A Deterministic Mathematical Model for the Control of the Spread of Schistosomiasis in Nigeria

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Abstract: The worldwide schistosomiasis epidemic is a global health crisis. Nigeria, a country that is part of Africa where the diseases were commonly found has her fare share of this crisis. In this study two equilibrium states were analyzed, the disease free equilibrium state was analyzed for stability or otherwise using Bellman and Cooke theorem and the disease endemic state was analyzed for stability or otherwise using the linearised stability theorem. It was established that the disease endemic state is locally asymptotically stable, while the disease free equilibrium state is stable if the birthrate (β) of the snails is from 0-20% and unstable if the birth rate (β) of the snails is from 30% and above.

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INTRODUCTION

Schistosomiasis, which is also known as bilharzia (bilharziasis or snail fever) is a water –borne parasitic infections caused by several species of flukes of the genus schistosoma. Schistosomiasis is the second most socioeconomically devastating parasitic diseases after malaria (the Carter centre 2008). This disease is most commonly found in Asia, Africa and South America, especially in areas where the water contains numerous fresh water snails. The disease affects many people in developing countries, particularly children who may acquire the disease by swimming or playing in infected water.

Schistosomiasis remains a major public health problem in many developing countries. The disease affects 207 million of people and poses a threat to 600 million people in more than 76 countries (WHO, 1993). The debilitating nature of schistosomiasis is a markedly deterrent factor in food and other production sector (Makanga B. 1991). Although it has a low mortality rate, schistosomiasis often is a chronic illness that can damage internal organs and, in children, impair growth and The cognitive development. urinary form of schistosomiasis which is cause by "schistosoma haematobium" is associated with increased risks for bladder cancer in adults (the Carter centre 2008).

Above all, schistosomiasis is a chronic disease. matures sexually and migrates to the liver where they Manifestations include: abdominal pain, cough, diarrhea, fever, fatigue, hepatosplenomegaly (which causes the enlargement of both the liver and the spleen), genital sores (which causes lesions that increase vulnerability to HIV infection). Lesions caused by schistosomiasis may continue to be a problem after control of the infection itself. Early treatment, especially of children, which is relatively inexpensive, prevents formation of the sore

(Donald, G. McNeil, J. 2009) and(Hotez et'al. 2009).At the start of infection, mild itching and a popular dermatitis of the feet and other part of the skin will manisfestate after swimming in polluted streams containing cercariae (James et'al. 2006).

Schistosomiasis is caused by parasitic flat worms of the genus schistosoma. Male and female mate within the human host and lay eggs in the blood vessels which line in the bladder and intestine. These eggs induce an immune reaction which causes the swelling of the spleen (hepatosplenic) and liver (liver fibrosis). A portion of the eggs leave the body with feaces or urine and find their way into freshwater supply where they hatch into a free swimming ciliated larva called a miracidium of about 0.2mm long.

If the miracidium reaches a fresh water snail of a suitable species, it penetrates and transforms into a sporocyst. The sporocyst begins an asexual phase of reproduction within the body of the snail producing thousands of daughter sporocyst. The infected snails then release a second form of free swimming larva called cercariae, of about 1mm long with a characteristic forked tail, into the water. The miricidia cannot infect a human being but the cercariae can. The cercariae eventually penetrate the skin of a human, Loses its tail and enters the blood vessels as a schistosomulum. It grows to adult size, matures sexually and migrates to the liver where they cause liver fluke, or migrate to the intestines or the stomach where they cause schistosomiasis. Most of the severe illnesses caused by schistosomiasis occur in children under 14 years of age. This was because they are commonly found wading or swimming in lakes, ponds and other bodies of water that are infested with snails (usually of the Biomphalaria, bulinus, or oncomelania genus) that

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(Kheir et'al. 1999).In this research, we developed a mathematical model for the control of the spread of schistosomiasis disease, analyze the equilibrium states for stability or otherwise.

MATERIALS AND METHODS

Model description: The model consisted of four (4) ordinary differential equations which specify the rate of change of the two compartments (susceptible and infected) of the individual in the human population and two compartments (susceptible and infected) of the snail population over time. The susceptible human compartment grows with new recruitment (Λ_h) into the population and the recovery rate from infected class decreases as a result of interaction with the infected snails (I_v) at the rate (α_1)

and natural death at the rate (μ_1).

