

## On the Stability and Threshold Analysis of an Epidemic Model

Muhammad A. Yau\*

Department of Mathematical Sciences, Nasarawa State University Keffi, Nigeria.

Received: 5 March 2013; Accepted: 26 July 2013.

---

**Abstract.** We consider a mathematical model of epidemic spread in which the population is partitioned into five compartments of susceptible  $S(t)$ , Infected  $I(t)$ , Removed  $R(t)$ , Prevented  $U(t)$  and the Controlled  $W(t)$ . We assume each of the compartments comprises of cohorts of individuals which are identical with respect to the disease status. We derive five systems of equations to represent each of the subpopulations. The general stability of the disease free equilibrium (DFE) and the endemic equilibrium states of the linearized model are established using the linear stability theory and the Routh-Hurwitz conditions are established and analyzed in the domain of interest. We find that the DFE is locally asymptotically stable when the infected individuals received ART and use the condom but the endemic state is unstable to initial perturbations. Also, we derive an expression for the basic reproduction number using the next generation matrix approach and find that for  $\mathcal{R}_0 < 1$  the DFE is stable but for  $\mathcal{R}_0 > 1$  is unstable.

---

**Keywords:** Stability, Threshold, Epidemic Model, HIV, AIDS.

### Index to information contained in this paper

1. Introduction
  - 1.1 Methodology
  - 1.2 The model equations
2. Stability analysis of the equilibrium states
  - 2.1 The sub-model without ART/Condom ( $\sigma = \lambda = 0$ )
  - 2.2 The sub model with the infected individuals using both ART/condom ( $\sigma \neq 0, \lambda \neq 0$ )
3. Transformation of the model equations to proportions
  - 3.1 Existence and stability of the steady states
4. Results and discussion
  - 4.1 Recommendation

## 1. Introduction

HIV and AIDS retard economic growth by destroying human capital and resources, UNAIDS has predicted outcomes for sub-Saharan countries for the year 2025 as decline in deaths beginning in the year 2012 to a catastrophic continual growth in death rate with about 90 million cases of infection by the years without proper nutrition and medical care [22]. Large numbers of people living in these countries are likely to fall victim to AIDS. They will not only be unable to work but will also acquire significant medical care. This scenario would cause collapse of many

---

\*Corresponding author. Email: yau4real2006@yahoo.com

countries and societies in the region. It increase mortality in this region which will result in a smaller skilled populations and labor force of mostly young people with reduced knowledge and work experience leading to reduced productivity. An increase in workers time off to look after sick family members or for sick leave will also lower productivity. Increase mortality will also weaken the mechanism that generates human capital and investment in people. This reduces the resources available for public expenditures such as education and health services resulting in increased pressure for the states finances and slower the growth of the economy. In general, the following are some of the negative impact of HIV/AIDS with high mortality rate, reduced rate of population growth, low life expectancy, high incidence of orphans and widows, drop out in school arising from the death of bread winner, slow rate of economic development arising from the use of economic resources to combat the disease, loss of highly productive and skilled segment of the population etc. This research dissertation work will help in determining the economic impact of the epidemic, which in turn helps us to develop reasonable, scientifically, and socially sound intervention plans in order to reduce the spread of the infection. It also serves as reference aid to government, NGOs and individuals in their effort to control the outbreak of HIV/AIDS and in providing adequate drugs and condom for the treatment and prevention of the epidemic [3], [8], [11], [16].

### 1.1 Methodology

To derive the modified model and to establish the general stability of the free equilibrium states of the model, three methods would be used. First we would use the method of Beltrami [4] then, we would use the Routh-Hurwitz stability condition and finally we would use the method of Diekmann [17], [16].

