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Full Length Research Paper

Mathematical Modelling for Screening and Migration in Horizontal and Vertical Transmission of HIV/AIDS

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This study proposed and analyzed a non-linear mathematical model for the dynamics of the effects of screening and migration in horizontal and vertical transmission of HIV/AIDS. The equilibriums point of the model was found and their stability is investigated. The model exhibited two equilibrium namely, the disease-free and the endemic equilibra. It is found that if the effective reproduction number Ro < 1, the disease free equilibrium is locally asymptomatically stable. If Ro ≤ 1, the disease free equilibrium is globally asymptomatically stable. If Ro > 1, a unique equilibrium exist which is locally, asymptotically stable.

Keywords: HIV/AIDS, Mathematical model, Screening and Transmission

INTRODUCTION

The Human Immuno-Deficiency Virus (HIV) infection which can lead to Acquired Immuno-Deficiency Syndrome (AIDS) has become an important infectious disease in both developed and developing nations. HIV/AIDS infection is a very fatal disease which weakens the body immune system leaving the victims into a state of life threatening neurological disorders (Al-sheikh *et al,* 2011). HIV/AIDS quickly developed into worldwide epidemic immediately its detection in USA, in 1981 (Naresh *et al.,* 2014). It is estimated that in 2007, out of the 33 millions HIV/AIDS infective worldwide, 22 million of them are from Sub-Sahara Africa (UNAIDS, 2008).In worldwide HIV/AIDS statistics, an HIV/AIDS epidemic is defined by the HIV prevalence in the general population. HIV prevalence is the percentage of the population living with HIV (Ma and Ma, 2006).

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Globally, more than 30 million people are living with HIV/AIDS and over 23 million have died since 1981. About 95% of people with HIV live in developing and moderate-income nations and over 25 million people with HIV living in poor and moderate-income countries should be on antiretroviral medication. Of this number, only about 30% of these people are getting the treatment (Nasidi *et al*., 1986).

In Nigeria, AIDS was first diagnosed in 1985 in a female teenaged less than 14 years but was reported in 1986. This case was diagnosed in Lagos, one of the most populous cities in Nigeria (Abdurrahman, 2009). Twenty seven years after, the disease has become a massive epidemic which has become not only a health burden but also a socio-economic problem (Usman *et al.,* 2012). According to Margaret Lampe of the US Centers for Disease Control and Prevention, the number of people living with HIV/AIDS in Nigeria increased by almost 500,000 in three years, while the number of AIDS-related

deaths also witnessed a marginal rise to 217,148 within the same period. Of particular interest to Jean Anderson of the Johns Hopkins Medical Institute, Maryland was the high rate of infection through blood transfusion and mother-to-child transmission, despite the fact that such forms of transmission are easily preventable. A United Nations report last year had also described Nigeria as the country with the highest number of children living with the virus in the world. The report said in part, "Nigeria has the largest number of children acquiring HIV infection, nearly 60,000 in 2012 — a number that has remained unchanged since 2009." (Adebayo, 2014).

In order to find an efficient way to control an infection, it is of great importance to establish its transmission dynamics. One main goal of mathematical epidemiology is to understand how to control and eradicate diseases (Akinwande, 2006). Mathematical models are used extensively in the study of ecological and epidemiological phenomena (Kaplan and Brandeau, 1994). They are particularly helpful as experimental tools with which to evaluate and compare control procedures and preventive strategies, and to investigate the relative effects of various sociological, biological and environmental factors on the spread of diseases.

The past century has witnessed the rapid emergence and development of a substantial theory of epidemics. Numerous mathematical models were developed to study a disease transmission, to evaluate the spread of epidemics and more importantly, to understand the mechanisms of epidemics in order to prevent them or minimize the transmission of disease via behavior change, vaccination, treatments, quarantine and other measures; Blower and Paul (2002), Simwa and Pokhariyal (2003), Bonzzette (2005), Tripathi *et al*, (2007), Geofrey (2009), Garba and Gummel, (2010), Al-Sheikh *et al,* (2011), Naresh and Dileep (2011), and Ofusuhene *et al*. (2014).

In the past years, Blower and Paul (2002), Al-Sheikh et al (2011), Naresh and Dileep (2011), and Ofusuhene *et al.,* (2014) have designed mathematical models on HIV/AIDS and provided long-term predictions regarding HIV/AIDS prevalence and control in various regions. Considering the works of the aforementioned authors, a new mathematical model was developed to complement and extend on their works by incorporating vertical and horizontal transmission, rate of recovering and treatment failure. These factors are very important in the transmission and control of HIV/AIDS especially in developing countries where the disease is prevalent. This necessitated a new research work to critically look into the dynamics of the effect of screening and migration in horizontal and vertical transmission of HIV/AIDS in order to come up with a feasible solution in controlling the disease.

