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Mathematical Modelling of the Dynamics of the Spread of Schistosomiasis Incorporating Protection of Humans

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Abstract: **Schistosomiasis, also known as Bilharziasis, is a parasitic disease of global concern, and is among the neglected tropical diseases. It is caused by the digenetic trematode flukes of the genus Schistosoma and transmitted by fresh water snails of the family Planorbidae. Its clinical manifestation depends on the immunity of the person, the warm burden and the duration of the same worms in the body. African countries suffer greatly from this disease. We have formulated a mathematical model with four compartments, Susceptible-Protected-Infected-Treated, (SPIT), and the corresponding ordinary differential equations in an attempt to investigate the impact of human protection. The disease free equilibrium and endemic equilibrium have been analyzed. The stability of the disease free equilibrium has also been determined. The reproduction number has been determined using the next Generation matrix. Mathematical simulation was carried out. The impact of human protection on the dynamics of the spread of the disease has been analyzed. Our results show that protection of the human being plays a great role in controlling the spread of this disease. It contributes significantly to reduction of infections and the stability of the susceptible population.**

Keywords: **Schistosomiasis modeling, Mathematical modeling, Reproduction number.**

1. INTRODUCTION

Bilharzia also known as Schistosomiasis or bilharziasis, is a parasitic disease caused by digenetic trematode parasitic flukes of the genus Schistosoma. It is mainly caused by five blood flukes species of medical and economic importance, which are transmitted through fresh water snails. These are S.mansoni, S.japonicum, S.Mekongi, S.intercalatum and S.Haematobium [2]. Debilitating disease arises from granulomas in the liver and intestines.

S.japonicum lives in the superior mesenteric vein of both large and small intestines producing clinical and pathological symptoms similar to those of S.mansoni [7]. Man gets infected by coming in contact with water containing cercariae (parasite stage infective to man released into water by snails). Snails of the family Planorbidae are the intermediate hosts since they habour the asexual stage of schistosomes. Human beings habour the sexual stage of the same parasite so they are the definitive hosts.

In 2019, 230 million people worldwide were affected by Schistosomiasis. A report from W.H.O in 2017 indicated that over 700 million people were at risk of infection and there were approximately 200,000 deaths from this infection alone. Migration and relocation increase the chances of infection [5].

Symptoms of Schistosomiasis are not caused by the worms themselves but by the body's reaction to the eggs. Many infections are asymptomatic. However, when the infection becomes acute it causes Katayama fever which is characterized by fever, cough, abdominal pain and diarrhea among others. If it is not treated it causes death.

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In Kenya the two most important species are S.mansoni and S.haematobium. S.mansoni is mainly found in Muranga'a, Kiambu, Mwea, Kirinyaga, Embu and along the shores of lake Victoria. S.hamatobium is found in Kwale, Kilifi, lower regions of Tana River, Machakos and Kitui Districts. It is also found in Western Kenya [1].

S.Haematobium is endemic in 53 countries in the Middle East and most African countries including Madagascar and Mauritius. S.japonicum is restricted to the Far East countries. It is endemic in China, Indonesia, Philippines and Thailand.

2. MODEL DESCRIPTION AND FORMULATION

The model chosen by the researcher has four compartments: Susceptible (S), Infected (I), Treated (T) and Protected (P). People are recruited to the susceptible compartment (S) by natural descent or immigration at the rate of Λ. If they obtain chemotherapy they join the "protected" compartment (P) at the rate of τ. When exposed to the contaminated environment this susceptible group is recruited to the infected compartment (I), at the rate of β, through mass action.

They could also join the susceptible compartment (S) at the rate of φ. When those infected are treated they join the "treated" compartment (T) at the rate of κ. There is no permanent immunity. So if the treated become exposed to the same contaminated environment they join the susceptible compartment (S) once again at the rate of ω. The protected people could also move to the Infected compartment, when the chemotherapy they have received wanes, at the rate of α.The population dies at a natural death rate of μ, while the infected die at the rate of σ, (due to the disease).

