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MATHEMATICAL ANALYSIS OF SCHISTOSOMIASIS WITH CASE DETECTION

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Abstract

Recently, WHO reported that 240 million people are infected with the schistosomiasis in the world with over 700 million people living in endemic areas. Reports also show that Nigeria has the highest cases of schistosomiasis in the world with over 30 million infected persons expected to be treated every year. Several deterministic population models have been formulated to study the dynamics of schistosomiasis in human, snails and the parasites population. In this study, we present a nonlinear mathematical model to provide mathematical and epidemiological insight to the effect of case detection on the transmission dynamics of schistosomiasis. The qualitative properties of the the model as well as the local and global asymptotic stability of equilibria are established. The existence of backward bifurcation is investigated. Furthermore, the disease-free equilibrium of the model was shown to be globally asymptotically stable (GAS) whenever the related effective reproduction number, $\mathfrak{R}_0$, is less than unity; this implies that schistosomiasis cannot prevail in the population. Moreover, we established the global asymptotic stability of the endemic equilibrium when the associated reproduction number $\mathfrak{R}_0$ is greater than one. This suggests that schistosomiasis will prevail in the population. Numerical simulations of the model showed the effect of varying some parameters of the model on the population dynamics of schistosomiasis.

Keywords: Disease free equilibrium, endemic equilibrium point, local asymptotic stability, global asymptotic stability, reproduction number, schistosomiasis.

1. Introduction

Schistosomiasis is a rapid, sensitive and chronic parasitic disease produced as a result of the blood flukes (trematode worms) of the genus *Schistosoma* [1-4]. Schistosomiasis being the incidence and susceptibility condition to death for developing nations in Africa, Caribbean, Middle East, South America, and Asia. The death toll from schistosomiasis is considered to be second only to malaria as the most devastating parasitic disease and one of the neglected tropical diseases (NTDs) [5]. WHO reported that about 240 million people are suffering from schistosomiasis in the world with 700 million people and above living in tropical and sub-tropical regions, in rural areas and areas with poor sanitation [1-4]. Africa has the highest cases of schistosomiasis with an estimated 85% of the world's schistosomiasis cases where more than 50% people live in endemic areas [6-8]. Nigeria is the leading country suffering from schistosomiasis in the sub-Saharan region of Africa and even in the entire world [8-11] with about 30 million Nigerians estimated to need treatment every year [9]. There are two major forms of schistosomiasis, intestinal and urogenital, and it is caused by 5 main species of blood fluke, namely: ***Schistosoma mansoni***, ***Schistosoma japonicum***, ***Schistosoma mekongi***, ***Schistosoma guineensis*** and ***Schistosoma haematobium*** [2-4]. These parasites possess in common some of their life-cycle characteristics, for example, eggs through excreta (urine or faeces) deposited by people infected with schistosomiasis into water bodies hatch on contact with water and then develop into **miracidia**; the miracidia are attracted to certain intermediate snails host through chemical sensors, and they must find and enter a suitable snail specie within 24 hours in order to survive, as the posibility of survival of the miracidia decreases quickly in all the schistosoma species after about ten hours [8]. The miracidia multiply inside the snail and the parasite progress into sporocysts, the sporocysts develop into cercaria, the free-swimming larval period of development of the parasite after the first to the second months within the snail. The cercaria is similar to the miracidia in terms of their short life span characteristics of about 2 days to penetrate man or a few distinctive sensitive mammalian

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explicitly host that come in contact with the infected water [12]. Schistosomiasis (or bilharzia) is unusually considered among helminth diseases because much of the origination and development of the disease is due to the eggs and not in larvae or adults stage with most of the pathology caused by delayed-type hypersensitivity and granulomatous reactions. The period of infection is frequently differentiated into Acute, Migratory and Chronic phase [6, 13]. Patients during migratory phase exhibit no symptons, but responsive patients may experience transient dermatitis (swimmers itch), occasionally pulmonary lesions and pneumonitis [13], while the acute phase (also known as Katayama syndrome) occurs at the same time with the first egg release and is characterized by excessively sensitive responses (serum sickness due to overwhelming immune complex formation) leading to lymphadenopathy, aches, gastrointestinal discomfort, pyrexia, eosinophilia and fatigue [6, 14] and the chronic phase occurs in response to the cumulative deposition of fluke eggs in tissues and the host responses that progress against them, although the eggs laid by female worms are not all successfully penetrating the gut or bladder walls, some are caught in organs where strong granulomatous responses was evoked and were taken away in the circulation [15, 16].

Schistosomiasis is diagnosed through the discovering of parasite eggs in urine or stool specimens, the antigens discovered in blood or urine samples are also signs of infection [3-5]. Urogenital schistosomiasis, which is geographically distributed in Africa and the Middle East countries is detected through the filtration technique diagnosed using paper, nylon, chemical reagents strips and polycarbonate filters [3-6, 16]. Fecal and urine specimens is used to detect the eggs of intestinal schistosomiasis through a practical method using methylene blue-stained cellophane immersed in glycerin or glass slides, known as the Kato-Katz technique and CCA (Circulating Cathodic Antigen) test [3-6, 17]. However, Polymerase chain reaction (PCR) tests and Blood tests are also effective in establishing the diagnosis of schistosomiasis but this test may not be positive until the parasites have penetrated into the skin for about two months because it usually takes time for the egg to develop and stimulate the system of an infected individual [16]. In some chronic cases of schistosomiasis, eggs are not always present in the fecal or in the urine specimens, other tests and procedures like liver biopsy, cytoscope, colonoscopy and endoscopy may be adopted to obtain the tissue biopsy materials [16]. In addition, CT scan, chest X-rays, ultrasound, echocardiograms, liver function tests , and complete blood count (CBC) may be used to ascertain the extent of the infection or if there has been damage caused by the parasites [16]. Praziquantel has been the recommended drug for the treatment of all types of schistosomiasis, because it is safe, its treatment is simple and it is not expensive [18]. Naji and Majeed [19] studied the transmission dynamics of schistosomiasis with the existence of treatment. They described a mathematical model where saturated treatment function $\frac{\beta y}{1+\alpha_0 y}$was used instead of the natural recovered term [19]. Chen et al. [20] proposed and analyzed a schistosomiasis mathematical model for human-cattle-snail transmission, emphasizing Hubei province, together with Anhui, Hunan, Jiangsu, and Jiangxi in the eastern and central China where schistosomiasis is endemic [20]. Chiyaka and Garira [21] developed a deterministic mathematical model to study schistosomiasis transmission behaviors in the Human-snail hosts population with the inclusion of the characteristics of the interaction between miracidia and cercariae. They concluded that the control strategies that is centered on the transmission of the disease from snail to human will be more effective in the control of the disease than preventing the transmission from man to snail [21]. Li et al. [22] constructed a periodic transmission rates deterministic model based on the monthly data delivered by the Chinese Center for Disease Control and Prevention (China CDC) on the cases of human schistosomiasis in the following provinces, Hubei, Hunan and Anhui (Lake and marshland region), they suggested to control human schistosomiasis, hygiene, education, treatment of at risk populations groups, snail control and improving sanitation will be an effective measures in these lakes and marshland region [22]. Remais [23] presented a mathematical model of the transmission of schistosomiasis involving several Environmental phases, he applied human understanding to the environmental determinants of the infectivity, viability, longevity and morbidity of these environmental stages to control the disease [23]. Adekiya et al. [24] examined the impact of rainfall and temperature on schistosomiasis; he showed that climatic change improves the reproduction number of schistosomes and the reproduction rate of snails [24]. Aboudrame et al. [25] considered a nonlinear deterministic model based on the transmission of schistosomiasis with two general incidence functions and delays.

In this paper, a deterministic mathematical model that investigates the impact of case detection on the dynamics of schistosomiasis in a given population is developed.

