



Article Info

Received: 28th January 2019

Revised: 14th February, 2019

Accepted: 21st February 2019

¹Department of Mathematics, Modibbo Adamu University of Technology, Yola. Adamawa State, Nigeria

²Department of Mathematics, College of Agriculture, Jalingo. Taraba State, Nigeria

³Department of Mathematics, Federal College of Education (T), Gombe. Gombe State, Nigeria

⁴Department of Biotechnology, Modibbo Adamu University of Technology, Yola. Adamawa State, Nigeria

*Corresponding author's email:

abduulfatai@mautech.edu.ng

Cite this: *CaJoST*, 2019, 1, 71-82

Mathematical Model for the Transmission Dynamics of HIV/Aids and TB Co-Infection

Abduulfatai A. Momoh^{1*}, Abubakar Isah², Nurudeen G. A. Abimbola³ and Deborah Mathias⁴

This paper presents a mathematical model for the transmission dynamics of HIV/AIDS and TB co-infection. The model is governed by seven (7) system of ordinary differential equations namely: Susceptible, Latent TB, TB infectious, Treated TB, HIV infected, HIV/AIDS and TB co-infected and AIDS. The disease free and endemic equilibrium points were obtained for HIV/AIDS sub model, TB sub model and HIV/AIDS and TB co-infection model. We also derived reproduction numbers for HIV/AIDS sub model, TB sub model and HIV/AIDS and TB co-infection model. It was established that the model is locally and globally asymptotically stable provided that $R_0 < 1$ and unstable when $R_0 > 1$. Numerical simulation shows that treatment of TB reduces the spread of TB among TB infected and HIV/TB individuals.

Keywords: Tuberculosis, HIV/AIDS, Co-infection, Basic reproduction number

1. Introduction

The prevalence of certain diseases such as HIV/AIDS, Tuberculosis (TB), Pneumonia, and Malaria etc. prompted enormous researchers to devise control strategies to curb these menaces in our society. The Human Immunodeficiency Virus (HIV) is the agent responsible for the acquired immunodeficiency syndrome (AIDS) [7]. HIV can be transmitted through unprotected sexual intercourse, transfusion of contaminated blood; and sharing of contaminated needles, syringes, surgical equipment or other sharp instruments. It may also be transmitted between a mother and her infant during pregnancy, childbirth and breastfeeding [18]. [12] reports that it targets the immune system of the infected individual and weakens it making the individual incapable of resisting attack from a wide range of infections such as TB, malaria and pneumonia [12]. HIV has claimed more than 35 million lives so far. In 2016, 1.0 million people died from HIV-related causes globally. There were approximately 36.7 million people living with HIV at the end of 2016 with 1.8 million people becoming newly infected globally. It is estimated that currently only 70% of people with HIV know their status. To reach the target of 90%, an additional 7.5 million people need to access HIV testing services. There is no cure for HIV infection. However, effective antiretroviral therapy (ART) drugs can control the virus and help prevent

transmission so that people with HIV, and those at substantial risk, can enjoy healthy, long and productive lives. 54% of adults and 43% of children living with HIV are currently receiving lifelong ART. Global ART coverage for pregnant and breastfeeding women living with HIV is high at 76%. In mid-2017, 20.9 million people living with HIV were receiving ART globally. Between 2000 and 2016, new HIV infections fell by 39%, and HIV-related deaths fell by one third with 13.1 million lives saved due to ART in the same period.

TB is a bacterial infectious disease of humans and animals caused by the pathogen *Mycobacterium Tuberculosis* (MTB). It is an airborne disease and one of the most common infectious diseases. TB is a disease with slow dynamics and therefore the epidemics must be studied over a long window in time [3], [11], [14]. Infected individuals may remain in the asymptomatic stage throughout their entire lives (exposed or latent TB) [14], [1]. Individuals progress from exposed stage to infectious or active stage and TB disease is as a result of this progression.

Although, the probability of progression towards active TB usually depends on age of infection [1]. Individuals in active TB stage show symptoms of infection and are infectious. We can group Active TB as pulmonary and extra-pulmonary kinds. Pulmonary TB is often seen in adults and transmitted by MTB, while extra-pulmonary is

more frequent among women, children and in HIV infected individuals [10]. We can classify TB within the period of five years after infection as primary TB, while TB after the period of five years from initial infection can be classified as secondary TB. The clinical observation of this disease reveals that the patient suffers from a latent fever, violent coughing, which expels thin purulent sputum, breathes with difficulty and has flushed cheeks. The entire body would turn ashen. The eyes have a weary expression; the patient is gaunt in appearance, sweating is seen on the upper parts of the chest. The patient loses appetite or suffers hunger pangs. The ends of the fingers swell and the fingernails curve abnormally. This infectious disease is so devastating that it has become a motivating force in the development of the fields of bacteriology and modern epidemiology [14].