The whole population is denoted by $N(t) = S(t) + P(t) + I(t) + T(t)$. Description of parameters used in the model

Table 1: Summary of compartment descriptions

Variable	Interpretation
S(t)	population at risk of infection.
P(t)	protected population
I(t)	infected population
T(t)	treated population

Table 2: Summary of parameter descriptions

Parameter	Interpretation
Λ	Recruitment rate
β	Rate of progression from Susceptible to Infected
τ	Rate of progression from Susceptible to Protected
α	Rate of progression from Protected to infected due to waning of protection.
κ	Rate at which Infected get treated.
ω	Rate at which treatment wanes
σ	Death rate due to disease
μ	Natural death rate

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Flow diagram for Schistosomiasis model

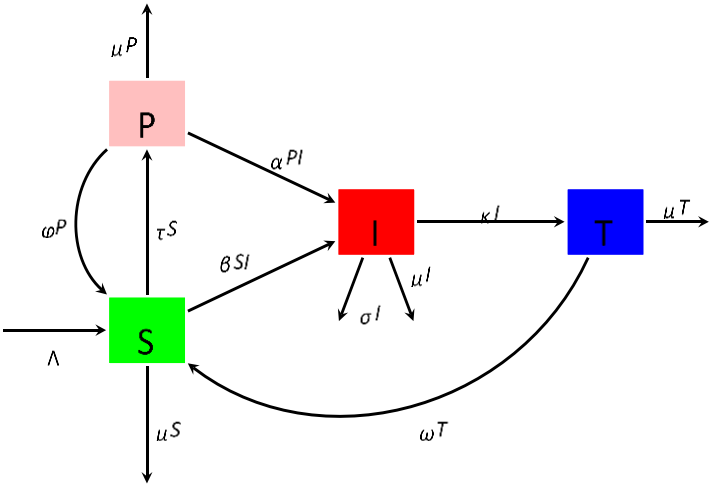

Figure 1: Model flow chart.

The corresponding differential equations are as follows.

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda + \phi P + \omega T - (\tau + \mu)S - \beta SI \\
\frac{dI}{dt} &= \beta SI + \alpha PI - (\sigma + \mu + \kappa)I \\
\frac{dT}{dt} &= \kappa I - (\mu + \omega)T \\
\frac{dP}{dt} &= \tau S - \alpha PI - (\mu + \phi)P
\end{aligned}\right\}$$

3. BASIC PROPERTIES OF THE MODEL

Considering our set of equations

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda + \phi P + \omega T - (\tau + \mu)S - \beta SI \\
\frac{dI}{dt} &= \beta SI + \alpha PI - (\sigma + \mu + \kappa)I \\
\frac{dT}{dt} &= \kappa I - (\mu + \omega)T \\
\frac{dP}{dt} &= \tau S - \alpha PI - (\mu + \phi)P
\end{aligned}\right\} \qquad (3.1)$$

Adding all the equations in 3.1 we have

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt}+\frac{dP(t)}{dt}+\frac{dI(t)}{dt}+\frac{dT(t)}{dt},$$

$$= \Lambda-\phi(S+I+T+P)-\delta I \le \Lambda-\rho N.$$

Since $N(t)=S(t)+P(t)+I(t)+T(t)$. Therefore,

$$\frac{dN}{dt} \le \Lambda-\Psi N.$$

Using separation of variable technique, we have

$$\frac{dN}{\Lambda-\Psi N} \le dt,$$

we let

$$u=\Lambda-\mu N,$$

such that

$$du=-\mu dN,$$

Implying that

$$dN=\frac{-du}{\mu},$$

therefore,

$$-\frac{du}{\mu u} \le dt.$$

As such,

$$\frac{du}{u} \ge -\mu dt,$$

upon integrating both sides we obtain

$$\ln u \ge -\mu t + c,$$

therefore taking exponential on both sides we obtain

$$e^{\ln(\Lambda-\mu N)} \ge e^{(c)}e^{(-\mu t)} = Ae^{(-\mu t)},$$

Rearranging equation 3 we get

$$\Lambda-\mu N \ge Ae^{(-\mu t)},$$

$$N(t) \le \frac{\Lambda}{\mu}-\frac{A}{\mu}e^{-\mu t},$$

At the initial condition we have $N(t=0)=N(0)$. Thus

$$N(0) \le \frac{\lambda}{\mu}-\frac{A}{\mu}e^{0}=\frac{\Lambda}{\mu}-\frac{A}{\mu}.$$

