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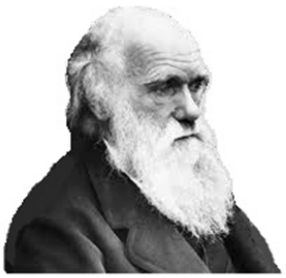

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Frequency Dependent Incidence Model for Acute and Chronic Schistosomiasis

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ABSTRACT

In this research work, a deterministic mathematical model for *schistosomiasis* transmission dynamics is presented. The model consists of five non-liniar ordinary differential equations incorporating the acute and chronic infectious compartments. The basic reproductive number, (the number of secondary infections when a single infectious individual is introduced into a population where everyone is susceptible) was obtained. Furthermore, we gained and analyzed for stability, the disease-free and endemic equilibrium. The qualitative feature of the model shows that the long-term behavior of the model is independent of initial conditions. Numerical simulation of the various state variables were obtained using matlab software.

Keyword**:** *Schistosomiasis*, stability, equilibrium, endemic, acute, chronic

1. INTRODUCTION

Schistosomiasis is an acute and chronic parasitic disease caused by blood flukes (trematodes worms) of the genus Schistosomia [1]. Intestinal *Schistosomiasis* has five main species; *Schistosoma mansoni, Schistosoma japonicum, Schistosoma mekongi, Schistosoma guineensis* and *S. intercalatum*, which afflicts at least 243 million people world over [2], among which *Schistosoma mansoni* is the most prevalent in Africa [3]. Globally, Schistosomiasis

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account for 200,000 deaths [4]. The risk of infection is highest amongst those who lived near lakes or rivers [5]. In Uganda, almost no transmission was found to have occurred at altitudes greater than 1400 m or where the annual rainfall was less than 900 mm [5].

Hygiene and playing in mud and water make children vulnerable to infection. Forty million women of childbearing age are infected [6] and approximately 10 million women in Africa have schistosomiasis during pregnancy [6].

In endemic areas, the infection is usually acquired as a child. The intensity and prevalence of infection rises with age and peaks usually between ages 15 and 20 years. In older adults, no significant change is found in the prevalence of disease, but the parasite burden or the intensity decreases [7].

Human beings become infected with schistosomiasis when larval forms of the parasite, released by freshwater snails, penetrate their skin during contact with infested water. In the body, the larvae develop into adult schistosomes. Adult worms live in the blood vessels, where the females release eggs. Some of the eggs are passed out of the body in the feces or urine to continue the parasite life cycle. Others can end up in the skin, brain, muscle, adrenal glands, and eyes. As the eggs penetrate the urinary system, they can find their way to the female genital region and form granulomas in the uterus, fallopian tube, and ovaries. Central nervous system (CNS) involvement occurs because of embolization of eggs from the portal mesenteric system to the brain and spinal cord via the paravertebral venous plexus [8, 9].

Acute schistosomiasis (Katayama syndrome) with incubation period of 14 – 84 days, is a systemic, serum sickness-like illness that develops after several weeks in some individuals with new schistosomal infections. It may correspond to the first cycle of egg deposition and is associated with marked peripheral eosinophilia and circulating immune complexes. It is most common with *S. japonicum* and *S. mansoni* infections and is most likely to occur in heavily infected individuals after primary infection. Chronic schistosomiasis, which is far more common than the acute form of the infection, results from egg-induced immune response, granuloma formation, and associated fibrotic changes. Chronic intestinal *schistosomiasis* can present with acute complications of appendicitis, perforation, and bleeding long after travel-related (or endemic) exposure [10]. Rectal perforation caused by *S. haematobium* has also been described in a case report [11].

The use of mathematical model to combat the menace of *schistosomiasis* dated back to 1982 when [12] developed a deterministic mathematical model to study the role of density-dependent fecundity on population biology and dynamics of soil-transmitted *helminthiases*, in Particular *ascariasis* and hookworm infection. In [13], a deterministic model to study prevalence of intestinal *schistosomiasis* in human and snail was develpoed. The model also was used to evaluate possible control strategies, limitations and uncertainties. [14], used stochastic model to investigate the role of immunity in observed patterns in endemic communities. Chemotherapy; vaccination; snail control; larval stage control; improved water and sanitation; health education effects on infection intensity in humans was studied by [15]. Deterministic model was developed by [16] to study infection intensity in humans and prevalence in snails. The findings reveals that infected snails suffer excess mortality and no reproduction. [17], developed a deterministic mathematical model to study the effect of time delay on mating structure.

More recently, [18] developed a mathematical model of *schistosomiasis* transmission under flood in Anhui province. The delay of *schistosomiasis* outbreak under flood was considered. The impact of flood on the stability of the endemic equilibrium was studied and the

results imply that flood can destabilize the system and periodic solutions can arise by Hopf bifurcation. In [19] a mathematical model is defined to encompass two different delivery strategies for the vaccination of the population, namely, infant (cohort) and mass vaccination. The paper focused on vaccination delivered in a cohort immunisation programme where infants are immunised within the first year of life before acquiring infection.

The major difference in this paper and the aforementioned ones is the inclusion of acute and chronic compartment. The paper also used the standard incidence (frequency dependent) as against mass action where it is assumed that the rate at which infection passes to the population is jointly proportional to the product of number of persons with infection and number susceptible to the infection.

2. MODEL FORMULATION

The total human population N_1 is divided into the following epidemiological classes; susceptible individuals (S_1), exposed individuals (i.e. who are infected but do not yet discharge *schistosoma* egg to the fresh water via their urine or faeces) (E_1), acute (transient) infected individual (I_{1A}), chronic infected individual (I_{1C}), and recovered individual (R_1) such that $N_1 = S_1 + E_1 + I_{1A} + I_{1C} + R_1$. The total snail population N_2 is divided into the following; susceptible snail (S_2), infected snail (i.e. snails that produce the parasitic fluke, *cercariae* which is passed unto human) (I_2), such that $N_2 = S_2 + I_2$.