MATERIALS AND METHODS

Model Formulation and Description

A mathematical model on the dynamics of the effect of screening and migration in horizontal and vertical transmission of HIV/AIDS was developed, improving on the existing model by incorporating the effect of treatment failure, screening and migration in horizontal and vertical transmission. Figure 1 is a schematic representation of the model.

The model contains five (5) state variables, namely: Susceptibles, (S), representing people who are likely to become infected with HIV; Unscreened HIV infectives, (I_u), Screened HIV infectives (I_s), Treated HIV infectives, (T); and AIDS individuals (A). There is constant inflow of susceptible at a rate of π. Both the treated infective and AIDS patients are assumed to be sexually inactive, and therefore non-infectious. The natural mortality rate is μ in all cases and the disease-induced death is η. It is also assumed that susceptibles become HIV infected via effective sexual contacts with infectives with force of infection $\Gamma = (\beta_1 I_u + \beta_2 I_s) S$. This sexual contact with infectives may also lead to the birth of children that are infected at birth, and hence, are directly recruited into the unscreened infective class at a rate $(1-\varepsilon)\theta$ and others are recruited into the screened infectives class at the rate $\varepsilon\theta$ with $(0 \le \varepsilon \le 1)$.

We considered the direct recruitment of immigrants into both the screened and unscreened infective classes at the rates $m(1-\pi)$ and $m\pi$ respectively. δ is a fraction of immigrants who left the population and ρ is the period of stay the individual is permitted to stay in the country.

The corresponding mathematical equations of the schematic diagram can be described by a system of Ordinary Differential Equations (ODEs) given below:

$$\frac{dS}{dt} = \pi - (\beta_1 I_u + \beta_2 I_s) S - (\mu + \rho\delta) S \quad (1a)$$

$$\frac{dI_u}{dt} = \left[(1-\varepsilon)\theta + m(1-\pi)\right] I_u + (\beta_1 I_u + \beta_2 I_s) S - \left[\alpha e^{\psi} + \mu + \rho\delta + \alpha(1-\sigma)\right] I_u \quad (1b)$$

$$\frac{dI_s}{dt} = (\varepsilon\theta + m\pi) I_s + \alpha\sigma e^{\psi} I_u - (\tau + \gamma_1 + \mu + \rho\delta) I_s \quad (1c)$$

$$\frac{dT}{dt} = \tau I_s - (\gamma_2 + \mu + \rho\delta) T \quad (1d)$$

$$\frac{dA}{dt} = \alpha(1-\sigma) I_u + \gamma_1 I_s + \gamma_2 T - (\mu + \rho\delta + \eta) A \quad (1e)$$

