

A Mathematical Model for the Transmission Dynamics of Schistosomiasis with Treatment

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Abstract: Schistosomiasis remains one of the major health problems in tropical and subtropical countries with school aged children usually the most affected. Urinary schistosomiasis is endemic in Wasai, a town located near Wasai dam in Minjibir Local Government area of Kano state. Different types of water contact activities takes place in the water body which have different snail intermediate hosts attached to water hyacinth. A nonlinear model that analyzed the pattern of transmission of urinary schistosomiasis for children from Wasai and Dingim towns and snails in Jakara dam with a single dose treatment with Praziquantel was designed. Picard Lindelof theorem was used to show the existence of a unique solution to the system. The disease free equilibrium was found to be locally asymptotically stable and due to the nonautonomous nature of the system, Floquet theory was applied to obtain the basic reproduction number. These results have been illustrated by numerically simulating the model with estimated parameter values, and sensitivity analysis was carried out.

Keywords: Asymptotic, Basic reproduction number, Mathematical modeling, Re-infection, Schistosomiasis, Treatment.

I. INTRODUCTION

Schistosomiasis is a parasitic disease that affects people in tropical and subtropical countries particularly children who acquire the disease during recreational activities in snail infested water. Coming in contact directly with open freshwater bodies through bathing, swimming, and wading has been reported to be a risk factor of schistosomiasis [1,2,3,4]. An estimated 779 million individuals are at risk of acquiring schistosomiasis and more than 200 millions were infected in mid- 2003 [5]. Observations for both visible and non visible blood (macrohaematuria) and (microhaematuria) in urine were found to be associated with the presence of *S. haematobium* eggs in urine. It has also been demonstrated as a good indicator of *S. haematobium* infection [6,7,8]. Although not all haematuria are related to *S. haematobium* infection, presence of blood in urine gives an early indication of infection. The presence of blood in urine is caused by granulomatous inflammation resulting from the lodging of *S. Haematobium* eggs in the bladder and urogenital system [9], and is increasingly becoming an important criterion for assessing *S. haematobium* infection in studies. Self reported blood in urine is even used to identify potential *S. haematobium* infection in people living in endemic areas [10,8]. Praziquantel (PZQ) remains the only drug for clinical management and community-based control of schistosomiasis [11, 12, 13, 14,15]. [16] described a mathematical model for the control of schistosomiasis in Hubei china. [17] presented a mathematical model that was used to study the transmission dynamics, control and vaccination of schistosomiasis with a variable population size. [18] formed a model of Schistosomiasis under flood in Anhui Province. [19] designed a mathematical model and Analyses the transmission Dynamics and Control of Schistosomiasis. Most of the models in the literature considered a more optimistic situation, where reinfection parameters are neglected. Schistosomiasis disease is endemic and can resurface at anytime as long as people revisit the dams. Even vaccination programs might fail completely or partially especially in developing countries since finances play a major role in the number of people who receive the vaccines.

2. MATERIALS AND METHODS

Table 1: Parameter description of the model

Variable	Description
S	Population of susceptible individuals
I	Population of infected individuals
T_F	Population of treated individuals who failed treatment
T	Population of treated individuals
R	Population of recovered individuals

Table 2: Parameter description of the model

Parameter	Description
Π	Recruitment rate of individuals
$\delta(t)$	Time varying contact rate of schistosomiasis
Ξ	Reinfection rate
Γ	Treatment dose rate
μ	Natural death rate of humans
Σ	Recovery rate of individuals who failed treatment
A	Recovery rate of treated individuals
δ_0	Baseline contact rate
δ_1	Fluctuating contact rate amplitude