The susceptible human population (S_1) is generated by birth and immigration at a constant rate Λ_1. It is reduced by natural death at the rate μ_1, and as members acquire the *schistosoma* parasites at the rate α_1. The exposed human population is generated upon a successful contact with *cercariae* at the rate α_1, and is decreased as the *cercariae* who survival the first 2 – 4 weeks takes the affected individual to infected stage at the rate σ_1 and by natural death at the rate μ_1. The acute infection population is generated as exposed individuals begin to show symptoms and discharge the *schistosoma* egg in fresh water at the rate σ_1. It is decreased as members recover at the rate of γ_1, progress to chronic infection at the rate σ_2, die naturally at the rate μ_1 or due to the infection at the rate δ_1. The chronic infection population is generated as acute infected individuals lasts for months and years without treatment at the rate σ_2. It is decreased as members recover from the infection at the rate γ_2, die naturally at the rate μ_1 or due to the infection at the rate δ_1. The recovered individual is generated as acute and chronic infected individuals recover at the rate of γ_1 and γ_2. It is in turn decreased by loss of immunity and natural death at the rate μ_1.

The susceptible snail population (S_2), is generated at a constant rate Λ_1. It is decreased as its members come in contact with *cercariae* discharged by acute and chronic infected individuals at the rates α_2 and α_3, and by natural death μ_2. The infected snail population is

generated by successful contact with the eggs introduced into their habitat by the acute and chronic infected human at the rate α_2 and α_3. It is reduced by natural death at the rate μ_2.

Form the above, we come up with the following differential equation to represent the dynamics of *schistosomiasis* within human and snail population.

$$\frac{dS_1}{dt} = \Lambda_1 - \frac{\alpha_3 I_2 S_1}{N_1} - \mu_1 S_1,$$

$$\frac{dE_1}{dt} = \frac{\alpha_3 I_2 S_1}{N_1} - (\mu_1 + \sigma_1)E_1,$$

$$\frac{dI_{1A}}{dt} = \sigma_1 E_1 - (\gamma_1 + \sigma_2 + \mu_1 + \delta_1)I_{1A},$$

$$\frac{dI_{1C}}{dt} = \sigma_2 I_{1A} - (\gamma_2 + + \mu_1 + \delta_1)I_{1C}, \qquad (1)$$

$$\frac{dR_1}{dt} = \gamma_1 I_{1A} + \gamma_2 I_{1C} - \mu_1 R_1,$$

$$\frac{dS_2}{dt} = \Lambda_2 - \frac{(\alpha_1 I_{1A} + \alpha_2 I_{1C})S_2}{N_1} - \mu_2 S_2,$$

$$\frac{dI_2}{dt} = \frac{(\alpha_1 I_{1A} + \alpha_2 I_{1C})S_2}{N_2} - \mu_2 I_2.$$

Basic Properties

In this section, the basic dynamical features of the model (1) will be explored. We Claim the following:

Lemma 1

$$D = \left\{(S_1, E_1, I_{1A}, I_{1C}, R_H, S_2, I_2) \in R_+^7 : S_1 + E_1 + I_{1A} + I_{1C} + R_1 \le \frac{\Lambda_1}{\mu_1}; S_2 + I_2 \le \frac{\Lambda_2}{\mu_2}\right\},$$

is positively invariant and attracting with respect to the basic model equations (1).

Proof

Adding equations four equations of (1) gives;

$$\frac{dN_1}{dt} = \Lambda_1 - \delta_1(I_{1A} + I_{1C}) - \mu_1 N_H. \quad (2)$$

Also, adding the last two equations of (1) gives;

$$\frac{dN_2}{dt} = \Lambda_2 - \mu_2 N_2. \quad (3)$$

Since $\frac{dN_1}{dt} < \Lambda_1 - \mu_1 N_1$, and $\frac{dN_2}{dt} < \Lambda_2 - \mu_2 N_2$, it follows that $\frac{dN_1}{dt} < 0$ and $\frac{dN_2}{dt} < 0$ if, $N_1(t) > \frac{\Lambda_1}{\mu_1}$ and $N_2(t) > \frac{\Lambda_2}{\mu_2}$ respectively.

Thus a standard comparison theorem, [20] can be used to show that,

$$N_1(t) < N_1(0)e^{-\mu_1(t)} + \frac{\Lambda_1}{\mu_1}\left[1 - e^{-\mu_1(t)}\right] \text{ and } N_2(t) < N_2(0)e^{-\mu_2(t)} + \frac{\Lambda_2}{\mu_2}\left[1 - e^{-\mu_2(t)}\right].$$

In particular, $N_1(t) \le \frac{\Lambda_1}{\mu_1}$ and $N_2(t) \le \frac{\Lambda_2}{\mu_2}$ if $N_2(0) \le \frac{\Lambda_1}{\mu_1}$ and $N_2(0) \le \frac{\Lambda_2}{\mu_2}$ Thus, D is positively invariant. Further, if $N_1(t) > \frac{\Lambda_1}{\mu_1}$ and $N_2(t) > \frac{\Lambda_2}{\mu_2}$ then either the solution enters D in finite time or $N_2(t)$ approaches $\frac{\Lambda_1}{\mu_1}$, and $N_2(t)$ approaches $\frac{\Lambda_2}{\mu_2}$ and the infected variables E_1, I_{1A}, I_{1C} approaches zero. Hence, all solutions R_+^7 eventually enters D. Thus in D, the basic model (1) is well posed epidemiologically and mathematically [21]. Hence, it is sufficient to study the dynamics of the model equations in D.