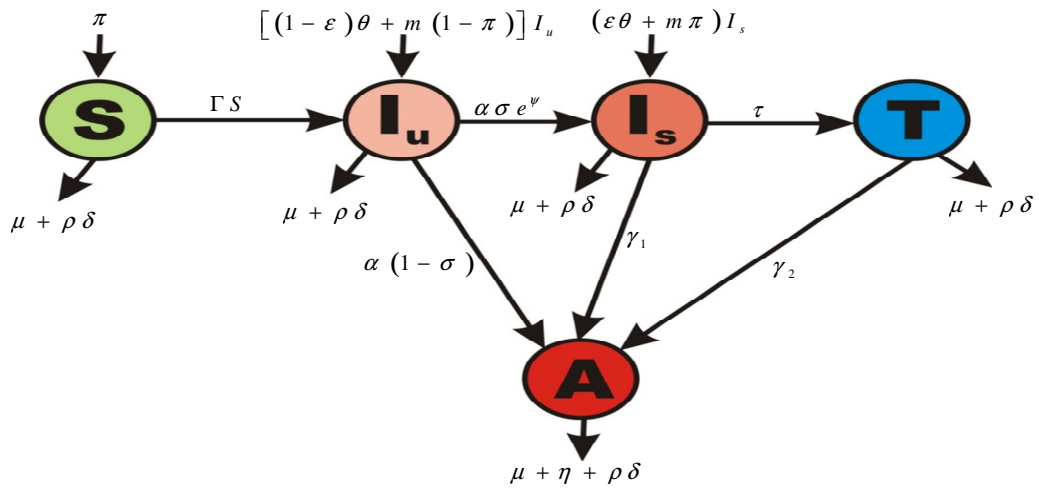

Figure 1 Schematic Diagram of the Model

Table 1 Definition of Parameters of the Model

Parameter	Interpretation
π	Recruitment rate of susceptible individuals
μ	Natural death rate
η	AIDS-induced death
θ	Rate of giving birth to offspring by infected pregnant mothers I_u and I_s
ε	Proportion of HIV-positive birth by infected pregnant mothers I_u and I_s
m	Rate at which immigrants enters I_u and I_s
ψ	Rate of screening individuals in class I_u
α	Rate of progression from I_u to I_s or A
σ	Proportion of I_u which progresses to I_s while $1-\sigma$ is the proportion of I_u which progresses to A
γ_1	Rates of progression to I_s to A
γ_2	Rates of progression to T to A
ρ	Period of stay the immigrant is permitted to stay in the country
δ	Fraction of immigrants who left the country
τ	Rate of treating screened infected individuals

Basic Assumptions for the Model

The following assumptions were taken into account in the model construction:

(1) The force of infection is given as $\left(\beta_1 I_u + \beta_2 I_s\right) S$

(2) Birth rate is not equal to death

(3) The rate of progression of HIV infectives to AIDS class satisfies $\alpha > \gamma_1 > \gamma_2$ since the higher the viral load of infectives, the faster the progression rate to AIDS

(4) Susceptible becomes infected via sexual contact

(1.2) – (1.10) are substituted into the system of equations (1.1) in order to efficiently simplify the equations.

$$k_1 = \mu + \rho\delta \quad (1.2)$$

$$k_2 = \alpha e^{\psi} + \mu + \rho\delta + \alpha(1-\sigma) \quad (1.3)$$

$$k_3 = \tau + \gamma_1 + \mu + \rho\delta \quad (1.4)$$

$$k_4 = \gamma_2 + \mu + \rho\delta \quad (1.5)$$

$$k_5 = \mu + \rho\delta + \eta \quad (1.6)$$

$$\vartheta_1 = (1-\varepsilon)\theta + m(1-\pi) \quad (1.7)$$

$$\vartheta_2 = \varepsilon\theta + m\pi \quad (1.8)$$

$$\vartheta_3 = \sigma e^{\psi} \quad (1.9)$$

$$\vartheta_4 = 1-\sigma \quad (1.10)$$

Hence, (1.1a) to (1.1e) becomes

$$\frac{dS}{dt} = \pi - (\beta_1 I_u + \beta_2 I_s) S - k_1 S \quad (1.11a)$$

$$\frac{dI_u}{dt} = \vartheta_1 I_u + (\beta_1 I_u + \beta_2 I_s) S - k_2 I_u \quad (1.11b)$$

$$\frac{dI_s}{dt} = \vartheta_2 I_s + \alpha\vartheta_3 I_u - k_3 I_s \quad (1.11c)$$

$$\frac{dT}{dt} = \tau I_s - k_4 T \quad (1.11d)$$

$$\frac{dA}{dt} = \alpha\vartheta_4 I_u + \gamma_1 I_s + \gamma_2 T - k_5 A \quad (1.11e)$$

Existence and Uniqueness of Solution for the Model

To check the validity and usability of any mathematical model, we have to confirm that the system of equations has a solution, if it has, is the solution unique? This subsection is concerned with finding if the system of equations has a solution and if the solution to the system is unique.

Theorem (Derick and Grossman, 1976)

Let D denotes the region:

$$\begin{aligned} &|t - t_0| \le a \\ &\| x - x_0 \| \le b \\ &x = (x_1, x_2, x_3 \cdots, x_n) \\ &x_0 = (x_{10}, x_{20}, x_{30} \cdots, x_{n0}) \end{aligned} \quad (1.12)$$

And suppose that $f(t,x)$ satisfies the Lipschitz condition

$$\left\| f(t,x_1) - f(t,x_2) \right\| \le k \left\| x_1 - x_2 \right\| \quad (1.13)$$

Whenever the pairs (t,x_1) and (t,x_2) belong to D, where k is a positive constant. Then there is a constant $\delta > 0$ such that there exist a unique continuous vector solution $x(t)$ of the system in the interval $t - t_0 < \delta$. It is important to note that the condition (3.6) is satisfied by the requirement that $\frac{\partial f_i}{\partial x_j}, i, j = 1, 2, 3, \ldots$ is continuous and bounded in D.

We now return to our model equations (1.1a) – (1.1e). We are interested in the region: $0 \le \xi \le R$. We look for a bounded solution in this region and whose partial derivatives satisfy $0 \le R < \infty$ where ξ and δ positive constants are.