### 1.2 The model equations

We derive the following five systems of equations representing each compartment as follows:

$$\begin{aligned}
 \frac{dS}{dt} &= Nb - (B + \mu)S, \\
 \frac{dI}{dt} &= BS - (\mu + \sigma + \lambda + \alpha_0)R, \\
 \frac{dR}{dt} &= \sigma I - (\mu + \rho + \alpha_1)R, \\
 \frac{dU}{dt} &= \lambda I - (\mu + \pi + \alpha_2)U, \\
 \frac{dW}{dt} &= \rho R + \pi U - (\mu + \alpha)W,
 \end{aligned} \tag{1}$$

where  $\alpha = \alpha_0 e^{-KT}$  and  $N = S + I + R + U + W$ . The incidence rate  $B(t)$  at time  $t$  is given as in [18] and [16]:

$$B(t) = \frac{c_0 \beta I + c \beta W}{N}. \tag{2}$$

## 2. Stability analysis of the equilibrium states

Here we would establish the general stability of the disease free equilibrium (DFE) states, by considering the model parameters and using the model equations. Since we have five systems of nonlinear equations, we know that it is almost impossible to obtain an analytical solution to these systems; therefore, we use the idea of [4] and [8].

### 2.1 The sub-model without ART/Condom ( $\sigma = \lambda = 0$ )

The sub model equations are:

$$\begin{aligned}\frac{dS}{dt} &= bN - \frac{c_0\beta_0SI}{N} - \mu S, \\ \frac{dI}{dt} &= \frac{c_0\beta_0SI}{N} - (\mu + \alpha_0)I.\end{aligned}\quad (3)$$

The equilibrium point of the above system is thus:  $E_0 = (bN/\mu, 0)$ , since  $I = 0$  thus the DFE state exists. We examine its stability using Routh-Hurwitz stability condition [7]. The Jacobian matrix is:

$$J_{E_0} = \begin{pmatrix} -\mu & -\frac{c_0\beta_0b}{\mu} \\ 0 & \frac{c_0\beta_0b}{\mu} - (\mu + \alpha_0) \end{pmatrix}\quad (4)$$

If  $\lambda_i (i = 1, 2)$  are the eigenvalues of the matrix  $J_{E_0}$  then, We would have for stability

$$\begin{aligned}\det(J_{E_0}) &= -c_0\beta_0b + (\mu + \alpha_0)\mu \\ &= (\mu + \alpha_0)\mu - c_0\beta_0b > 0\end{aligned}\quad (5)$$

only when  $(\mu + \alpha_0)\mu > c_0\beta_0b$ , since all the parameters are non-negative. And the trace of the matrix is

$$\begin{aligned}tr(J_{E_0}) &= \frac{c_0\beta_0b}{\mu} - (2\mu + \alpha_0) < 0 \\ \text{if } \frac{c_0\beta_0b}{\mu} &< (2\mu + \alpha_0).\end{aligned}\quad (6)$$

Even then, the DFE is conditionally stable. That is, if the above conditions fail then the DFE will be unstate.

### 2.2 The sub model with the infected individuals using both ART/condom ( $\sigma \neq 0, \lambda \neq 0$ )

The resulting equations are:

$$\begin{aligned}
\frac{dS}{dt} &= Nb - \frac{(c_0\beta_0I + c\beta W)S}{N} - \mu S, \\
\frac{dI}{dt} &= \frac{(c_0\beta_0I + c\beta W)S}{N} - (\mu + \sigma + \lambda + \alpha_0)R, \\
\frac{dR}{dt} &= \sigma I - (\mu + \rho + \alpha_1)R, \\
\frac{dU}{dt} &= \lambda I - (\mu + \pi + \alpha_2)U, \\
\frac{dW}{dt} &= \rho R + \pi U - (\mu + \alpha)W,
\end{aligned} \tag{7}$$