Lemma 2. Let the initial data $F(0) \ge 0$, where $F(t) = (S_1, E_1, I_{1A}, I_{1C}, R_1, S_2, I_2$. Then the solution $F(t)$ of the *schistosomiasis* model (1) are non- negative for all $t \ge 0$. Furthermore form (2) and (3),

$$\underset{t\to\infty}{Lim}\sup N_1(t) = \frac{\Lambda_1}{\mu_1} \text{ and } \underset{t\to\infty}{Lim}\sup N_2(t) = \frac{\Lambda_2}{\mu_2}$$

Proof

$t_1 = \sup\{t > 0 : F(t) > 0 \in [0,t]\}$. Thus $t_1 > 0$. it follows from (1) that

$$\frac{dS}{dt} = \Lambda_H + \psi V_H - \frac{\alpha_1 b_m S_H I_M}{N} - (\nu + \mu_1) S_H,$$

which can be written as follows

$$\frac{\mathrm{dS_1}}{\mathrm{dt}} = \Lambda_1 + \tau R - \frac{\alpha_3 I_2 S_1}{N_1} - \mu_1 S_1,$$

$$\frac{d}{dt}\left\{ S_1(t) \exp\left[\alpha_3 \int_0^{t_1} \frac{I_2}{N_1}(\xi) d\xi + (\mu_1) t \right] \right\} = (\Lambda_1 + \tau R_1) \exp\left[\alpha_3 \int_0^{t_1} \frac{I_2}{N_1}(\xi) d\xi + (\mu_1) t \right].$$

So that,

$$\frac{dS_1(t_1)}{dt} \exp\left[\alpha_3 \int_0^{t_1} \frac{I_2}{N_1}(\xi) d\xi + (\mu_1) t_1 \right] - S_1(0) = \int_0^{t_1} (\Lambda_1 + \tau R_1) \exp\left[\alpha_3 \int_0^{P} \frac{I_2}{N_H}(\xi) d\xi + (\mu_1) p \right] dp.$$

Hence,

$$S_1(t_1) = S_1(0) \exp\left[-\alpha_3 \int_0^{t_1} \frac{I_2}{N_1}(\xi) d\xi + (\mu_1) t_1 \right]$$

$$+ \exp\left[-\alpha_3 \int_0^{t_1} \frac{I_2}{N_1}(\xi) d\xi + (\mu_1) t_1 \right] \cdot \int_0^{t_1} (\Lambda_H + \psi V_H) \exp\left[\alpha_1 b_M \int_0^{P} \frac{I_M}{N_H}(\xi) d\xi + (\nu + \mu_1) p \right] dp > 0.$$

Similarly, it can be shown that $F > 0$, for all $t > 0$.
For the second part of the proof, note that

$0 < E_1(t) \le N_1(t)$, $0 < I_{1A}(t) \le N_1(t)$, $0 < I_{1C}(t) \le N_1(t)$, $0 < R_1(t) \le N_1(t)$, $0 < S_2(t) \le N_2(t)$, $0 < I_2(t) \le N_2(t)$.

From equations (2) and (3)

$$\frac{\Lambda_1}{\mu_1} \le \underset{t\to\infty}{Lim}\inf\, N_1(t) \le \underset{t\to\infty}{Lim}\sup N_1(t) = \frac{\Lambda_1}{\mu_1},$$

and

$$\frac{\Lambda_2}{\mu_2} \le \underset{t\to\infty}{Lim}\inf\, N_2(t) \le \underset{t\to\infty}{Lim}\sup N_2(t) = \frac{\Lambda_2}{\mu_2}, \text{ as required.}$$

Local stability of Disease free equilibrium (DFE)

Disease free equilibrium is equilibrium where there is no infection. Therefore, the infected classes will be zero that means that the whole population will be susceptible. To find the disease free equilibrium (E_0) of our model equations (1), we equate the rate of change of our state variables to zero i.e.

$$\frac{dS_1(t)}{dt}=\frac{dE_1(t)}{dt}=\frac{dI_{1A}(t)}{dt}=\frac{dI_{1C}(t)}{dt}=\frac{dR_1(t)}{dt}=\frac{dS_2(t)}{dt}=\frac{dI_2(t)}{dt}=0.$$

Solving the resulting algebra equations we have:

$$E_0=(S_1^*,,E_1^*,I_{1A}^*,I_{1C}^*,R_1,S_2^*,I_M^*)=\left(\frac{\Lambda_1}{(\mu_1},0,0,0,0\frac{\Lambda_2}{\mu_2},0\right) \tag{4}$$

The linear stability of E_0 can be established using the next generation Matrix operator method on the system (I). Using the notation in [22], the matrices F and V for the new infection terms and the remaining transfer terms, are, respectively, given by:

$$F=\begin{pmatrix} 0 & 0 & 0 & \frac{\alpha_3 S_1^*}{N_1} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\alpha_1 S_2^*}{N_2} & \frac{\alpha_2 S_2^*}{N_2} & 0 \end{pmatrix}, \tag{5}$$

$$V=\begin{pmatrix} (\sigma_1+\mu_1) & 0 & 0 & \frac{\alpha_3 S_1^*}{N_1} \\ \sigma_1 & (\sigma_2+\mu_1+\gamma+\delta_1) & 0 & 0 \\ 0 & \sigma_2 & (+\mu_1+\gamma+\delta_1) & 0 \\ 0 & 0 & 0 & \mu_2 \end{pmatrix} \tag{7}$$

for

$$\begin{aligned} &K_1=\mu_1, K_2=(\sigma_1+\mu_1), K_3=(\sigma_2+\gamma+\delta_1+\mu_1), \\ &K_4=(\gamma+\delta_1+\mu_1), K_5=\mu_1, K_6=K_7=\mu_2. \end{aligned} \tag{8}$$

$$V' = \begin{pmatrix} \frac{1}{K_2} & 0 & 0 & 0 \\ \frac{-\sigma_1}{K_2 K_3} & \frac{1}{K_3} & 0 & 0 \\ \frac{1}{K_2 K_3 K_4} & \frac{-\sigma_2}{K_3 K_4} & \frac{1}{K_4} & 0 \\ 0 & 0 & 0 & \frac{1}{K_7} \end{pmatrix}, \tag{9}$$

$$FV' = \begin{pmatrix} 0 & 0 & 0 & \frac{\alpha_3 S_1^*}{N_1 K_7} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ \frac{-\sigma_1 \alpha_1 S_2^*}{N_2 K_2 K_3} + \frac{\alpha_2 S_2^*}{N_2 K_2 K_3 K_4} & \frac{\alpha_1 S_2^*}{N_2 K_3} - \frac{\sigma_2 \alpha_2 S_2^*}{N_2 K_3 K_4} & \frac{\alpha_2 S_2^*}{N_2 K_4} & 0 \end{pmatrix}, \tag{10}$$