2.1 Model Formulation

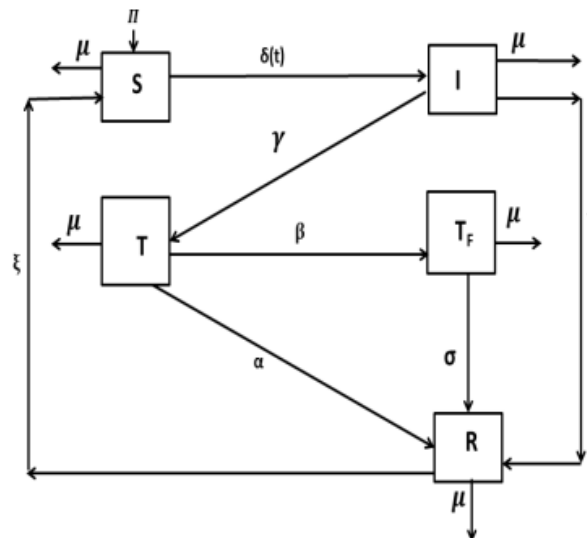


Figure 1: Schematic diagram of the model

In order to formulate the model equations the total population N is divided into five classes. The fully susceptible class (S), the infected class (I), treated individuals (T), treated individuals who failed treatment T_F , and recovered individuals (R). The class S of susceptible is increased either by birth, immigration at the rate Π or by reinfection at the rate ξ . It is decreased by a time varying infection rate $\delta(t)$, and by death at the rate μ . The class I of infected individuals is derived through infection of fully susceptible, this class is decreased either by death at the rate μ , treatment dose γ , or by recovery at the rate τ . The class of treated individuals T is generated a perfect treatment dose γ and decreased by death at the rate μ or by recovery at the rate α it is also decreased at a lower rate β which happens when the treatment dose fails. The class of individuals who failed treatment T_F is obtained when the treatment dose fails at the rate β and is decreased by death and by natural recovery at the rate σ . The class of recovered individuals is increased when the treatment dose works at the α or by natural recovery at the rate τ . The class is diminished by reinfection and natural death, at the rates ξ and μ respectively.

Model Assumptions

1. Based on the fact that children in Wasai and Digim swim more during dry season, the contact rate is assumed to be time varying.
2. Children who failed treatment and those who did not receive any treatment can still recover from disease at a lower rate.
3. The model also assumes that the recovered class is not protected, that is children can be infected again and again. The transitions between model classes is expressed by the following differential equations.

$$\frac{dS}{dt} = -\delta(t)SI - \mu S + \zeta R + \Pi \quad (0.2.1)$$

$$\frac{dI}{dt} = \delta(t)SI - (\gamma + \mu + \tau)I \quad (0.2.2)$$

$$\frac{dT}{dt} = \gamma I - (\mu + \alpha + \beta)T \quad (0.2.3)$$

$$\frac{dT_F}{dt} = \delta T - (\mu + \sigma)T_F \quad (0.2.4)$$

$$\frac{dR}{dt} = \tau I + \alpha T + \sigma T_F - (\mu + \zeta)R \quad (0.2.5)$$

Existence and Uniqueness of Solution

The equations 0.2.1 to 0.2.5 can be written as:

$$F(x) = Ax + g(t, x) + b \quad (0.3.1)$$

$$A = \begin{bmatrix} -\mu & 0 & 0 & 0 & \xi \\ 0 & -(\mu + \gamma + \tau) & 0 & 0 & 0 \\ 0 & \gamma & -(\mu + \beta + \alpha) & 0 & 0 \\ 0 & \beta & 0 & -(\mu + \sigma) & 0 \\ 0 & \tau & \alpha & \sigma & -(\mu + \zeta) \end{bmatrix}$$

$$g(t, x) = \begin{bmatrix} -\delta(t)SI \\ \delta(t)SI \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

$$b = \begin{bmatrix} \Pi \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

Theorem 0.3.1 :The system 0.3.1 satisfies Lipschitz conditions.

Proof:

$$\|f(x) - f(x^*)\| = \|A(x - x^*) + g(x) - g(x^*)\| \quad (0.3.5)$$

$$\leq (\|A\| + \tilde{k})(x - x^*), \quad (0.3.6)$$

$$\|A\| = \max\{\mu + \beta + \alpha, \alpha + \sigma + \mu + \zeta + \tau\},$$

$$\tilde{k} = 5\max\{\delta I + \mu + \tau + \gamma, \delta S^*, \zeta\}$$

0.3.2 Boundedness and Positivity of Solution

Since the model describes human population, it is necessary to show that all the state variables S, I, T_F, T and R are non-negative for all t ≥ 0, solutions with non-negative initial data remains non-negative for all t ≥ 0 and are bounded. Let N represent the total population, that is:

$$N = S + I + T + T_F + R \quad (0.3.7)$$

From 0.3.7 $N \rightarrow \frac{\Pi}{\mu}$ as $t \rightarrow \infty$

This shows that the feasible region

$$D = \left\{ (S, I, T, T_F, R) : S, I, T, T_F, R \geq 0, S + I + T + T_F + R = \frac{\Pi}{\mu} \right\} \quad (0.3.8)$$

Is positively invariant set for the model.