Please observe that here the entire model equations are involved, therefore, we use the basic reproduction ratio of the infective to discuss the stability of the general model to avoid algebraic complexity involved in the computation of the Routh-Hurwitz stability method. In adopting this approach we use the idea of the next generation operator by [12], [11]. We recategorize the population into two classes as follows:

$$X = (S, W) \quad Z = (I, R, U) \tag{8}$$

Then we have thus:

$$\begin{aligned}
X = f(X, Z) &= bN - (c_0\beta_0Z + c\beta X)\frac{X}{N} + (\rho + \lambda)Z - (2\mu + \alpha)X, \\
&\text{and}
\end{aligned} \tag{9}$$

$$Z = h(X, Z) = (c_0\beta_0Z + c\beta X)\frac{X}{N} + (\sigma + \lambda)Z - (3\mu + \sigma + \lambda + \rho + \pi + \alpha_i)Z.$$

Now let

$$H = \frac{\partial h}{\partial Z} \rightarrow H = (c_0\beta_0 + \sigma + \lambda) - (3\mu + \sigma + \lambda + \rho + \pi + \alpha_i), \tag{10}$$

where  $\alpha_i = \alpha_0 + \alpha_1 + \alpha_2 + \alpha$ .

Letting  $H = M^* - d^*$ , with  $M^* > 0, D^* > 0$  are diagonal matrices. Then,  $M^* = (c_0\beta_0 + \sigma + \lambda)$  and  $D^* = (3\mu + \sigma + \lambda + \rho + \pi + \alpha_i)$ . The basic reproduction number of the infective is defined as the spectral radius (dominant eigenvalues) of the matrix  $M^*D^{*-1}$ , [10]. Thus,

$$\mathcal{R}_0 = \phi(M^*D^{*-1}) = \frac{(c_0\beta_0 + \sigma + \lambda)}{(3\mu + \sigma + \lambda + \rho + \pi + \alpha_i)}, \tag{11}$$

for stability of the DFE state, we have;

$$\mathcal{R}_0 = \frac{(c_0\beta_0 + \sigma + \lambda)}{(3\mu + \sigma + \lambda + \rho + \pi + \alpha_i)} < 1, \tag{12}$$

which is consistent. Thus the DFE state is locally asymptotically stable and the disease would not persist, it would die out, if the threshold parameters satisfy this condition, while for  $\mathcal{R}_0 > 1$ , we have an EE state which is also locally asymptotically stable following the same analysis.

### 3. Transformation of the model equations to proportions

The above stability analysis is a more general approach and gives a little inside to the DFE and EE states of the general model. Therefore, another approach is needed to give a more inside to the DFE and EE states so as to ascertain whether or not the DFE and the EE states are locally or globally asymptotically stable. In this quest we nondimensionalize the model equations to their equivalence in proportions.

Now let,  $s = \frac{S}{N}$ ,  $i = \frac{I}{N}$ ,  $r = \frac{R}{N}$ ,  $u = \frac{U}{N}$ ,  $w = \frac{W}{N}$ , this implies that  $s = 1 - i - r - u - w$ .

$$\begin{aligned} \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} + \frac{dU}{dt} + \frac{dW}{dt} \\ &= Nb - (B + \mu)S + BS - (\mu + \alpha_0 + \sigma + \lambda)I + \sigma I - (\mu + \rho + \alpha_1)R \\ &\quad + \lambda I - (\mu + \pi + \alpha_2)U + \rho R + \pi U - (\mu + \alpha)W \\ &= (b - \mu)N - (\alpha_0 I + \alpha_1 R + \alpha_2 U + \alpha W). \end{aligned} \quad (13)$$

And the governing equations in proportions  $r$ ,  $u$  and  $w$  are as follows:

$$\begin{aligned} \frac{dr}{dt} &= \sigma i - [(b + \alpha_1 + \rho) - (\alpha_0 i + \alpha_1 r + \alpha_2 u + \alpha w)]r \\ \frac{du}{dt} &= \lambda i - [(b + \alpha_2 + \pi) - (\alpha_0 i + \alpha_1 r + \alpha_2 u + \alpha w)]u \\ \frac{dw}{dt} &= \rho r + \pi u - [b - (\alpha_0 i + \alpha_1 r + \alpha_2 u + \alpha w)]w. \end{aligned}$$