Here, the basic reproductive number $\Re_0$ is the spectral radius (dominant eigenvalue) of the product matrix FV^{-1}, hence model (1) has:

$$\Re_0 = \rho(FV') = \sqrt{\frac{1}{2}\left(\frac{\sigma_1 \alpha_1 S_2^*}{N_2 K_2 K_3} + \frac{\sigma_2 \alpha_2 S_2^*}{N_2 K_2 K_3 K_4}\right)\frac{\sigma_1 \alpha_3 S_1^*}{N_1 K_7}} \tag{11}$$

Theorem 1

The disease free equilibrium of the schistosomiasis model (1) is locally asymptotically stable when $\Re_0 < 1$ and unstable when $\Re_0 > 1$.

Proof

The local stability of the disease free equilibrium is a direct consequence of [22] and shows that the model equations (1) to (7) satisfies five assumptions $A_1 - A_5$ in [22], and that ends the proof.

Theorem 2

The disease free equilibrium of the schistosomiasis model (1) is globally asymptotically stable when $\Re_0 < 1$ and unstable when $\Re_0 > 1$

Proof

For the prove of the globally asymptotic stability of DFE of model (1), we use the comparison theorem in [20, 24] p.31. To apply this theorem we re-write the equations for the infected compartments in (1) as follows:

$$\begin{pmatrix} E_1' \\ I_{1A}' \\ I_{1C}' \\ I_2' \end{pmatrix} = (F-V)\begin{pmatrix} E_1 \\ I_{1A} \\ I_{1C} \\ I_2 \end{pmatrix} - \begin{pmatrix} \frac{\alpha_3 I_2}{N_1^*}(S_1^* - S_1) \\ 0 \\ 0 \\ \frac{\alpha_1 I_{1A} + \alpha_2 I_{1C}}{N_2^*}(S_2^* - S_2) \end{pmatrix} \quad (12)$$

where F and V are as defined in (5) and (7). Further, since $S_1 < S_1^* = \frac{\Lambda_1}{\mu_1}$, for all $t > 0$, it follows that

$$\begin{pmatrix} E_1' \\ I_{1A}' \\ I_{1C}' \\ I_2' \end{pmatrix} \leq (F-V)\begin{pmatrix} E_1 \\ I_{1A} \\ I_{1C} \\ I_2 \end{pmatrix} \quad (13)$$

Since the eigenvalues of the matrix $(F-V)$ have negative real parts as in Lemma 1 of [22], then the model (1) is stable whenever $\Re_0 < 1$. So, $(E_1, I_{1A}, I_{1C}, I_2), t \to (0,0,0,0)$ t as $t \to \infty$, By comparison theorem, it follows that $(E_1, I_{1A}, I_{1C}, I_2), t \to (0,0,0,0)$ and $S_1 \to \frac{\Lambda_1}{\mu_1}$ as $t \to \infty$, then, $(S_1, E_1, I_{1A}, I_{1C}, R_1, S_2, I_2), t \to E_0, t \to \infty$. So E_0 is globally asymptotically stable for $\Re_0 < 1$.

Endemic equilibrium state/stability

This is an equilibrium state where at least one of the infected compartments is non-zero. In order to find the Endemic equilibrium for our model equations (1) to (7), the following steps are taken. We let $E_1 = \left(S_1^{**}, E_1^{**}, I_{1A}^{**}, I_{1C}^{**}, R_1^{**}, S_2^{**}, I_2^{**}\right)$ represent any arbitrary point of the endemic equilibrium of our model equations (1) to (7), further, let; we let

$$, \lambda_1^{**} = \frac{\alpha_3 I_2^{**}}{N_1^{**}}, \lambda_2 = \frac{\alpha_1 I_{1A}^{**} + \alpha_2 I_{1C}^{**}}{N_2^{**}}. \quad (14)$$

Substituting (8) and (14) into the right hand side of (1) and equating to zero, we have

$$\Lambda_1 + -\lambda_1^{**} S_1^{**} - K_1 S_1^{**} = 0,$$

$$\lambda_1^{**} S_1^{**} - K_2 E_1^{**} = 0,$$

$$\sigma_1 E_1^{**} - K_3 I_{1A}^{**} = 0,$$

$$\sigma_2 I_{1A}^{*} - K_4 I_{1C}^{**} = 0, \qquad (15)$$

$$\gamma_1 I_{1A}^{**} + \gamma_1 I_{1C}^{**} - K_5 R_1^{**} = 0,$$

$$\Lambda_2 - \lambda_2^{**} S_2^{**} - K_6 S_2^{**} = 0,$$

$$\lambda_2^{**} S_2^{**} - K_6 I_2^{**} = 0.$$

Solving (15) we have:

$$E_1 = \left(S_1^{**}, E_1^{**}, I_{1A}^{**}, I_{1C}^{**}, R_1^{**}, S_2^{**}, I_2^{**}\right) = \left(\begin{array}{l} \dfrac{\Lambda_1}{\lambda_1^{**} + K_1}, \dfrac{\Lambda_1 \lambda_1^{**}}{K_1(\lambda_1^{**} + K_1)}, \\ \dfrac{\Lambda_1 \sigma_1}{K_1 K_3 (\lambda_1^{**} + K_1)}, \dfrac{\Lambda_1 \sigma_1 \sigma_2}{K_1 K_3 K_4 (\lambda_1^{**} + K_1)}, \\ \dfrac{\alpha_1 (\Lambda_1 \sigma_1 (K_4 + \sigma_2))}{K_1 K_3 K_4 K_5 (\lambda_1^{**} + K_1)}, \dfrac{\Lambda_2}{(\lambda_2^{**} - K_6)}, \\ \dfrac{\lambda_2^{**} \Lambda_2}{K_7 (\lambda_2^{**} - K_6)}. \end{array} \right) \qquad (16)$$