2. The Mathematical Model

This population model is divided into five human sub-populations, two-snail sub-populations and two parasites states. The human sub-populations are susceptible individuals (S_h) who are open to contract the disease, exposed (individuals who are already infected but not infectious) (E_h), undetected infected individuals (infectious but are not yet detected of the disease) (I_{h1}), detected infected (individuals who have been infected, infectious and also detected of the disease) (I_{h2}), and treated humans (individuals who have been treated of the disease) (T_h). The two snail epidemiological state are susceptible snails (S_s) that is capable of contracting the parasite miracidia and infected snails (I_s) that has contracted miracidia and is able to

release another parasite called cercaria. There are two parasites states; miracidia (free-swimming ciliated larval) class (M_s) and cercariae class (J_s). The recruitment into the population is through influx of individuals into the human susceptible class at the rate of Λ_h and influx of snails into the susceptible snails compartment at the rate of Λ_s. The population get infected with the disease when the parasites (cercaria) discharged by the snails penetrate a susceptible human skin during contact with infected fresh water at the rate λ_j, given by

$$\lambda_j = \frac{\beta_j J_s}{J_0 + \epsilon J_s} \qquad (1)$$

and the snail population also get infected when the free-swimming ciliated larval (miracidia) penetrate the snail at the rate λ_m given by,

$$\lambda_m = \frac{\beta_m M_s}{M_0 + \epsilon M_s}, \qquad (2)$$

where β_j is the cercaria penetration rate, β_m is the miracidia infection rate, J_0 is the carrying capacity for cercaria, M_0 is the carrying capacity for miracidia and ϵ is the limiting growth factor for cercaria and miracidia. The population is infected with schistosomiasis when the parasites (cercaria) discharged by the snails penetrate a susceptible human skin during contact with infected fresh water, the newly infected susceptible human and a portion ω of treated human re-infected with schistosomiasis are assumed to progressively move to the Exposed class E_h, individuals in this class of the population is believed to progressively move out from the latent periods to the active infected periods at the rate v. A fraction of the detected cases of the active infected persons with schistosomiais (pvE_h) moved to the detected infected class I_{h2} with schistosomiasis per unit time. The remaining fraction of the active infected persons with schistosomiaisis $(1-p)vE_h$ progressed to the undetected infected class I_{h1} of the population and the population reducing as a result of the disease-induced deaths (d_0, d_1) and the proportion (η_0) of the detected schistosomiasis patient commencing treatment. The population of individuals treated of schistosomiaisis (T_h) increases due to treatment of detected ($\eta_0 I_{h2}$) persons, the treated individuals may be re-infected at the rate ($\omega\lambda_j$) and the miracidia (free-swimming ciliated larval) population increases as a portion n_e of the parasites egg secreted by both the undetected and detected infected individuals, contaminate the fresh water with their feaces or urine which hatch into miracidia, the free-swimming ciliated larval at the rate ζ and all the human population dies naturally at the rate μ_h. The miracidia finds and penetrate its suitable species of a fresh water snail and change it into a sporocyst, if in anyway the miracidia failed to find a suitable snail, it dies naturally at the rate μ_m. The snail population is infected at the rate λ_m as the free-swimming larval known as miracidia penetrate into the susceptible snails and also die naturally at the rate μ_s. The infected snails will then release another form of free swimming larva called cercaria at a rate θ which will infect humans and may also die naturally at the rate μ_j.

The assumptions above are combined to form the model for the dynamics of the schistosomes parasite transmission as

$$\begin{aligned}
S_h' &= \Lambda_h - \frac{\beta_j S_h J_s}{J_0 + \epsilon J_s} - \mu_h S_h,\\
E_h' &= \frac{\beta_j J_s}{J_0 + \epsilon J_s}(S_h + \omega T_h) - (v + \mu_h)E_h,\\
I_{h1}' &= (1-p)vE_h - (d_0 + \mu_h)I_{h1},\\
I_{h2}' &= pvE_h - (\eta_0 + d_1 + \mu_h)I_{h2},\\
T_h' &= \eta_0 I_{h2} - \frac{\omega\beta_j J_s T_h}{J_0 + \epsilon J_s} - \mu_h T_h, \qquad (3)\\
M_s' &= n_e\zeta(I_{h1} + I_{h2}) - \mu_m M_s,\\
S_s' &= \Lambda_s - \frac{\beta_m M_s S_s}{M_0 + \epsilon M_s} - \mu_s S_s,\\
I_s' &= \frac{\beta_m M_s S_s}{M_0 + \epsilon M_s} - \mu_s I_s\\
J_s' &= \theta I_s - \mu_j J_s.
\end{aligned}$$

The total human population is represented by $N_h = S_h + E_h + I_{h1} + I_{h2} + T_h$ while the total snail population in the environment is accounted for by $N_s = S_s + I_s$.

A schematic representations of the system is given in figure

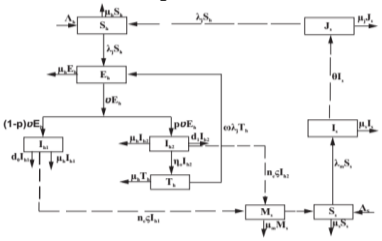

Figure 1: Schematic diagram of our schistosomiasis model (3).

2.1 Positivity and Boundedness of Solutions

Theorem 1: Let the initial data for the tuberculosis-schistosomiasis co-infection model be given as $S_h(0) > 0, E_h(0) > 0, I_{h1}(0) > 0, I_{h2}(0) > 0, T_h(0) > 0, M_s(0) > 0, S_s(0) > 0, I_s(0) > 0$ and $J_s(0) > 0$. Then the orbits $(S_H(t), E_h(t), I_{h1}(t), I_{h2}(t), T_h(t), M_s(t), S_s(t), I_s(t), J_s(t))$ of the model with positive initial conditions, will continue to be positive for all time $t > 0$.

Proof: Let $t_1 = \sup\{t > 0: S_h(0) > 0, E_h(0) > 0, I_{h1}(0) > 0, I_{h2}(0) > 0, T_h(0) > 0, M_s(0) > 0, S_s(0) > 0, I_s(0) > 0, J_s(0) > 0\}$. Consider the first equation of model (3), given below as

$$\frac{dS_h(t)}{dt} = \Lambda_h - (\lambda_j + \mu_h)S_h(t), \tag{4}$$

which can be re-expressed as

$$\frac{d}{dt}[S_h(t)\exp\{\mu_h t + \int_0^t \lambda_j(\tau)\ d\tau\}] \geq \Lambda_j \exp\left\{\mu_j t + \int_0^t \lambda_j(\tau) d\tau\right\}. \tag{5}$$

$$S_h(t_1)\exp\{\mu t_1 + \int_0^{t_1} \lambda_j(\tau)\ d\tau\} - S_h(0) \geq \int_0^{t_1} \Lambda_h \left[\exp\left\{\mu_h y + \int_0^y \lambda_j(\tau) d\tau\right\}\right] dy, \tag{6}$$

So that,

$$S_h(t_1) \geq S_h(0)\exp\ [-\mu_h t_1 - \int_0^{t_1} \lambda_j(\tau) d\tau] + [\exp\{-\mu_h t_1 - \int_0^{t_1} \lambda_j(\tau) d\tau\}] \times \int_0^{t_1} \Lambda_h \left[\exp\left\{\mu_h y + \int_0^y \lambda_j(\tau) d\tau\right\}\right] dy > 0. \tag{7}$$

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

Similarly, it can be shown that $E_h(t) > 0,\ I_{h1}(t) > 0,\ I_{h2}(t) > 0,\ T_h(t) > 0\ ,\ M_s(t) > 0,\ S_s(t) > 0,\ I_s(t) > 0,\ J_s(t) > 0, \forall\ t > 0$.

