http://dx.doi.org/10.4314/bajopas.v12i1.10



Bayero Journal of Pure and Applied Sciences, 12(1): 70 - 80 Received: October, 2018 Accepted: May, 2019 ISSN 2006 – 6996

MATHEMATICAL MODELING OF THE TRANSMISSION DYNAMICS, CONTROL AND VACCINATION OF SCHISTOSOMIASIS WITH A VARIABLE POPULATION SIZE

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ABSTRACT

In this paper, a compartmental mathematical model for the transmission dynamics of schistosomiasis in human, cattle and snail populations with a variable population size; and vaccination as a control strategy has been studied. The basic reproduction number R_0 of the model has been computed. Disease free- equilibrium state and its local stability

using next generation matrix and linearization method were used respectively; the model was found out to be locally asymptotically stable (LAS) given that $R_0 < 1$. The numerical

results revealed that, high rate of vaccination use decreases both susceptible and infected populations in both human and cattle. It is therefore sufficient to adhere to the vaccination exercise on both susceptible and infected human and cattle populations to exterminate schistosomiasis.

Keywords: schistosomiasis, Vaccination; variable population; Stability; strategy

INTRODUCTION

One of the most serious public health problems in the tropics and subtropics is human schistosomiasis (Bilharziasis), а parasitic infection caused by flatworms of the family Schistosoma that live in fresh water habitats. Schistosomiasis is characterized by long-term disability and is estimated to affect over 200 million people mostly in underdeveloped countries where the disease is endemic. According to the survey done in 2003 by World Health Organization (WHO), more than 200 million people are infected and over 600 million people in 74 countries are at risk of the infection. Mortality rate exceeds 100,000 annually and schistosomiasis remains formidable to humans because of the complexities of parasitic adjustment to two or more different hosts (Garrett, 1994; McNeill, 1977).

The persistence of schistosomiasis infection in a locality depends on a complex cycle involving humans and possibly additional mammalian species called definite hosts, such includes some particular species of snails and certain parasitic flatworms (schistosomes). Schistosomes are digenetic trematodes that spend their adult life in humans and a previous stage in aquatic snails (Jordan *et al.*, 1993). Anderson and May (1992)

confirmed that Schistosomes live inside blood vessels; the adult schistosome worms mate heterosexually, laying hundreds of eggs and these eggs are deposited in intestine or bladder; eventually passed out as faeces or urine in fresh water bodies. In the fresh water bodies, snail asexually produces cercariae, at maturity human comes with contact with the cercariae and subsequently pairing with the opposite sex, copulation and oviposition which begins the cycle over again

The transmission of schistosomiasis is associated with water development projects such as dams for irrigation systems and fish-farming, as the snail intermediate hosts of the parasites breed in them and human water contact (Klump and Webbe, 1987; WHO, 1989; WHO, (1993)). Schistosomiasis, being a water-based disease is spread through contact with water in which snails harbouring and shedding the infective stage (cercariae) of the parasite (schistosome) are present (Costa *et al.*, 1993).

Schistosomiasis has been classified as a neglected tropical disease (NTD), although an estimated 779 million people in the world are at risk of the infection according to recent surveys (Steinmann *et al.*, 2006; Hotez *et al.*, 2007).

Human schistosomiasis is caused by five species flatworms: Schistosoma of mansoni, Schistosoma intercalatum, Schistosoma japonicum, Schistosoma mekongi, and Schistosoma haematobium. Three of these species (S. mansoni, S. haematobium and S. intercalatum) are endemic in Nigeria, which led to the formation of a national schistosomiasis control program in the late 1980s. Estimates in the mid-1990s suggested that more than 100 million people were at risk of this disease and that 25.8 million people were actually infected (Chitsulo et al., 2000). More recently the latter figure was updated to 29 million infections (Steinmann et al., 2006; Moné et al., 2010), which corresponds to 14% of the global number of schistosomainfections and puts Nigeria at the top of the list of endemic countries.

One of the control strategy of schistosomiasis is vaccination, and vaccines that would specifically reduce parasite reproduction and egg viability may also be a desirable goal. Silvera *et al., 2004* confirmed that, an alternative vaccinology approach, is inducing immunity with attenuated parasites has provided the strongest animal proof-of-concept that vaccines against schistosomiasis are feasible.