Substituting (16) into (14) and solving, we have:

$$\lambda_1^{**} = a_1 (\lambda_1^{**})^2 + b_1 \lambda_1^{**} - c_1, \qquad (17)$$

where

$$\begin{aligned} a_1 &= N_2^{**} K_1 K_3 K_4 K_6, \\ b_1 &= N_1^{**} K_1 \left[\left(\Lambda_1 \sigma_1 (K_4 \alpha_1 + \alpha_2 \sigma_2) \right) - N_2^{**} (K_1)^2 K_3 K_4 K_6 \right], \\ c_1 &= \alpha_3 \Lambda_2 \left(\left(\Lambda_2 \sigma_1 (K_4 \alpha_1 + \alpha_2 \sigma_2) \right) \right). \end{aligned} \qquad (18)$$

It can be seen that the coefficient a_1 of (17) is always positive while c_1 is positive which occurs only when $\Re_0$ is less than unity and negative if $\Re_0$ is greater than unity. Thus the following result is claimed.

Theorem

The basic model given by (1) is characterized by

(i) one unique endemic equilibrium if $b_1 < 0$, and $c_1 = 0$ or $(b_1)^2 - 4a_1 c_1 = 0$,

(ii) one unique endemic equilibrium if $c_1 < 0 \Leftrightarrow \Re_0 > 1$,

(iii) two endemic equilibrium if $c_1 > 0, (b_1)^2 < 0$ and $(b_1)^2 - 4a_1 c_1 > 0$,

(iv) no endemic equilibrium otherwise.

Case (iii) above suggest the possibility of backward bifurcation where local stability of DFE co-exist with endemic equilibrium even when $\Re_0 < 1$. This prompted the bifurcation analysis.

Local stability of Endemic Equilibrium and Bifurcation analysis

Theorem The endemic equilibrium of model (1) is locally asymptotically stable when

$c_2 c_2 - c_0 > 0$

where c_2, c_1, and c_0 are given in the proof of the theorem.

Proof

The variational matrix, M^{**} corresponding to the endemic equilibrium point is given by

$$M^{**} = \begin{pmatrix} \frac{\alpha_3 I_2^{**}}{N_1} + K_1 & 0 & 0 & 0 & 0 & 0 & \frac{\alpha_3 S_2^{**}}{N_1} \\ -\frac{\alpha_3 I_2^{**}}{N_1} & K_1 & 0 & 0 & 0 & 0 & -\frac{\alpha_3 S_2^{**}}{N_1} \\ 0 & -\sigma_1 & K_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\sigma_2 & K_4 & 0 & 0 & 0 \\ 0 & 0 & -\gamma_1 & -\gamma_2 & K_5 & 0 & 0 \\ 0 & 0 & \frac{\alpha_1 S_2^{**}}{N_2} & \frac{\alpha_2 S_2^{**}}{N_2} & 0 & \frac{\alpha_1 I_A^{**} + \alpha\ I_C^{**}}{N_2} + K_6 & 0 \\ 0 & 0 & -\frac{\alpha_1 S_2^{**}}{N_2} & -\frac{\alpha_2 S^{**}}{N_2} & 0 & 0 & K_7 \end{pmatrix}$$

To prove that the endemic equilibrium (16) of the model (1) is locally asymptotically stable, we perform bifurcation analysis at the DFE using the Centre Manifold theorem as described in [23], and given in appendix A.

In order to apply this theorem, we first make the following change of variables. Let

$S_1 = x_1, E_1 = x_{2,}, I_{1A} = x_3 = I_{1C} = x_4, R_1 = x_5, S_2 = x_6, I_2 = x_7$, so that

$N_1 = x_1 + x_{2,} + x_3 + x_4 + x_5$

and $N_2 = x_6 + x_7$, such that $N_x = N_1 + N_2 = x_6 + x_7$ further, using the vector notation,

$X = (x_1, x_{2,}, x_3 x_4, x_5, x_6, x_7)^T$.

Then our model equations (1) to (7) can be written in the form

$\frac{dx}{dt} = (f_1, f_{2,}, f_3, f_4, f_5, f_6, f_7)^T$, such that:

$$\begin{aligned}
\frac{dx_1}{dt} &= f_1 = \Lambda_1 - \frac{\alpha_3 x_1 x_7}{N_x} - k_1 x_1 \\
\frac{dx_2}{dt} &= f_2 = \frac{\alpha_3 x_1 x_7}{N_x} - k_2 x_2 \\
\frac{dx_3}{dt} &= f_3 = \sigma_1 x_2 - k_3 x_3 \\
\frac{dx_4}{dt} &= f_4 = \sigma_2 x_3 - k_4 x_4 \\
\frac{dx_5}{dt} &= f_5 = \gamma_1 x_3 + \gamma_2 x_4 - k_5 x_5 \\
\frac{dx_6}{dt} &= f_6 = \Lambda_1 - \frac{\alpha_1 x_3 x_6 + \alpha_2 x_4 x_6}{N_x} - k_6 x_6 \\
\frac{dx_7}{dt} &= f_7 = \frac{\alpha_1 x_3 x_6 + \alpha_2 x_4 x_6}{N_x} - k_7 x_7
\end{aligned} \tag{19}$$

Suppose that $\alpha_3 = \alpha_3^*$ is taken as the bifurcation parameter, and considering where the basic reproductive number $\Re_0 = 1$, and $\alpha_3 = \alpha_3^* \Re_0$, in (11). Then linearizing (19) at DFE point E_0 when $\alpha_3 = \alpha_3^*$ gives the Jacobia J_0^* which has a trivial zero eigenvalues, while the rest eigenvalues has negative real part.