Theorem 2: Let $(S_h(t), E_h(t), I_{h1}(t), I_{h2}(t), T_h(t), M_s(t), S_s(t), I_s(t), J_s(t))$ be trajectories of the system with initial conditions and the biological feasible region given by the set $\mathcal{D}_1 = \mathcal{D}_h \times \mathcal{D}_m \times \mathcal{D}_s \times \mathcal{D}_j \subset \mathbb{R}_+^5 \times \mathbb{R}_+^1 \times \mathbb{R}_+^2 \times \mathbb{R}_+^1 \subset \mathbb{R}_+^9$, where:

$$\mathcal{D}_h = \{(S_h, E_h, I_{h1}, I_{h2}, T_h) \in \mathbb{R}_+^5 : N_h \leq \frac{\Lambda_h}{\mu_h}\}$$

$$\mathcal{D}_m = \{M_s \in \mathbb{R}_+^1 : L \leq \frac{n_e \zeta \Lambda_h}{\mu_m \mu_h}\}$$

$$\mathcal{D}_s = \{(S_s, I_s) \in \mathbb{R}_+^2 : N_s \leq \frac{\Lambda_s}{\mu_s}\}$$

$$\mathcal{D}_j = \{J_s \in \mathbb{R}_+^1 : J_s \leq \frac{\theta \Lambda_s}{\mu_j \mu_s}\}$$

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

Proof: Adding up the right flank of the vector field for the human population in (3), yields

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - d_0 I_{h1} - d_1 I_{h2}. \tag{8}$$

From (8), it follows that $\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h$. Hence, $\frac{dN_h}{dt} \leq 0$ if $N_h(t) \geq \frac{\Lambda_h}{\mu_h}$. Employing a standard comparison theorem [26], we prove that $N_h(t) \leq N_h(0)e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}(1 - e^{-\mu_h t})$. In particular, if $N_h(0) \leq \frac{\Lambda_h}{\mu_h}$, thus $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ for every $t > 0$. Hence, the set $\mathcal{D}_h$ is positively invariant. Moreover, if $N_h(0) > \frac{\Lambda_h}{\mu_h}$, then either the orbits enters the domain $\mathcal{D}_h$ in finite time or $N_h(t)$ asymptotically tends towards $\frac{\Lambda_h}{\mu_h}$ as $t \to \infty$. Thus, the domain $\mathcal{D}_h$ attracts every orbit in $\mathbb{R}_+^5$.

$$\frac{dM_s}{dt} = n_e\zeta(I_{h1} + I_{h2}) - \mu_s M_s. \quad (9)$$

From (9), which follows that $\frac{dM_s}{dt} \leq \frac{n_e\zeta\Lambda_h}{\mu_h} - \mu_m M_s$ since $N_h = S_h + E_h + I_{h1} + I_{h2} + T_h \leq \frac{\Lambda_h}{\mu_h} \Rightarrow I_{h1} + I_{h2} \leq \frac{\Lambda_h}{\mu_h}$. Hence, $\frac{dM_s}{dt} \leq 0$ if $M_s(t) \geq \frac{n_e\zeta\Lambda_h}{\mu_s\mu_h}$. Employing a standard comparison theorem [26], we prove that $M_s(t) \leq M_s(0)e^{-\mu_m t} + \frac{n_e\zeta\Lambda_h}{\mu_s\mu_h}(1 - e^{-\mu_m t})$. In particular, if $M_s(0) \leq \frac{n_e\zeta\Lambda_h}{\mu_s\mu_h}$, then $M_s(t) \leq \frac{n_e\zeta\Lambda_h}{\mu_s\mu_h}$ for all $t > 0$. Hence, the set $\mathcal{D}_m$ is positively invariant. Moreover, if $M_s(0) > \frac{n_e\zeta\Lambda_h}{\mu_s\mu_h}$, then either the orbits enters the domain $\mathcal{D}_m$ in finite time or $M_s(t)$ asymptotically approaches $\frac{n_e\zeta\Lambda_h}{\mu_s\mu_h}$ as $t \to \infty$. Thus, the domain $\mathcal{D}_m$ attracts every trajectory in $\mathbb{R}^1_+$.

For the snail population, we add up the right flank of the vector field of the snail population in (3), which gives

$$\frac{dN_s}{dt} = \Lambda_s - \mu_s N_s. \quad (10)$$

From (10), it follows that $\frac{dN_s}{dt} \leq 0$ if $N_s(t) \geq \frac{\Lambda_s}{\mu_s}$. Consequently, $N_s(t) = N_s(0)e^{-\mu_s t} + \frac{\Lambda_s}{\mu_s}(1 - e^{-\mu_s t})$. Then the $\limsup_{t\to\infty} N_s(t) = \frac{\Lambda_s}{\mu_s}$. In particular, if $N_s(0) \leq \frac{\Lambda_s}{\mu_s}$, then $N_s(t) \leq \frac{\Lambda_s}{\mu_s}$ for every $t > 0$. Hence, the set $\mathcal{D}_s$ is positively invariant. Moreover, if $N_s(0) > \frac{\Lambda_s}{\mu_s}$, then either the orbits enters the domain $\mathcal{D}_s$ in finite time or $N_s(t)$ asymptotically approaches $\frac{\Lambda_s}{\mu_s}$ as $t \to \infty$. Thus, the domain $\mathcal{D}_s$ attracts every trajectory in $\mathbb{R}^2_+$.

For the concentration of the cercariae, we consider the right flank of the vector field J in (3), yields

$$\frac{dJ_s}{dt} = \theta I_s - \mu_j J_s. \quad (11)$$

From (11), $\frac{dJ_s}{dt} = \theta I_s - \mu_j J_s$ which follows that $\frac{dJ_s}{dt} \leq \frac{\theta\Lambda_s}{\mu_s} - \mu_j J_s$ since $N_s = S_s + I_s \leq \frac{\Lambda_s}{\mu_s} \Rightarrow I_s \leq \frac{\Lambda_s}{\mu_s}$. Hence, $\frac{dJ_s}{dt} \leq 0$ if $J(t) \geq \frac{\theta\Lambda_s}{\mu_j\mu_s}$. Employing a standard comparison theorem [26], we prove that $J_s(t) \leq J_s(0)e^{-\mu_j t} + \frac{\theta\Lambda_s}{\mu_j\mu_s}(1 - e^{-\mu_j t})$. In particular, if $J_s(0) \leq \frac{\theta\Lambda_s}{\mu_j\mu_s}$, then $J_s(t) \leq \frac{\theta\Lambda_s}{\mu_j\mu_s}$ for all $t > 0$. Hence, the set $\mathcal{D}_j$ is positively invariant. Moreover, if $J_s(0) > \frac{\theta\Lambda_s}{\mu_j\mu_s}$, then either the orbits enters the domain $\mathcal{D}_j$ in finite time or $J_s(t)$ asymptotically approaches $\frac{\theta\Lambda_s}{\mu_j\mu_s}$ as $t \to \infty$. Thus, the domain $\mathcal{D}_j$ attracts every trajectory in $\mathbb{R}^1_+$.

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

3 Mathematical Analysis of the Model

We establish the Local and global stability of the disease-free equilibrium (DFE) and endemic equilibrium (EEP) in this section.

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

At the disease-free state, the cercaria and miracidia states do not exist, then the host and the intermediate host do not have any infection. Thus, the disease-free equilibrium for (3) is

$$U_0 = (S_h^0,\ E_h^0,\ I_{h1}^0,\ I_{h2}^0,\ T_h^0,\ M_s^0,\ S_s^0,\ I_s^0,\ J_s^0) = \left(\frac{\Lambda_h}{\mu_h},\ 0,\ 0,\ 0,\ 0,\ 0,\ \frac{\Lambda_s}{\mu_s},\ 0,\ 0\right) \quad (12)$$