0.3.3 Stability Analysis of DFE

In the absence of infection, the model has a unique disease free equilibrium

$E_0 = (S^0, I^0, T^0, T_F^0, R^0) = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0 \right)$. To analyze the disease free equilibrium, the model 0.2.1 – 0.2.5 is linearized about E_0 by setting:

$S(t) = \bar{S}(t) + S^0, I(t) = i, T(t) = \check{t}(t), T_F(t) = t_F(t), R(t) = r(t)$, then we have the Jacobian matrix,

$$J = \begin{bmatrix} -\mu & \frac{-\delta(t)\Pi}{\mu} & 0 & 0 & \xi \\ 0 & \frac{\delta(t)\Pi}{\mu} - (\mu + \gamma + \tau) & 0 & 0 & 0 \\ 0 & \gamma & -(\mu + \beta + \alpha) & 0 & 0 \\ 0 & \beta & 0 & -(\mu + \sigma) & 0 \\ 0 & \tau & \alpha & \sigma & -(\mu + \xi) \end{bmatrix}$$

Since the Jacobian matrix J is a non constant matrix, we shall apply Floquet theory (20) to find its eigen values.

$$\lambda_1 = \exp[-\mu T], \lambda_2 = \exp \left\{ \int_0^T \frac{\Pi \delta(s)}{\mu} - (\mu + \gamma + \tau) ds \right\}, \lambda_3 = \exp[-(\mu + \alpha)T], \lambda_4 = \exp[-(\mu + \sigma)T], \lambda_5 = \exp[-(\mu + \xi)T]$$

It is easy to see that $0 < \lambda_1, \lambda_3, \lambda_4, \lambda_5 < 1$, which means that DFE is locally asymptotically stable if $\lambda_2 < 1$. We can see that $\lambda_2 < 1$ if and only if:

$$\frac{1}{T} \int_0^T \delta(s) ds < \frac{\mu + \gamma + \tau}{\Pi} \quad (0.3.10)$$

Since $(t) = \delta_0[1 + \delta_1 \sin(2\pi t)]$, the inequality (0.3.10) can be expressed as $R_0 < 1$, where

$$R_0 = \frac{\delta_0 \Pi}{(\mu + \gamma + \tau)} \quad (0.3.11)$$

0.2 Numerical Simulation

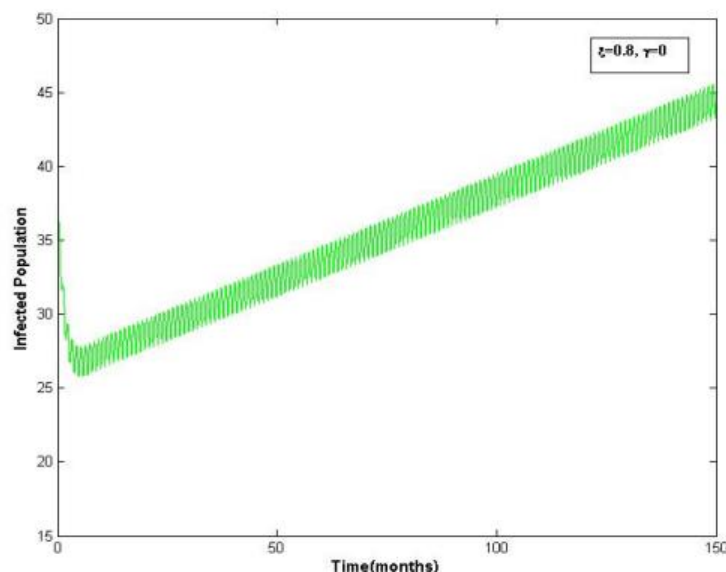


Figure 2:

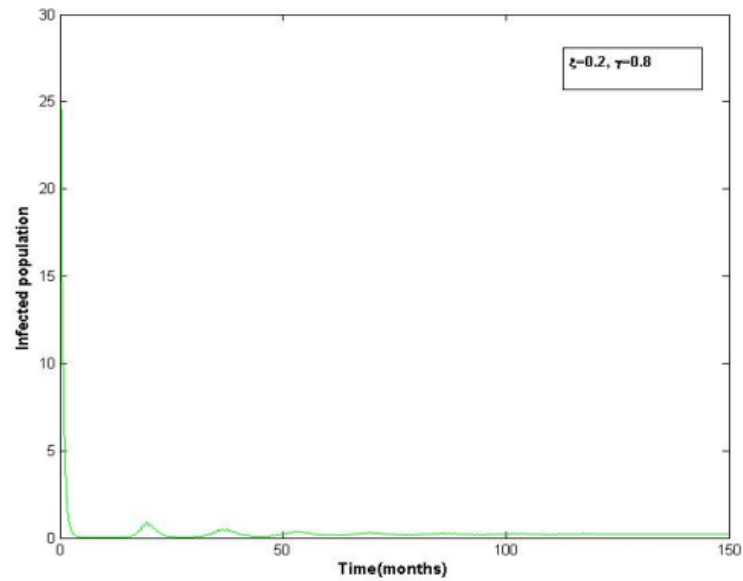


Figure 3:

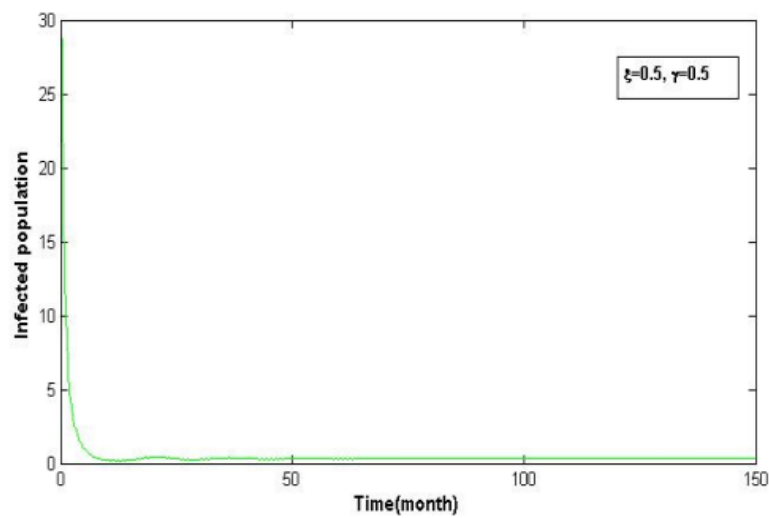


Figure 4:

3. DISCUSSION OF RESULTS

The figures 4,5,6 represents the profile of infected population with different rates of reinfection and treatment dose..

Table 3: Parameter description of the model

Parameter	Values	References
Ξ	0,0.5,0.8	Assumed
Γ	0.67	(21)
μ	0.1	Assumed
Σ	0.15	Assumed
A	0.79	(21)
T	0.25	Assumed
B	0.35	Assumed
δ_0	0.3	Assumed
δ_1	0.4	Assumed

Figure 4 assumes no treatment dose is given that is $\gamma = 0$ and rate of reinfection is taken to be $\zeta = 0.8$. The figure shows how the infected population increasing towards its limit.

In figure 5 treatment dose is given at a very high rate $\gamma = 0.8$ and $\zeta = 0.2$, which makes the infected population decrease and approach zero.

Figure 6 assumes reinfection rate is as good as the given dose and we can see the infected population almost vanishing.

4. CONCLUSION

This result shows how sensitive the reinfection parameter could be. Treatment doses alone are not sufficient when it come to eradication of schistosomiasis. Vaccination and sensitization programs must be adopted.