The infected human compartment grows with those susceptible human that moves out of susceptible compartment as a result of interaction with the infected snails (I_v), and decreases with natural death (μ_1) and death due to infections (μ_0). The susceptible snail compartment grows with birthrate of the snails at rate (β) and decreases as a result of interaction with the infected human (I_h) at rate (α_3) and natural death at rate (μ_2).

The last compartment is the snails infected compartment. This compartment grows with those susceptible snails that moves out of the susceptible compartment as a result of interaction with the infected human (I_h) at rate (α_3) and decreases as a result of natural death (μ_2). In this study, record of reported cases of water borne diseases (schistosomiasis) from the year 2003 to 2005 was collected from federal ministry of water Resources Survey, Abuja.



$$\frac{dS_{\nu}}{dt} = \beta S_{\nu} - (\mu_2 + \alpha_3 I_h) S_{\nu}$$
(3)
$$\frac{dI_{\nu}}{dt} = \alpha_3 I_h S_{\nu} - \mu_2 I_{\nu}$$
(4)

Equilibrium solutions: We now solve the model equations to obtain the equilibrium states as by Enagi (2011). At equilibrium state:

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dS_v}{dt} = \frac{dI_v}{dt} = 0$$

Let:

 $(S_h(t), I_h(t), S_v(t), I_v(t)) = (w, x, y, z)$ Then the system of equations becomes

$$\Lambda_h - (\alpha_1 z + \mu_1) w - \alpha_2 x = 0$$
(5)

$$\alpha_{1}zw - (\mu_{1} + \mu_{0} + \alpha_{2})x = 0$$
(6)

$$\beta y - (\mu_2 + \alpha_3 x) y = 0 \tag{7}$$

$$\alpha_3 x y - \mu_2 z = 0 \tag{8}$$

For the disease free equilibrium state, we have: From equation (8), we have;

$$\alpha_3 xy - \mu_2 z = 0$$

$$\alpha_3 xy = \mu_2 z$$

$$z = \frac{\alpha_3 xy}{\mu_2}$$
(9)

From equation (7), we have;

$$\left[\beta - \left(\mu_2 + \alpha_3 x\right)\right] y = 0 \Longrightarrow y = 0 \tag{10}$$

Substituting y = 0 in equation (9) yields: z = 0 (11) From equation (6), we have;

$$\alpha_1 z w = (\mu_1 + \mu_0 + \alpha_2) x$$

$$x = \frac{\alpha_1 z w}{\mu_1 + \mu_0 + \alpha_2}$$
But from (11), z = 0, hence
$$x = 0$$
(12)

Also from equation (5), we have; $\Lambda_{h} - (\alpha_{1}z + \mu_{1})w - \alpha_{2}x = 0$

$$\Lambda_h - \alpha_1 z w - \mu_1 w - \alpha_2 x = 0$$

 $\Lambda_h - \mu_1 w - \alpha_2 x = \alpha_1 z w$

$$z = \frac{\Lambda_h - \mu_1 w - \alpha_2 x}{\alpha_1 w} \tag{13}$$

Again from equation (6), we have;

$$\alpha_1 z w - \left(\mu_1 + \mu_0 + \alpha_2\right) x = 0$$

$$\alpha_1 z w = (\mu_1 + \mu_0 + \alpha_2) x$$
$$z = \frac{(\mu_1 + \mu_0 + \alpha_2) x}{\alpha_1 w}$$
(14)

Equating equation (13) and (14) yields

$$\frac{\Lambda_{h} - \mu_{1}w - \alpha_{2}x}{\alpha_{1}w} = \frac{(\mu_{1} + \mu_{0} + \alpha_{2})x}{\alpha_{1}w}$$

$$\Lambda_{h} - \mu_{1}w - \alpha_{2}x = (\mu_{1} + \mu_{0} + \alpha_{2})x$$

$$\Lambda_{h} - \mu_{1}w - \alpha_{2}x - (\mu_{1} + \mu_{0} + \alpha_{2})x = 0$$

$$\Lambda_{h} - \alpha_{2}x - (\mu_{1} + \mu_{0} + \alpha_{2})x = \mu_{1}w$$

$$w = \frac{\Lambda_{h} - \alpha_{2}x - (\mu_{1} + \mu_{0} + \alpha_{2})x}{\mu_{1}}$$

$$w = \frac{\Lambda_{h} - (2\alpha_{2} + \mu_{0} + \mu_{1})x}{\mu_{1}}$$
(15)

