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# Mathematical Assessment of the Transmission Dynamics of HIV/AIDS with Treatment Effects

## <sup>1</sup>Agbata B.C, <sup>2</sup>Ode O.J, <sup>3</sup>Ani B.N <sup>4</sup>Odo C.E <sup>5</sup>Olorunnishola O.A

<sup>1,3</sup>Department of Mathematics University of Nigeria Nsukka, Nigeria
 <sup>2</sup>Department of Mathematics/Computer science Benue State University, Makurdi, Nigeria
 <sup>4</sup>Department of Mathematics Federal Polytechnic Bida,Nigeria
 <sup>5</sup>Department of Science Education Kogi State University, Anyingba, Nigeria

**ABSTRACT:** This article examines the transmission dynamics of HIV/AIDS with treatment effects. The total population ( $N_t$ ) was grouped into four compartments namely susceptible individuals at time t ( $S_t$ ), infected humans at time t ( $I_t$ ), individuals on treatment at time t ( $T_t$ ), and the AIDS carriers at time t ( $A_t$ ). A first order deterministic mathematical model for the case is formulated and analyzed to gain insight into the qualitative features of the local stability and disease free equilibrium which enable us to understand the transmission dynamics of the disease. The numerical simulation of the model was carried out to investigate the sensitivity of some threshold parameters on spread rate of the disease. We also investigated the impact of early detection of HIV cases and compliant behavior of patients on treatment. Our results from numerical simulation and early detection of HIV case/ compliant behavior of patients on treatment show that early detection of HIV case and compliant behavior of patients on treatment reduce spread of the disease and minimize rate at which people develop AIDS.

**KEYWORDS**: Transmission dynamics, treatment, HIV/AIDS, mathematical assessment, modeling.

#### INTRODUCTION

The human immunodeficiency virus (HIV) is retrovirus that infects cells of the immune system, damaging or impairing their functions in the body. As the infection progresses, the immune system becomes weak and the infected individual becomes more susceptible to infections [2]. If an individual is infected with HIV, the HIV virus attacks and destroys the CD4 cells of the immune system. CD4 cells are also called T – helper cells and they a type of white blood cells help the body to fight against diseases[7]. This virus was discovered among homosexuals in the United States of America in 1983; but another counter report claimed that the HIV was actually discovered among apes in 1982 in Kenya [3]. HIV causes serious illness because the cells the virus uses to replicate itself are the same cells that help the body to fight infections, damaging these cells leads to serious illness by the infected persons[7]. The main causes of HIV include Virginal intercourse with a person who has HIV while not using a condom or PIEP, sharing equipment for inject able illicit drugs hormones and Steroids with a person who has HIV [10]. It can also be spread through eating food that has been pre – chewed by a person with HIV, being bitten by person with HIV,

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contact between broken skin, wounds or HIV infected blood[4].A pregnant woman living with HIV or has recently given birth might transfer the disease to her new born child during child birth breastfeeding, or during blood transfusion, though the risk of HIV transmitting through blood transfusion is extremely low in countries that have effective screening procedures in place for donating of blood. HIV is not spread by mosquitoes, ticks or other insects, tears, hugging, shaking hands, sharing toilets, sharing dishes, other sexual activities that don't involve the exchange of body fluids[4]. The spread of HIV has reduced because to transmit HIV, these fluids must contain enough of the virus. If an infected individual has "undetectable" HIV they will not transmit HIV to another person, even if after a transfer of fluids. Undetectable HIV is occurs when the HIV in the infected individual is so low that cannot be detected by blood test. HIV carriers may be able to achieve undetectable levels of HIV by strictly adhere to prescribe course of treatment [10]. The progression HIV to AIDS depends on many factors, including:

The age of the HIV carrier, ability of the body immune system to fight HIV, complaint behavior to high quality sanitary healthcare, the individuals genetic inheritance resistance to contain strains of HIV [8, 10].

Since the discovery of HIV/AIDS many researchers have formulated different mathematical models to aid in the prevention and control of the epidemic. [5]Studied Modeling of HIV/AIDS Dynamics with Treatment and Vertical Transmission using a non linear deterministic Mathematics Model with five Compartments. It was established that the disease free equilibrium is locally asymptotically stable with Ro < 1; but did not consider disease induce death rate in the necessary compartments. [6]Investigated mathematics analysis of HIV/AIDS prophylaxis treatment model with five compartment diagram of the local stability of disease free equilibrium was carried out and analyzed, disease death rate was only considered in AIDS class. [11] Studied mathematical modeling of HIV prevention measures including pre – Exposure Prophylaxis on HIV incidence in South Korea.

