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Mathematical Model for Vertical Transmission of HIV/IDS, in a Homogeneous Mixing Population

Umar Mallam Abdulkarim and H.S. Ndakwo
Department of Mathematical Sciences, Nasarawa State University Keffi,
Nasarawa State, Nigeria

Abstract: In this study, we studied the Susceptible-Exposed-Infectious-Aids, (SEIA) Epidemic model for vertical transmission of HIV/AIDS, along the line of Michael *et al.* assumptions with other additional considerations and employed the Routh-Hurwitz stability conditions and the threshold parameter, to examine the equilibrium states, (disease-freeequilibrium, DFE and the endemic equilibrium, EE states,). The threshold conditions for the stability of the disease-fee equilibrium and endemic equilibrium states are obtained and the Biological interpretations are also provided.

Key words: Basic reproductive number, HIV/AIDS, DFE, EE, vertical transmission, latency, infectious period, mortality rate

INTRODUCTION

The term vertical transmission of Human Immunodeficiency Virus and Acquired Immunodeficiency Syndrome (HIV/AIDS), is described as Mother to Child Transmission, (MTCT), (Olowofeso, 2005; Mugisha *et al.*, 2003). This means transmission of HIV/AIDS from infected mothers to their unborn or newly born babies, (Mugisha *et al.*, 2003). The Human Immunodeficiency Virus (HIV) infection in children is generally more serious then in adult due to faster disease complications and progression. However, there are three major mechanism of vertical transmission of HIV, (Rachel Waema and Olowofeso, 2005). These are, infection through the placenta, known as in utero infection; infection during birth known as intra-partum infection and infection through breast-feeding, known as post-partum. The concentration of infective HIV (high viral load) in the blood and genital secretions of HIV infected pregnant women appear to be the factor best associated with the risk of vertical transmission and this appears to be highest during labour and delivering. Approximately 25-38% of vertical transmission occurs in utero, (Mugisha *et al.*, 2003) and at least 50% of HIV infected children have been infected either peri-or post natally by infected mothers milk as observed by Mugisha *et al.* (2003). The major risk of infection through breast-feeding appears in the period early after birth, between 1 and 2 years, third of breast-fed children of HIV-infected mothers get infected (Mugisha *et al.*, 2003). Vertical transmission of HIV/AIDS has been the principal cause of 80-90% of HIV-infected children (Mugisha *et al.*, 2003), however there is no doubt that the treatment of pregnant women with their children with antiretroviral recombinants has reduced the transmission to some low levels in most developed countries, (Mugisha *et al.*, 2003), This reduction has not been significant in Africa, Asia and Latin America; perhaps this may be due to less access to and no systematic use of drugs, which may be due poverty and corruption of most government of developing countries. These problems couple with lack of discovery of possible vaccine continues to put the population at risk.

Some early published research articles that studied vertical transmission of epidemic diseases includes the works of Michael *et al.* and that of Pugliese *et al.* While Michael *et al.* studied the spread of the virus in a constant population, using both horizontal and vertical transmission, with incidence term that of bilinear mass-action, death and birth rates assumed, the same and also non disease mortality. He developed a SEIR model using these assumptions.

Pugliese *et al.* studied vertical transmission of the virus in a varying population, with density depended mortality and a disease induced mortality, not density depended. In this research, we would adopt Michael *et al.* definition with modifications consistent with HIV and AIDS transmission.

Corresponding Author: Umar Mallam Abdulkarim, Department of Mathematical Sciences, Nasarawa State University Keffi, Nasarawa State, Nigeria

THE MODEL

Assume that the population is homogeneous random mixing and partitioned into compartments of densities, as susceptible S(t), Exposed, (infective without symptoms) E(t), infectives, (can infect others) I(t) and AIDS, (those that have progressed from HIV to AIDS). Suppose the natural birth and death rates are assumed to be identical and denoted by μ and that HIV-infectious who developed full-blown AIDS are easily identifiable and are no longer sexually active as such, they don't contribute to human reproduction process. They are quarantine from infecting others. Suppose a fraction p and a fraction q of offspring from the exposed and the infectious classes, respectively are born into the exposed class. Then the birth flux into the exposed class is given by $p\Delta E+q\Delta I$ and the birth flux into the susceptible class is given by-$p\Delta E$- $q\Delta I$, in line with Michael *et al*), where. $0 \le p \le 1$ and $0 \ge q \ge 1$.