$$J_0^* = \begin{bmatrix}
-K_1 & 0 & 0 & 0 & 0 & 0 & -\frac{\alpha_3}{N_x^*} \\
0 & -K_2 & 0 & 0 & 0 & 0 & \frac{\alpha_3}{N_x^*} \\
0 & \sigma_1 & -K_3 & 0 & 0 & 0 & 0 \\
0 & 0 & \sigma_2 & -K_4 & 0 & 0 & 0 \\
0 & 0 & \gamma_1 & \gamma_2 & -K_5 & 0 & 0 \\
0 & 0 & -\frac{\alpha_1}{N_x^*} & -\frac{\alpha_2}{N_x^*} & 0 & -K_6 & 0 \\
0 & 0 & \frac{\alpha_1}{N_x^*} & \frac{\alpha_2}{N_x^*} & 0 & 0 & -K_7
\end{bmatrix} \tag{20}$$

The Jacobia J_0^* (20) evaluated $\alpha_3 = \alpha_3^*$ has right eigenvector associated with the trivial eigenvalues given by

$w = [w_1, w_2, w_3, w_4, w_5, w_6, w_7]^T$, such that:

$$\left.\begin{aligned}
-Kw_1 - \frac{\alpha_3}{N_x^*} w_7 &= 0 \\
-K_2 w_2 + \frac{\alpha_3}{N_x^*} w_7 &= 0 \\
\sigma_1 w_2 - K_3 w_3 &= 0 \\
\sigma_2 w_3 - K_4 w_4 &= 0 \\
\gamma_1 w_3 + \gamma_2 w_4 - K_5 w_5 &= 0 \\
-\frac{\alpha_1}{N_x^*} w_3 - \frac{\alpha_2}{N_x^*} w_4 - K_6 w_6 &= 0 \\
\frac{\alpha_1}{N_x^*} w_3 + \frac{\alpha_2}{N_x^*} w_4 - K_7 w_7 &= 0
\end{aligned}\right\} \qquad (21)$$

Solving (21), we have;

$$\begin{aligned}
w_1 &= -\frac{\alpha_3}{N_x^* K_1} w_7 \\
w_2 &= \frac{\alpha_3}{N_x^* K_2} w_7 \\
w_3 &= \frac{\sigma_1 \alpha_3}{N_x^* K_2 K_3} w_7 \\
w_4 &= \frac{\sigma_2 \sigma_1 \alpha_3}{N_x^* K_2 K_3 K_4} w_7 \qquad (22) \\
w_5 &= \frac{\sigma_1 \alpha_3 w_7}{N_x^* K_2 K_3 K_5} \left(\frac{\gamma_2 \sigma_2}{K_4} + \gamma_1 \right) \\
w_6 &= -\frac{\sigma_1 \alpha_3 w_7}{(N_x^*)^2 K_2 K_3 K_6} \left(\frac{\alpha_2 \sigma_2}{K_4} + \alpha_1 \right) \\
w_7 &= w_7 > 0
\end{aligned}$$

Similarly the left eigenvalues of J_0^* evaluated at $\alpha_3 = \alpha_3^*$ corresponding to the trivial eigenvalue is $v = [v_1, v_2, v_3, v_4, v_5, v_6, v_7]$. Such that:

$$\left.\begin{aligned} -K_1 v_1 &= 0,\\ -K_2 v_2 + \sigma_1 v_3 &= 0,\\ -K_3 v_3 + \sigma_2 v_4 + \gamma_1 v_5 - \frac{\alpha_1}{N_x^*} v_6 + \frac{\alpha_1}{N_x^*} v_7 &= 0,\\ -K_4 v_4 + \gamma_2 v_5 - \frac{\alpha_2}{N_x^*} v_6 + \frac{\alpha_2}{N_x^*} v_7 &= 0,\\ -K_5 v_5 &= 0,\\ -K_6 v_6 &= 0,\\ -\frac{\alpha_3}{N_x^*} v_1 + \frac{\alpha_3}{N_x^*} v_2 - K_7 v_7 &= 0. \end{aligned}\right\} \qquad (23)$$

By solving (23), we have;

$$v_1 = 0,$$

$$v_2 = \frac{\sigma_1 v_3}{K_2},$$

$$v_3 = v_3,$$

$$v_4 = \frac{\alpha_2 \alpha_3 \sigma_1 v_3}{(N_x^*)^2 K_2 K_4 K_7},$$

$$v_5 = 0,$$

$$v_6 = 0,$$

$$v_7 = \frac{\alpha_3 \sigma_1 v_3}{N_x^* K_2 K_7}.$$

Computation of a and b

We consider $k = 2 \text{ and } 7$, that is, the following functions will be used to find a and b from the system.

$$f_2 = \frac{\alpha_3 x_1 x_7}{N_X} - k_2 x_2$$

$$f_7 = \frac{\alpha_1 x_3 x_6 + \alpha_2 x_4 x_6}{N_x^*} - k_7 x_7$$

but at DFE, $N_x^* = x_1^* + x_6^*$.

Hence, the associated non-zero partial of f at the DFE for $f = f_2, f_7$ are given by;

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_7} = \frac{\partial^2 f_2}{\partial x_7 \partial x_1} = \frac{\alpha_3^* x_6^*}{(x_1^* + x_6^*)^2},$$
$$\frac{\partial^2 f_7}{\partial x_3 x_6} = \frac{\partial^2 f_7}{\partial x_6 x_3} = \frac{\alpha_1 x_1^*}{(x_1^* + x_6^*)^2},$$
$$\frac{\partial^2 f_7}{\partial x_4 x_6} = \frac{\partial^2 f_7}{\partial x_6 x_4} = \frac{\alpha_2 x_1^*}{(x_1^* + x_6^*)^2}.$$