Substituting equation (12) into (15) yields:-

$$w = \frac{\Lambda_h}{\mu_1} \tag{16}$$

Hence, the disease free equilibrium state is:

$$(w, x, y, z) = \left(\frac{\Lambda_h}{\mu_1}, 0, 0, 0\right)$$

For the disease Endemic state, Recall that equation (7) is given by:-

$$\beta y - (\mu_2 + \alpha_3 x) y = 0$$

$$[\beta - (\mu_2 + \alpha_3 x)]y = 0$$

Either $\beta - (\mu_2 + \alpha_3 x) = 0$ or y = 0. Hence $\beta = (\mu + \alpha r) = 0$

$$p - (\mu_2 + \alpha_3 x) = 0$$

 $\beta - \mu_2 - \alpha_3 x = 0$

 $\beta - \mu_2 = \alpha_3 x$

$$x = \frac{\beta - \mu_2}{\alpha_3} \tag{17}$$

Substituting equation (17) into (15) yields to:

$$w = \frac{\Lambda_{h} - \alpha_{2} \left(\frac{-(-\beta + \mu_{2})}{\alpha_{3}}\right) - (\mu_{1} + \mu_{0} + \alpha_{2}) \left(\frac{-(-\beta + \mu_{2})}{\alpha_{3}}\right)}{\mu_{1}}$$
$$w = \frac{\Lambda_{h} \alpha_{3} - 2\alpha_{2}\beta + 2\alpha_{2}\mu_{2} - \mu_{1}\beta - \mu_{0}\beta + \mu_{1}\mu_{2} + \mu_{0}\mu_{2}}{\mu_{1}\alpha_{3}}$$
(18)

From equation (14), we have

$$z = \frac{\left(\mu_1 + \mu_0 + \alpha_2\right)x}{\alpha_1 w}$$

$$z = \frac{(\mu_{1} + \mu_{0} + \alpha_{2})\left(\frac{(-(-\beta + \mu_{2})}{\alpha_{3}}\right)}{\alpha_{1}\left[\frac{\Lambda_{h}\alpha_{3} - 2\alpha_{2}\beta + 2\alpha_{2}\mu_{2} - \mu_{1}\beta - \mu_{0}\beta + \mu_{1}\mu_{2} + \mu_{0}\mu_{2}}{\mu_{1}\alpha_{3}}\right]}$$
$$z = -\frac{(\mu_{1} + \mu_{0} + \alpha_{2})(-\beta + \mu_{2})\mu_{1}}{\alpha_{1}(\Lambda_{h}\alpha_{3} - 2\alpha_{2}\beta + 2\alpha_{2}\mu_{2} - \mu_{1}\beta - \mu_{0}\beta + \mu_{1}\mu_{2} + \mu_{0}\mu_{2})}$$
(19)

Also from equation (8), we have;

$$\alpha_3 x y - \mu_2 z = 0 \qquad \qquad y = \frac{\mu_2 z}{\alpha_3 x} \tag{20}$$

Substituting equation (17) and (19) in (20) yields:

$$y = \frac{\frac{\mu_2 \lfloor (\mu_1 + \mu_0 + \alpha_2) (-(-\beta + \mu_2) \mu_1 \rfloor}{\alpha_1 (\Lambda_h \alpha_3 - 2\alpha_2 \beta + 2\alpha_2 \mu_2 - \mu_1 \beta - \mu_0 \beta + \mu_1 \mu_2 + \mu_0 \mu_2)}}{\alpha_3 \left(\frac{(-(-\beta + \mu_2))}{\alpha_3}\right)}$$

$$y = \frac{\mu_{1}\mu_{2}(\mu_{1} + \mu_{0} + \alpha_{2})}{\alpha_{1}(\Lambda_{h}\alpha_{3} - 2\alpha_{2}\beta + 2\alpha_{2}\mu_{2} - \mu_{1}\beta - \mu_{0}\beta + \mu_{1}\mu_{2} + \mu_{0}\mu_{2})}$$
(21)

Hence, the endemic equilibrium state is given by:

$$w = \frac{\Lambda_h \alpha_3 - 2\alpha_2 \beta + 2\alpha_2 \mu_2 - \mu_1 \beta - \mu_0 \beta + \mu_1 \mu_2 + \mu_0 \mu_2}{\mu_1 \alpha_3}$$
$$y = \frac{\mu_1 \mu_2 (\mu_1 + \mu_0 + \alpha_2)}{\alpha_1 (\Lambda_h \alpha_3 - 2\alpha_2 \beta + 2\alpha_2 \mu_2 - \mu_1 \beta - \mu_0 \beta + \mu_1 \mu_2 + \mu_0 \mu_2)}$$

$$z = -\frac{(\mu_{1} + \mu_{0} + \alpha_{2})(-\beta + \mu_{2})\mu_{1}}{\alpha_{1}(\Lambda_{h}\alpha_{3} - 2\alpha_{2}\beta + 2\alpha_{2}\mu_{2} - \mu_{1}\beta - \mu_{0}\beta + \mu_{1}\mu_{2} + \mu_{0}\mu_{2})}$$

Having established the equilibrium states. We now investigate the stability of the equilibrium states. To obtain this, we examine the behaviour of the model population near the equilibrium states.