In this study, the formulation of HIV/AIDS mathematical model is based on the fact that susceptible individual can be infected as a result of body contact with fluid of infected individuals, the infected individual can therefore undergo treatment after detection, poor quality treatment migrate people to AIDS.

#### **Model Formulation**

The total population N(t) is portioned into four epidemiological groups, the individuals who have not come into contact with HIV virus known as susceptible humans at time t S(t). The susceptible humans become infected through the rate of infection  $\propto$ ,  $\theta$  is the rate at which infected individuals develop AIDS and  $\beta$  the rate at which the infected individual undergo treatment.  $u_1$  and  $u_2$  are the natural death rate and disease induced death rate respectively.

#### **Model Assumptions**

The model is formulated based on the following mathematical assumptions.

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- Only infected individuals receive treatment and individuals undergoing treatment and infected humans can develop AIDS
- > The all have equal probability of being infected if they come in contact with infected fluids.
- > The total population N(t) is heterosexual.
- > There is no recovery in the A(t) compartment
- > There is no migration and emigration, new recruits enter the population birth and the population reduces by natural or disease induced death rate.
- > Disease induced mortality occurs in every compartment except the susceptible class.



Figure (1) Schematic Diagram of the Model

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VARIABLE / PARAMETER	DESCRIPTION	
S(t)	Susceptible humans at time t	
I(t)	Infected humans at time t	
T(t)	Individuals undergoing treatment at time t	
A(t)	Individuals infected with AIDS at time t	
π	Recruitment rate into susceptible individual	
×	Force of infection	
$u_2$	Death rate due to infection	
$u_1$	Natural death rate	
θ	The proportion of infected individuals that	
	developed AIDS	
β	Proportion of infected individual undergoing	
	treatment	
ω	Rate at which individual under treatment develop	
	AIDS	
Ν	Total population	
С	Probability of been infected	
K	Per Contact rate	

Table 1 : Description of variable and parameter used in this articles

From the above description and schematics diagram we establish the following ordinary differential equations.

$$\dot{S}(t) = \pi - (u_1 + \alpha)S(t)$$

$$\dot{I}(t) = \alpha S(t) - (u_1 + u_2)I(t) - (\beta + \theta)I(t)$$

$$\dot{I}(t) = \beta I(t) - (u_1 + u_2 + \omega)T(t)$$

$$\dot{A}(t) = \theta I(t) + \omega T(t) - (u_1 + u_2)A(t)$$
Where  $\alpha = \frac{CK(I + T + A)}{N}$ 
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With initial conditions

st(0) > 0, It(0) > 0Tt(0) > 0Tt(0) > 0At(0) > 0N(t) = S(t) + I(t)

Therefore the overall population N(t) = S(t) + T(t) + I(t) + A(t)

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#### POSITIVITY SOLUTIONS FOR HIV / AIDS TRANSMISSION DYNAMICS

#### THEOREM 1

Given the initial data { $St(0) \ge 0$   $It(0) \ge 0$   $Tt(0) \ge 0$   $At(0) \ge 0$ }

Then solution set  $\{S(t), I(t), T(t), A(T)\}$  of the non – linear system of dinary differential equations 1 to 4 are positive for all t > 0

$$\{St(0) \ge 0 \ It(0) \ge 0 \ Tt(0) \ge 0 \ At(0) \ge 0\}$$

Proof

From equation (1)

$$\begin{split} \dot{S}(t) &= \pi - (u_1 + \alpha)S(t) \\ \dot{S}(t) &= \pi - (u_1 + \alpha)S(t) \leq -(u_1 + \alpha)S(t) \\ \dot{S}(t) &\leq -(u + \alpha)S(t) \end{split}$$

Integrating and applying the initial condition t = 0 S(0) $\leq K_0$ 

We have

$$S(t) \le S(0)e^{-(u_1 + \alpha)t} \tag{6}$$

From equation (2)

Integrate and apply  $t = 0 I(0) = K_1$ 

We have

$$I(t) \le I(0)e^{-(u_1+u_2+\beta+\theta)t}$$
From equation (3)
$$\dot{T}(t) = \theta I(t) - (u_1+u_2+\omega)T(t)$$

$$7$$

$$\dot{T}(t) \le \theta I(t) - (u_1 + u_2 + \omega)T(t) \le -(u_1 + u_2 + \omega)T(t)$$
  
 $\dot{T}(t) \le -(u_1 + u_2 + \omega)T(t)$ 

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Integrate and apply t = 0  $T(0) \le K_2$   $T(t) < T(0)e^{-(u_1+u_2+\omega)t}$ From equation (4)  $\dot{A}(t) = \theta I(t) + \omega T(t) - (u_1 + u_2)A(t)$  $\dot{A}(t) \le \theta I(t) + \omega T(t) - (u_1 + u_2)A(t) \le -(u_1 + u_2)A(t)$ 

$$\dot{A}(t) \le -(u_1 + u_2)A(t)$$
  
Integrate and apply  $t = 0 A(0)$ 
$$A(t) \le A(0)e^{-(u_1 + u_2)t}$$

$$A(t) \le A(0)e^{-(u_1+u_2)}$$
  
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It has been verified that solutions of the models are positive for all t > 0.

