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), a parasitic infection caused by flatworms of the family Schistosomatidae 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 of flatworms: *Schistosoma 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 schistosomiasis infections 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

### 2.0 MATERIALS AND METHODS

Table 1: Modified Model State Variable and their Description

| Variable | Description                        |
|----------|------------------------------------|
| $V_H(t)$ | Vacinated compartment for human $t$|
| $V_C(t)$ | Vacinated compartment for cattle $t$|
| $S_H(t)$ | Susceptible human population at time $t$|
| $S_S(t)$ | Susceptible snail population at time $t$|
| $S_C(t)$ | Susceptible cattle population at time $t$|
| $I_H(t)$ | Infected human population at time $t$|
| $I_S(t)$ | Infected snail population at time $t$|
| $I_C(t)$ | Infected cattle population at time $t$|
Table 2: Modified Model Parameters and their Description

| Parameters | Descriptions |
|------------|--------------|
| $b_h$      | Natural birth rate of human |
| $\beta_{sh}$ | transmission rate from infected snail to human |
| $r_h$      | recovery rate of infectes human |
| $b_c$      | natural birth rate of cattle |
| $\beta_{sc}$ | transmission rate from infected snail to cattle |
| $d_c$      | death rate of infected snails |
| $(b_c - d_c)$ | carrying capacity of cattle |
| $k_c$      | |
| $r_c$      | recovery rate of infectes cattle |
| $b_s$      | natural birth rate of snails |
| $\beta_{hs}$ | transmission rate from infected human to snail |
| $\beta_{cs}$ | transmission rate from infected cattle to snail |
| $d_s$      | transmission rate from infected cattle to snails |
| $(b_s - d_s)$ | carrying capacity of snails |
| $k_s$      | |
| $d_h$      | Death due to the disease in human |
| $\mu_h$    | Death due to natural causes in human |
| $\mu_s$    | Death due to natural causes in Snail |
| $\mu_c$    | Death due to natural causes in cattle |
| $\upsilon_h$ | Vaccination rate for human |
| $\epsilon_h$ | Rate of loss of immunity in human |
| $\upsilon_c$ | Vaccination rate for Cattle |
| $\epsilon_c$ | Rate of loss of immunity in cattle |

### 2.1 Zimin et al. (2010) Assumptions

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

i. Human, cattle and snail populations are all positive, i.e., $N_H > 0$, $S_c + I_c > 0$ and $S_s + I_s > 0$.

ii. The birth rate is greater than the death rate for both cattle and snails, i.e., $b_c - d_c > 0$ and $b_s - d_s > 0$.

### 2.2 Zimin et al. (2010) Model Equations

\[
\frac{dS_h}{dt} = \beta_{sh} S_h I_s + r_h I_h 
\]

\[
\frac{dI_h}{dt} = \beta_{sh} S_h I_s - r_h I_h 
\]

\[
\frac{dS_c}{dt} = b_c(S_c + I_c) - \beta_{sc} S_c I_s + r_c I_c - d_c S_c - k_c S_c (S_c + I_c) + I_c 
\]

\[
\frac{dI_c}{dt} = \beta_{sc} S_c I_s - r_c I_c - d_c I_c - k_c I_c (S_c + I_c) 
\]

\[
\frac{dS_s}{dt} = b_s(S_s + I_s) - \beta_{hs} S_s I_h - \beta_{cs} S_s I_c - d_s S_s - k_s S_s (S_s + I_s) 
\]

\[
\frac{dI_s}{dt} = \beta_{hs} S_s I_h + \beta_{cs} S_s I_c - d_s I_s - k_s I_s (S_s + I_s) 
\]
2.3 Modified Model Assumptions

iii. Human, cattle and snail populations are all positive, i.e. \( N_H > 0, S_C > 0 \) and \( S_S > 0 \).

iv. The birth rate is greater than the death rate for both cattle and snails, i.e., \( b_C - d_C > 0 \) and \( b_S - d_S > 0 \).

v. The recruitment rate of human into the susceptible class by natural birth.

vi. The vaccinated chambers as vaccines availability for both human and cattle are feasible.

vii. Death due to natural death can occur in both susceptible and infected humans, cattle, and snails.