The effective reproduction number $\Re_0$, is defined as the expected number of secondary cases produced in a totally sensitive population by a typical infective individual during infectious period at a disease free equilibrium. The effective reproduction number is used to ascertain the transmission ability of a disease. The reproduction number is affected by the rate of contacts in the host population, the probability of infection transmission during contact and the contagious duration, hence, it follows that matrices f and k, representing transfer rate of individuals into new compartments and out of the compartments at DFE can be expressed as,

$$F_0 = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & \frac{\beta_j\Lambda_h}{J_0\mu_h} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_m\Lambda_s}{M_0\mu_s} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (13)$$

and

$$K = \begin{pmatrix} (v+\mu_h) & 0 & 0 & 0 & 0 & 0 \\ (-1+p)v & (d_0+\mu_h) & 0 & 0 & 0 & 0 \\ -pv & 0 & (d_0+\eta_0+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & \mu_s & 0 & 0 \\ 0 & -\zeta n_e & -\zeta n_e & 0 & \mu_m & 0 \\ 0 & 0 & 0 & -\theta & 0 & \mu_j \end{pmatrix} \quad (14)$$

respectively. Thus, it follows that the effective reproduction number of the model (3) denoted by $\mathfrak{R}_0$, is obtained as,

$$\mathfrak{R}_0 = \rho(F_0K^{-1}) = \sqrt{\frac{\zeta\theta v n_e \beta_j \beta_m \Lambda_h \Lambda_s((1-p)(\eta_0+d_1+\mu_h)+p(d_0+\mu_h))}{J_0 M_0 \mu_h (v+\mu_h)(d_0+\mu_h)(d_1+\eta_0+\mu_h)\mu_j \mu_m \mu_s^2}} \quad (15)$$

where, $\rho(F_0K^{-1})$ is the matrix (F_0K^{-1}) spectral radius [27]. Thus, it is observed in this case that,

$$\mathfrak{R}_0 = \sqrt{\mathfrak{R}_{0,h}\mathfrak{R}_{0,s}} \quad (16)$$

where,

$$\mathfrak{R}_{0,h} = \frac{\zeta v n_e \beta_j \Lambda_h((1-p)(\eta_0+d_1+\mu_h)+p(d_0+\mu_h))}{J_0\mu_h(v+\mu_h)(d_0+\mu_h)(d_1+\eta_0+\mu_h)\mu_m} \text{ and } \mathfrak{R}_{0,s} = \frac{\beta_m\theta\Lambda_s}{\mu_j\mu_s^2 M_0} \quad (17)$$

From the above generated effective reproduction number $\mathfrak{R}_0$, it is observed that the $\mathfrak{R}_0$ is obtained as a geometric mean of two quantities because an infected individual introduced into an entirely susceptible population generates $\mathfrak{R}_0$ infections during his/her infectious period [27]. Schistosomiasis passes through a snail before another human can get infected, therefore the number of new infections is obtain by the geometric mean of $\mathfrak{R}_{0,h}$ and $\mathfrak{R}_{0,s}$. If we consider an infectious individual, present in a population at the disease free equilibrium, then the likely number of snails infected by this person, that is, the man-snail transmission coefficient is approximately

$$\mathfrak{R}_{0,s} = \frac{\beta_m\theta\Lambda_s}{\mu_j\mu_s^2 M_0} \quad (18)$$

Similarly, if we consider an infectious snail entering and present in a population at the disease free equilibrium, then the likely number of humans infected by this snail that is, the snail-man transmission coefficient, is approximately

$$\boldsymbol{\mathfrak{R}_{0,h} = \frac{\zeta v n_e \beta_j \Lambda_h((1-p)(\eta_0+d_1+\mu_h)+p(d_0+\mu_h))}{J_0\mu_h(v+\mu_h)(d_0+\mu_h)(d_1+\eta_0+\mu_h)\mu_m}} \quad (\mathbf{19})$$

Lemma 3: *The disease-free equilibrium point of the system (3), U_0, is LAS whenever $\mathfrak{R}_0 < 1$ and unstable otherwise. Thus we can deduce that there is possibility of eliminating the disease from the population when the effective reproduction number $\mathfrak{R}_0 < 1$.*

3.1.1 Examination of $\mathfrak{R}_0$

The impact of some parameters is evaluated by examining the effective reproduction number with respect to the sensitivity of the progression rate from latently to actively infected with schistosomiasis v, the case detection p, and the schistosomiasis treatment rate η_0. If we consider the change of $\mathfrak{R}_0^2$ with respect to v, we have

$$\frac{\partial\mathfrak{R}_0^2}{\partial v} = \frac{\zeta\theta n_e\beta_j\beta_m\Lambda_h\Lambda_s(p(d_0+\mu_h)+(1-p)(\eta_0+d_1+\mu_h))}{J_0M_0(v+\mu_h)^2(d_0+\mu_h)(\eta_0+d_1+\mu_h)\mu_j\mu_m\mu_s^2} > 0 \quad (20)$$

Clearly, from (12), $\frac{\partial\mathfrak{R}_0^2}{\partial v} > 0$, this implies that schistosomiasis infection progression rate from latently to actively infected cases on the dynamics of schistosomiasis will have negative impact.

Also, if we consider how $\mathfrak{R}_0^2$ changes relative to p, we have

$$\frac{\partial\mathfrak{R}_0^2}{\partial p} = \frac{v\zeta\theta n_e\beta_j\beta_m(d_0-d_1-\eta_0)\Lambda_h\Lambda_s}{J_0M_0\mu_h(v+\mu_h)(d_0+\mu_h)(\eta_0+d_1+\mu_h)\mu_j\mu_m\mu_s^2} \quad (21)$$

Thus,

1. If $d_0 < (d_1+\eta_0)$ (i.e. $d_0-d_1 < \eta_0$), then $\frac{\partial\mathfrak{R}_0^2}{\partial p} < 0$; since $\mathfrak{R}_0$ is a decreasing function of p, then detection (as well as quick commencement of treatment) of a proportion of persons infected with schistosomiasis will have a positive impact (reduced the burden of the disease) in a population.
2. If $d_0-d_1 > \eta_0$, then $\frac{\partial\mathfrak{R}_0^2}{\partial p} > 0$; since $\mathfrak{R}_0$ is an increasing function of p, then case detection of schistosomiasis will have a negative impact (will not reduce the burden of the disease) in the population.
3. If $d_0 = d_1$, this implies that $\frac{\partial\mathfrak{R}_0^2}{\partial p} < 0$; since $\mathfrak{R}_0$ is a decreasing function of p. Hence, case detection of schistosomiasis will have a positive impact on the dynamics of the disease.

Also if we consider how $\Re_0^2$ changes in respect to η_0, that is,

$$\frac{\partial \Re_0^2}{\partial \eta_0} = \frac{-pv\zeta\theta n_e \beta_j \beta_m \Lambda_h \Lambda_s}{J_0 M_0 \mu_h (v+\mu_h)(\eta_0 + d_1 + \mu_h)^2 \mu_j \mu_m \mu_s^2} < 0. \quad (22)$$

Clearly, $\frac{\partial \Re_0^2}{\partial \eta_0} < 0$, this implies that if the proportion of detected persons with schistosomiasis, promptly treated is large, it will result into reduction in schistosomiasis burden in a population.

3.2 Existence of the Endemic Equilibrium Point (EEP)

Let the endemic equilibrium point (EEP) corresponding to the system (3) be defined as

$$U^{**} = (S_h^{**},\ E_h^{**},\ I_{h1}^{**},\ I_{h2}^{**},\ T_h^{**},\ M_s^{**},\ S_s^{**},\ I_s^{**},\ J_s^{**}). \quad (23)$$

and the non-trivial endemic equilibrium as,

$$\begin{aligned} S_h^{**} &= \frac{\Lambda_h}{\lambda_j^{**} + \mu_h}, \quad E_h^{**} = \frac{\lambda_j^{**}(S_h^{**} + \omega T_h^{**})}{(v+\mu_h)}, \quad I_{h1}^{**} = \frac{(1-p)vE_h^{**}}{(d_0+\mu_h)} \\ I_{h2}^{**} &= \frac{pvE_h^{**}}{(\eta_0 + d_1 + \mu_h)}, \quad T_h^{**} = \frac{\eta_0 I_{h2}^{**}}{\omega\lambda_j^{**} + \mu_h}, \quad M_s^{**} = \frac{n_e\zeta(I_{h1}^{**} + I_{h2}^{**})}{\mu_m} \\ S_s^{**} &= \frac{\Lambda_s}{(\lambda_m^{**} + \mu_s)}, \quad I_s^{**} = \frac{\lambda_m^{**} S_s^{**}}{\mu_s}, \quad J_s^{**} = \frac{\theta I_s^{**}}{\mu_j} \end{aligned} \quad (24)$$