 $\leq K_3$ 

#### **Model Analysis**

#### **Equilibrium States**

The disease free Equilibrium State of the model is obtained below,

at disease free equilibrium the I(t) = 0, T(t) = 0, A(t) = 0

Therefore the disease free equilibrium is  $(S^*(t), I^*(t), T^*(t), A^*(t)) = \left[\frac{\pi}{(u_1 + \alpha)}, 0, 0, 0\right]$ 

#### The Endemic State of The Model Equation

$$S^{*}(t) = \frac{\pi N}{\mu_{1} + k\gamma(l+T+A)}$$

$$I^{*}(t) = \frac{\pi \alpha N}{(\mu_{1} + \mu_{2} + \theta + \beta)[\mu_{1} + k\gamma(l+T+A)]}$$

$$T^{*}(t) = \frac{\pi \alpha \beta N}{(\mu_{1} + \mu_{2} + \omega)(\mu_{1} + \mu_{2} + \theta + \beta)[\mu_{1} + k\gamma 6(l+T+A)]}$$

$$A^{*}(t) = \frac{\pi \alpha \theta N}{(\mu_{1} + \mu_{2})(\mu_{1} + \mu_{2} + \theta + \beta)[\mu_{1} + k\gamma(l+T+A)]} + \frac{\omega \pi \alpha \beta N}{(\mu_{1} + \mu_{2})(\mu_{1} + \mu_{2} + \theta + \beta)[\mu_{1} + k\gamma(l+T+A)]}$$

## The Basic Reproduction Number $(R_0)$

It is the number of secondary cases which one case would produce in a completely susceptible population and it depends on the duration of the infection period. [1]

By the method of next generation matrix, method defined by  $FV^{-1}$ 

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$$fi = \begin{bmatrix} \frac{\kappa_{\gamma}}{N} (I + T + A)S_0 \\ 0 \\ 0 \end{bmatrix} \qquad Vi = \begin{bmatrix} (\mu_1 + \mu_2 + \beta + \theta)I_{(t)} \\ -\beta I_{(t)} + (\mu_1 + \mu_2 + \omega)T_{(t)} \\ -\theta I_{(t)} + (\mu_1 + \mu_2)A_{(t)} \end{bmatrix}$$

Differentiating *fi* and *Vi* w.r.t *I*, *T* and *A* we have

$$F = \begin{bmatrix} \frac{krso}{N} & \frac{krso}{N} & \frac{krso}{N} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \qquad V = \begin{bmatrix} (u_1 + u_2 + \beta + \theta) & 0 & 0 \\ -\beta & +(\mu_1 + \mu_2 + \omega) & 0 \\ -\theta & 0 & (\mu_1 + \mu_2) \end{bmatrix}$$
$$V^{-1} = \begin{bmatrix} \frac{1}{(u_1 + u_2 + \beta + \theta)} & 0 & 0 \\ -\beta & \frac{1}{(\mu_1 + \mu_2 + \omega)} & 0 \\ -\theta & 0 & \frac{1}{(\mu_1 + \mu_2)} \end{bmatrix}$$

The next generation matrix  $FV^{-1}$  is given by

$$FV^{-1} = \begin{vmatrix} \frac{krso(u_1 + u_2 + \beta + \theta)}{N} - \frac{\beta krso}{N} - \frac{\theta krso}{N} & \frac{krso(\mu_1 + \mu_2 + \omega)}{N} & \frac{krso(\mu_1 + \mu_2)}{N} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{vmatrix}$$

 $R_0$  Is the spectra radius of  $|FV^{-1} - I\lambda| = 0$ 

Therefore  $R_0 = \frac{krso}{N} [(u_1 + u_2 + \beta + \theta) - \beta - \theta]$ 