#### 3.1 Existence and stability of the steady states

It is easy to see that  $(0, 0, 0)$  is an equilibrium state of the model (14). The Jacobian matrix associated with the equilibrium state  $(0, 0, 0)$  is given by

$$J_{E_0} = \begin{pmatrix} -(b + \alpha_0 + \rho) & 0 & 0 \\ 0 & (b + \alpha_2 + \pi) & 0 \\ \rho & \pi & -b \end{pmatrix} \quad (14)$$

Let  $\mathcal{R}_0$  be given as equation (11) and we define the region  $D$  as follows:

$$D = \{(r, u, w) : r > 0, u > 0, w > 0, r + u + w = 1\}. \quad (15)$$

**THEOREM 3.1**

Let  $\mathcal{R}_0$  be given by (11), then the DFE for the sub-model (14) is locally asymptotically stable if  $\mathcal{R}_0 < 1$ .

*Proof*

Since all our parameters are non-negative, the community matrix of system (14) is given by thus:

$$J_{E_0} = \begin{pmatrix} -(b + \alpha_1 + \rho + \lambda) & 0 & 0 \\ 0 & (b + \alpha_2 + \pi + \lambda) & 0 \\ \rho & \pi & -(b + \lambda) \end{pmatrix} \quad (16)$$

Here we need to show that the determinant of the jacobian matrix at the equilibrium points is non-negative and also the trace of the contact matrix is negative. If these two conditions are satisfied then it implies that the DFE is stable. Now let our contact matrix be represented by:

$$J_{E_0} = \begin{pmatrix} -(b + \alpha_0 + \pi) & 0 \\ \pi & -b \end{pmatrix} \quad (17)$$

$$\begin{aligned} \det(J_{E_0}) &= [-(b + \alpha_0 + \pi)(-b) - (0)(\pi)] \\ &= b(b + \alpha_0 + \pi) > 0 \end{aligned} \quad (18)$$

Clearly the determinant of  $J_{E_0}$  is non-negative since all the parameters are non-negative. Next we show that the trace of  $J_{E_0}$  is negative.

$$\begin{aligned} \text{tr}(J_{E_0}) &= [-(b + \alpha_0 + \pi) + (-b)] \\ &= -(2b + \alpha_0 + \pi) < 0 \end{aligned} \quad (19)$$

Since all the parameters are nonnegative, this means the trace will always be negative. Therefore, the DFE is locally asymptotically stable and hence the epidemic would die out. ■

**THEOREM 3.2** Let  $\mathcal{R}_0$  be given by (11), then the endemic equilibrium state for the model equations (14) is locally asymptotically stable if  $\mathcal{R}_0 > 1$ .

*Proof* To prove that the EE state is stable, we only need to show that the region  $D$  is invariant, containing no periodic solutions of the systems (14), so that all solutions tend to the endemic equilibrium state. First, we shall show that  $D$  is a positive invariant region. We do this by showing, as in [? ], [? ], that the inner product of the vector field defined by system (14) with the inward normal to  $D$  is non-negative.

Let  $f_1(r, u, w)$ ,  $f_2(r, u, w)$  and  $f_3(r, u, w)$ , denote respectively, the rhss of system (14). Then, the inward normal to the  $r$ -axis is  $(1, -1, 0)$ , therefore,

$$\begin{aligned}
 (1, -1, 0) \begin{pmatrix} f_1 \\ f_2 \\ f_3 \end{pmatrix} &= f_1 - f_2 \\
 &= (\sigma - \lambda)i + [(b + \alpha_2 + \pi) - (\alpha_0i + \alpha_1r + \alpha_2u)]u \\
 &\quad - [(b + \alpha_1 + \pi) - (\alpha_0i + \alpha_1r + \alpha_2u)]r > 0
 \end{aligned}
 \tag{20}$$