As clearly shown above, the partial derivatives of the whole system(1.11) exist, they are finite and bounded as shown in (1.10) – (1.11) above. Hence, by Theorem 1, the model system (1.1) has a unique solution.

Disease-free Equilibrium State (E^0)

At the disease-free equilibrium state there is absence of disease. Thus, all the infected classes will be zero and the entire population will be made up of susceptible individuals.

Lemma 1.1: A disease-free equilibrium state of the model (1.1) exists at the point

$$\begin{pmatrix} S^0 \\ I_u^0 \\ I_s^0 \\ T^0 \\ A^0 \end{pmatrix} = \begin{pmatrix} \frac{\pi}{\mu + \rho\delta} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

Effective Reproduction Number R_O

The next generation operator approach described by Van de Driessche and Watmough (2002) is a better method used in finding R_T and it is widely accepted because it reflects the biological meaning of R_O. Using this method we obtained the basic reproduction number, R_T of the system (1.1) which is the spectral radius (ρ) of the next generation matrix, G, i.e $R_T = \rho(FV^{-1})$. F is the matrix of the new infection terms and V the matrix of the transition terms.

Then,

$$F = \begin{pmatrix} \beta_1 S^0 & \beta_2 S^0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} k_2 - \vartheta_1 & 0 & 0 & 0 \\ -\alpha\vartheta_3 & k_3 - \vartheta_2 & 0 & 0 \\ 0 & -\tau & k_4 & 0 \\ -\alpha\vartheta_4 & -\gamma_1 & -\gamma_2 & k_5 \end{pmatrix}$$

Thus

$$R_T = \frac{(k_3 - \vartheta_2)\beta_1 S^0 + \alpha\vartheta_3\beta_2 S^0}{(k_2 - \vartheta_1)(k_3 - \vartheta_2)}$$

Global stability of disease-free equilibrium

Global stability of equilibrium removes the restrictions on the initial conditions of the model variables. In global asymptotic stability, solutions approach the equilibrium for all initial conditions. There are many ways of proving the global stability of disease-free equilibrium which include among others the Lyapunov theorem and the Castillo-Chavez (2002) global stability theorem. We used the later in this work.

Theorem: The disease-free equilibrium, E^0 of system (1.11) is globally asymptotically stable (GAS) if $R_o < 1$.

Proof: To establish the global stability of the disease-free equilibrium, the two conditions (H1) and (H2) as in Castillo-Chavez *et al.* (2002) must be satisfied for $R_o < 1$,The model system (3.1) can be written in the form

$$X_1'(t) = F(X_1, X_2)$$

$$X_2'(t) = G(X_1, X_2);\ G(X_1, 0) = 0$$

where

$$\left.\begin{aligned} X_1 &= (S^0) \\ X_2 &= (I_u{}^0, I_s{}^0, T^0, A^0) \end{aligned}\right\}$$

with the components of $X_1 \in \mathbb{R}^1$ denoting the uninfected individuals and the components of $X_2 \in \mathbb{R}^4$ denoting the infected individuals.

The disease-free equilibrium is now denoted as

$$E^0 = (X_1^*, 0)$$

where

$$X_1^* = (S^0)$$

Now, to proof that the first condition, $(H1)$ for $X_1'(t) = F(X_1^*, 0)$ is true, i.e X_1^* is a globally asymptotically stable.

We have linear differential equations as thus

$$X_1'(t) = F(X_1, 0) = (\pi - k_1 S^0)$$

Solving, gives

$$S^0(t) = \frac{\pi}{k_1} - \frac{\pi}{k_1} e^{-k_1 t} + S^0(0) e^{-k_1 t}$$

Now, clearly from (3.34), we have that $I_u{}^0(t) + I_s{}^0(t) + T^0(t) + A^0(t) \to S^0(t)$ as $t \to \infty$ regardless of the value of $S^0(0)$. Thus, $X_1^* = (S^0, 0)$ is globally asymptotically stable.