RESULTS AND DISCUSSION Stability analysis of disease free equilibrium state The characteristic equation:

Recall that the system of the equations in this model at

equilibrium state is given by:-

$$\Lambda_h - (\alpha_1 z + \mu_1) w - \alpha_2 x = 0$$

$$\alpha_1 z w - (\mu_1 + \mu_0 + \alpha_2) x = 0$$

$$\beta_y - (\mu_2 + \alpha_3 x) y = 0$$

$$\alpha_3 x y - \mu_2 z = 0$$

The Jacobian matrix of this system of equations is given by:-

$$J = \begin{bmatrix} -\alpha_1 z + \mu_1 & -\alpha_2 & 0 & -\alpha_1 w \\ \alpha_1 z & -(\mu_1 + \mu_0 + \alpha_2) & 0 & \alpha_1 w \\ 0 & \alpha_3 y & \beta - (\mu_2 + \alpha_3 x) & 0 \\ 0 & \alpha_3 y & \alpha_3 x & -\mu_2 \end{bmatrix}$$

The characteristic equation obtained from the Jacobian determinant with the Eigen values λ at

$$(w, x, y, z) = \left(\frac{\Lambda_h}{\mu_1}, 0, 0, 0\right)$$
 is:-

 $(\mu_{1} - \lambda)\{\beta\mu_{1}\mu_{2} + \mu_{1}\beta\lambda - \mu_{1}\mu_{2}^{2} - 2\mu_{1}\mu_{2}\lambda - \mu_{1}\lambda^{2} + \beta\mu_{0}\mu_{2} + \beta\lambda\mu_{0} - \mu_{0}\mu_{2}^{2} - 2\mu_{0}\mu_{2}\lambda + \lambda^{2}\mu_{0} + \beta\mu_{2}\alpha_{2} + \beta\alpha_{2}\lambda - \mu_{2}^{2}\alpha_{2} - 2\mu_{2}\lambda\alpha_{2} - \lambda^{2}\alpha_{2} + \beta\mu_{2}\lambda + \beta\lambda^{2} - \mu_{2}^{2}\lambda - 2\mu_{2}\lambda^{2} - \lambda^{3}\} = 0$ Hen

ce the characteristic equation for the disease free equilibrium state is:-

$$\lambda^{4} - [\mu_{1} - \mu_{1} - \mu_{0} - \alpha_{2} + \beta - 2\mu_{2}]\lambda^{3} - [\mu_{1}^{2} + \mu_{0}\mu_{1} + \mu_{0}\alpha_{2} - \mu_{1}\beta + 2\mu_{1}\mu_{2} + \mu_{0}\beta - 2\mu_{1}\mu_{2} - 2\mu_{0}\mu_{2} + \beta\alpha_{2} - 2\mu_{2}\alpha_{2} + \beta\mu_{2} - \mu_{2}^{2}]\lambda^{2} + [\mu_{1}^{2}\beta - 2\mu_{1}^{2}\mu_{2} + \beta\mu_{0}\mu_{1} - 2\mu_{0}\mu_{2}\mu_{1} + \beta\alpha_{2}\mu_{2} - 2\mu_{1}\alpha_{2}\mu_{2} + \mu_{1}\beta\mu_{2} - \mu_{1}\mu_{2}^{2} - \mu_{1}\mu_{2}\beta + \mu_{1}\mu_{2}^{2} - \mu_{0}\mu_{2}\beta + \mu_{0}\mu_{2}^{2} - \beta\alpha_{2}\mu_{2} + \alpha_{2}\mu_{2}^{2}]\lambda + \mu_{1}^{2}\beta\mu_{2} - \mu_{1}^{2}\mu_{2}^{2} + \beta\mu_{1}\mu_{2}\mu_{0} - \mu_{1}\mu_{0}\mu_{2}^{2} + \beta\mu_{2}\mu_{0}\alpha_{2} - \mu_{1}\alpha_{2}\mu_{2}^{2} = 0$$

