

A Mathematical Model of an Aids Epidemic in a Homosexual Population

H. O. Adagba and G. C. Okpala

¹Department of Industrial Mathematics/Applied Statistics, Ebonyi State University, Abakaliki.

²Department of Statistics, Institute of Management and Technology, Enugu.

Abstract

We propose a mathematical model of an AIDS epidemic in a homosexual population. Asymptotic analysis of the model under the prescribed parameters we obtained the stability of the equilibrium state. The solution of the system and the numerical simulation agrees with the existing literature. However, the presence of the homosexual factor and the differentiated recruitment rate makes the reduction more realistic.

1.0 Introduction

In this paper, we propose a mathematical model of an AIDS epidemic in a homosexual population. Many of the models in the literature uses a two compartment approach dividing the population into susceptible and infectious with different infectivity and staged progression [1], [2], [3]. Some authors [4], [5], [6] have capture heterosexual activities of disease in those communities where it spread by heterosexual contact.

Keywords: AIDS epidemic, homosexual factor, intrinsic growth rate, characteristic equation, Steady state, stability

Infection by the virus HIV-1, the most common Variety, has many highly complex characteristics most of white are still not understood [7], [8], [9]. HIV primarily infects a class of white blood cells or lymphocytes, called CD4T-cells, but also infects other cells such as dendritic cells.

Since the mid – 1980s numerous models, deterministic and stochastic, have been developed to describe the immune system and its interaction with HIV. Most models have been deterministic such as [10], [6], [7] and example of stochastic is [11].

Here we are interested in the development of an AIDs epidemic in a homosexual population. Let us assume there is a constant immigration rate B of susceptible males into a population of size $N(t)$, where $X(t)$, $Y(t)$, $A(t)$ and $Z(t)$ denote respectively the number of susceptible, infectious males, AIDS patients and the number of HIV Positive or seropositive men who are noninfectious.

We assume susceptible die naturally at a rate μ and AIDS patient die at a rate d .

1.1 The Model Equation

The model equations are given by

$$X' = B - \mu X - \lambda c X, \quad \lambda = \frac{\beta Y}{N} \tag{1}$$

$$Y' = \lambda c X - (v + \mu) Y \tag{2}$$

$$A' = P v Y - (d + \mu) A \tag{3}$$

$$Z' = (1 - P) v Y - \mu Z \tag{4}$$

$$N(t) = X(t) + Y(t) + A(t) + Z(t) \tag{5}$$

With the parameters given by

B – Recruitment rate of susceptible

μ – Natural death rate (non-AIDS related)

λ – Probability of acquiring infection from partner

β – Transmission probability

c – Number of sexual partners

d – Death rate for AIDS patients

p – Proportion of HIV positive who are infectious

R_0 – Basic reproductive rate i.e. number of secondary infections which arises from a primary infection

v – Rate of conversion from infection to AIDS

$X(t)$ – Number of susceptible

Corresponding author: *H. O. Adagba* E-mail: -, Tel.: +2348036258406

$Y(t)$ – infectious males
 $A(t)$ – AIDS patients
 $Z(t)$ – HIV positive or seropositive men
 $N(t)$ – Population size

2.0 Solution of the Equations

When the epidemic starts, the system (1) – (5) evolves to a steady state with the solution given by

$$X(t) = \frac{B}{\mu + \lambda c} [1 - e^{-(\mu + \lambda c)t}] + X(0)e^{-(\mu + \lambda c)t} \tag{6}$$

$$Y(t) = \frac{B\lambda c}{(\mu + \lambda c)(v + \mu)} [1 - (1 - x(0))e^{-(\mu + \lambda c)t}] [1 + e^{-(v + \mu)t}] \tag{7}$$

An epidemic ensues if the basic reproductive rate $R_0 > 0$: that is the number of secondary infections which arise from a primary infection is greater than 1. In (5) if, $t = 0$ an infected individual is introduced into an otherwise infection free population of the susceptible, we have initially $x \cong N$ and so near $t = 0$.

$$\frac{dy}{dt} \cong (\beta c - v - \mu)Y \cong v(R_0 - 1)y \tag{8}$$

Since the average incubation time $\frac{1}{v}$ from infection to development of the disease is very much shorter than the average life expectancy, $\frac{1}{\mu}$ of the susceptible: that is $v \gg \mu$.