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as such

$$\frac{A}{\mu} \leq \frac{\Lambda}{\mu} - N(0),$$

we obtain the following expression

$$A \leq \Lambda - \mu N(0).$$

substituting 3 in 3 we have the following expression

$$N(0) \leq \frac{\Lambda}{\mu} - \frac{(\Lambda - \mu N(0))}{\mu},$$

$$N(1) \leq \frac{\Lambda}{\mu} - \frac{A}{\mu} e^{-\mu t} - N(0)e^{-\mu t},$$

$$N(1) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}\left(1 - e^{-\mu t}\right).$$

As, $\lim_{t\to\infty} N(t) = \lim_{t\to\infty}\left(N(0)e^{-\mu t} + \frac{\Lambda}{\mu}\left(1-e^{-\mu t}\right)\right) = \frac{\Lambda}{\mu}$, It should be noted that $N(t) \leq \frac{\Lambda}{\mu}$ if $N(0) \leq \frac{\Lambda}{\mu}$ for all $t > 0$ and $\frac{dN}{dt} \leq 0$ if $N \leq \frac{\Lambda}{\mu}$. This region Π generated by such a flow is positively invariant

4. THE DISEASE-FREE EQUILIBRIUM (DFE)

At equilibrium point,all equations are equated to zero.

$$0 = \Lambda + \phi P + \omega T - (\tau + \mu)S - \beta SI,$$

$$0 = \beta SI + \alpha PI - (\sigma + \mu + \kappa)I,$$

$$0 = \kappa I - (\mu + \omega)T,$$

$$0 = \tau S - \alpha PI - (\mu + \phi)P.$$

At Disease free equilibrium $I = 0$ and $T = 0$ since there is no disease in the community [8].This implies that,

$$0 = \Lambda + \phi P - (\tau + \mu)S,$$

$$0 = \tau s - (\mu + \phi)P,$$

From this equation 4 and making P the subject;

$$\tau S = (\mu + \phi)P,$$

$$P = \frac{\tau S}{(\mu + \phi)},$$

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Using equation 4 in equation 4

$$0 = \Lambda + \phi(\frac{\tau S}{(\mu+\phi)} - (\tau+\mu)S,$$

$$0 = \Lambda + (\frac{\phi\tau}{\mu+\phi})S - (\tau-\mu)S,$$

$$-\Lambda = \phi\tau - (\tau+\mu)S,$$

$$S^* = -\frac{\Lambda}{\frac{\phi\tau}{(\mu+\phi)}} - (\tau+\mu),$$

$$S^* = \frac{\Lambda(\mu+\phi)}{(\phi\tau - (\tau+\mu)(\mu+\phi)},$$

$$S^* = \frac{\Lambda(\mu+\phi)}{\mu(\tau+\mu+\phi)},$$

Using equation 4 in equation 4

$$P^* = \frac{\tau\Lambda}{\mu(\tau+\mu+\phi)},$$

At disease free equilibrium,

$$DFE = (S^*, P^*, I^*, T^*) = \left(\frac{\Lambda(\mu+\phi)}{\mu(\tau+\mu+\phi)}, \frac{\tau\Lambda}{\mu(\tau+\mu+\phi)}, 0, 0\right).$$