Enormous research has been going on to device control strategies to deal with the menace, more

importantly transmission dynamics of schistosomiasis via mathematical model that brought substantial insight on the control strategies. Since 1973, there have been many mathematical models of the transmission dynamics of schistosomes examples are works done by Anderson and May (1985, 1992); Kimbir (1997); Wu and Feng (2002); Feng *et al.*, (2001); Riley *et al.* (2008); Mangal *et al.*, (2008); Zimin *et al.* (2010) and among others.

Zimin *et al.* (2010) proposed a mathematical model for the human–cattle–snail transmission of schistosomiasis in Hubei Province of China. The compartmental model consists of human, cattle and snail populations and each populations entails susceptible and infected compartments. The results suggested that, to control or eradicate schistosomiasis in the studied region, a more comprehensive approach is needed to consider environmental factors in order to break the cattle-snail transmission.

The aim of this paper is to modify the model due to Zimin *et al.* (2010) by incooperating vaccination as a control strategy and considered a variable population size. Also, Death due to natural death is accounted for in the model considering the fact that death due to natural death can occur in both susceptible and infected humans, cattle, and snails

Table 1: Modified Model State Variable and their Description				
Description				
Vacinated compartment for human t				
Vacinated compartment for cattle t				
Susceptible human population at time t				
Susceptible snail population at time t				
Susceptible cattle population at time t				
Infected human population at time t				
Infected snail population at time t				
Infected cattle population at time t				

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2.0 MATERIALS AND METHODS

BAJOPAS Volume 12 Number 1, June, 2019 Table 2: Modified Model Parameters and their Description

Table 2: Modified Model Parameters and their Description				
Parameters	Descriptions			
$b_{\scriptscriptstyle H}$	Natural birth rate of human			
$eta_{_{SH}}$	transmisson rate from infected snail to human			
r _H	recovery rate of infectes human			
b_{C}	natural birth rate of cattle			
$oldsymbol{eta}_{\scriptscriptstyle SC}$	transmisson rate from infected snail to cattle			
d_{c}	death rate of infected snails			
$(b_C - d_C)$	carrying capacity of cattle			
$\frac{\left(b_{C}-d_{C}\right)}{k_{C}}$				
r_{C}	recovery rate of infectes cattle			
b_s	natural birth rate of snails			
$eta_{\scriptscriptstyle HS}$	transmisson rate from infected human to snail			
eta_{cs}	transmisson rate from infected cattle to snail			
d_s	transmisson rate from infected cattle to snails			
$\frac{\left(b_{s}-d_{s}\right)}{k_{s}}$	carrying capacity of snails			
k_s				
$d_{\scriptscriptstyle H}$	Death due to the disease in human			
$\mu_{\scriptscriptstyle H}$	Death due to natural causes in human			
μ_{s}	Death due to natural causes in Snail			
μ_{c}	Death due to natural causes in cattle			
$\mathcal{U}_{_{H}}$	Vaccination rate for human			
\mathcal{E}_{H}	Rate of loss of immunity in human			
v_{c}	Vaccination rate for Cattle			
\mathcal{E}_{C}	Rate of loss of immunity in cattle			

2.1 Zimin *et al.* (2010) Assumptions Zimin *et al.* (2010) made the following assumptions that:

Human, cattle and snail populations are all positive, i.e $N_H > 0$, $S_C + I_C > 0$ and $S_S + I_S > 0$. The birth rate is greater than the death rate for both cattle and snails, i.e., $b_C - d_C > 0$ i. ii.

and $b_s - d_s > 0$. Zimin *et al.* (2010) Model Equations 2.2

$$\frac{dS_H}{dt} = \beta_{SH}S_HI_S + r_HI_H \tag{*1}$$

$$\frac{dI_H}{dt} = \beta_{SH} S_H I_S - r_H I_H \tag{*2}$$

$$\frac{dS_{c}}{dt} = b_{c} \left(S_{c} + I_{c} \right) - \beta_{sc} S_{c} I_{s} + r_{c} I_{c} - d_{c} S_{c} - k_{c} S_{c} \left(S_{c} + I_{c} \right) + I_{c}$$
(*3)

$$\frac{dI_c}{dt} = \beta_{sc}S_cI_s - r_cI_c - d_cI_c - k_cI_c\left(S_c + I_c\right)_c \tag{*4}$$

$$\frac{dS_s}{dt} = b_s \left(S_s + I_s \right) - \beta_{Hs} S_s I_H - \beta_{Cs} S_s I_C - d_s S_s - k_s S_s \left(S_s + I_s \right)$$
(*5)

$$\frac{dI_s}{dt} = \beta_{HS}S_sI_H + \beta_{CS}S_sI_C - d_sI_s - k_sI_s\left(S_s + I_s\right)$$
(*6)