Therefore

$$a = v_2 w_7 w_1 \frac{\alpha_3^* \Lambda_2 (\mu_1 \mu_2)^2}{\mu_2 (\Lambda_1 \mu_2 + \mu_1 \Lambda_2)^2} + v_7 w_3 w_6 \frac{\alpha_1 \Lambda_1 (\mu_1 \mu_2)^2}{\mu_1 (\Lambda_1 \mu_2 + \mu_1 \Lambda_2)^2} + v_7 w_4 w_6 \frac{\alpha_2 \Lambda_1 (\mu_1 \mu_2)^2}{\mu_1 (\Lambda_1 \mu_2 + \mu_1 \Lambda_2)^2}$$
$$= \frac{(\mu_1 \mu_2)^2}{(\Lambda_1 \mu_2 + \mu_1 \Lambda_2)^2} \left[\frac{\Lambda_2}{\mu_2} \alpha_3^* v_2 w_1 w_7 + \frac{\Lambda_1}{\mu_1} v_7 w_6 (w_3 \alpha_1 + w_4 \alpha_2) \right]$$

$$b = \sum_{k,i,j=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial P^*} = v_2 w_7 \frac{\Lambda_1 (\mu_1 \mu_2)}{\mu_1 (\Lambda_1 \mu_2 + \mu_1 \Lambda_2)}$$

As a corollary to the above when $a > 0, b > 0,$ the bifurcation at $\phi = 0$ is subcritical (backward bifurcation).

Numerical simulation

In this section we carried out numerical simulation of the model using the parameter values provided in Table 1.

Parameter and their meaning	Value	Reference
Λ_1 - Recruitment rate of susceptible human population.	100	[25]
Λ_2 - Recruitment rate of susceptible snail population.	200	[25]
μ_1 - Natural death rate of human.	0.0004	[26]
μ_2 - Natural death rate of snail.	0.000056	[27]
α_1 - Transmission rate from acute infected individual to snail.	0.004	[27]
α_2 - Transmission rate from chronic infected individual snail.	0.0046	Assumed
α_3 - Transmission rate from infected snail to susceptible human	0.046	[27]

γ_1 - Recovery rate of acute infected individual.	0.05	[28]
γ_2 - Recovery rate of chronic infected individual.	0.06	Assumed
σ_1 - Progression rate from exposed to acute infection.	0.2	Assumed
σ_2 - Progression rate from acute to chronic infection	0.03	Assumed
δ - *Schistosomiasis* induced death rate.	0.0039	[29]

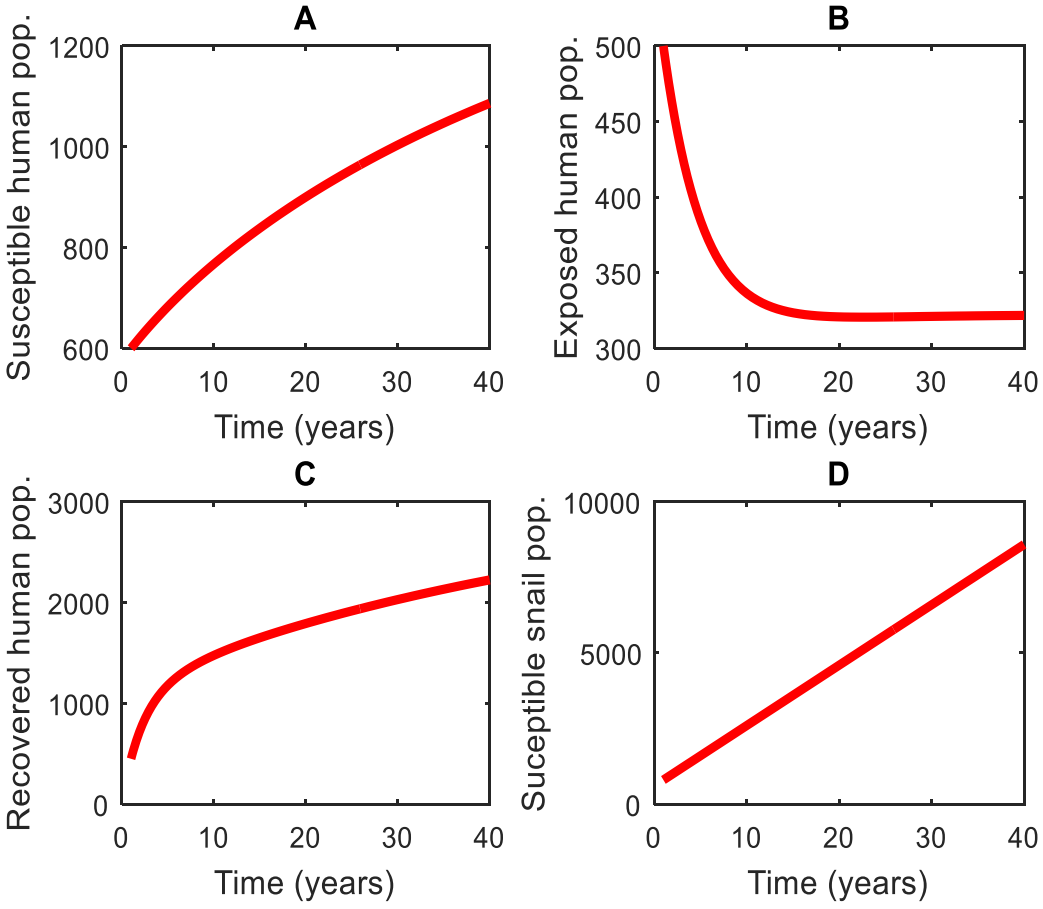

Figure 1. Simulation of some compartments with time. Figure 1 (A) is the simulation of susceptible human population with time. The figure indicates an initial increase in the population, then a decrease in the population due to the infection and natural death. Figure 1(B) is the simulation of exposed human population, the figure shows that the population is been depleted due to graduation to acute population and natural death. Figure 1©, simulation of recovered human population, the figure shows initial increase in the population due to treatment. Figure 1(D), simulation of susceptible snail population, this figure show a steady increase in the population.