2.4 Modified Model Equations

\[
\frac{dS_H}{dt} = b_H N_H - \beta_{SH} S_H I_S + r_H I_H - v_H S_H + \epsilon_H V_H - \mu_H S_H \tag{1}
\]

\[
\frac{dI_H}{dt} = \beta_{SH} S_H I_S - (d_H + \mu_H) I_H - r_H I_H \tag{2}
\]

\[
\frac{dS_C}{dt} = b_C N_C - \beta_{SC} S_C I_C + r_C I_C - \mu_C S_C - k_C S_C (S_C + I_C) - \mu_C S_C + \epsilon_C V \tag{3}
\]

\[
\frac{dI_C}{dt} = \beta_{SC} S_C I_S - r_C I_C - (d_C + \mu_C) I_C - k_C I_C (S_C + I_C) \tag{4}
\]

\[
\frac{dS_S}{dt} = b_S N_S - \beta_{HS} S_S I_H + \beta_{CS} S_C I_C - \mu_S S_S - k_S S_S (S_S + I_S) \tag{5}
\]

\[
\frac{dI_S}{dt} = \beta_{HS} S_S I_H + \beta_{CS} S_C I_C - (d_S + \mu_S) I_C - k_S I_S (S_S + I_S) \tag{6}
\]

\[
\frac{dV_H}{dt} = v_H S_H - \epsilon_H V_H - \mu_H V_H \tag{7}
\]

\[
\frac{dV_C}{dt} = v_C S_C - \epsilon_C V_C - \mu_C V_C \tag{8}
\]

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

\[
\dot{i}_H = B_{SH} (1 - v_h) - (B_{SH} + r_H + d_H + b_H - d_H i_h) i_h \tag{9}
\]

\[
v_h' = v_H (1 - i_h) - (v_H + \epsilon_H + b_H - d_H i_h) v_h \tag{10}
\]

\[
\dot{i}_C = B_{SC} (1 - v_c) - (B_{SC} + r_C + d_C + b_C - d_C i_c) i_c \tag{11}
\]

\[
v_c' = v_C (1 - i_c) - (v_C + \epsilon_C + b_C - d_C i_c) v_c \tag{12}
\]

\[
i_S' = B_{HS} + B_{CS} - (B_{HS} + B_{CS} + d_S + b_S - d_S i_s) i_s \tag{13}
\]

3.0 RESULTS

3.1 Disease Free Equilibrium (DFE) State of the Model

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:

\[
v_h = \frac{v_H}{v_H + \epsilon_H + b_H}, \quad \text{and} \quad v_c = \frac{v_C}{v_C + \epsilon_C + b_C}
\]

Remember that the following equations hold throughout this study

\[
B_{SH} = \beta_{SH} I_S, B_{SC} = \beta_{SC} I_S, B_{CS} = \beta_{CS} I_C, B_{HS} = \beta_{HS} I_H \tag{14}
\]

Hence, the disease free equilibrium point is given as:

\[
(i_h, v_h, i_c, v_c, i_s) = \left( 0, \frac{v_H}{v_H + \epsilon_H + b_H}, 0, \frac{v_C}{v_C + \epsilon_C + b_C}, 0 \right) \tag{15}
\]
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:

\[
R_0 = \sqrt{\frac{(r_H + d_H + b_H)\beta_{CS}^2 + \beta_{HS}^2 (r_C + d_C + b_C)}{(r_H + d_H + b_H)(r_H + d_H + b_H)(d_s + b_s)}}
\]

(19)