$$(22)$$

We then apply the result of Bellman and Cooke (1963) as applied by Enagi (2011) to analyse the disease free equilibrium state for stability or otherwise. Bellman and Cooke theorem is given by:

$$F(0)G'(0) - F'(0)G(0) > 0$$

But,
$$F(0) = \mu_1^2 \beta \mu_2 - \mu_1^2 \mu_2^2 + \beta \mu_1 \mu_2 \mu_0 - \mu_1 \mu_0 {\mu_2}^2 + \beta \mu_2 \mu_1 \alpha_2 - \mu_1 \alpha_2 {\mu_2}^2.$$
 (23)

$$G(0) = 0$$
(24)

$$F'(0) = 0.$$
(25)

$$G'(0) = \mu_{1} - \mu_{1} - \mu_{0} - \alpha_{2} + \beta - 2\mu_{2} + \mu_{1}^{2}\beta - 2\mu_{1}^{2}\mu_{2}$$

$$+\beta\mu_{0}\mu_{1} - 2\mu_{0}\mu_{2}\mu_{1} + \beta\alpha_{2}\mu_{2} - 2\mu_{1}\alpha_{2}\mu_{2} + \mu_{1}\beta\mu_{2}$$

$$-\mu_{1}\mu_{2}^{2} - \mu_{1}\mu_{2}\beta + \mu_{1}\mu_{2}^{2} - \mu_{0}\mu_{2}\beta + \mu_{0}\mu_{2}^{2} - \beta\alpha_{2}\mu_{2}$$

$$+\alpha_{2}\mu_{2}^{2}.$$
(26)

Using hypothetical values for the parameters in equation (23) and (26) to analyse the stability or otherwise of the disease free equilibrium state with the help of mathematical software (maple) which lead us to the table below.

μ_o	μ_{l}	μ_2	β	α2	F(0)	$G^{1}(0)$	Remark
0.2	0.1	0.3	0.0	0.0	-0.0027	-0.794	Stable
0.2	0.1	0.3	0.1	0.1	-0.0024	-0.792	Stable
0.2	0.1	0.3	0.2	0.2	-0.0015	-0.790	Stable
0.2	0.1	0.3	0.3	0.3	0.0273	-0.788	Unstable
0.2	0.1	0.3	0.4	0.4	0.0021	-0.786	Unstable
0.2	0.1	0.3	0.5	0.5	0.0048	-0.784	Unstable
0.2	0.1	0.3	0.6	0.6	0.0081	-0.782	Unstable
0.2	0.1	0.3	0.7	0.7	0.0120	-0.780	Unstable
0.2	0.1	0.3	0.8	0.8	0.0165	-0.778	Unstable
0.2	0.1	0.3	0.9	0.9	0.0216	-0.776	Unstable
0.2	0.1	0.3	1	1	0.0273	-0.774	Unstable

Where S = Stable, U = Unstable.

Stability Analysis of the Disease Endemic State

To obtain the stability analysis of the disease endemic state, we use the principle of linearised stability, as applied by (lanka 2007).

The condition for the linearised stability is given by:-

Let
$$J = \begin{pmatrix} f_x(x_*, y_*) & f_y(x_*, y_*) \\ g_x(x_*, y_*) & g_y(x_*, y_*) \end{pmatrix}$$

Be the Jacobian matrix of the non-linear system

$$\frac{dx}{dt} = f\left(x, y\right)$$

$$\frac{dy}{dt} = g\left(x, y\right)$$

Which can be evaluated at the critical point X_*, Y_*), Then

the critical point (X_*, Y_*) is

(i) Asymptotically stable if trace (J) < 0 and Det (J) > 0.

(ii) Stable but not asymptotically stable if trace (J) = 0 and Det (J) > 0.

(iii) Unstable if either, trace (J) > 0 and Det (J) < 0.