2.3 **Modified Model Assumptions**

- iii.
- Human, cattle and snail populations are all positive, i.e $N_H > 0$, $S_C + I_C > 0$ and $S_S + I_S > 0$. The birth rate is greater than the death rate for both cattle and snails, i.e., $b_c d_c > 0$ iv. and $b_s - d_s > 0$.
- The recruitment rate of human into the susceptible class by natural birth. ٧.
- The vaccinated chambers as vaccines availability for both human and cattle are feasible. vi.
- Death due to natural death can occur in both susceptible and infected humans, cattle, and vii. snails

2.4 **Modified Model Equations**

$$\frac{dS_H}{dt} = b_H N_H - \beta_{SH} S_H I_S + r_H I_H - \upsilon_H S_H + \varepsilon_H V_H - \mu_H S_H$$
(1)

$$\frac{dI_{H}}{dt} = \beta_{SH} S_{H} I_{S} - (d_{H} - \mu_{H}) I_{H} - r_{H} I_{H}$$
(2)

$$\frac{dS_C}{dt} = b_C N_C - \beta_{SC} S_C I_S + r_C I_C - \mu_C S_C - k_C S_C \left(S_C + I_C\right) - \mu_C S_C + \varepsilon_C V$$
(3)

$$\frac{dI_{C}}{dt} = \beta_{SC}S_{C}I_{S} - r_{C}I_{C} - (d_{C} + \mu_{C})I_{C} - k_{C}I_{C}(S_{C} + I_{C})_{C}$$
(4)

$$\frac{dS_s}{dt} = b_s N_s - \beta_{HS} S_s I_H - \beta_{CS} S_s I_C - \mu_s S_s - k_s S_s \left(S_s + I_s\right)$$
(5)

$$\frac{dI_{s}}{dt} = \beta_{HS}S_{s}I_{H} + \beta_{CS}S_{s}I_{C} - (d_{s} + \mu_{s})I_{C} - k_{s}I_{s}(S_{s} + I_{s})$$
(6)

$$\frac{dV_H}{dt} = v_H S_H - \varepsilon_H v_H - \mu_H V_H$$
⁽⁷⁾

$$\frac{dV_c}{dt} = v_c S_c - \varepsilon_c v_c - \mu_c V_c \tag{8}$$

Therefore equations (1)-(8) are transform into proportions, and hence our reduced model equations are given below:

$$i'_{h} = B_{SH} \left(1 - v_{h} \right) - \left(B_{SH} + r_{H} + d_{H} + b_{H} - d_{H} i_{h} \right) i_{h}$$
(9)

$$v'_{h} = v_{H} \left(1 - i_{h} \right) - \left(v_{H} + \varepsilon_{H} + b_{H} - d_{H} i_{h} \right) v_{h}$$

$$\tag{10}$$

$$i'_{c} = B_{SC} \left(1 - v_{c} \right) - \left(B_{SC} + r_{C} + d_{C} + b_{C} - d_{C} i_{c} \right) i_{c}$$
(11)

$$\mathbf{v}'_{c} = \mathbf{v}_{C} \left(1 - i_{c} \right) - \left(\mathbf{v}_{C} + \mathbf{\varepsilon}_{C} + b_{C} - d_{C} i_{c} \right) \mathbf{v}_{c}$$

$$(12)$$

$$i'_{s} = B_{HS} + B_{CS} - (B_{HS} + B_{CS} + d_{s} + b_{s} - d_{s}i_{s})i_{s}$$
(13)

3.0 RESULTS

Disease Free Equilibrium (DFE) State of the Model 3.1

To obtain the disease free equilibrium (DFE) of the model, set the right hand side of equations (9)-(13) to zero, and letting $i_h = i_c = i_s = 0$ at disease free equilibrium. Resolving the equations yield the followings:

$$v_{h} = \frac{v_{H}}{v_{H} + \varepsilon_{H} + b_{H}}, \text{ and } v_{c} = \frac{v_{C}}{v_{C} + \varepsilon_{C} + b_{C}}$$
Remember that the following equations hold throughout this study
$$B_{SH} = \beta_{SH}I_{S}, B_{SC} = \beta_{SC}I_{S}, B_{CS} = \beta_{CS}I_{S}, B_{HS} = \beta_{HS}I_{H}$$
(14)

$$(i_h, v_h, i_c, v_c, i_s) = \left(0, \frac{v_H}{v_H + \varepsilon_H + b_H}, 0, \frac{v_C}{v_C + \varepsilon_C + b_C}, 0\right)$$
(15)