With solution

$$y(t) = y(0)e^{v(R_0 - 1)t} = y(0)e^{rt} \tag{9}$$

The intrinsic growth rate $r = v(R_0 - 1)$, is positive only if the epidemic exists ($R_0 > 0$). Hence we replace equation (7) by equation (9).

To solve for the AIDS patients, we substitute (9) into (3), to get,

$$\frac{dA}{dt} = Pvy(0)e^{rt} - (d + \mu)A \tag{10}$$

Initially, in the epidemic there are no AIDS patient, i.e. $A(0) = 0$ and so the solution is given by

$$A(t) = Pvy(0) \left[\frac{e^{rt} - e^{-(d + \mu)t}}{r + d + \mu} \right] \tag{11}$$

For the number of HIV patients or seropositive men, we solve equation (4) to obtain

$$Z(t) = \frac{(1-p)vy(0)e^{rt}}{\mu} [1 + e^{-\mu t}] \tag{12}$$

$Z(0) = 0$, since at $t = 0$, there is no infection in the system.

Here we can get some interesting information from the analysis of the system such as $\lambda = \frac{BY}{N}$ and v the rate of conversion from infection to AIDS here taken to be constant, $\frac{1}{v}$ equal to D say, is then the average incubation time of the disease. (Actually, λ here is more appropriately $\frac{BY}{(X+Y+Z)}$ but A is considered small in comparison with N .)

The model total population $N(t)$ is not constant, and as such, if we add equations (1) – (5) we will get

$$\frac{dN}{dt} = B - \mu N - dA \tag{13}$$

And the solution is given by

$$N(t) = \frac{B}{\mu} (e^{\mu t} - 1) - dpvy(0) \left[\frac{e^{(r+\mu)t} + e^{rt}}{r + d + \mu^2} + \frac{2e^{-(\mu+d)t}}{r + \mu + d^2} \right] + N(0) \tag{14}$$

Estimate for the parameters were calculated as in [12] and the period of the epidemic outbreak was of the order of 30 to 40 years. It is unrealistic to think that the parameters characterizing the social behaviors associated with the disease would remain unchanged over that time span. The life expectancy of people with HIV has dramatically increased since then due to new medicine such as AZT and protease inhibitor. The estimates for the parameters are as follows:

$R_{0=5.15}$, $B = 13333.3$, $V = 0.2$, $P = 0.3$, $N(0) = X(0) = 1000$, $Y(0) = 6875$, $C = 2$, $\mu = \frac{1}{32}$, as in [3].

2.1 Characteristic Equation

An equilibrium point, of system of equations (1) – (5) is a steady solution, therefore the derivatives are equal to zero. Hence the characteristic values to satisfy the equation

$$JU = SU$$

We determine the eigenvectors and eigenvalues.

$u = [\mu, v]^T$ of 4 x 4 matrix, where

$$J = \begin{pmatrix} -(\mu + \lambda c) & 0 & 0 & 0 \\ \lambda c & -(\mu + v) & 0 & 0 \\ 0 & pv & -(d + \mu) & 0 \\ 0 & (1 - p)v & 0 & -\mu \end{pmatrix}$$

Therefore, $0 = \det(j - SI)$

$$0 = \begin{pmatrix} -(\mu + \lambda c) - s & 0 & 0 & 0 \\ \lambda c & -(\mu + v) - s & 0 & 0 \\ 0 & pv & -(d + \mu) - s & 0 \\ 0 & (1 - p)v & 0 & -\mu - s \end{pmatrix}$$

Solving and upon simplification, yields

$$s^3 - \alpha s^2 + \beta s - \gamma = 0 \tag{15}$$

Where

$$\begin{aligned} \alpha &= (3\mu + \lambda c + d + v) \\ \beta &= v\lambda c + d\lambda c + \mu^2 + 2\mu\lambda c - dv \\ \gamma &= (vd + 2\mu^2 + \mu d)(\mu + \lambda c) \end{aligned}$$

Substituting $s = y + \frac{\alpha}{3}$ into (15) upon simplification we get,

$$y^3 + py = q \tag{16}$$

Where $p = \frac{\alpha^3 + 3\beta}{3}$ and $q = \frac{2\alpha^3 + 9\alpha\beta + 27\gamma}{27}$