The endemic cercaria force of infection polynomial λ_j^{**} is computed by considering

$$\lambda_j^{**} = \frac{\beta_j J_s^{**}}{J_0 + \epsilon J_s^{**}} \quad (25)$$

and

$$\lambda_m^{**} = \frac{\beta_m M_s^{**}}{M_0 + \epsilon M_s^{**}} \quad (26)$$

then the EEP associated with the system (3) satisfy the following polynomial written as a function of λ_j^{**} as,

$$C_1^0 \lambda_j^{**2} + C_2^0 \lambda_j^{**} + C_3^0 = 0 \quad (27)$$

where,

$$\begin{aligned} C_1^0 &= J_0 M_0 \beta_j ((1-p)v\omega\eta_0 + v\omega(d_1+\mu_h) + \omega\mu_h(\eta_0 + d_1 + \mu_h))\mu_j\mu_s^2 \\ &+ J_0 M_0 \beta_j ((1-p)v\omega\eta_0 + v\omega(d_1+\mu_h) + \omega\mu_h(\eta_0 + d_1 + \mu_h)) \\ &+ \omega J_0 \beta_m \mu_h (v+\mu_h)(\eta_0 + d_1 + \mu_h)\big(\epsilon\theta\Lambda_s + J_0\mu_j\mu_s\big)\Re_{0,h} \end{aligned} \quad (29)$$

$$\begin{aligned} C_2^0 &= J_0 M_0 \beta_j (\mu_h(v+\mu_h)(\eta_0 + d_1 + \mu_h))\mu_j\mu_s^2 \\ &+ \mu_h J_0 M_0 \beta_j ((1-p)v\omega\eta_0 + v\omega\eta_0 + v\omega(d_1+\mu_h) + \omega\mu_h(\eta_0 + d_1 + \mu_h))\mu_j\mu_s^2 \\ &- \theta\omega J_0 \beta_j \beta_m \Lambda_s \mu_h (v+\mu_h)(\eta_0 + d_1 + \mu_h)\Re_{0,h} \\ &+ \epsilon J_0^2 \mu_h^2 (v+\mu_h)(\eta_0 + d_1 + \mu_h)\mu_j\mu_s^2\Re_{0,h} \\ &+ J_0 \beta_m \mu_h^2 (v+\mu_h)(\eta_0 + d_1 + \mu_h)\big(\epsilon\theta\Lambda_s + J_0\mu_j\mu_s\big)\Re_{0,h} \end{aligned} \quad (30)$$

$$C_3^0 = J_0 M_0 \beta_m \mu_h^2 (v+\mu_h)(\eta_0 + d_1 + \mu_h)\mu_j\mu_s^2(1 - \Re_0^2). \quad (31)$$

We solved the polynomial in (26) to obtain the components of the EEP. Clearly, the coefficient C_1^0 is always positive (since $0 \le p \le 1$) , it also follows from (26) that the coefficient C_3^0 is always positive if the reproduction number $\Re_0^2$ is less than one and always negative if the reproduction number $\Re_0^2$ is greater than one, therefore we establish the following theorem.

Theorem 4: Together with the above statement, the system (3) has:

1. a unique endemic equilibrium whenever $\Re_0^2 > 1$ and $C_2^0 < 0$
2. no endemic equilibrium whenever $\Re_0^2 < 1$ and $C_2^0 > 0$

3.2.1 Backward Bifurcation Analysis

Set the variables such that

$$S_h = x_1,\ E_h = x_2,\ I_{h1} = x_3,\ I_{h2} = x_4,\ T_h = x_5,\ M_s = x_6,\ S_s = x_7,\ I_s = x_8,\ J_s = x_9,$$

and rewrite equation (3) as

$$\frac{dX}{dt} = G = (g_1, g_2, g_3, g_4, g_5, g_6, g_7, g_8, g_9)^T,$$

such that,

$$
\begin{aligned}
x_1'(t) &= g_1 = \Lambda_h - \frac{\beta_j x_1 x_9}{J_0 + \epsilon x_9} - \mu_h x_1,\\
x_2'(t) &= g_2 = \frac{\beta_j x_9 (x_1 + \omega x_5)}{J_0 + \epsilon x_9} - (v + \mu_h) x_2,\\
x_3'(t) &= g_3 = (1-p) v x_2 - (d_0 + \mu_h) x_3,\\
x_4'(t) &= g_4 = p v x_2 - (\eta_0 + d_1 + \mu_h) x_4,\\
x_5'(t) &= g_5 = \eta_0 x_4 - \frac{\omega \beta_j x_5 x_9}{J_0 + \epsilon x_9} - \mu_h x_5,\\
x_6'(t) &= g_6 = n_e \zeta (x_3 + x_4) - \mu_m x_6,\\
x_7'(t) &= g_7 = \Lambda_s - \frac{\beta_m x_6 x_7}{M_0 + \epsilon x_6} - \mu_s x_7,\\
x_8'(t) &= g_8 = \frac{\beta_m x_6 x_7}{M_0 + \epsilon x_6} - \mu_s x_8,\\
x_9'(t) &= g_9 = \theta x_8 - \mu_j x_9.
\end{aligned} \tag{31}
$$

Suppose $\beta_j = \beta_j^*$ is chosen to be the bifurcation parameter for the system (31) then when $\Re_0 = 1$,we obtain

$$
\beta_j = \beta_j^* = \frac{J_0 M_0 \mu_h \mu_j \mu_m \mu_s^2 (v + \mu_h)(d_0 + \mu_h)(\eta_0 + d_1 + \mu_h)}{\beta_m \Lambda_h \Lambda_s n_e \zeta v \theta ((1-p)(\eta_0 + d_1 + \mu_h) + p(d_0 + \mu_h))} \tag{32}
$$

The Jacobian matrix associated to the model (31) at the DFE, is obtained as

$J(U_0)|_{\beta_j = \beta_j^*} =$

$$
\begin{bmatrix}
-\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_j^* \Lambda_h}{\mu_h J_0}\\
0 & -(v+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_j^* \Lambda_h}{\mu_h J_0}\\
0 & (1-p)v & -(d_0+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0\\
0 & pv & 0 & -(\eta_0 + d_1 + \mu_h) & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & \eta_0 & -\mu_h & 0 & 0 & 0 & 0\\
0 & 0 & n_e\zeta & n_e\zeta & 0 & -\mu_h & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & -\frac{\beta_m \Lambda_s}{\mu_s M_0} & -\mu_s & 0 & 0\\
0 & 0 & 0 & 0 & 0 & \frac{\beta_m \Lambda_s}{\mu_s M_0} & 0 & -\mu_s & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \theta & -\mu_j
\end{bmatrix} \tag{33}
$$

By the Centre Manifold Theorem in [28, 29], we investigate the possibility of backward bifurcation at $\Re_0 = 1$. We can show that the Jacobian $(J_{\beta_j^*})$ at $\beta_j = \beta_j^*$ of the system (31) possesses a right eigenvector given as $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9)^T$ by multiplying the Jacobian matrix (25) with, we further express each of the vectors in terms of w_9

$$
\begin{aligned}
w_1 &= \frac{-\beta_j \Lambda_h w_9}{\mu_h^2 J_0} \qquad w_2 = \frac{\beta_j \Lambda_h w_9}{\mu_h (v+\mu_h) J_0}, \qquad w_3 = \frac{(1-p) v \beta_j \Lambda_h w_9}{\mu_h (v+\mu_h)(d_0+\mu_h) J_0}\\
w_4 &= \frac{p v \beta_j \Lambda_h w_9}{\mu_h (v+\mu_h)(\eta_0 + d_1 + \mu_h) J_0}, \qquad w_5 = \frac{\eta_0 p v \beta_j \Lambda_h w_9}{\mu_h^2 (v+\mu_h)(\eta_0 + d_1 + \mu_h) J_0}\\
w_6 &= \frac{\mu_s^2 M_0 \mu_j w_9}{\theta \beta_m \Lambda_s}, \qquad w_7 = \frac{-\mu_j w_9}{\theta}, \qquad w_8 = \frac{\mu_j w_9}{\theta}
\end{aligned} \tag{34}
$$