5. THE BASIC REPRODUCTION NUMBER

The Basic reproduction number is the measure of secondary infections that arise from one infected individual [8]. We used the next generation matrix to determine this R_0. The model equations are:

$$\frac{dS}{dt} = \Lambda + \phi P + \omega T - (\tau+\mu)S - \beta SI,$$

$$\frac{dI}{dt} = \beta SI + \alpha PI - (\sigma+\mu+\kappa)I,$$

$$\frac{dT}{dT} = \kappa I - (\mu+\omega)T,$$

$$\frac{dP}{dt} = \tau S - \alpha PI - (\mu+\phi)P,$$

Considering the infectious compartments and non-infectious compartments denoted as vectors X and Y respectively, we have

$$X = I \qquad \text{and} \qquad Y = \begin{bmatrix} S \\ T \\ P \end{bmatrix}.$$

$$F(X,Y)=[\beta SI+\alpha PI]\rightarrow f_1,$$

$$F=\frac{df}{dI}=[\beta S+\alpha P],$$

At DFE

$$DFE=\left(\frac{\Lambda}{\mu}.\frac{(\mu+\phi)}{(\mu+\phi+\tau)},\frac{\Lambda}{\mu}.\frac{\tau}{(\mu+\phi+\tau)},0,0\right),$$

$$F=\left[\frac{\beta\Lambda}{\mu}.\frac{(\mu+\phi)}{(\mu+\phi+\tau)}+\frac{\alpha\Lambda}{\mu(\mu+\phi+\tau)}\right].$$

$$V(X,Y)=[\sigma+\mu+\kappa]I\rightarrow v,$$

$$V=\frac{dv}{dI}=[\sigma+\mu+\kappa],$$

$$V=[(\sigma+\mu+\kappa)],$$

$$V^{-1}=\frac{1}{(\sigma+\mu+\kappa)}.$$

The next generation matrix will be given by,

$$FV^{-1}=\left[\frac{\beta\Lambda}{\mu}.\frac{(\mu+\phi)}{\mu(\mu+\phi+\tau)}+\frac{\alpha\Lambda}{\mu}.\frac{\tau}{\mu+\phi+\tau)}\right]\frac{1}{(\sigma+\mu+\kappa)},$$

Simplifying, we have,

$$\frac{\Lambda}{\mu(\mu+\phi+\tau)}(\beta(\mu+\phi)+\alpha\tau)\cdot\frac{1}{(\sigma+\mu+\kappa)}=\frac{\Lambda(\beta(\mu+\phi)+\alpha\tau)}{\mu(\mu+\phi+\tau)(\sigma+\mu+\kappa)}=R_0$$

Therefore the reproduction number R_0 of the disease dynamics is given by

$$R_0=\frac{\Lambda(\beta(\mu+\phi)+\alpha\tau)}{\mu(\mu+\phi+\tau)(\sigma+\mu+\kappa)}. \qquad (5.1)$$

6. LOCAL STABILITY OF DFE

Considering the model equations,

$$\frac{dS}{dt}=\Lambda+\phi P+\omega T-(\tau+\mu)S-\beta SI.$$

$$\frac{dI}{dt}=\beta SI+\alpha PI-(\sigma+\mu+\kappa)I.$$

$$\frac{dT}{dT}=\kappa I-(\mu+\omega)T.$$

$$\frac{dP}{dt}=\tau S-\alpha PI-(\mu+\phi)P.$$

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we have the Jacobian matrix below,

$$J=\begin{bmatrix} -(\tau+\mu)-\beta I & \phi & -\beta S & \omega \\ \tau & -\alpha I-\mu-\phi & \alpha P & 0 \\ \beta I & \alpha I & \beta S+\alpha P-(\sigma+\mu+\kappa) & 0 \\ 0 & 0 & \kappa & -\omega-\mu \end{bmatrix}$$