Since $p \neq 0$, we set $y = hz$ and multiply equation (16) through by k,

Where $h = \sqrt{\frac{4|p|}{3}}$ and $k = \frac{3}{h|p|}$

Equation (16) reduces to one of the forms

$$4z^3 + 3z = cor 4z^3 - 3z = c \tag{17}$$

Using trigonometric identity, if $c \geq 1$,

$$sinh 3\theta = 4sinh^3\theta + 3sinh\theta$$

Whence, $z = sinh[\frac{1}{3}sinh^{-1}c]$ (18a)

To solve the second equation, if $c \geq 1$, we use

$$cosh 3\theta = 4cosh^3\theta - 3cosh\theta, \text{ to get}$$

$z = Cosh[\frac{1}{3}Cosh^{-1}c]$ (18b)

Substituting back into $y = hz$

$$y = \frac{\sqrt{4\frac{|\alpha^3+3\beta|}{3}}}{3} sinh[\frac{1}{3}sinh^{-1}c] \tag{19a}$$

or

$$y = \frac{\sqrt{4\frac{|\alpha^3+3\beta|}{3}}}{3} cosh[\frac{1}{3}cosh^{-1}c] \tag{19b}$$

Hence

$$S = \frac{\sqrt{4\frac{|\alpha^3+3\beta|}{3}}}{3} sinh[\frac{1}{3}sinh^{-1}c] + \frac{\alpha}{3} - \tag{20a}$$

or

$$S = \frac{\sqrt{4\frac{|\alpha^3+3\beta|}{3}}}{3} cosh[\frac{1}{3}cosh^{-1}c] + \frac{\alpha}{3}, \tag{20b}$$

Hence there is a linear stability if the eigenvector ($s < 0$) for all the eigenvalues i.e. if $-(\mu + \lambda c)$, $-(v + \mu)$ and $-(d + \mu) < 0$ and instability otherwise.

From the calculation, equation (20a) gives,

$$s = -(0.1396 + 0.08233i) \tag{21a}$$

and equation (20b) gives

$$s = -(0.1396 + 0.8974i) \tag{21b}$$

In both cases, $s < 0$ which show stability of the system.

3.0 Concluding Remarks

Numerical simulation using the given estimate of the parameters as in [3] for the model give a clear picture of the epidemic development after the introduction of the HIV into the susceptible homosexual population. Fig. 1.shows that HIV has some unfortunate unique properties even within this retrovirus family such as using the mRNA processing of the cell it invades to synthesize its own RNA. However, [6] have shown the dynamics of the viral replication is very high in vivo the immune system can counteract this replication. Hence, the susceptible will battle from zero to 5 years before its stabilizes which explains the graph in figure 1.

Infection by the virus HIV-1, i.e. when the infectious males (Y-class) is introduce into the susceptible class, progression can last more than 10years from the first day of infection. Also, immune response can briefly control the HIV which primarily infects a class of the white blood cells or lymphocytes, called CD4 T-cells. This account for the static progression until after some time and increase in progression is noticed in figure 2.

In figures (3) and (4) we observed that after the window period, there is a sharp increase in the progression for the graph of AIDS patient and seropositive men as time progresses which means that it is only the anti-retroviral therapy that can control or maintained the viral loads to be low [12]