Let us look at our Jacobian matrix which is given

by:
$$J = \begin{bmatrix} -\alpha_1 z + \mu_1 & -\alpha_2 & 0 & -\alpha_1 w \\ \alpha_1 z & -(\mu_1 + \mu_0 + \alpha_2) & 0 & \alpha_1 w \\ 0 & \alpha_3 y & \beta - (\mu_2 + \alpha_3 x) & 0 \\ 0 & \alpha_3 y & \alpha_3 x & -\mu_2 \end{bmatrix}$$

We can easily see that the trace of the matrix is given by:-

 $T = -(\alpha_1 z - \mu_1 + \mu_1 + \mu_0 + \alpha_2 - \beta + \mu_2 - \alpha_3 x + \mu_2) < 0 \text{ and its}$ determinant is also given by:- $D = \alpha_1 z + \mu_2 z + \alpha_2 z + \alpha_3 z + \alpha_4 z + \alpha_5 z + \alpha_$

$$D = \alpha_{1}z\mu_{1}\mu_{2}^{2} + \alpha_{1}z\mu_{1}\mu_{2}\alpha_{3}x + \alpha_{1}z\alpha_{1}w\alpha_{3}y\beta - \alpha_{1}z\mu_{1}\mu_{2}\beta$$

- $\alpha_{1}z\alpha_{1}w\alpha_{3}y\alpha_{3}x - \alpha_{1}z\alpha_{1}w\alpha_{3}y\mu_{2} - \alpha_{1}z\alpha_{1}w\alpha_{3}y\alpha_{3}x - \mu_{1}^{2}\mu_{2}\beta$
+ $\mu_{1}^{2}\mu_{2}^{2} + \mu_{1}^{2}\mu_{2}\alpha_{3}x - \mu_{1}\alpha_{1}w\alpha_{3}y\alpha_{3}x + \mu_{1}\alpha_{1}w\alpha_{3}y\beta$
- $\mu_{1}\alpha_{1}w\alpha_{3}y\mu_{2} - \mu_{1}\alpha_{1}w\alpha_{3}y\alpha_{3}x - \alpha_{2}\alpha_{1}z\mu_{2}\beta + \alpha_{2}\alpha_{1}z\mu_{2}^{2}$
+ $\alpha_{2}\alpha_{1}z\mu_{2}\alpha_{3}x + \alpha_{1}w\alpha_{1}z\alpha_{3}y\alpha_{3}x - \alpha_{1}w\alpha_{1}z\beta\alpha_{3}y$
- $\alpha_{1}w\alpha_{1}z\alpha_{3}y\mu_{2} + \alpha_{1}w\alpha_{1}z\alpha_{3}y\alpha_{3}x > 0.$

Therefore the roots of the characteristic equation have negative real parts. According to the principle of linearised stability; the disease endemic state is locally asymptotically stable; Infact, the endemic steady state is globally asymptotically stable.

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REFERENCES

- 1. Bellman, R. and Cooke K.L. Differential Difference Equations, Academic Press London, (1963)11 45.
- Carter Center. "Schistosomiasis ControlProgram" <u>http://www.cartercenter.org/health/</u> Schistosomiasis/index, html, retrieved 2008-07-17.

2/14/12

- Carter Center. "How is Schistosomiasis Treated" <u>http://www.cartercenter.org/health/</u>Schistosomiasis /treatment.html retrieved 2008-07-17.
- 4. Donald, G. McNeil Jr (2009). "Parasites: Giving a Deworming Drug to Girls could cut HIV Transmission in Africa" Article in the New York Times.
- 5. Enagi A.I Development and Some Applications of New Deterministic Models For Analysing the Spread and Eradication of Tuberculosis in Nigeria. A Ph.D thesis (Unpublished), (2011). Submitted to the Department of Mathematics, Usmanu Danfodiyo University Sokoto.
- Hotez, P.J, Fenwick A. and Kjetland E.F Africa's 32 cents solution for HIV/AIDS. Plos NeglTropdis (2009). (5).e430.doi:10:137/journal.pntd.0000430.
- 7. James, William, D.Berger, Timothy, GAndrews Disease of the Skin: Clinical Dermatology Sauder's Elsevier. (2006). ISBN 0-7216-2921-0.
- Kheir, M.M, Eltoum, A., Sa'ad, A.M, Ali, M.M, Baraka, O.Z, Homeida, M.M. Mortality due to Schistosomiasis Mansoni: A field study in Sudan A.M.J Trop.med.Hyg (1999). 60(2): 307-10. PMID10072156.
- 9. Lanka, B.. The mathematics of infectious disease. M.sc thesis published, (2007)Comenius University Bratislava.
- Makanga, B.. The molluscicidal properties of balanites wilsoniana dawe and Sprague and Khaya grandifoliola C.D.C. Ph.D thesis, Department of zoology, Makerere University Kampala. (1991).
- World Health Organization). The Control of Schistosomiasis, Second Report of theWHO Expert Committee. World health organ tech Rep ser. (1993)830:1-86.