At DFE we have,

$$\left(\left(\frac{\Lambda}{\mu}\right)\left(\frac{\mu+\phi}{\tau+\mu+\phi}\right),\frac{\tau\Lambda}{\mu(\tau+\mu+\phi)},0,0\right)$$

since $I = 0$ and $T = 0$ We obtain the Jacobian matrix at DFE as,

$$J_{DFE}=\begin{bmatrix} -\tau-\mu & \phi & \dfrac{\beta\Lambda(\mu+\phi)}{\mu(\mu+\phi+\tau)} & \omega \\ \tau & -\mu-\phi & -\dfrac{\alpha\tau\Lambda}{\mu(\mu+\phi+\tau)} & 0 \\ 0 & 0 & \dfrac{\alpha\tau\Lambda}{\mu(\mu+\phi+\tau)}+\dfrac{\beta\Lambda(\mu+\phi)}{\mu(\mu+\phi+\tau)}-\kappa-\mu-\sigma & 0 \\ 0 & 0 & \kappa & -\omega-\mu \end{bmatrix}$$

The eigenvalues are,

$$\begin{bmatrix} -\mu \\ -\mu-\phi-\tau \\ -\omega-\mu \\ \dfrac{-\mu^3+(-\phi-\tau-\kappa-\sigma)\mu^2+((-\kappa-\sigma)\phi+(-\kappa-\sigma)\tau+\beta\Lambda)\mu+\Lambda(\alpha\tau+\beta\phi)}{\mu(\mu+\phi+\tau)} \end{bmatrix}$$

For local stability of the DFE,

$$\frac{-\mu^3-(\phi+\tau+\kappa+\sigma)\mu^2-(\kappa+\sigma)\phi\mu-(\kappa+\sigma)\tau\mu+\beta\Lambda\mu+\Lambda(\alpha\tau+\beta\phi)}{\mu(\mu+\phi+\tau)}<0$$

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Rearranging the equation we have,

$$\frac{\beta\Lambda\mu+\Lambda(\alpha\tau+\beta\phi)}{\mu(\mu+\phi\tau)}<\frac{(\mu^3+(\phi+\tau+\kappa+\sigma)\mu^2+(\kappa+\sigma)\phi\mu+(\kappa+\sigma)\tau+\mu}{\mu(\mu+\phi+\tau)}.$$

simplifying gives

$$\frac{\beta(\beta(\mu+\phi)+\alpha\tau)}{\mu(\mu+\phi+\tau)}<\frac{(\mu^2(\mu+\phi+\tau)+(\kappa+\sigma)(\mu+\phi+\tau)\mu}{\mu(\mu+\phi+\tau)}.$$

further simplification leads to

$$\frac{\beta(\beta(\mu+\phi)+\alpha\tau)}{\mu(\mu+\phi+\tau)}<\frac{\mu(\mu+\phi+\tau)(\mu+\kappa+\sigma)}{\mu(\mu+\phi+\tau)}.$$

Simplifying the right hand side and dividing all through by $\mu+\kappa+\sigma$ we get

$$\frac{\beta(\beta(\mu+\phi)+\alpha\tau)}{\mu(\mu+\phi+\tau)(\mu+\kappa+\sigma)}<1.$$

However, the left hand side give the R_0 thus the system is stable when $R_0 < 1$

7. ENDEMIC EQUILIBRIUM

The existence of an endemic equilibrium indicates the fact that the disease is spreading in a certain population [9]. By equating all model equations to zero, we can solve to find the endemic equilibrium. We shall now investigate whether a unique endemic equilibrium exists. From the model equations,

$$\begin{aligned}
\dot{S} &= \Lambda+\phi P+\omega T-(\tau+\mu)S-\beta SI,\\
\dot{I} &= \beta SI+\alpha PI-(\sigma+\mu+\kappa)I,\\
\dot{T} &= \kappa I-(\mu+\omega)T,\\
\dot{P} &= \tau S-\alpha PI-(\mu+\phi)P.
\end{aligned}\tag{7.1}$$