REFERENCES

- [1] Couto LD, Tibiric,a SH, Pinheiro IO, Mitterofhe A, Lima AC, Castro MF, et al. Neglected tropical diseases: prevalence and risk factors for schistosomiasis and soil-transmitted helminthiasis in a region of Minas Gerais State, Brazil. *Trans R Soc Trop Med Hyg.* 2014; 108(6):363–371. <https://doi.org/10.1093/trstmh/tru054> PMID: 24781377
- [2] Amuta EU, Houmsou RS (2014). Prevalence, intensity of infection and risk factors of urinary schistosomiasis in pre-school and school aged children in Guma Local Government Area, Nigeria. *Asian Pac J Trop Med.*; 7: 34–9. [https://doi.org/10.1016/S1995-7645\(13\)60188-1](https://doi.org/10.1016/S1995-7645(13)60188-1) PMID: 24418080
- [3] Ismail HA, Hong ST, Babiker AT, Hassan RM, Sulaiman MA, Jeong HG, et al (2014). Prevalence, risk factors, and clinical manifestations of schistosomiasis among school children in the White Nile River basin,Sudan. *Parasit Vectors*; 7:478. <https://doi.org/10.1186/s13071-014-0478-6> PMID: 25312470
- [4] Zida A, Briegel J, Kabre´ I, Sawadogo MP, Sangare´ I, Bamba S, et al (2016). Epidemiological and clinical aspects of urogenital schistosomiasis in women, in Burkina Faso, West Africa. *Infect Dis Poverty.*; 5(1): 81. <https://doi.org/10.1186/s40249-016-0174-1> PMID: 27581074
- [5] Steinmann, P., Keiser,J., Bos,R., Tanner,M .and Utzinger,J.(2006). Schistosomiasis and water resources development: Systematic review, metaanalysis, and estimates of people at risk. *Lancet Infectious Diseases* 6,411425
- [6] Zida A, Briegel J, Kabre´ I, Sawadogo MP, Sangare´ I, Bamba S, et al. Epidemiological and clinical aspects of urogenital schistosomiasis in women, in Burkina Faso, West Africa. *Infect Dis Poverty.* 2016;5(1): 81. <https://doi.org/10.1186/s40249-016-0174-1> PMID: 27581074
- [7] Hessler MJ, Cyrs A, Krenzke SC, Mahmoud ES, Sikasunge C, Mwansa J, et al. Detection of duo-schistosome infection from filtered urine samples from school children in Zambia after MDA. *PLoS ONE.* 2017; 12(12): e0189400. <https://doi.org/10.1371/journal.pone.0189400> PMID: 29228024
- [8] Duwa,M.R and Oyeyi,T.I and Bassey S E (2009). Prevalence and intensity of urinary schistosomiasis among Primary school pupils in minjibir local government area of Kano state. *Bayero journal of pure and applied sciences*, 2(1):75 - 78
- [9] Colley DG, Bustinduy AL, Secor WE, King CH. Human schistosomiasis. *Lancet (London, England).* 2014; 383: 2253–64. [https://doi.org/10.1016/S0140-6736\(13\)61949-2](https://doi.org/10.1016/S0140-6736(13)61949-2) PMID: 24698483
- [10] Gray DJ, Ross AG, Li YS, McManus DP. Diagnosis and management of schistosomiasis. *BMJ.* 2011; 342:d2651. <https://doi.org/10.1136/bmj.d2651> PMID: 21586478.
- [11] Cioli, D. (2000). Praziquantel: is there real resistance and are there alternatives *Current Opinion in Infectious Diseases* 13,659663.
- [12] Fenwick, A., Savioli, L., Engels, D., Bergquist, N. R. and Todd, M. H. (2003). Drugs for the control of parasitic diseases:current status and development in schistosomiasis.*Trends in Parasitology* 19,509515.
- [13] Utzinger, J.and Keiser, J.(2004). Schistosomiasis and soil-transmitted helminthiasis: Common drugs for treatment and control. *Expert Opinion on Pharmacotherapy*, 5,263285.
- [14] Caffrey,C.R.(2007).Chemotherapy of schistosomiasis : Present and future. *Current Opinion in Chemical Biology* 11,433439.

- [15] Doenhoff, M. J., Cioli, D. and Utzinger, J. (2008). Praziquantel: mechanisms of action, resistance and new derivatives for schistosomiasis. *Current Opinion in Infectious Diseases* 21,659667.
- [16] Zimin Chen., Lan Zou., Dingwen Shen., Weinian Zhang., Shigui Ruan (2010). Mathematical modelling and control of Schistosomiasis in Hubei Province, China. *Acta Tropica* (115) pp119–125
- [17] Musa.S., Bello.N., Umar.A. (2019) Mathematical Modeling of The Transmission Dyanamics, Control and Vaccination of Schistosomiasis With aVariable. *Bayero Journal of Pure and Applied Sciences* 12(1): 70 - 80
- [18] Longxing Qi, Jing-an Cui, Tingting Huang, Fengli Ye, and Longzhi Jiang (2014). Mathematical Model of Schistosomiasis under Flood in Anhui Province. *Abstract and Applied Analysis* Vol. 2014, Article ID 972189
- [19] Ebrima, Kanyi., Ayodeji, Sunday Afolabi., Nelson Owuor Onyango (2021). Mathematical Modeling and Analysis of Transmission Dynamics and Control of Schistosomiasis. *Hindawi Journal of Applied Mathematics*, vol.2021.1-20
- [20] James D Meiss: Differential dynamical systems (2007) Copyright by the *Society for Industrial and Applied Mathematics*.
- [21] Rabi Musa Duwa (2008). Urinary Schistosomiasis in Kano state: Pattern of human infection, treatment outcome of single dose praziquantel, and distribution of snail intermediate host in two dam sites communities. PhD Thesis Bayero University Kano (2008)