3.2 **Basic Reproduction Number of the Model**

The basic reproduction number denoted by R_0 could be computed by using next-generation matrix. This method is given by Driessche and Watmough, (2002). Therefore, get F and V as given below:

$$F = \begin{bmatrix} \beta_{SH} I_{S} \\ \beta_{SC} I_{S} \\ \beta_{HS} I_{H} + \beta_{CS} I_{C} \end{bmatrix}$$
(16)
$$V = \begin{bmatrix} (B_{SH} V_{h} - (B_{SH} + r_{H} + d_{H} + b_{H} - d_{H} i_{h}) i_{h}) \\ (B_{SC} V_{C} - (B_{SC} + r_{c} + d_{c} + b_{c} - d_{c} i_{c}) i_{c}) \\ (B_{HS} + B_{CS} + d_{s} + b_{s} - d_{s} i_{s}) i_{s} \end{bmatrix}$$
(17)

So, taking partial derivatives of equations (16) - (17) and evaluated at disease free equilibrium state gives the followings:

$$F = \begin{bmatrix} 0 & 0 & \beta_{SH} \\ 0 & 0 & \beta_{SC} \\ \beta_{HS} & \beta_{CS} & 0 \end{bmatrix}, \quad V = \begin{bmatrix} r_H + d_H + b_H & 0 & 0 \\ 0 & r_C + d_C + b_C & 0 \\ 0 & 0 & d_S + b_S \end{bmatrix}$$
(18)

Solving FV^{-1} of the two equations in (18) with the largest eigen value is given below:

$$\boldsymbol{R}_{0} = \sqrt{\frac{(r_{H} + d_{H} + b_{H})\beta^{2}_{CS} + \beta^{2}_{HS}(r_{C} + d_{C} + b_{C})}{(r_{H} + d_{H} + b_{H})(r_{C} + d_{C} + b_{C})(d_{S} + b_{S})}}$$
(19)

Local stability of the disease free equilibrium (DFE) State 3.3

Linearization approach is used to examine the local stability of the disease free equilibrium (DFE) state, this is done by obtaining the Jacobian matrix of the model equations in proportion given by (9) to (13). Thus, the Jacobian evaluated at disease free equilibrium is given below:

$$J = \begin{bmatrix} -(B_{HS} + r_{H} + d_{H} + b_{H}) & -B_{HS} & 0 & 0 & 0 \\ -v_{H} + \frac{d_{H}(B_{SH} + v_{H})}{B_{HS} + v_{H} + \mathcal{E}_{H} + b_{H}} & -(v_{H} + \mathcal{E}_{H} + b_{H}) & 0 & 0 & 0 \\ 0 & 0 & -(B_{LS} + r_{C} + d_{C} + b_{C}) & -B_{CS} & 0 \\ 0 & 0 & -v_{C} + \frac{d_{C}(B_{CS} + v_{C})}{B_{LS} + v_{C} + \mathcal{E}_{C} + b_{C}} & -(v_{C} + \mathcal{E}_{C} + b_{C}) & 0 \\ 0 & 0 & 0 & 0 & -(B_{HS} + B_{CS} + d_{S} + b_{S}) \end{bmatrix}$$
(20)

For simplification purpose, let's denote

0

0

0

$$q = (B_{HS} + r_H + d_H + b_H), \ p = v_H + \frac{d_H (B_{SH} + v_H)}{B_{HS} + v_H + \varepsilon_H + b_H}, \ \omega = (v_H + \varepsilon_H + b_H), \ k = (B_{CS} + r_C + d_C + b_C),$$

$$P = v_C + \frac{d_C (B_{CS} + v_C)}{B_{CS} + v_C + \varepsilon_C + b_C}, \qquad l = (v_C + \varepsilon_C + b_C), \qquad n = (B_{HS} + B_{CS} + d_S + b_S),$$

$$f = v_C + \frac{d_C (B_{CS} + v_C)}{B_{CS} + v_C + \varepsilon_C + b_C}$$
Therefore equation (20) becomes
$$|J - \lambda I| = \begin{vmatrix} -q - \lambda & -B_{HS} & 0 & 0 & 0 \\ P & -\omega - \lambda & 0 & 0 & 0 \\ 0 & 0 & -k - \lambda & -B_{CS} & 0 \\ 0 & 0 & 0 & 0 & -n - \lambda \end{vmatrix} = 0$$
(21)