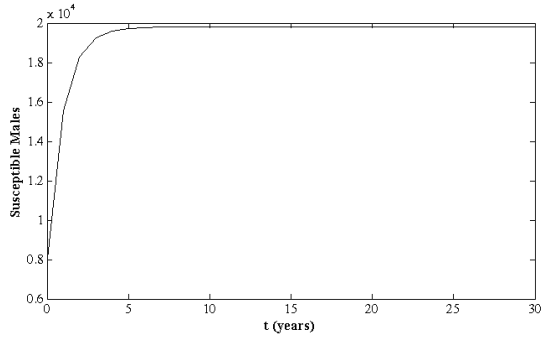


Fig.1: Susceptible Males with Time

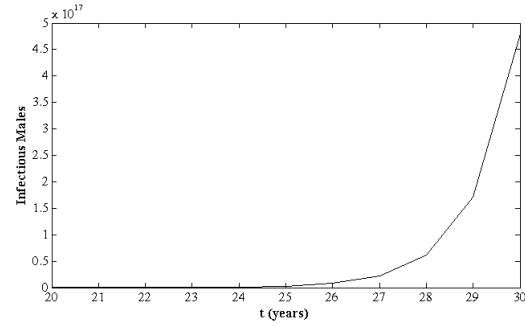


Fig.2: Infectious Males with Time Curve

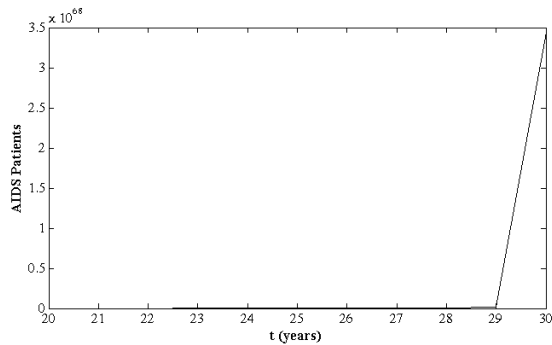


Fig.3: AIDS Patients with Time

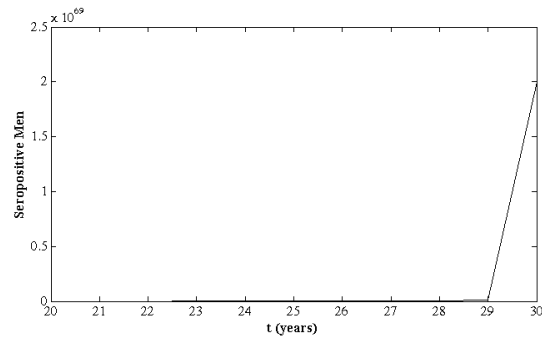


Fig.4: Seropositive Men with Time

References

- [1] Dietz K. and Haderl K. P. Epidemiological of Models for Sexually Transmitted Diseases. *J. Math. Biol.* 26: 1-25; 1988.
- [2] Essunger P. and Polelson A.S. Modeling HIV Infection of CD4+T-Cells Subpopulation. *J. Theor. Biol.* 170: 367 – 391; 1994.
- [3] Perelson A. S., and Nelson P.W. Mathematical Models of HIV-1 Dynamics in Vivo. *SIAM Rev.* 41: 3 – 44; 1999.
- [4] Anderson R. M. The Epidemiology of HIV Infection: Variable incubation plus infectious period and heterogeneity in sexual activity. *J. Roy. Stat. Soc. (A)* 151: 66-93, 1988.
- [5] Ho D. D., Neumann A. U. Perelson A. S. C, Chen W., Leonard J. M. and Markowitz M., Rapid Turnover of Plasma Virious and CD4 Lymphocytes in HIV-1 Infection, *Nature*, 373: 123 – 126; 1995
- [6] Kimbir A. R. A Two-Sex Model for HIV/AIDS Transmission Dynamics in a Polygamous Female Dominant Population, *NMS. J.* 24: 24 – 29; 2005.
- [7] Perelson A. S., Kirshner D. E., and De Boer R. Dynamic of HIV Infection of CD4+T-Cells. *Maths.Biol.* 114: 81- 125; 1993.
- [8] Perelson A. S., Neumann A. U., Markowitz M., Leonard J. M., and Ho D.D. HIV-1 Dynamics in vivo: Virion clearance rate, infected life-span, and viral generation time. *Science*, 27: 1582 – 1586; 1996
- [9] Wein L. M., Amato R.M.D., and Perelson A. S. Mathematical Consideration of antiretroviral therapy aimed at HIV-1 eradication or Maintenance of low Viral Loads. *J. Theor. Biol.* 192: 81 – 98, 1998.
- [10] Akinwande N. I. A Mathematical Model of the Chaotic Dynamics of the AIDS Disease Pandemic. *NMS .J.* 24: 8-16, 2005
- [11] Stilianakis N. I., Boucher C. A. B, DeJong M. D., Vanleeuwen R., Schuurman R., and De Boer R. J., Clinical data sets on human immunodeficiency virus type 1. Reverse transcriptase resistance mutants explained by a mathematical model. *J. Virol.* 71: 161 – 168; 1997
- [12] Anderson R. M., Medley G. F., May R. M. and Johnson A. M. A preliminary study of the transmission dynamics of the human immunodeficiency virus (HIV), The causative agent of AIDS, *IMA. J. Maths. Appl. In Medicine and Biol.* 3: 229-263; 1986