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

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:

\[
J = \begin{bmatrix}
-(B_{SH} + r_H + d_H + b_H) & -B_{HS} & 0 & 0 & 0 \\
-d_H (B_{SH} + v_H) & B_{SH} + v_H + e_H + b_H & (v_H + e_H + b_H) & -B_{HS} & 0 \\
-v_H & B_{HS} + v_H + e_H + b_H & -(v_H + e_H + b_H) & 0 & 0 \\
0 & 0 & 0 & 0 & -(B_{HS} + B_{CS} + d_s + b_s)
\end{bmatrix}
\]

(20)

For simplification purpose, let’s denote

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

\[
, \quad q = v_c + \frac{d_C (B_{CS} + v_c)}{B_{CS} + v_c + e_c + b_C}, \quad \omega = (v_c + e_c + b_C), \quad n = (B_{HS} + B_{CS} + d_s + b_s)
\]

Then equation (20) becomes

\[
J = \begin{bmatrix}
-q - \lambda & -B_{HS} & 0 & 0 & 0 \\
0 & -\omega - \lambda & 0 & 0 & 0 \\
0 & 0 & -k - \lambda & -B_{CS} & 0 \\
0 & f & -f - \lambda & 0 & 0 \\
0 & 0 & 0 & 0 & -n - \lambda
\end{bmatrix}
\]

(21)

Solving equation (21) for eigen values gives the followings:
Therefore, the disease free equilibrium state is stable if and only if

$$B_{HS} \left( \nu_c + \frac{d_c (B_{CS} + \nu_{cs})}{B_{CS} + \nu_c + \nu_{cs} + b_c} \right) < (\nu_{hs} + \nu_{hc} + b_h).$$

### 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

| Parameters/Variables | Values        | References                |
|----------------------|---------------|---------------------------|
| $V_H(0)$             | 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                   |
| $\beta_{SH}$         | $2.23 \times 10^{-7}$ | Allen and Victory, (2003) |
| $r_H$                | $4.47 \times 10^{-7}$ | Allen and Victory, (2003) |
| $b_C$                | $1.20 \times 10^{-3}$ | Allen and Victory, (2003) |
| $\beta_{SC}$         | $2.00 \times 10^{-3}$ | Allen and Victory, (2003) |
| $d_C$                | $5.00 \times 10^{-6}$ | Allen and Victory, (2003) |
| $(b_C - d_C)$         | $7.00 \times 10^{-3}$ | Allen and Victory, (2003) |
| $k_C$                | $2.4 \times 10^{-2}$   | Allen and Victory, (2003) |
| $b_S$                | $6.00 \times 10^{-2}$   | Allen and Victory, (2003) |
| $\beta_{HS}$         | $1.04 \times 10^{-5}$  | Allen and Victory, (2003) |
| $\beta_{CS}$         | $1.05 \times 10^{-7}$  | Allen and Victory, (2003) |
| $d_S$                | $8.86 \times 10^{-3}$  | Allen and Victory, (2003) |
| $(b_S - d_S)$         | $7.00 \times 10^{-3}$  | Allen and Victory, (2003) |
### Table 3 Continue

| Parameter | Value          | Source                      |
|-----------|----------------|-----------------------------|
| $b_H$     | 0.312          | Kbenesh et al. (2009)       |
| $\mu_H$  | $4.0 \times 10^{-5}$ | Hyun (2001)               |
| $d_H$     | $5.0 \times 10^{-4}$ | WHO (2003)                 |
| $\mu_S$  | $8.86 \times 10^{-3}$ | Allen and Victory, (2003) |
| $\mu_C$  | $5.00 \times 10^{-3}$ | Allen and Victory, (2003) |
| $\nu_H$  | $0.00 - 0.75$   | Assumed                    |
| $\varepsilon_H$ | 0.31 | Assumed                   |
| $\nu_C$  | $0.00 - 0.75$   | Assumed                    |
| $\varepsilon_C$ | 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
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 figure 9 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, 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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