At the equilibrium point, $\frac{d}{dt}=0$ thus equation 7.1 reduces to;

$$0=\Lambda+\phi P^*+\omega T^*-(\tau+\mu)S^*-\beta S^*I^*,$$

$$0=\beta S^*I^*+\alpha P^*I^*-(\sigma+\mu+\kappa)I^*,$$

$$0=\kappa I^*-(\mu+\omega)T^*,$$

$$0=\tau S^*-\alpha P^*I^*-(\mu+\phi)P^*.$$

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Considering 7.4 we have

$$0 = \kappa I^* - (\mu + \omega)T^*.$$

Making T^* the subject of the equation

$$\frac{(\mu+\omega)T^*}{(\mu+\omega)} = \frac{(\kappa I^*}{(\mu+\omega)}$$

Therefore, equation 7.7 reduces to,

$$T^* = \frac{\kappa I^*}{\mu+\omega}$$

Considering 7.5 and making P^* the subject we have

$$P^* = \frac{\tau S^*}{\alpha I^* + \mu + \phi}$$

$$P^* = \frac{\tau\left(\Lambda\mu + \omega\left(I^*\kappa + \Lambda\right)\right)}{\left(\mu^2 + \left(\left(\beta+\alpha\right)I^* + \phi + \tau\right)\mu + I^*\left(\alpha\beta I^* + \alpha\tau + \beta\phi\right)\right)\left(\mu+\omega\right)}$$

Considering 7.2 and substituting 7.7 and 7.9 into the equation we get the expression of S^* as follows

$$S^* = \frac{\left(\alpha I^* + \mu + \phi\right)\left(\Lambda\mu + \omega\left(I^*\kappa + \Lambda\right)\right)}{\left(\mu^2 + \left(\left(\beta+\alpha\right)I^* + \phi + \tau\right)\mu + I^*\left(\alpha\beta I^* + \alpha\tau + \beta\phi\right)\right)\left(\mu+\omega\right)}$$

Equation 7.3 gives the expression of I^* which can be summarized as

$$f(I^*) = c_1 I^{*2} + c_2 I^* + c_3.$$

Where

$$c_1 = -\beta\alpha b_1,$$
$$c_2 = a_2\mu^3 - b_2\mu^2 + \Lambda\beta - a_5\omega - \tau\alpha a_6 - \beta\mu, b_3 + \omega\alpha, b_4 - \phi\beta\sigma,$$
$$c_3 = a_8(\mu^3 + a_9\mu^2 + a_{10}a_6 - \Lambda\beta)\mu - \Lambda(\alpha\tau + \beta\phi).$$
$$b_1 = \mu^2 + a_1\mu + \omega\sigma,$$
$$b_2 = a_3\alpha + \beta a_4.$$
$$b_3 = a_7\omega + \phi a_6.$$
$$b_4 = \Lambda\beta - \sigma\tau.$$
$$a_1 = \kappa + \omega + \sigma.$$

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$$a_2 = \beta + \alpha.$$
$$a_3 = \tau + \kappa + \omega + \sigma.$$
$$a_4 = \phi + \kappa + \omega + \sigma.$$
$$a_5 = \tau + \kappa + \sigma.$$
$$a_6 = \kappa + \sigma.$$
$$a_7 = \phi + \sigma.$$
$$a_8 = \mu + \omega.$$
$$a_9 = \phi + \tau + \kappa + \sigma.$$
$$a_{10} = \tau + \phi.$$

8. NUMERICAL SOLUTIONS

a. PARAMETER DATA

Model parameter values were contextualized and estimated in drawing the graphs. The final rates were summarized as follows:

Table 3: Summary of data and its sources

Parameter	Value	period	Source
Λ	3077	per annum	[3][4]
β	0.000011315	per annum	[6]
τ	0.02	per annum	Estimated
α	0.0000014144	per annum	Calculated from [6]
κ	0.848837211	per annum	[6]
ω	0.1	per annum	Estimated
σ	0.1095	per annum	[6]
μ	0.005448	per annum	[5]
φ	0.0005	per annum	Estimated