Next, to prove that the second condition $(H2)$ is true, That is

$$\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2)$$

We have

$$A = \begin{pmatrix} -(k_2 - \vartheta_1 - \beta_1 S^0) & \beta_2 S^0 & 0 & 0 \\ \alpha\vartheta_3 & -(k_3 - \vartheta_2) & 0 & 0 \\ 0 & \tau & -k_4 & 0 \\ \alpha\vartheta_4 & \gamma_1 & \gamma_2 & -k_5 \end{pmatrix}$$

Since then $k_2 > \vartheta_1 + \beta_1 S^0$ and $k_3 > \vartheta_2$, it is clear that matrix A is an M-matrix (the off-diagonal elements of A are non-negative).

$$G(X_1, X_2) = \begin{pmatrix} \vartheta_1 I_u{}^0 + (\beta_1 I_u{}^0 + \beta_2 I_s{}^0) S^0 - k_2 I_u{}^0 \\ \vartheta_2 I_s{}^0 + \alpha\vartheta_3 I_u{}^0 - k_3 I_s{}^0 \\ \tau I_s{}^0 - k_4 T^0 \\ \alpha\vartheta_4 I_u{}^0 + \gamma_1 I_s{}^0 + \gamma_2 T^0 - k_5 A^0 \end{pmatrix}$$

Hence

$$\hat{G}(X_1, X_2) = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

It is thus obvious that $\hat{G}(X_1, X_2) = 0$. Hence, the proof is complete.

RESULTS AND DISCUSSION

The system of equations were tested for existence and uniqueness of solution, analyzed for criteria under which it is stable and the basic reproduction number was computed. This section simulates the system of equations using the data in Table 2 and Table 3 below.

Using the data from Table 2 below, we calculate the basic reproduction number and presented the result in

Table 2 Table of Parameters and Values for Model 1 and Model II

Parameter	Value	Source
π	2000	Naresh *et al., 2009*
μ	0.004	Shabani *et al., 2011*
η	0.00197	Shabani *et al., 2011*
θ	(0,1)	Varied for computational reason
ε	0.6	Apenteng and Ismail 2014
m	(0,1)	Varied for computational reason
ψ	(0,1)	Varied for computational reason
α	0.1461574	Apenteng and Ismail 2014
σ	(0,1)	Varied for computational reason
γ_1	0.56	Shabani *et al., 2011*
γ_2	0.36	Assumed
ρ	0.8596852	Apenteng and Ismail 2014
δ	14 days	Apenteng and Ismail 2014
τ	(0,1)	Varied for computational reason
ω	(0,1)	Rate of treatment failure

Table 3 Results for Model

ψ	τ	σ	θ	m	R_0	$\mathrm{Re}\,mark$
0.25	0.25	0.08	0.02	0.01	6.895901765	Stable
0.50	0.25	0.25	0.02	0.01	6.870396937	Stable
0.75	0.75	0.50	0.02	0.01	6.818078087	Stable
0.75	0.25	0.50	0.02	0.08	1.294849381	Stable
0.80	0.25	0.50	0.08	0.02	4.250042846	Stable
0.80	0.75	0.50	0.08	0.25	0.4358412149	Unstable
0.80	0.75	0.01	0.25	0.25	0.4359018664	Unstable
0.80	0.75	0.75	0.75	0.25	0.4360680763	Unstable
0.80	0.75	0.75	0.25	0.75	0.1476865392	Unstable
0.80	0.75	0.75	0.75	0.75	0.1477060850	Unstable

Table 3 above.

The simulation result table presented above established the positivity of solutions of the model. Table 2 shows the value of R_0 at constant infection rate but varying the rates among susceptible offspring by infected mothers, screened infected offspring, progression to AIDS and recruitment to AIDS while other parameters were kept constant. Table 3 shows the value of R_0, establishing the effect of contact rates on a population while varying infection varying the rates among susceptible offspring by infected mothers, treatment failure, screened infected offspring, progression to AIDS and recruitment to AIDS while other parameters were kept constant These were done to verify the effect of an infected pregnant woman, treatment failure, screened offspring, progression to AIDS and recruitment to AIDS.

Since the reproduction number is defined as the number of secondary case(s) generated by an infectious individual, hence the tables established that if a pregnant woman has HIV, the unborn baby has high possibility of contacting it from its mother. Also, the basic reproduction number from the simulated table established the importance of screening and migration and treatment failure as a means of reducing the breakout HIV/AIDS.

CONCLUSION

The numerical simulation result clearly shows that varying the parameters in combination such as screening and migration lead to the reduction of basic reproduction number. Thus, reduction in susceptible population, HIV/AIDS epidemics constitutes one of the health and developmental challenges in the absence of cure and there is need to develop effective strategies in the prevention and control of the infection.

Government and NGO's should subsidize the cost of HAART drugs, and made easily accessible for infective individuals, Infected individuals should be encouraged to use HAART drugs as prescribed by doctors through counseling and HIV-related public health education programs should be encouraged with the aim of emphasizing on the model of transmission, prevention and control measures of the disease.

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