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 $R_0 < 1$, the disease free equilibrium is locally asymptotically stable. If $R_0 \leq 1$, the disease free equilibrium is globally asymptotically stable. If $R_0 > 1$, 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).

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
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 \(\pi\). Both the treated infective and AIDS patients are assumed to be sexually inactive, and therefore non-infectious. The natural mortality rate is \(\mu\) in all cases and the disease-induced death is \(\eta\). 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 screened infective class at a rate \((1-\varepsilon)\theta\) and others are recruited into the screened infectives class at the rate \(\varepsilon\theta\) with \(0 \leq \varepsilon \leq 1\).

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. \(\delta\) is a fraction of immigrants who left the population and \(\rho\) 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:

\[
\begin{align*}
\frac{dS}{dt} &= \pi - (\beta_1 I_u + \beta_2 I_s) S - (\mu + \rho \delta) S \\
\frac{dI_u}{dt} &= -(1-\varepsilon)\theta + m(1-\pi) I_u + \beta_1 I_u + \beta_2 I_s S - (\alpha \varepsilon + \mu + \rho \delta + \alpha(1-\sigma)) I_u \\
\frac{dI_s}{dt} &= (\varepsilon\theta + m\pi) I_s + \alpha \sigma \varepsilon \psi I_u - (\tau + \gamma_1 + \mu + \rho \delta) I_s \\
\frac{dT}{dt} &= \tau I_s - (\gamma_2 + \mu + \rho \delta) T \\
\frac{dA}{dt} &= \alpha(1-\sigma) I_u + \gamma_1 I_s + \gamma_2 T - (\mu + \rho \delta + \eta) A
\end{align*}
\]
Table 1  Definition of Parameters of the Model

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

Basic Assumptions for the Model

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

1. The force of infection is given as (β_1 I_u + β_2 I_s) S
2. Birth rate is not equal to death
3. The rate of progression of HIV infectives to AIDS class satisfies α > γ_1 > γ_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.

\[ k_1 = μ + ρδ \]

Hence, (1.1a) to (1.1e) becomes
\[
\frac{dS}{dt} = \pi - (\beta_1 I_u + \beta_2 I_s) S - k_1 S \tag{1.11a}
\]
\[
\frac{dI_u}{dt} = \beta_1 I_u + (\beta_1 I_u + \beta_2 I_s) S - k_2 I_u \tag{1.11b}
\]
\[
\frac{dI_s}{dt} = \beta_2 I_s + \alpha \beta_3 I_u - k_3 I_s \tag{1.11c}
\]
\[
\frac{dI}{dt} = \tau I_s - k_4 I \tag{1.11d}
\]
\[
\frac{dA}{dt} = \alpha \beta_3 I_u + \gamma_1 I_s + \gamma_2 T - k_5 A \tag{1.11e}
\]

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:

\[
|t - t_0| \leq a
\]
\[
\|x - x_0\| \leq b
\]
\[
x(t) = (x_1, x_2, x_3, \ldots, x_n)
\]
\[
x(t) = (x_{10}, x_{20}, x_{30}, \ldots, x_{n0})
\]

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

\[
\|f(t, x_1) - f(t, x_2)\| \leq k\|x_1 - x_2\| \tag{1.13}
\]

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 \( \frac{\partial f_j}{\partial x_i} \), \( i, j = 1, 2, 3, \ldots \) is continuous and bounded in D.

We now return to our model equations (1.1a) - (1.1e). We are interested in the region: \( 0 \leq \xi \leq R \). We look for a bounded solution in this region and whose partial derivatives satisfy \( 0 \leq R < \infty \) where \( \xi \) and \( \delta \) 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

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

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 \( R_o \) and it is widely accepted because it reflects the biological meaning of \( R_o \). Using this method we obtained the basic reproduction number, \( R_o \) of the system (1.1) which is the spectral radius \( (\rho) \) of the next generation matrix, \( G \), i.e \( R_o = \rho(FV^{-1}) \). \( F \) 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

\[
V = \begin{pmatrix}
k_2 - \varphi_1 & 0 & 0 & 0 \\
-\varphi_3 & k_3 - \varphi_2 & 0 & 0 \\
0 & -\tau & k_4 & 0 \\
-\varphi_4 & -\gamma_1 & -\gamma_2 & k_5
\end{pmatrix}
\]
Thus \( R_f = \frac{(k_3 - \varphi_2)\beta_3S^0 + \alpha\varphi_3\beta_2S^0}{(k_2 - \varphi_1)(k_3 - \varphi_2)} \)

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 \( R_0 < 1 \).

**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 \( R_0 < 1 \), 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

\[
X_1 = (S^0) \quad X_2 = (I_u^0, I_s^0, T^0, A^0) \]

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, \( (H 1) \) 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

\[
X'_1(t) = F(X_1, 0) = (\pi - k_3S^0)
\]

Solving, gives

\[
S^0(t) = \frac{\pi}{k_1} - \frac{\pi}{k_1}e^{-k_3t} + S^0(0)e^{-k_3t}
\]

Now, clearly from (3.34), we have that

\[
I_u^0(t) + I_s^0(t) + T^0(t) + A^0(t) \to S^0(t) \quad \text{as} \quad t \to \infty \quad \text{regardless} \quad \text{of the value} \quad S^0(0).
\]

Thus, \( X^*_1 = (S^0, 0) \) is globally asymptotically stable.