(since  $w = 0, r > 0, u < 0$  on this axis).  
 Next the inward normal to the  $u$ -axis is  $(-1, 1, 0)$ , so that

$$\begin{aligned}
 (-1, 1, 0) \begin{pmatrix} f_1 \\ f_2 \\ f_3 \end{pmatrix} &= f_2 - f_1 \\
 &= (\lambda - \sigma)i + [(b + \alpha_1 + \rho) - (\alpha_0i + \alpha_1r + \alpha_2u)]r \\
 &\quad - [(b + \alpha_2 + \pi) - (\alpha_0i + \alpha_1r + \alpha_2u)]u > 0
 \end{aligned}
 \tag{21}$$

(since  $w = 0, r < 0, u > 0$  on this axis). Also, on the lines  $r+w = 1$  and  $u+w = 1$  we have

$$\frac{1}{\sqrt{2}}(-1, 0, -1) \begin{pmatrix} f_1 \\ f_2 \\ f_3 \end{pmatrix} = -\frac{1}{\sqrt{2}}(f_1 + f_3) > 0
 \tag{22}$$

$$\frac{1}{\sqrt{2}}(0, -1, -1) \begin{pmatrix} f_1 \\ f_2 \\ f_3 \end{pmatrix} = -\frac{1}{\sqrt{2}}(f_2 + f_3) > 0
 \tag{23}$$

and

$$\frac{1}{\sqrt{2}}(-1, -1) \begin{pmatrix} f_1 \\ f_2 \end{pmatrix} > 0,
 \tag{24}$$

using similar arguments. Thus we have proved that  $D$  is invariant.  
 It remains to prove, using the Bendixon-Dulac criterion, as in [? ], that there are no periodic solutions of the system (14). Let the dulac function be  $g = \frac{1}{ruw}$ , then we have thus;

$$\frac{\partial}{\partial r}(gf_1) + \frac{\partial}{\partial u}(gf_2) + \frac{\partial}{\partial w}(gf_3) < 0.
 \tag{25}$$

Therefore there are no periodic solutions of the system in  $D$ , hence the proof.  
 This implies that the EE state is locally asymptotically stable and hence by this threshold condition the disease would not persists. ■

#### 4. Results and Conclusion

We have proved the general stability of the system in absent and presence of disease. Our analysis revealed that the disease free steady state is always stable in our domain  $D$ , while the endemic state is unstable. We have derived an expression for the basic reproduction number and found that the epidemic model is always stable whenever  $\mathcal{R}_0 < 1$  and unstable for  $\mathcal{R}_0 > 1$ . This agreed with the theory and conclude that to control the epidemic we have to keep  $\mathcal{R}_0 < 1$ . This result further confirmed our theoretical assumption. Thus, for an effective ART/condom programme, it may be necessary to also reduce the transmission probability and the average number of sexual partners of the infected individuals. These can be done through counselling and education.

##### 4.1 Recommendation:

We make the following recommendations:

- ✓ The national response to the HIV/AIDS epidemic must be strengthened and expanded to ensure balance in interventions between urban and rural areas, as well as in intervention strategies-Prevention, Treatment and Care for people living with HIV/AIDS.
- ✓ Definite intervention should be designed to target people with primary and secondary level education especially using mass media campaigns that they will be responsive to.
- ✓ The extent of adoption and implementation of HIV/AIDS education curricula should be assessed and strengthened in order to reduce the prevalence among people with primary and secondary level education that is in school.
- ✓ Emphasis should continue to focus on the youth to ensure a sustained downward trend in new infections.
- ✓ There should be increased efforts to expand quality comprehensive HIV/AIDS prevention, treatment, care and support services.
- ✓ In view of the large number of AIDS orphans with its attendant burden, comprehensive care and support programmes should be packaged and adequately delivered on sustainable basis.
- ✓ Focused research in sites/states with consistently low and high prevalence would facilitate the identification of possible factors for appropriate intervention strategies.
- ✓ Further research can improve on this model to incorporate a situation where susceptible individuals use the preventive measure.