Similarly, we can obtain that the Jacobian $(J_{\beta_j^*})$ at $\beta_j = \beta_j^*$ of the system (31) possesses a left eigenvector given as $u = [u_1,\ u_2,\ u_3,\ u_4,\ u_5,\ u_6,\ u_7,\ u_8,\ u_9]$ by multiplying the Jacobian matrix (33) with u and express each of the vectors in terms of u_9 as

$$
\begin{aligned}
&u_1 = u_7 = 0,\ u_2 = \frac{\mu_j \mu_h J_0 u_9}{\beta_j \Lambda_h},\ u_3 = \frac{\theta n_e \zeta \beta_m \Lambda_s u_9}{\mu_m \mu_s^2 (d_0 + \mu_h) M_0}\\
&u_4 = \frac{\theta n_e \zeta \beta_m \Lambda_s u_9}{\mu_m \mu_s^2 (\eta_0 + d_1 + \mu_h) M_0},\ u_5 = 0,\ u_6 = \frac{\theta \beta_m \Lambda_s u_9}{\mu_m \mu_s^2 M_0},\ u_8 = \frac{\theta u_9}{\mu_s}
\end{aligned} \tag{35}
$$

Using the expression derived by [29] for computation of a and b for the system (31), the corresponding non-zero partial derivatives is given as,

$$a = \sum_{k,i,j=1}^{n} u_k w_i w_j \frac{\partial^2 g_k(0,0)}{\partial x_i \partial x_j} \quad (36)$$

and

$$\text{b} = \sum_{k,i=1}^{n} u_k w_1 \frac{\partial^2 g_k(0,0)}{\partial x_i \partial \beta_j^*} \quad (37)$$

thus

$$a = 2u_2 w_9 w_1 \frac{\beta_j^*}{J_0} + 2u_2 w_5 w_9 \omega \frac{\beta_j^*}{J_0} - \frac{2\epsilon \beta_j^* x_1^* u_2 w_9^2}{J_0^2} - \frac{2\epsilon \beta_m x_7^* u_8 w_6^2}{M_0^2} + 2u_8 w_6 w_7 \frac{\beta_j^*}{M_0},$$

$$= \frac{2\omega \beta_j^* \mu_j \eta_0 p v u_9 w_9^2}{\mu_h (v+\mu_h)(d_1+\eta_0+\mu_h) J_0}$$
$$- \left(\frac{2\mu_j \beta_j^* u_9 w_9^2}{\mu_h J_0} + \frac{2\mu_j \mu_h \epsilon x_1^* u_9 w_9^2}{\Lambda_h J_0} + \frac{2\epsilon\theta \mu_s^3 \mu_j^2 x_7^* u_9 w_9^2}{\theta^2 \beta_m \Lambda_s^2} + \frac{2\mu_s \mu_j^2 u_9 w_9^2}{\theta \Lambda_s} \right) \quad (38)$$

Thus, $a > 0$ if and only if

$$\omega > \frac{H_6}{H_5}(H_1 + H_2 + H_3 + H_4) \quad (39)$$

where,

$$H_1 = \frac{2\mu_j \beta_j^* u_9 w_9^2}{\mu_h J_0},\ H_2 = \frac{2\mu_j \mu_h \epsilon x_1^* u_9 w_9^2}{\Lambda_h J_0},\ H_3 = \frac{2\epsilon\theta \mu_s^3 \mu_j^2 x_7^* u_9 w_9^2}{\theta^2 \beta_m \Lambda_s^2}$$
$$H_4 = \frac{2\mu_s \mu_j^2 u_9 w_9^2}{\theta \Lambda_s},\ H_5 = 2\beta \mu_j \eta_0 p v u_9 w_9^2,\ H_6 = \mu_h (v+\mu_h)(d_1+\eta_0+\mu_h) J_0 \quad (40)$$

and

$$b = \frac{u_2 w_9 x_1^*}{J_0} = \frac{\mu_j \mu_h x_1^* u_9 w_9}{\beta_j \Lambda_h} > 0. \quad (41)$$

Hence, we establish these results with the following theorem:.

Theorem 5: If $\Re_0 = 1$, and

$$\omega > \frac{H_6}{H_5}(H_1 + H_2 + H_3 + H_4) \quad (42)$$

where $w_1, w_2, \cdots, w_9$ and $u_1, u_2, \cdots u_9$ are given in equation (34) and (35) respectively, then the system (31) undergoes a backward bifurcation, otherwise it undergoes a transcritical bifurcation.

3.3 Global Asymptotic Stability (GAS) of DFE

Consider the system (3) without re-infection (i.e. $\omega = 0$), we have the following sub-model

$$\begin{aligned}
S_h' &= \Lambda_h - \lambda_j S_h - \mu_h S_h, \\
E_h' &= \lambda_j S_h - (v+\mu_h) E_h, \\
I_{h1}' &= (1-p) v E_h - (d_0+\mu_h) I_{h1}, \\
I_{h2}' &= p v E_h - (\eta_0 + d_1 + \mu_h) I_{h2}, \\
T_h' &= \eta_0 I_{h2} - \mu_h T_h, \\
M_s' &= n_e \zeta (I_{h1} + I_{h2}) - \mu_h M_s, \\
S_s' &= \Lambda_s - \lambda_m S_s - \mu_s S_s, \\
I_s' &= \lambda_m S_s - \mu_s I_s, \\
J_s' &= \theta I_s - \mu_j J_s.
\end{aligned} \quad (43)$$

where,

$$\lambda_j = \frac{\beta_j J_s}{J_0 + \epsilon J_s} \quad (44)$$

$$\lambda_m = \frac{\beta_m M_s}{M_0 + \epsilon M_s} \quad (45)$$

Theorem 6: The disease-free state of the system (43), without re-infection (i.e., $\omega = 0$) is globally asymptotically stable (GAS) in $\mathcal{R}$ whenever $\Re_0 \leq 1$ and unstable when $\Re_0 > 1$.

Consider the following Lyapunov function associated with system (34)

$$L_1 = G_1E_h + G_2I_{h1} + G_3I_{h2} + G_4I_s + G_5M_s + G_6J_s \quad (46)$$

where $X^T = (E_h, I_{h1}, I_{h2}, I_s, M_s, J_s)$ and

$$\begin{aligned} G_1 &= \frac{\beta_m \Lambda_s \theta \zeta \nu n_e((1-p)(\eta_0 + d_1 + \mu_h) + p(d_0 + \mu_h))}{M_0\mu_j\mu_m\mu_s^2(\nu + \mu_h)(d_0 + \mu_h)(\eta_0 + d_1 + \mu_h)}, \\ G_2 &= \frac{\beta_m \Lambda_s \theta \zeta n_e}{M_0\mu_j\mu_m\mu_s^2(\nu + \mu_h)(d_0 + \mu_h)}, \\ G_3 &= \frac{\beta_m \Lambda_s \theta \zeta n_e}{M_0\mu_j\mu_m\mu_s^2(\nu + \mu_h)(\eta_0 + d_1 + \mu_h)}, \\ G_4 &= \frac{\theta \Re_0}{\mu_j\mu_s}, \\ G_5 &= \frac{\beta_m \Lambda_s \theta}{M_0\mu_j\mu_m\mu_s^2}, \\ G_6 &= \frac{\Re_0}{\mu_j} \end{aligned} \quad (47)$$

The time derivative of the Lyapunov function at DFE is given as

$$L_1' = G_1E_h' + G_2I_{h1}' + G_3I_{h2}' + G_4I_s' + G_5M_s' + G_6J_s' \quad (48)$$

Substituting the right-hand side of (43) and (47) into (48) gives,

$$\begin{aligned} L_1' &= \frac{\beta_m \nu \zeta \theta n_e \Lambda_s S_h \lambda_j((1-p)(\eta_0 + d_1 + \mu_h) + p(d_0 + \mu_h))}{M_0\mu_j\mu_m\mu_s^2(\nu + \mu_h)(d_0 + \mu_h)(\eta_0 + d_1 + \mu_h)} \\ &\quad - \frac{\theta\beta_m M_s \Lambda_s}{M_0\mu_j\mu_s^2} + \frac{\theta\lambda_m S_s R_0}{\mu_j\mu_s} - J_s\Re_0 \\ &= \frac{\beta_m \nu \zeta \theta n_e \Lambda_s \lambda_j((1-p)(\eta_0 + d_1 + \mu_h) + p(d_0 + \mu_h))}{M_0\mu_j\mu_m\mu_s^2(\nu + \mu_h)(d_0 + \mu_h)(\eta_0 + d_1 + \mu_h)}\left(\frac{\beta_j J_s}{J_0 + \epsilon J_s}\right)S_h \\ &\quad - J_s\Re_0 + \frac{\theta\Re_0}{\mu_j\mu_s}\left(\frac{\beta_m M_s}{M_0 + \epsilon M_s}\right)S_s - \frac{\beta_m\theta\Lambda_s}{M_0\mu_j\mu_s}M_s \end{aligned} \quad (49)$$