The dynamics of transmission of HIV and AIDS can be described by the transfer diagram: Fig. 1.

Equation of the model: Applying the assumptions and the inter-relations between the variables and parameters as described in the above compartmental model, the density dynamics of HIV are described by the following equations:

$$\dot{S} = \Delta - \beta\frac{IS}{N} - P\Delta E - q\Delta I - \mu S$$

$$\dot{E} = \beta\frac{IS}{N} + P\Delta E + q\Delta I - (m+\mu)E$$

$$\dot{I} = mE - (k+\mu)I$$

$$\dot{A} = \quad = kI - (\mu + r)A$$

Where N = S + E + I + A, is the total population size and Δ is a function of the total population size entering the susceptible class and the birth rate, μ same as the death rate.

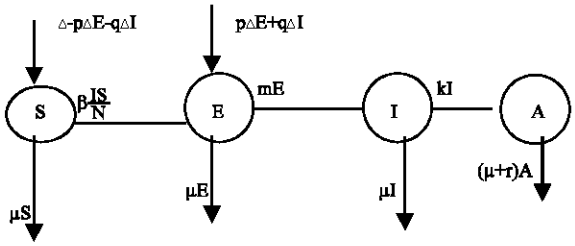

Fig. 1: Dynamics of transmission of HIV and AIDS

ANALYSIS OF THE MODEL

1/m is the mean latent period. When m approaches infinity the latent period is negligible and the model reduces to a SIA epidemic with bilinear incidence and no vertical transmission. When k = 0 then susceptible simply develop HIV and does not become AID patients and the model reduces to an SEI model. We would be considering the two models. One for progression of HIV to AIDS, with above assumptions and the other with progression from latency to HIV-infection. We would also examine the stability of the disease-free equilibrium state E_0 and the endemic equilibrium state E_1, using the Routh-Hurwitz stability conditions and the basic reproductive number, R_0. The disease-free equilibrium state is obtained as $E_0 = (\frac{\Delta}{\mu}, 0, 0, 0)$ and the Jacobian at the disease-free equilibrium is given by,

$$J_{E_0} = \begin{pmatrix} -b, & -p\Delta & -q\Delta & 0 \\ 0 & p\Delta - (m+\mu) & \beta C + q\Delta & 0 \\ 0 & m & -(k+\mu) & 0 \\ 0 & 0 & 0 & -(\mu+r) \end{pmatrix}$$

The characteristics equation associated with this matrix is also obtained as,

$$a_0\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0,$$

where $a_1 = m + k + r + 4\mu - p\Delta$

$$a_2 = \mu(\mu+r)(k+\mu)[p\Delta + (m+\mu)] + (2\mu+r)(m+k+2\mu - p\Delta) - m(\beta + q\Delta)$$

$$a_3 = (k+\mu)(2\mu+r)[p\Delta + (m+\mu)] + (m+k+2\mu - p\Delta)\mu(\mu+r) - m(2\mu+r)(\beta+q\Delta)$$

$$a_4 = [p\Delta + (m+\mu)](k+\mu) - m(\beta+q\Delta)$$

For the stability of the disease free equilibrium state, the coefficient of the characteristics equation must satisfy thefollowingconditions,

