_1}^{**}\kappa_{s_1}}{P_4P_5(\lambda_{s_1}^{**} + \mu_s)},$$
$$I_{s_2}^{**} = \frac{\Lambda_{s_2}\lambda_{s_2}^{**}\kappa_{s_2}}{P_9P_{10}(\lambda_{s_2}^{**} + \mu_{s_2},)},$$
$$C_1^{**} = \frac{\Lambda_{s_1}\lambda_{s_1}^{**}\kappa_{s_1}\theta_{C_1}}{P_4p_5\mu_s(\lambda_{s_1}^{**} + \mu_s)},$$
$$C_2^{**} = \frac{\Lambda_{s_2}\lambda_{s_2}^{**}\kappa_{s_2}\theta_{C_2}}{P_9P_{10}\mu_s(\lambda_{s_2}^{**} + \mu_s)},$$
$$\lambda_{C_1}^{**} = \frac{A_1\beta_{h_1}\Lambda_{s_1}\lambda_{s_1}^{**}\kappa_{s_1}\theta_{C_1}}{G_{16}\lambda_{s_1}^{**} + C_0\mu_s\mu_C P_4P_5},$$
$$\lambda_{C_2}^{**} = \frac{A_2\beta_{h_2}\Lambda_{s_2}\lambda_{s_2}^{**}\kappa_{s_2}\theta_{C_2}}{G_{17}\lambda_{s_2}^{**} + C_0\mu_{s_2}\mu_C P_9P_{10}},$$
$$\lambda_{s_1}^{**} = \frac{A_3\beta_{s_1}\theta_{M_1}(G_{10}\lambda_{C_1}^{**}\lambda_{C_2}^{**} + G_{11}\lambda_{C_1}^{**} + G_{12}\lambda_{C_2}^{**})}{G_{18}\lambda_{C_1}^{**}\lambda_{C_2}^{**} + G_{19}\lambda_{C_1}^{**} + G_{20}\lambda_{C_2}^{**} + M_0\mu_M G_1G_2G_9},$$
$$\lambda_{s_2}^{**} = \frac{A_4\beta_{s_2}\theta_{M_2}(G_{13}\lambda_{C_1}^{**}\lambda_{C_2}^{**} + G_{14}\lambda_{C_1}^{**} + G_{15}\lambda_{C_2}^{**})}{G_{21}\lambda_{C_1}^{**}\lambda_{C_2}^{**} + G_{22}\lambda_{C_1}^{**} + G_{23}\lambda_{C_2}^{**} + M_0\mu_M G_1G_2G_9}.$$

where: $G_1 = P_{13} = P_1P_6 - \eta_{12}\eta_{21} > 0, \quad G_2 = P_{14} = P_2P_7 - a_{12}a_{21} > 0,$
$G_3 = \Lambda_{h_1}P_7 + \Lambda_{h_2}a_{21}, \quad G_4 = P_7(\Lambda_{h_1}P_6 + \Lambda_{h_2}\eta_{21}),$
$G_5 = a_{21}(\Lambda_{h_1}\eta_{12} + \Lambda_{h_2}P_1), \quad G_6 = P_2\Lambda_{h_2} + a_{12}\Lambda_{h_1},$
$G_7 = P_6\Lambda_{h_1} + \Lambda_{h_2}\eta_{21}, \quad G_8 = P_2(\Lambda_{h_1}\eta_{12} + \Lambda_{h_2}P_1),$
$G_9 = P_3P_8 - b_{12}b_{21} > 0, \quad G_{10} = \kappa_{h_1}P_8G_3 + b_{21}\kappa_{h_2}G_6,$
$G_{11} = \kappa_{h_1}P_8G_4 + b_{21}\kappa_{h_2}G_7, \quad G_{12} = \kappa_{h_1}P_8G_5 + b_{21}\kappa_{h_2}G_8,$
$G_{13} = P_3\kappa_{h_2}G_6 + b_{12}\kappa_{h_1}G_3, \quad G_{14} = P_3\kappa_{h_2}G_7 + b_{12}\kappa h_1G_4,$