At DFE,

$$\begin{aligned} L_1' &\leq \frac{\beta_j\beta_m \nu \zeta \theta n_e \Lambda_h \Lambda_s((1-p)(\eta_0 + d_1 + \mu_h) + p(d_0 + \mu_h))}{J_0 M_0\mu_j\mu_m\mu_h\mu_s^2(\nu + \mu_h)(d_0 + \mu_h)(\eta_0 + d_1 + \mu_h)}J_s \\ &\quad + \left(\frac{\beta_m\theta\Lambda_s\Re_0}{M_0\mu_j\mu_s^2}\right)M_s - \left(\frac{\beta_m\theta\Lambda_s}{M_0\mu_j\mu_s^2}\right)M_s - J_s\Re_0 \\ &\leq \left(\frac{\beta_m\Lambda_s\theta}{M_0\mu_j\mu_s^2}M_s + J_s\Re_0\right)(\Re_0 - 1) \\ &\leq (\Re_{0,s}M_s + J_s\Re_0)(\Re_0 - 1) \end{aligned} \quad (50)$$

Hence, $L_1' \leq 0$ whenever $\Re_0 \leq 1$ with $L_1' = 0$ if and only if $M_s = J_s = 0$. Therefore, it follows that L_1 is a Lyapunov function in the domain $\mathcal{D}$. Hence, from LaSalle's Invariance Principle [30], we have that

$$(E_h(t), I_{h1}(t), I_{h2}(t), I_S(t), M_s(t), J_s(t)) \to (0,0,0,0,0,0) \quad \text{as} \quad t \to \infty. \quad (51)$$

This result shows that in a population *where there is effective case-detection and treatment for active schistosomiasis cases*, on the condition that there is negligible re-infection, that is, $\omega = 0$, the DFE will be GAS whenever $\Re_0 \leq 1$. Hence, schistosomiasis can be eradicated from the population whenever $\Re_0 \leq 1$, irrespective of the initial sizes of the sub-populations.

3.4 Global Asymptotic Stability of Endemic Equilibrium Point (EEP)

Assume that the stable manifold of the DFE of the model system (34) is

$$\mathcal{D}_0 = \{(S_h, E_h, I_{h1}, I_{h2}, T_h, M_s, S_s, I_s, J_s) \in \mathcal{D}_*: E_h = I_{h1} = I_{h2} = M_s = I_s = J_s = 0\}. \quad (52)$$

We claim the following result.

Theorem 7: The unique EEP, U^{**}, of model (34) with $\omega = 0$ is globally asymptotically stable in $\mathcal{D}_* \setminus \mathcal{D}_0$ at any time $\Re_0 > 1$

Proof: Consider the system (43) together with the force of infection and the Reproduction number $\mathfrak{R}_0$. Hence whenever $\mathfrak{R}_0 > 1$, the unique EEP (U^{**}) exist. We represent the Lyapunov function L_2 for the EEP as

$$\begin{aligned} L_2 &= S_h - S_h^{**} ln \frac{S_h}{S_h^{**}} + E_h - E_h^{**} ln \frac{E_h}{E_h^{**}} + G_1 \left(I_{h1} - I_{h1}^{**} ln \frac{I_{h1}}{I_{h1}^{**}} \right) \\ &+ G_2 \left(I_{h2} - I_{h2}^{**} ln \frac{I_{h2}}{I_{h2}^{**}} \right) + G_3 \left(T_h - T_h^{**} ln \frac{T_h}{T_h^{**}} \right) \\ &+ G_4 \left(M_s - M_s^{**} ln \frac{M_s}{M_s^{**}} \right) + S_s - S_s^{**} ln \frac{S_s}{S_s^{**}} + I_s - I_s^{**} ln \frac{I_s}{I_s^{**}} \\ &+ G_5 \left(J_s - J_s^{**} ln \frac{J_s}{J_s^{**}} \right) \end{aligned} \quad (53)$$

where

$$\begin{aligned} G_1 &= \frac{(v+\mu_h)(\eta_0 + d_1 + \mu_h)}{v((1-p)(\eta_0 + d_1 + \mu_h) + p(d_0 + \mu_h))}, \\ G_2 &= \frac{(v+\mu_h)(d_0 + \mu_h)}{v((1-p)(\eta_0 + d_1 + \mu_h) + p(d_0 + \mu_h))}, \\ G_3 &= 0 \\ G_4 &= \frac{(v+\mu_h)(d_0 + \mu_h)(\eta_0 + d_1 + \mu_h)}{v\zeta n_e((1-p)(\eta_0 + d_1 + \mu_h) + p(d_0 + \mu_h))}, \\ G_5 &= \frac{\mu_s}{\theta}. \end{aligned} \quad (54)$$

Taking the time derivatives of L_2 in (53), we have

$$\begin{aligned} L_2' &= S_h' - \left(\frac{S_h^{**}}{S_h}\right) S_h' + E_h' - \left(\frac{E_h^{**}}{E_h}\right) E_h' + G_1 \left(I_{h1}' - \left(\frac{I_{h1}^{**}}{I_{h1}}\right) I_{h1}' \right) \\ &+ G_2 \left(I_{h2}' - \left(\frac{I_{h2}^{**}}{I_{h2}}\right) I_{h2}' \right) + G_3 \left(T_h' - \left(\frac{T_h^{**}}{T_h}\right) T_h' \right) \\ &+ G_4 \left(M_s' - \left(\frac{M_s^{**}}{M_s}\right) M_s' \right) + S_s' - \left(\frac{S_s^{**}}{S_s}\right) S_s' + I_s' - \left(\frac{I_s^{**}}{I_s}\right) I_s' \\ &+ G_5 \left(J_s' - \left(\frac{J_s^{**}}{J_s}\right) J_s' \right) \end{aligned} \quad (55)$$

substituting the right hand side of (3) into (55) and manipulating the result, we have

$$\begin{aligned} L_2' &= \mu_h S_h^{**} \left(2 - \frac{S_h}{S_h^{**}} - \frac{S_h^{**}}{S_h} \right) + \lambda_j^{**} S_h^{**} \times \\ &\left[4 - \frac{S_h^{**}}{S_h} - \left(\frac{I_{h1}^{**2}}{I_{h1}(I_{h1}^{**} + I_{h2}^{**})} + \frac{I_{h2}^{**}}{I_{h2}(I_{h1}^{**} + I_{h2}^{**})} \right) \frac{E_h}{E_h^{**}} - \left(\frac{I_{h1} + I_{h2}}{I_{h1}^{**} + I_{h2}^{**}} \right) \frac{M_s^{**}}{M_s} \right] \\ &+ \mu_s S_s^{**} \left(2 - \frac{S_s^{**}}{S_s} - \frac{S_s}{S_s^{**}} \right) + \mu_s I_s^{**} \left(2 - \frac{I_s J_s^{**}}{I_s^{**} J_s} - \frac{S_h E_h^{**} J_s}{S_h^{**} E_h J_s^{**}} \right) + \lambda_m^{**} S_s^{**} \left(1 - \frac{S_s^{**}}{S_s} \right) \end{aligned} \quad (56)$$

Since arithmetic mean is greater than or equal to geometric mean, we conclude that the following inequalities hold

$$\begin{aligned} &\left(2 - \frac{S_h}{S_h^{**}} - \frac{S_h^{**}}{S_h} \right) \leq 0, \; 4 - \frac{S_h^{**}}{S_h} - \left(\frac{I_{h1}^{**2}}{I_{h1}(I_{h1}^{**} + I_{h2}^{**})} + \frac{I_{h2}^{**}}{I_{h2}(I_{h1}^{**} + I_{h2}^{**})} \right) \leq 0 \\ &\left(\frac{I_{h1} + I_{h2}}{I_{h1}^{**} + I_{h2}^{**}} \right) \leq 0, \; \left(2 - \frac{S_s^{**}}{S_s} - \frac{S_s}{S_s^{**}} \right) \leq 0, \left(2 - \frac{I_s J_s^{**}}{I_s^{**} J_s} - \frac{S_h E_h^{**} J_s}{S_h^{**} E_h J_s^{**}} \right) \leq 0, \\ &\left(1 - \frac{S_s^{**}}{S_s} \right) \leq 0. \end{aligned} \quad (57)$$

Therefore, whenever $\mathfrak{R}_0 > 1$, $L_2' \leq 0$.