 $-n-\lambda$

Solving equation (21) for eigene values gives the followings:

$$\lambda_4, \lambda_5 = \frac{-(k+l) \pm \sqrt{(k+l)^2 - 4(B_{CS}f + lk)}}{2}$$
(22)

$$\lambda_4 = \frac{-(k+l) + \sqrt{(k+l)^2 - 4(B_{CS}f + lk)}}{2}$$
(23)

$$\lambda_{5} = \frac{-(k+l) - \sqrt{(k+l)^{2} - 4(B_{CS}f + lk)}}{2}$$
(24)

Therefore, the disease free equilibrium state is stable if and only if $B_{HS}\left(\nu_{C} + \frac{d_{C}\left(B_{CS} + \nu_{C}\right)}{B_{CS} + \nu_{C} + \varepsilon_{C} + b_{C}}\right) < \left(\nu_{H} + \varepsilon_{H} + b_{H}\right).$ (25)

4.0 Simulation Results

In this section, graphical solutions in Figure 1 to 10 are presented to show the effects of vaccination and variable population on the transmission dynamics of schistosomiasis. The parameters values used in the simulations are presented in Table 3

Table 3:values for state variables and Parameters

Parameters/Variables	Values	Referrences
$V_{H}(0)$	15	Assumed
$V_c(0)$	30	Assumed
$S_{H}(0)$	90	Assumed
$S_{s}(0)$	100	Assumed
$S_{C}(0)$	200	Assumed
$I_{H}(0)$	20	Assumed
$I_{s}(0)$	30	Assumed
$I_c(0)$	50	Assumed
$eta_{_{SH}}$	2.23×10 ⁻⁷	Allen and Victory, (2003)
r_{H}	4.47×10^{-7}	Allen and Victory, (2003)
b_{c}	1.20×10^{-3}	Allen and Victory, (2003)
$oldsymbol{eta}_{SC}$	2.00×10 ⁻³	Allen and Victory, (2003)
d_{C}	5.00×10 ⁻⁶	Allen and Victory, (2003)
$\frac{\left(b_{C}-d_{C}\right)}{k_{C}}$	7.00×10^{-3}	Allen and Victory, (2003)
k _c		
r_{c}	2.4×10^{-2}	Allen and Victory, (2003)
b_s	6.00×10^{-2}	Allen and Victory, (2003)
$eta_{\scriptscriptstyle HS}$	1.04×10^{-5}	Allen and Victory, (2003)
eta_{cs}	1.05×10^{-7}	Allen and Victory, (2003)
d_s	8.86×10 ⁻³	Allen and Victory, (2003)
$(b_s - d_s)$	7.00×10^{-3}	Allen and Victory, (2003)
k_{s}		

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Table 3 Continue		
b_{H}	0.312	Kbenesh <i>et al.</i> (2009)
$\mu_{\scriptscriptstyle H}$	4.0×10^{-5}	Hyun (2001)
$d_{_H}$	5.0×10^{-4}	WHO (2003)
μ_s	8.86×10 ⁻³	Allen and Victory, (2003)
μ_{c}	5.00×10^{-3}	Allen and Victory, (2003)
\mathcal{U}_{H}	0.00 - 0.75	Assumed
\mathcal{E}_{H}	0.31	Assumed
v_c	0.00 - 0.75	Assumed
\mathcal{E}_{C}	0.23	Assumed