b. SCHISTOSOMIASIS DISEASE DYNAMICS

The schistosomiasis disease dynamics were summarized by the figure 2 below

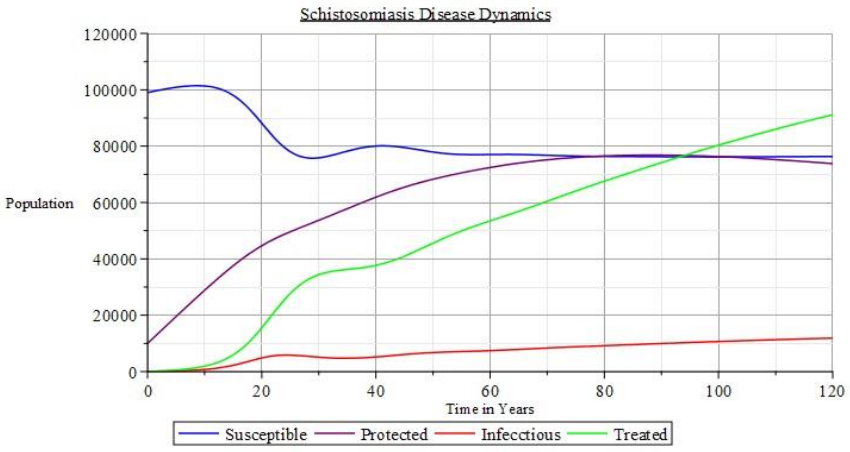

Figure 2: Schistosomiasis disease dynamics

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In figure 2 the susceptible population stabilizes and then decreases for a while, and then stabilizes again. Over 80% of the population remains susceptible to the disease at any given time.

The protected population increases gradually and then stabilizes for a length of time. At the stability point the population is dominated by the protected, treated and susceptible rises a bit and then levels off, almost going to zero. The treated population increases gradually over the whole period of time. At stability point the treated are more than the susceptible and the protected.

The infection rates remain generally low. It takes a long period of time before the spread of the disease gains momentum. Less than 1% are infected at any given time.

c. PROTECTED AND INFECTIOUS POPULATIONS

The comparison between the protected individuals and the infectious individuals is summarized by the graph in fig 3 below

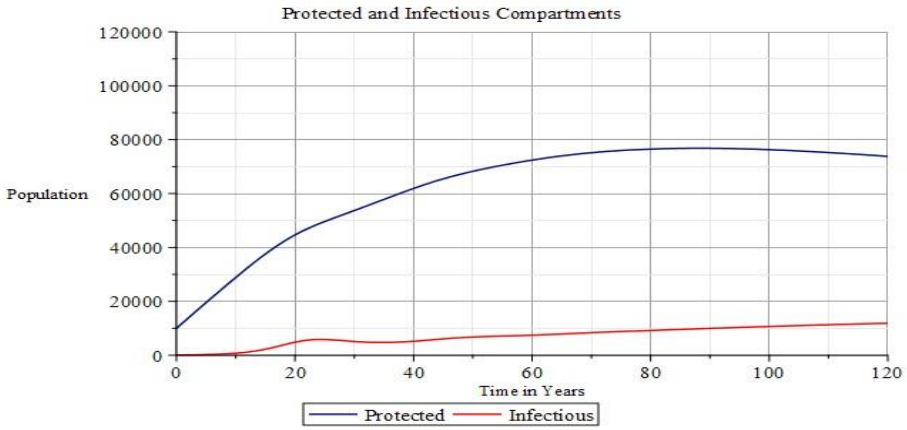

Figure 3: Protected and infectious populations

The graph in fig 3 illustrates the dynamics of the protected and the infected. As the protected population increases the Infectious population increases as well but at a low rate over a long period of time. The protected population increases with time, as well, but at a far much higher rate. This is the graph of great interest in this study as it covers the population of greatest interest to the study.

d. INFECTIOUS POPULATION AT DIFFERENT PROTECTION RATES

The infectious rates are directly affected by the interventions that enhance the protection of the infectious populations as illustrated by the graph in fig 4 below