$G_{15} = P_3\kappa_{h_2}G_8 + b_{12}\kappa_{h_1}G_5, \quad G_{16} = C_0\mu_C P_4 P_5 + \varepsilon\Lambda_{s_1}\kappa_{s_1}\theta_{C_1},$
$G_{17} = C_0\mu_C P_9 P_{10}\varepsilon\Lambda_{s_2}\kappa s_2\theta_{C_2}, \quad G_{18} = M_0\mu_M G_2 G_9 + \varepsilon\theta_{M_1}G_{10},$
$G_{19} = M_0\mu_M G_2 G_9 P_6 + \varepsilon\theta_{M_1}G_{11}, \quad G_{20} = M_0\mu_M G_2 G_9 P_1 + \varepsilon\theta_{M_1}G_{12},$
$G_{21} = M_0\mu_M G_2 G_9 + \varepsilon\theta_{M_2}G_{13}, \quad G_{22} = M_0\mu_M G_2 G_9 P_6 + \varepsilon\theta_{M_2}G_{14},$
$G_{23} = M_0\mu_M G_2 G_9 P_1 + \varepsilon\theta_{M_2}G_{15}.$

CONCLUSION

In this paper, we formulated a novel deterministic mathematical model which investigated the impact of metapopulation on the transmission dynamics of Schistosomiasis in a population. The sixteen (16) state variables of the model were shown to be non-negative and bounded for all time, t and that the trajectories generated by the metapopulation 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 metapopulation Schistosomiasis model was derived and was shown to be LAS whenever the corresponding reproduction number is below one ($\mathcal{R}_0^2 < 1$), which suggested that Schistosomiasis can be eradicated from the entire population in a two patch 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 two patch population where control measures are available would not generate large outbreaks, and unstable and if $\mathcal{R}_0^2 > 1$, which implied that Schistosomiasis will become endemic in the two patch population. The Endemic Equilibrium Point (EEP) was derived.

REFERENCE

[1] Inobaya, M. T., Remigio M Olveda,R. M., Chau, T. N., Olveda, D. U., and Ross, A. G.(2014). Prevention and control of schistosomiasis: a current perspective. *Res Rep Trop Med.*, vol(5): pp 65-75.

[2] Hotez P.J., and Kamath, A (2009). Neglected tropical diseases in Sub-Saharan Africa: review of their prevalence, distribution, and disease burden. *PLoS Negl Trop Dis* 3: e412.

[3] Hotez, P. J., Asojo, O. A., Adesina, A. M. (2012). Nigeria: ‘‘Ground Zero’’ for the High Prevalence Neglected Tropical Diseases. *PLoS Negl Trop Dis* 6(7).

[4] World Health Organization (WHO) (2017). Schistosomiasis Fact sheet 2017. *World Health Organization Press; Geneva*, Switzerland.

[5] Steinmann, P., Keiser, J., Bos, R., Tanner, M., and Utzinger, J. (2006). Schistosomiasis and water resources development: systematic review, meta analysis and estimates of people at risk. *Lancet Infect Dis* 6: 411-425.

[6] Allen, E.J., and Victory, H.D (2003). Modelling and simulation of a schistosomiasis infection with biological control. *Acta Tropica* vol 87 : pp 251-267.

[7] Anderson, R.M., and May, R.M. (1985). Helminth infections of humans: mathematical models, population dynamics, and control. *In: Baker, J.R., Muller, R. (Eds.), Advances in Parasitology*, vol. 24, pp. 1-101.

[8] Anderson, R.M., and May, R.M. (1992). Infectious diseases of humans: Dynamics and control. *Oxford University Press*, Oxford.

[9] 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 , pp 119-125.