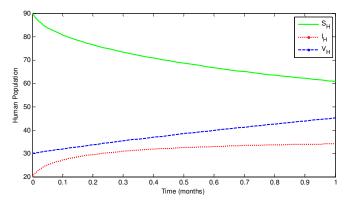


Figure 1: Human population with vaccination and variable population size

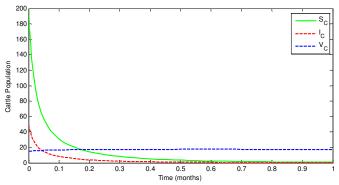


Figure 2: Cattle population with vaccination and variable population size

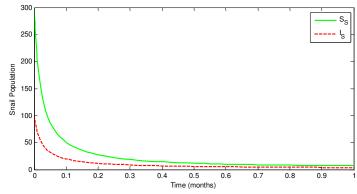


Figure 3: Snail population for the model with variable population size

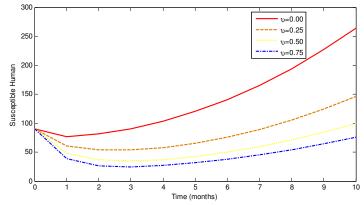


Figure 4: Effects of vaccination rate on susceptible human

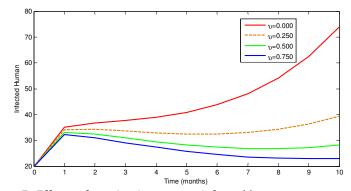


Figure 5: Effects of vaccination rate on infected human

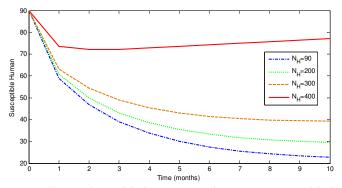


Figure 6: Effects of variable human population on susceptible human

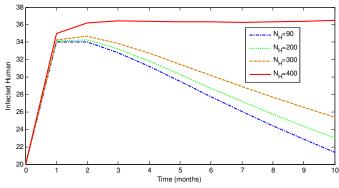


Figure 7: Effects of variable human population on infected human

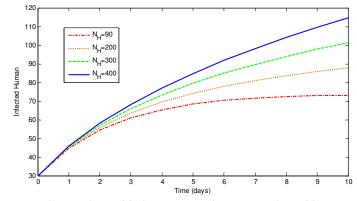


Figure 8: Effects of variable human population on infected human

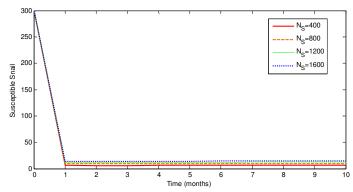


Figure 9: Effects of variable snail population on susceptible snail

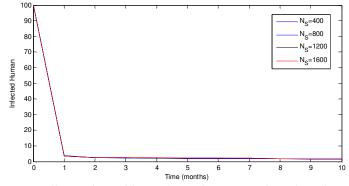


Figure 10: Effects of variable snail population on infected snail

5.0 DISCUSSION OF RESULTS

In this experiment, it is observed that in Figure 1 both infected human and vaccinated human populations gradually increase over time while susceptible humans' population dropped sharply as time goes on. Moreover, in figure 2 both susceptible and infected populations of cattle decrease whereas vaccinated population increases over time. In another experiment in figure 3, both susceptible and infected snail populations decrease over time.

Figure 4 shows the effect of vaccination on susceptible human, it is observed that as vaccination rate increases from 0.00 to 0.75,

susceptible human population decreases. Similarly, figure 5 shows the effect of vaccination on infected human, it is observed that as vaccination rate increases at 0.00, 0.25, 0.50, and 0.75 there was a corresponding decrease in the infected human population over time.

The effect of variable human populations on susceptible and infected human, show that as the total population of human increases; both susceptible human and infected human correspondingly increase; see figure 6 and figure 7 respectively.

On the contrary, figure 8 shows that, as the variable human population increases, vaccinated human population decrease steadily. Meanwhile, variable snail population on both susceptible and infected snails has no effect whatsoever as depicted in figure9 and figure 10

6.0 CONCLUSION

The results in this paper agree with previous works showing the importance of the use of vaccine in checking the spread of schistosomiasis in human, cattle and snail populations. In such work, high rate of vaccination use decreases both susceptible and infected population in human, cattle and snail. In addition, results in this study revealed that,

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variable human populations on both susceptible and infected human has positive effect while it has negative effect on vaccinated human population. Conversely, effect of variable snail population on both susceptible and infected snails remain trivial. Hence, to pull stop to the menace of schistosomiasis, vaccination is an essential control strategy.

Contributions of the authors

Musa, S. and Bello, N. jointly formulated the model equations and carried out the stability analysis, while Umar, A. performed the numerical experiments using MATLAB R2016a.

Conflict of Interests

The authors declare that there is no conflict of interests.

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