Since the relevant variables in the equation of I_{h1} and I_{h2} are at the endemic equilibrium, they can be substituted into the equations representing I_{h1} and I_{h2} in the model (43) so that

$I_{h1}(t) \to I_{\mathrm{h1}}^{**}, \quad I_{h2}(t) \to I_{\mathrm{h2}}^{**}$ as $t \to \infty$.

Therefore, L_2 represents a Lyapunov function in $\mathcal{D}_* \backslash \mathcal{D}_0$.

This outcome shows that in a population with endemic schistosomiasis, if $\omega = 0$, the EEP will be GAS whenever $\Re_0 > 1$. Thus, schistosomiasis will persist in the population regardless of the initial sizes of the sub-populations whenever $\Re_0 > 1$.

4 Numerical simulations

The model (3) is simulated in this section with the parameter values set as given in Table 1. Some of the epidemiological parameters are set related to Nigeria.

Table 1: Parameter values used in model (3)

Parameters	Values	Reference
μ_h	0.02041	[31]
ϵ	0.2	[21]
ω	0.1	Assumed
Λ_h	4,147,316	Estimated
n_e	500	[32]
v	6.5	Assumed
Λ_s	73000	[21]
μ_s	0.72	[10]
p	0.1	Assumed
β_j	4.19	Assumed
μ_j	0.504	[10]
η_0	0.23	Assumed
β_m	1.475	Assumed
μ_m	0.75	[33]
d_0	0.02	Assumed
M_0	100,000,000	[21]
ζ	0.835	[13]
d_1	0.0201	Assumed
J_0	90,000,000	[21]
θ	0.9	[13]

Nigeria population is estimated to be 203,200,191 as at August 2019 [34]. The mean natural mortality rate for human was estimated to be $\mu_h = 0.02041$ [31, 35] and the mean annual rate of recruitment into both human and snails population is $\Lambda_h = 4{,}147{,}316$ and $\Lambda_s = 73{,}000$ respectively. For the initial conditions, we obtain the following values from: $S_h(0) = 22{,}000{,}600$, $E_h(0) = 101{,}400{,}000$, $I_{h1}(0) = 30{,}000{,}000$, $I_{h2} = 29{,}000{,}000, T_h(0) = 20{,}089{,}0000$, $M_s(0) = 70{,}000{,}000$, $S_s(0) = 50{,}000{,}000$, $I_s(0) = 30{,}000{,}000$, $J_s(0) = 10{,}000{,}000$.

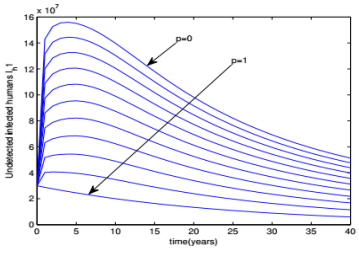

Figure 2: *Graph of undetected infected human population with varying case detection (p)*

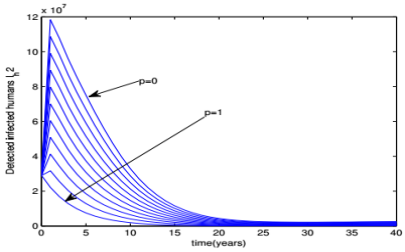

Figure 3: *Graph of detected infected human population with varying case detection (p)*

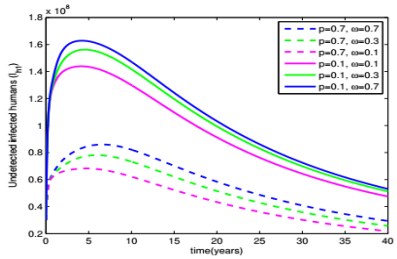

Figure 4: *Graph of undetected infected human population with high and low case detection (p) and varying re-infection (ω)*

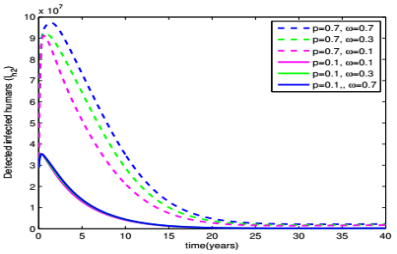

Figure 5: *Graph of detected infected human population with high and low case detection (p) and varying re-infection (ω)*

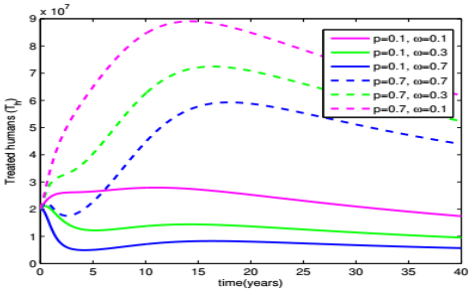

Figure 6: *Graph of treated human population with high and low case detection (p) and varying re-infection (ω)*

Figure 2 shows the result of varying the case detection parameter p. The undetected infected human population reaches its maximum value after about 3 years and then gradually declines until it attains a value where it remains asymptotically stable for all time. This implies that it will take a much longer time for the disease to be eradicated from the population. Meanwhile, Figure 3 shows that the detected infected human population reaches an maximum after 2 years, and then decreases rapidly as a result of the treatment given to the detected infected population. Figure 4 represents the impact of varied re-infection rate at high and low rate of case detection. It shows that the presence of the re-infection parameter has very little effect on the number of the undetected infected human population. The undetected infected human population increases at varying rates of re-infection within the first 5 years and decreases slowly thereafter. Figure 5 shows the propagation of the detected infected human population at high and low case detection with varying re-infection rates. The detected cases of the infected human population at low rate of re-infection, unlike the propagation of the population at high detection rate, shows no significant difference as re-infection rate increases. The propagation of the treated human population is shown in Figure 6. The treated human population increases at high detection rate as the re-infection rate is varied and the detected cases of schistosomiasis increases up until the 15^{th} year after which it begins to decline and then becomes asymptotically stable.

5. Conclusion

Schistosomiasis transmission and infection remains a threat to developing countries in the world. Detection of this disease performs a very significant role in treating and controlling the disease. In this work we have developed a mathematical model to examine the effect of case detection on the transmission dynamics of schistosomiasis, a very severe neglected tropical disease (NTD). This study presented a novel schistosomiasis population model by considering two infected classes: undetected class of infected human population and the detected class of infected human population. Another class is also introduced for the treatment of detected infected human population. The effective reproduction number $\mathfrak{R}_0 = \sqrt{\mathfrak{R}_{0h}\mathfrak{R}_{0s}}$ is obtained and the DFE of the formulated system was shown to be locally asymptotically stable (LAS) whenever the associated reproduction number is less than unity. Furthermore, the DFE of the system was shown to be globally asymptotically stable (GAS) with negligible re-infection rates when the corresponding reproduction number $\mathfrak{R}_0$ is less than unity. We established the case to have backward bifurcation and the GAS of the EEP.

Numerical simulation results from the system (3) showed that a significant decrease in the schistosomiasis cases could be achieved in the population if there is an increase in the proportion of the detected human cases of schistosomiasis which are set for treatment immediately. It is obvious from the result of this work that schistosomiasis can be controlled in a population if the public health control programmes provide and implement strategies for detection as well as the timely treatment of a very large number of persons infected with schistosomiasis.It is also important to mention that continuous contact of the treated population with the infected water with cercaria is one of problem in eradicating the burden of schistosomiasis. Therefore an increase in the proportion of detected cases of schistosomiasis infection, treated as soon as possible, would reduce the cases of schistosomiasis and possibly eradicate the disease from the population.

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