$$a_1 > 0, \quad a_3 > 0, \quad a_4 > 0, \quad a_1a_2a_3 > a_3^2 + a_1^2a_4,$$

$$a_1 > 0 \Rightarrow \mu < \frac{p\Delta - (m+k+r)}{4}$$

If $a_{21} = \mu(\mu+r)(k+\mu)[p\Delta + (m+\mu)]$,

$a_{22} = (2\mu+r)(m+k+2\mu - p\Delta)$, $a_{23} = m(\beta + q\Delta)$

$$a_{31} = (k+\mu)(2\mu+r)[p\Delta+(m+\mu)],$$
$$a_{32} = (m+k+2\mu-p\Delta)\mu(\mu+r)$$

$$a_{33} = m(2\mu+r)(\beta+q\Delta),$$
$$a_{41} = p\Delta+(m+\mu)(k+\mu),\ \ a_{42} = m(\beta+q\Delta)$$

It is obvious that

$$a_{21}>0,\ a_{22}>0,\ a_{31}>0,\ a_{32}>0,\ a_{33}>0,\ a_{41}>0,\ a_{42}>0$$

Then

$$a_1(a_{21}+a_{22}+a_{23})(a_{31}+a_{32}+a_{33})(a_{41}+a_{42}) = a_1a_2a_3>0$$

Where

$a_2 = (a_{21}+a_{22}+a_{23}),\ a_3 = (a_{31}+a_{32}+a_{33}),\ a_4 = (a_{41}+a_{42})$

Also, since $a_4 > 0$, we would $a_3^2 + a_1^2a_4 > 0$ have,

$$a_1a_2a_3 - (a_3^2 + a_1^2a_4) = a_1(a_{21}+a_{22})+a_{23})(a_{31}+a_{32}$$
$$+a_{33})(a_{41}+a_{42}) - [(a_{31}+a_{32}+a_{33})^2 + a_1^2(a_{41}+a_{42})]$$
$$= a_1(a_{21}+a_{22}+a_{23}) - \frac{(a_{31}+a_{32}+a_{33})}{(a_{41}+a_{42})} + a_1^2 > 0,$$
$$\text{if}\ \ (a_{21}+a_{22}+a_{23}) + a_1 > \frac{(a_{31}+a_{32}+a_{33})}{a_1(a_{41}+a_{42})}$$

Substitution gives,

$$\mu(\mu+r)(k+\mu)[p\Delta+(m+\mu)+(2\mu+r)$$
$$(m+2\mu-p\Delta)+m(\beta-q\Delta)+m+k+r+4\mu-p\Delta>$$

$$\frac{(k+r)(2\mu+r)[p\Delta+(m+\mu)]+(m+k+2\mu-p\Delta)}{\mu(\mu+r)+m(2\mu+r)(\beta+q\Delta)}$$
$$(m+k+r+4\mu-p\Delta)[(p\Delta+(m+r)(k+\mu)+m(\beta+q\Delta)$$

This is condition holds for these positive values of the model parameters.
Also,

$$a_4 > 0\ \text{ if } (k+\mu)[p\Delta+(m+b)] > m(\beta+q\Delta)$$
$$\text{i.e.,}\ \ \beta < \frac{(k+\mu)[p\Delta+(m+\mu)]-mq\Delta}{m}$$

Where β is the transmission rate of the HIV-infection. It also follows that the condition, $a_1a_2a_3>a_3^2+a_1^2a_4$, holds for positive values of the model parameters. Thus the disease-free equilibrium state is locally asymptotically stable and the infection will die out.