#### References

- [1] Akinwande, N.I, *A mathematical model of the dynamics of the HIV/AIDS disease pandemic*, J. Nig. Math. Soc. **25** 99-108.
- [2] Arazoza, H. D. and Lounes, R., *A nonlinear model for a sexually transmitted disease with contact tracing*, IMA Journal of Mathematics Applied in Medicine and Biology **19** 221-234.
- [3] Bergquist, L. M. and Pogolian, B., *Microbiology Principles and Health Science Applications*, Sanders Company, USA, (2000).
- [4] Beltrami, E., *Mathematics for dynamic modeling*, Academic Press, N.Y, (1989).
- [6] Centers for Disease Control and Prevention, *Perspectives in disease prevention and health promotion condoms for prevention of sexually transmitted diseases*, WHO, (1989).
- [6] Centers for Disease Control and Prevention, *Basic facts about condoms and their use in preventing HIV infection and other STDs*, WHO, (1989).
- [7] Hethcote, H.W. and J.W. Van Ark, *Epidemiological models for heterogeneous populations, proportionate mixing, parameter estimation, and immunization programs*. Math. Biosci., **84** (1987) 85-118.
- [8] Hethcote, H. W. and J. A. Yorke, *Gonorrhoea, transmission dynamics, and control*, Math. Biosci.,(1984).



- [9] Zikore, K.U, *Lecture Notes in Biomathematics*, Springer-Verlag, Berlin, (2001).
- [10] Hethcote, H. W., *Three basic epidemiological Models*, In Applied Mathematical Ecology (S. A. Levin, T. G. Hallam and L. J. Gross, editors), (1980)119-144.
- [11] Hsieh, H.Y. Velasco-Hernandez, J.X., *Community treatment of HIV-1 initial stage and asymptotic analysis*, Bio-systems, **25** (1995) 75-81.
- [12] Hsieh, H.Y., *A two sex model for treatment of AIDS and behavior change in a population of varying size*, IMA. J. Math. Appl. Bio. & Medicine, **13** (1996) 151-173.
- [13] Hsu-Schmitz, S.F., *Effects of treatment or/and vaccination on HIV transmission in homosexuals with genetic heterogeneity*, Math. Biosc. **167** (2000) 1-18.
- [14] Kaosimore, M. and Lungu, E.M, *Effects of vaccination and treatment on the spread of HIV/AIDS*, J. Biol. Systems., **12** (4) (2004) 399-417.
- [15] Kimbir, A.R. and Aboiyar, T., *A mathematical model for the prevention of HIV/AIDS in a varying population*, J. Nig. Math. Soc. **22** (2003) 43-55.
- [16] Kimbir, A.R., Musa, S. and Bassey, E.B., *On a two-sex mathematical model for the prevention of HIV/AIDS in a varying population*, ABACUS, (J. Math. Assoc. Nig.) **33** (201) (2006) –13.
- [17] Kimbir, A.R and Oduwole, H.K, *A mathematical model of HIV transmission dynamics considering the use of Antiretroviral Therapy and counseling*, J. of Modern maths. and statistic, **2** (5) (2008).
- [18] Leigh Johnson, *An introduction to the mathematics of HIV/AIDS modeling*, lecture note, Center for Actuarial Research.
- [19] Mastro, T.D. and Limpakarnjanarat, K., *Condom use in Thailand: How much is it slowing down the HIV/AIDS epidemic?*, AIDS, **9** (1995) 523-525.
- [20] May, R.M. and R.M. Anderson, *Transmission dynamics of HIV infection*, Nature **326** (1987) 137-142.
- [21] May, R.M. and R.M. Anderson, *Transmission dynamics of human immunodeficiency virus (HIV)*, Phil. Trans. Roy. Soc. (in press), (1991).
- [22] Prescott, Harly, and Kleins, *Microbiology*, seventh edition. Joanne M. Willey Hofstra University, (1994).

Archive of SID