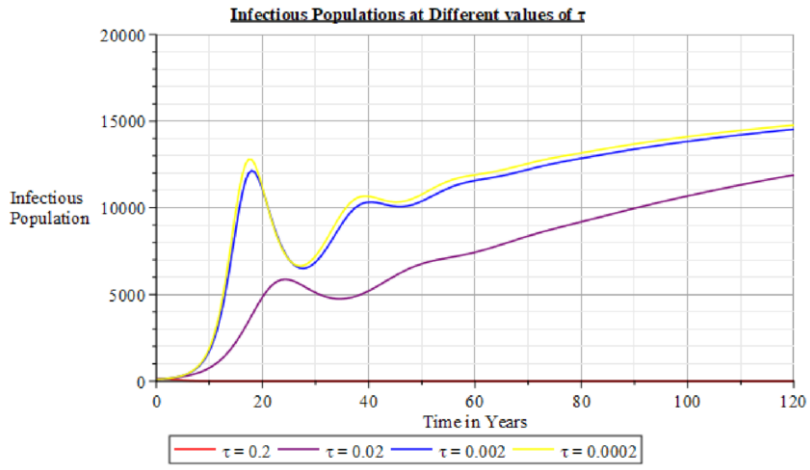

Figure 4: Infectious population at different protection rates

The graph in fig 4 illustrates the fact when τ = 0.2 the infectious population is very low. When τ = 0.02 the infectious population increases at a high rate. When τ is0.002 the infectious population increases again. When τ = 0.0002 the infectious population increases, though not at a significant rate.

e. PROTECTED POPULATION AT DIFFERENT PROTECTION RATES

The protected population dynamics are illustrated in fig 5 below

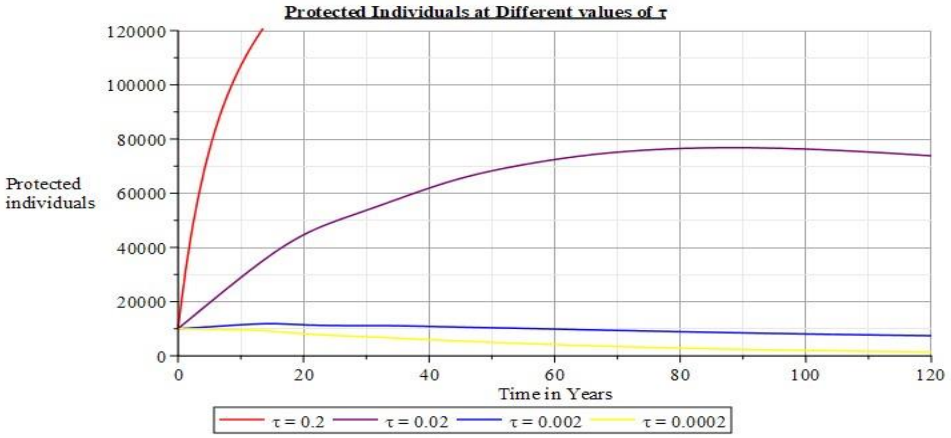

Figure 5: Protected population at different protection rates

In figure 5 it was noted that the graph the population of protected population is large when τ is 0.2 .When τ is 0.02 the protected population decreases about 25%. When τ is 0.002 this population decreases again greatly. When τ is 0.0002, this protected population again decreases to a very great extent.

9. CONCLUSIONS AND RECOMMENDATIONS

a. Conclusions

An SPIT model for the transmission dynamics of schistosomiasis was developed and the corresponding differential equations were formulated based on the thesis assumptions. The disease free equilibrium of the differential equations was determined. The stability of the DFE was determined and it was concluded that it is stable when $R_0 < 1$. The endemic equilibrium of schistosomiasis disease dynamics was also determined. The numerical solution was determined and the corresponding graphs analyzed.

b. Recommendations

It is recommended that further research be conducted on introduction of irradiated cercariae which cannot infect the human being therefore providing more protection to humans by minimizing contact with snails bearing schistosomiasis. This intervention will ensure that R_0 is kept less than 1. Strategies of enhancing both long-term and short-term protection should be enhanced. Long-term strategies include investment in infrastructure such as bridges and drainage systems, while short-term strategies include provision of personal protective gears, chlorination of water, and sensitization of communities.

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