Next, to prove that the second condition \( (H 2) \) is true, That is

\[
\tilde{G}(X_1, X_2) = AX_2 - G(X_1, X_2)
\]

We have

\[
A = \begin{pmatrix}
-k_2 - \beta S^0 & \beta S^0 & 0 & 0 \\
\alpha\varphi_3 & -(k_3 - \varphi_2) & 0 & 0 \\
0 & \tau & -k_4 & 0 \\
\gamma_1 & \gamma_2 & -k_5 & 0
\end{pmatrix}
\]

Since then \( k_2 > \varphi_1 + \beta S^0 \) and \( k_3 > \varphi_2 \), it is clear that matrix \( A \) is an M-matrix (the off-diagonal elements of \( A \) are non-negative).

Hence

\[
\tilde{G}(X_1, X_2) = \left( \begin{pmatrix}
\varphi_3 I_u^0 + (\beta I_u^0 + \beta_2 I_s^0)S^0 - k_2 I_u^0 \\
\varphi_2 I_s^0 + \alpha\varphi_3 I_u^0 - k_3 I_s^0 \\
\tau I_s^0 - k_4 T^0 \\
\alpha\varphi_3 I_u^0 + \gamma_1 I_s^0 + \gamma_2 T^0 - k_5 A^0
\end{pmatrix}
\right)
\]

It is thus obvious that \( \tilde{G}(X_1, X_2) = 0 \). 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 I and Model II

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\pi$</td>
<td>2000</td>
<td>Naresh et al., 2009</td>
</tr>
<tr>
<td>$\mu$</td>
<td>0.004</td>
<td>Shabani et al., 2011</td>
</tr>
<tr>
<td>$\eta$</td>
<td>0.00197</td>
<td>Shabani et al., 2011</td>
</tr>
<tr>
<td>$\theta$</td>
<td>(0,1)</td>
<td>Varied for computational reason</td>
</tr>
<tr>
<td>$\varepsilon$</td>
<td>0.6</td>
<td>Apenteng and Ismail 2014</td>
</tr>
<tr>
<td>$m$</td>
<td>(0,1)</td>
<td>Varied for computational reason</td>
</tr>
<tr>
<td>$\psi$</td>
<td>(0,1)</td>
<td>Varied for computational reason</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>0.1461574</td>
<td>Apenteng and Ismail 2014</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>(0,1)</td>
<td>Varied for computational reason</td>
</tr>
<tr>
<td>$\gamma_1$</td>
<td>0.56</td>
<td>Shabani et al., 2011</td>
</tr>
<tr>
<td>$\gamma_2$</td>
<td>0.36</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\rho$</td>
<td>0.8596852</td>
<td>Apenteng and Ismail 2014</td>
</tr>
<tr>
<td>$\delta$</td>
<td>14 days</td>
<td>Apenteng and Ismail 2014</td>
</tr>
<tr>
<td>$\tau$</td>
<td>(0,1)</td>
<td>Varied for computational reason</td>
</tr>
<tr>
<td>$\omega$</td>
<td>(0,1)</td>
<td>Rate of treatment failure</td>
</tr>
</tbody>
</table>

Table 3 Results for Model

<table>
<thead>
<tr>
<th>$\psi$</th>
<th>$\tau$</th>
<th>$\sigma$</th>
<th>$\theta$</th>
<th>$m$</th>
<th>$R_0$</th>
<th>Remark</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.25</td>
<td>0.25</td>
<td>0.08</td>
<td>0.02</td>
<td>0.01</td>
<td>6.895901765</td>
<td>Stable</td>
</tr>
<tr>
<td>0.50</td>
<td>0.25</td>
<td>0.25</td>
<td>0.02</td>
<td>0.01</td>
<td>6.870396937</td>
<td>Stable</td>
</tr>
<tr>
<td>0.75</td>
<td>0.25</td>
<td>0.50</td>
<td>0.02</td>
<td>0.01</td>
<td>6.818078087</td>
<td>Stable</td>
</tr>
<tr>
<td>0.75</td>
<td>0.25</td>
<td>0.50</td>
<td>0.02</td>
<td>0.08</td>
<td>1.294849381</td>
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<tr>
<td>0.80</td>
<td>0.25</td>
<td>0.50</td>
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<td>0.08</td>
<td>4.250042846</td>
<td>Stable</td>
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<tr>
<td>0.80</td>
<td>0.75</td>
<td>0.50</td>
<td>0.02</td>
<td>0.25</td>
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<td>Unstable</td>
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<td>0.80</td>
<td>0.75</td>
<td>0.01</td>
<td>0.25</td>
<td>0.25</td>
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<td>Unstable</td>
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<tr>
<td>0.80</td>
<td>0.75</td>
<td>0.75</td>
<td>0.25</td>
<td>0.25</td>
<td>0.4360680763</td>
<td>Unstable</td>
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<tr>
<td>0.80</td>
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<td>0.25</td>
<td>0.75</td>
<td>0.1476865392</td>
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</tr>
<tr>
<td>0.80</td>
<td>0.75</td>
<td>0.75</td>
<td>0.75</td>
<td>0.75</td>
<td>0.1477060850</td>
<td>Unstable</td>
</tr>
</tbody>
</table>

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.
REFERENCES