## Local Stability Analysis of Disease Free Equilibrium State

Let 
$$H = \pi - (u_1 + \alpha)S(t)$$
  

$$\frac{dH}{dI} = \frac{dH}{dT} = \frac{dH}{dA} = 0$$

$$\frac{dH}{dS} = -(u_1 + \alpha)$$

$$\rho = \alpha S(t) - (u_1 + u_2 + \theta + \beta)It$$

$$\frac{d\rho}{dS} = \alpha \frac{d\rho}{dI} = -(u_1 + u_2 + \theta + \beta)$$

$$\frac{d\rho}{dT} = \frac{d\rho}{dA} = 0$$

$$R = \beta I(t) - (u_1 + u_2 + \omega)T(t)$$

$$\frac{dR}{dS} = \frac{dR}{dA} = 0$$

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$$\frac{dR}{dI} = \beta \quad \frac{dR}{dT} = -(u_1 + u_2 + \omega)$$
$$X = \theta I(t) + NT(t) - (u_1 + u_2)A(t)$$
$$\frac{dX}{dI} = \theta \frac{dX}{dT} = \omega$$
$$\frac{dX}{dA} = -(u_1 + u_2)\frac{dX}{dS} = 0$$

The Jacobian matrix is given below

$$J(\Sigma f) = \begin{bmatrix} -(u_1 + \alpha) & 0 & 0 & 0 \\ \alpha & -(u_1 + u_2 + \beta + \theta) & 0 & 0 \\ 0 & \beta & -(u_1 + u_2 + \omega) & 0 \\ 0 & \theta & \omega & -(u_1 + u_2) \end{bmatrix}$$

The characteristics matrix is

Determinant of the Jacobian matrix is

$$\{-(u_{1} + \alpha) - \lambda\}\{-(u_{1} + u_{2} + \beta + \theta) - \lambda\}\{-(u_{1} + u_{2} + \omega) - \lambda\}\{-(u_{1} + u_{2}) - \lambda\} = 0$$
  
$$-(u_{1} + \alpha) - \lambda = 0 - (u_{1} + u_{2} + \beta + \theta) - \lambda = 0, -(u_{1} + u_{2} + \omega) - \lambda = 0 - (u_{1} + u_{2}) - \lambda = 0$$
  
$$\lambda_{1} = -(u_{1} + \alpha)\lambda_{2} = -(u_{1} + u_{2} + \beta + \theta)\lambda_{3} = -(u_{1} + u_{2} + \omega) \text{ and } \lambda_{4} = -(u_{1} + u_{2})$$

 $\lambda_1, \lambda_2, \lambda_3, \lambda_4 < 0$ , The disease free equilibrium is stable.

#### CONCLUSION

Our main objective of this of this work is to investigate the dynamics of uncured life threatening disease HIV/AIDS in order to understand the epidemic situation and recommend remedies for its control.

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Based on the findings of this work, it can be concluded that the stability analysis of the disease free equilibrium state of the model is stable and that of the endemic equilibrium state is locally asymptotically stable. Our results from numerical simulations in figure 1 and figure 2 compliant behavior of patients on treatment show that increase in treatment rate reduces the rate at which develop AIDS

#### Recommendation

HIV/AIDS has been a life threatening disease all over the world. However, from the results of this research work, we recommend that:

- More awareness should be conducted in urban and rural areas to enable people to have good knowledge of HIV/AIDS, its mode of transmission, signs and symptoms, treatment and to avoid unhealthy sexual activities, sharing sharp objects like needles, clippers, blades
- Stigmatization, segregation or discrimination against people suffering HIV/AIDS should be discouraged to enable the infected class comply to treatment

## **Numerical Simulation**

Numerical simulation helps to understand the effects of every parameter used in the model and the control measures .The numerical simulation of our model was carried out with various set of parameters values in table 2 using maple 18. It was discovered that at high treatment the rate at which people develop AIDS decreases monotonically.

Parameters a	nd Value	Source
Ctata Manialalar	ilu value	Source
State Variables		
S	13307	Yang B.M et <i>al.</i> (2004)
Ι	500	Assumed
Т	500	Assumed
А	30000	Assumed
β	0.01	(CDC, 2004)
Ν	164077	Calculated
$\mu_{1}$	0.0000548	Olaniyi, 2013
$\pi$	5000	Assumed
ω	0.08	Abdalla et <i>al</i> .
$\mu_2$	0.1	Assumed
α	0.0.0024	Calculated
С	0.2	Assumed
Κ	0.2	Assumed

Table 2 shows initial conditions for each plot and parameters values.



Figure 1 is the graph of HIV infected individuals on treatment with time. It observed that the population of HIV infected individuals on treatment increases as the treatment rate increases.

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Figure 2 is the graph of Individuals leaving with AIDS against time. We notice that the number of AIDS infected individual's decreases as the HIV treatment rate increases and brought down the number of individuals infected with AIDS to zero when the HIV treatment rate is high i.e at 85% treatment coverage.



Figure 3 shows the graph of HIV infected individuals against time. It is observed that the population of infected HIV individuals' increases as the HIV contact parameter k increases.

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Figure 4 shows the graph of HIV infected individuals on treatment against time. It is observed that the population of infected HIV individuals' on treatment increases as the HIV contact parameter k increases.

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