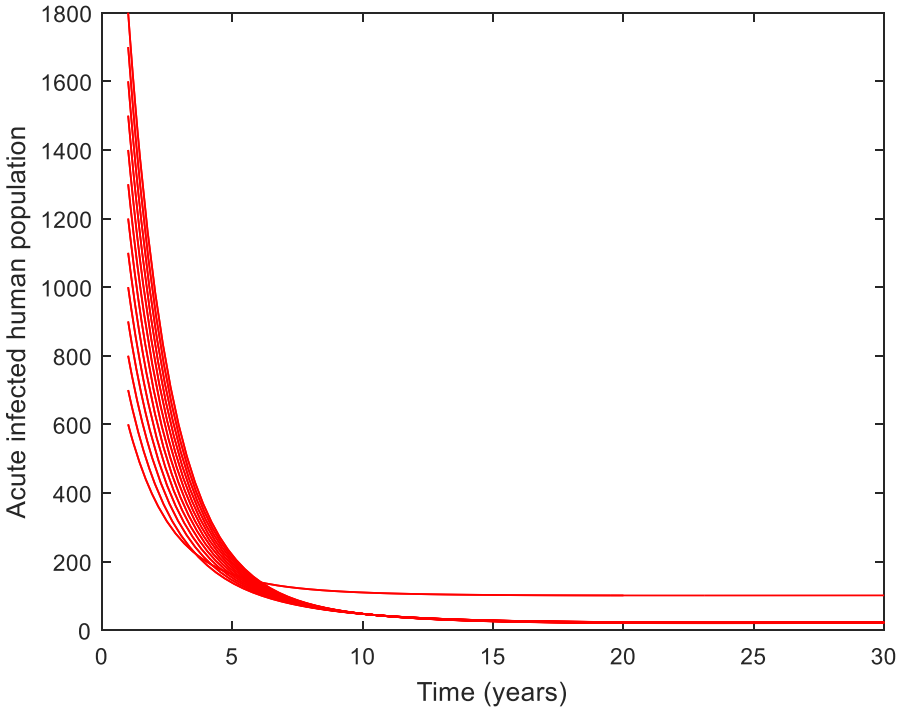

Figure 2. Simulation of the acute infected population with time.

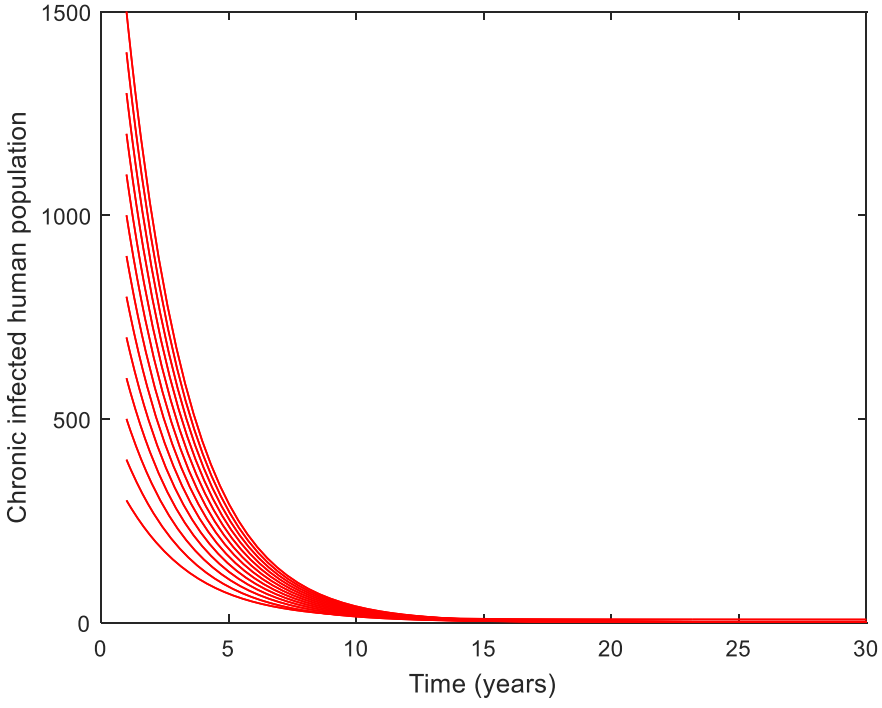

Figure 3. Simulation of the chronic infected population with different initial conditions

3. CONCLUSIONS

A *schistosomisis* transmission model within human and snail population was developed using standard incidence approach and analyzed. The disease free and endemic equilibria were obtained and analyzed for both local and global asymptotically stability. The analysis shows that the model undergoes backward bifurcation when the effective basic reproductive number $R < 1$.

The following results were obtained:

(i) The model reveals that at for certain small initial condition the disease will persist in the population (see Figure 2) which is rather unexpected result. This is however in line with the bifurcation result.

(ii) The simulation of the model suggests the need to carry out routine treatment in endemic areas since the long incubation period was obvious as symptoms did not manifest until after some weeks (see Figures 2 and 3).

(iii) The snail population figure 1, exhibits a kind of linear property which suggest that the population cannot go extinct. In other words control and eradication can will be guaranteed by provision of portable water for domestic purposes, avoidance of infected water bodies and sustained regular treatment.

(iv) The bifurcation analysis indicates that the model undergoes subcritical (backward bifurcation). When the associated basic reproductive number is less than one.

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Appendix A

Consider the following general system of ordinary differential equations with a parameter ϕ.

$$\frac{dx}{dt} = f(x,\phi): R^n \times R \to R^n \quad \text{and} \quad f \varepsilon C^2(R^n \times R) \qquad (24)$$

where 0 is an equilibrium point of the system (that is, $f(0,\phi) = 0$ for all ϕ) and

(A1) $A = D_x f(0,0) = \left(\frac{\partial f_i}{dx_j}(0,0) \right)$ is the linearization matrix of the system (24) around the equilibrium 0 with ϕ evaluated at 0;

(A2) Zero is a simple eigenvalues of A and other eigenvalues of A have negative real parts;

(A3) Matrix A has a right eigenvector w and left eigenvector v (each corresponding to zero eigenvalues).

Let f_k be the kth component of f and:

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

$$b = \sum_{k,i,=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial x_\phi}(0,0)$$

then, the local dynamics of the system (24) around equilibrium point 0 is totally determined by the signs of a and b, particularly,

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

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

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