Existence and stability of endemic equilibrium state: The endemic equilibrium state is obtained as,

$$E_1 = (\Delta - \frac{(m+\mu)}{\mu}E,\ \frac{\Delta-\mu S}{m+\mu},\ \frac{mE}{k+\mu},\ \frac{kmE}{(\mu+r)(k+\mu)})$$

$$\text{Where } S^* = \Delta - \frac{(m+\mu)}{\mu}E,\ E^* = \frac{\Delta-\mu S}{m+\mu},$$
$$I^* = \frac{mE}{k+\mu},\ A^* = \frac{kmE}{(\mu+r)(k+\mu)}$$

S*, E*, I* and A* are fraction of the population that are susceptible, exposed, with HIV-infection and has progressed to AIDS respectively. However, E* exists if $\Delta > \mu S$ or if $S < \frac{\Delta}{\mu}$. Meaning that for E* exists, if the number of susceptibles recruited per unit of time is greater than the product of number of susceptibles and the recruitment rate. Also the fraction of the population susceptibles depends on whether, $\frac{\Delta\mu}{m+\mu} > 1$ where is $\frac{1}{m+\mu}$ the average length of the infection. Thus the endemic equilibrium state exists if these conditions hold. The Jacobian at the endemic state is obtained as,

$$J_{E_1} = \begin{pmatrix} -\beta\frac{I^*}{N} - b, & -p\Delta & -q\Delta & 0 \\ \beta\frac{I^*}{N} & P\Delta-(m+\mu) & \beta\frac{S^*}{N}+q\Delta & 0 \\ 0 & m & -(k+\mu) & 0 \\ 0 & 0 & k & -(\mu+r) \end{pmatrix}$$

Using this approach may render the computations inconclusive. However Heffernan *et al.* (2005), Castillo-Chavez *et al* and Mugisha *et al.* (2005), described the use of the basic reproductive number of infection in analyzing the stability of the endemic equilibrium state. According to their work, when, $R_0 > 0$ the system has a unique endemic equilibrium state that is globally asymptotically stable. However whether the virus becomes persistent or dies out depends on the magnitude of the basic reproductive number, R_0 as observed by Mugisha *et al.* (2005). According to him, the diseases-free equilibrium point is locally asymptotically stable $R_0 < 1$ if and unstable if $R_0 > 1$. Adopting this approach in this section, using the next generation operator developed by Heffernan *et al.* (2005), we get the following results.

Let X = (S), Y = (E), Z = (I) and the disease-free state is $E_0 = (\frac{\Delta}{\mu},0,0,0)$. Then we derived the following systems of equations in line with Heffernan *et al.* (2005).

g (X, Y, Z) = 0 is assumed to implicitly determined the function, $Y = \bar{g}(X^*, Y)$ as in Mugisha *et al.* (2005) and Castillo-Chavez *et al.* Thus we get

$$h(X,Y,Z) = m\frac{(\beta + P\Delta)}{(m+\mu-P\Delta)}Z - (\mu+k)Z,$$

$$h_Z(X,Y,Z) = m\frac{(\beta + P\Delta)}{(m+\mu-p\Delta)} - (\mu+k)$$

Letting H = M-D with m ≥ 0 and D>0, a diagonal matrix, we get,

$$M = \frac{m(\beta + P\Delta)}{(m+\mu-p\Delta)} \quad \text{and} \quad D = (\mu+k)$$

The basic reproductive number is defined as the spectral radius (dominant eigenvalue) of the matrix MD^{-1}, (Heffernan *et al.*, 2005), $R_0 = \rho(MD^{-1})$ is given by

$$R_0 = \frac{m(\beta + P\Delta)}{(m+\mu-p\Delta)(\mu+k)}$$

where, $\frac{m}{(m+\mu-p\Delta)}$ is the probability of survival from latency into HIV infectious stage and $\frac{1}{(\mu+k)}$ is the effective infectious period.

If, $R_0 < 1$ then DFE is locally asymptotically stable and the HIV-infection will simply die out. This is the target of all public heath control measure. It is often directed at lowering R_0 below unity, so that the disease is eradicated.