[10] Chiyaka, E.T and Garira, W (2009). Mathematical Analysis of the Transmission Dynamics of schistosomiasis in the Human-Snail hosts. *Journal of Biological Systems*, Vol. 17, No. 3, pp 397-423.

[11] Chiyaka, E.T., Magombedze, G., and Lawrence Mutimbu, L (2010). Modelling within host parasite dynamics of schistosomiasis. *Computational and Mathematical Methods in Medicine*, Vol. 11, No. 3, pp 255-280.

[12] Feng, Z., Curtis, J., Minchella, D.J. (2001). The influence of drug treatment on the maintenance of schistosome genetic diversity. *J. Math. Biol.* 43, pp 52-68.

[13] Feng, Z., Li, C., Milner, F. A. (2002). Schistosomiasis models with density dependence and age of infection in snail dynamics. *Mathematical Biosciences* 177, pp 271-286.

[14] Feng,Z., Eppert, A., Milner, F. A., Minchella, D. J,(2004). Estimation of Parameters Governing the Transmission Dynamics of Schistosomes *Applied Mathematics Letters* 17, pp 1105-1112.

[15] Liang, S., Spear, R. C., Seto, E., Hubbard, A., and Qiu, D. (2005). A multi-group model of Schistosoma japonicum transmission dynamics and control: model calibration and control prediction. *Tropical Medicine and International Health*, volume 10 no 3, pp 263-278.

[16] Longxing, Q., Jing-an, C., Tingting, H., Fengli, Y., and Longzhi, J. (2014). Mathematical Model of Schistosomiasis under Flood in Anhui Province. *Hindawi Publishing Corporation Abstract and Applied Analysis*, Volume 2014, Article ID 972189.

[17] Luchsinger, C.J. (2001a). Approximating the long-term behavior of a model for parasitic infection. *J. Math. Biol.* 42, pp 555-581.
[18] Luchsinger, C.J. (2001b). Stochastic models of a parasitic infection, exhibiting three basic reproduction ratios. *J. Math. Biol.* 42, pp 532-554.
[19] Mouhamadou, D. (2015). Stability Analysis of a Schistosomiasis Transmission Model with Control Strategies. *Biomath 1*, 1504161,04.161.
[20] Murray, J. D. (1993). Mathematical Biology. *Springer-Verlag*, New York.
[21] Mushayabasa, S. and Bhunu, C.P. (2011). Modeling Schistosomiasis and HIV/AIDS Codynamics. *Hindawi Publishing Corporation Computational and Mathematical Methods in Medicine* Volume 2011, Article ID 846174.
[22] Ngarakana-Gwasiraa, E. T., Bhunua, C. P., Masochab, M., and Mashonjowa, E. (2016). Transmission dynamics of schistosomiasis in Zimbabwe: A mathematical and GIS Approach. *Commun Nonlinear Sci Numer Simulat* 35, pp 137-147.
[23] Pellegrinelli, A., and Gabriel, J.P. (1993). Eradication of helminthic infections. *Math. Biosci.* 117, 179-195.
[24] Woolhouse, M.E.J. (1991). On the application of mathematical models of schistosome transmission dynamics I. *Natural transmission. Acta Trop.* 49, pp 1241-1270.
[25] Woolhouse, M. E. J. (1992). On the application of mathematical models of schistosome transmission dynamics II. *Control. Acta Trop.* 50, pp 189-204.
[26] Moreno, V.,Espinoza, B., Bichara, D., Holechek, S. A., and Castillo-Chavez, C. (2016). Role of short-term dispersal on the dynamics of Zika virus. (unpublished).
[27] Hethcote, H.W. (2000). The mathematics of infectious diseases. *SIAM Review* 42(4): pp 599-653.
[28] Mishra, S., Fisman, N. P., and Boily, M.(2010). The ABC of terms used in mathematical models of infectious diseases. *J Epidemiol Community Health.* doi:10.1136/jech.2009.097113.
[29] Van den Driessche, P., and Watmough J., (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences* 180: pp 29-48.