A submodel without AIDS, (k= r = 0): The model takes the form,

$$\dot{S} = \Delta - \beta\frac{IS}{N} - P\Delta E - q\Delta I - \mu S$$

$$\dot{E} = \beta\frac{IS}{N} + P\Delta E + q\Delta I - (m+\mu)E$$

$$\dot{I} = mE - \mu I$$

The stability of this model is examined at the disease -free equilibrium state, using the basic reproductive number of the infection as in Mugisha *et al.* (2005) and Castillo-Chavez *et al.* The disease-free equilibrium point is obtained as.

$$E_0 = (\frac{\Delta}{\mu},0,0)$$

Let X = (S), Y = (E) and Z = (I). Also suppose,

$$f(X,Y,Z) = \Delta - \beta\frac{XZ}{N} - P\Delta Y - q\Delta Z - \mu X$$

$$g(X,Y,Z) = \beta\frac{XZ}{N} + P\Delta Y + q\Delta Z - (m+\mu)Y$$

$$h(X,Y,Z) = mY - \mu Z$$

$g(X,Y,Z) = 0$, implicitly determined the function. $Y = \bar{g}(X^*, Y)$. Thus

$$Y = \frac{(\beta + q\Delta)}{(m+\mu-p\Delta)}Z = \bar{g}(X^*, Y)$$

$$h(X^*,Y,Z) = m\frac{(\beta + q\Delta)}{(m+\mu-p\Delta)}Z - \mu Z \text{ and}$$

$$h_Z(X^*,Y,Z) = m\frac{(\beta + q\Delta)}{(m+\mu-p\Delta)} - \mu$$

Letting H = M- D, with and D>0 a diagonal matrix, we get,

$$M = \frac{m(\beta + q\Delta)}{(m+\mu-p\Delta)} \text{ and } D = \mu$$

The basic reproductive number is then defined as,

$$R_0 = \frac{m(\beta + q\Delta)}{\mu(m+\mu-p\Delta)} .$$

Where, $\frac{m}{m+k-p\Delta}$ is the probability of survival from latency into infectious stage, as in the case with progression to AIDS.

DISCUSSION

Here we have studied a compartmental model for the vertical transmission dynamics of HIV/AIDS in a homogeneous mixing population and also obtained

expressions in terms of the model parameters as threshold condition for the stability and existence of disease-free equilibrium states for two types of models. One with HIV and AIDS infectious compartments and the other without AIDS compartment. For the model with HIV and AIDS compartments, using the Routh-Hurwitz condition we observed that the disease- free equilibrium state is locally asymptotically stable if,

$$\frac{m}{m+k-p\Delta}\beta < \frac{1}{m}\{(k+\mu)[p\Delta+(m+\mu)]-mq\Delta\}$$

where $p\Delta < (m+k+r+4\mu)$ and holds, whenever it exists. Otherwise there exists an endemic equilibrium state, which is locally asymptotically stable.

This means that the transmission rate must be as small as possible, so as to obtain stability of the disease –free state. However using the basic reproductive number we observed that the disease-free equilibrium state is locally asymptotically stable whenever it exists, if

$$\mu < \frac{1}{4}(p\Delta-(m+k+r)$$

$$R_0 = \frac{m(\beta+P\Delta)}{(m+\mu)(2\mu+r)} < 1$$

For the submodel without AIDS, we see that the disease-free equilibrium state is locally asymptotically stable, provided, $R_0 = \frac{m(\beta+q\Delta)}{\mu(m+\mu-p\Delta)} < 1$, otherwise there is an endemic equilibrium which is globally asymptotically stable.

Thus in both models, reducing the transmission rate, may eradicate HIV and AIDS in a population with constant recruitment of susceptibles.

VARIABLES AND PARAMETERS

S(t) : The number of susceptible individuals at time t.
E(t) : The number of exposed/Latently infected individual at time t.
I (t) : The number of infectious individual at time t.
A (t) : The number of Aids infected individual at time t.
μ : The per capital natural mortality rate.
m : Rate of progression from latency to HIV.
k : Rate of progression from HIV to AIDS.
r : AIDS induced mortality rate.
β : The rate of transmission of HIV virus.
Δ : Number of new susceptible recruited per unit of time.

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