According to WHO, it is estimated that one third of the population of people in the world are infected with TB [19] and as a result leads to 2-3 million death each year [3], [4], [8] with about 8-9 million developing active TB [8]. Although, 90% - 95% of TB cases occur in developing countries and about 1.8 million new cases of TB occur per year in India [13]. Overall, a relatively small proportion (5–15%) of the estimated 1.7 billion people infected with MTB will develop TB disease during their lifetime. However, the probability of developing TB disease is much higher among people infected with HIV, and also higher among people affected by risk factors such as under-nutrition, diabetes, smoking and alcohol consumption [13]. Today, WHO reports TB as the leading cause of death among HIV-positive persons worldwide and accounts for 40 percent of AIDS deaths in Africa and Asia, up to 50 million people worldwide may be infected with multidrug-resistant Tuberculosis (MDRTB) [17].

TB disease can be treated by taking several drugs for 6 to 9 months. There are 10 drugs currently approved by the U.S. Food and Drug Administration (FDA) for treating TB. Of the approved drugs, the first-line anti-TB agents that form the core of treatment regimens are isoniazid (INH), rifampin (RIF), ethambutol (EMB) and pyrazinamide (PZA) [19].

The HIV epidemic has significantly impacted TB dynamic. Over the last 15 years, one third of the detected increases in active TB cases can be related to the HIV epidemic [5]. How HIV

increases the incidence of new MTB infections had been described by several reports. It aggravates the degree of infectiousness of TB and re-activates latent MTB [6]. Conversely, Immunocompromised HIV infected individuals are at higher risk of opportunistic infections like TB, TB tends to increase the HIV replication rate. Replication of HIV may lead to fast progression to AIDS [3].

However, the relationship between HIV and TB is a public health concern and this has propelled this study to examine the co-infection of TB and HIV/AIDS. As such, the model of HIV/AIDS transmission dynamic due to [15] and the model of TB transmission dynamic due to [2] were coined to give a transmission dynamics of HIV/AIDS and TB co-infections.

2. Model Formulation

The population is divided into seven compartments or classes namely: susceptible $S(t)$, latent TB $L_T(t)$, Active TB $I_T(t)$, treated TB $T_T(t)$, infected HIV $I_H(t)$, infected TB and HIV $I_{TH}(t)$, and AIDs $A(t)$. The susceptible class increases as a result of coming in of individuals by birth or immigration at the rate Λ_0 . The class reduces as a result of progression of individuals to the latent TB class with a proportion $\frac{\beta_1}{N}(I_T + I_{TH})S$, to the infected HIV class with a proportion $\frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)S$ and due to the natural death at a rate μS . The latent TB class increases as a result of coming in of susceptible individuals which are been infected by TB with a rate $\frac{\beta_1}{N}(I_T + I_{TH})S$, due to coming in of latent individuals by immigration at a rate Λ_1 , and as a result of coming in of treated individuals which are been infected by TB at a rate $\alpha c T_T \frac{I_T}{N}$. The

class reduces as a result of moving out of individuals from the class to Active TB class at a progression rate κL_T and as a result of natural death at a rate μL_T and death induced at a rate dL_T . The Active TB class increases as a result of coming in of individuals from the latent TB class at

a progression rate κL_T and as a result of incoming of Active TB individuals by immigration at a rate Λ_2 . The class reduces as a result of progression of individuals to the treated TB class at the treatment rate rI_T , to the TB and HIV/AIDS co-infected class at a rate $\delta \frac{\beta_2 C}{N} (I_H + I_{TH} + \eta A) I_T$, due to active TB induced death rate at a rate $d_T I_T$ and due to natural death at a rate μI_T . The treated TB class increases as a result of coming in of treated active TB individuals at a treatment rate rI_T . The class reduces as a result of having contact with infectious individuals and becomes latent at a rate $\alpha \tau T_T \frac{I_T}{N}$, due to progression of individuals to infected HIV with a proportion $\frac{\beta_2 C}{N} (I_H + I_{TH} + \eta A) I_T$ and due to natural death at rate μT_T . The HIV infected class increases as a result of coming in of individuals from Treated TB class with a proportion $\frac{\beta_2 C}{N} (I_H + I_{TH} + \eta A) I_T$, due to coming in of individuals from the susceptible with a proportion $\frac{\beta_2 C}{N} (I_H + I_{TH} + \eta A) S$ and due to coming in of HIV infectious individuals after being treated from TB at a rate τI_{TH} . The class reduces due to moving out of individuals to AIDS class at a rate σI_H , due to progression of HIV infected individuals to HIV/AIDS and TB class with a proportion $\psi \frac{\beta_1}{N} (I_T + I_{TH}) I_H$, and also due to HIV induced death at a rate $d_H I_H$, and also due to natural death at a rate μI_H . The infected TB and HIV class increases as a result of progression of individuals from the infected TB class with a proportion $\delta \frac{\beta_2 C}{N} (I_H + I_{TH} + \eta A) I_T$ and due to progression of individuals from Infected HIV class at a rate $\psi \frac{\beta_1}{N} (I_T + I_{TH}) I_H$. The class reduces as a result of moving out of HIV infectious individuals from I_{TH} class after being treated from

TB, due to TB induced death at a rate $d_T I_{TH}$, due to HIV induced death at a rate $d_H I_{TH}$ and as a result of natural death at a rate μI_{TH} . The full blunt AIDS class increases as a result of coming in of individuals from HIV infected class at a rate σI_H . The class reduces as a result of death due to full blunt AIDS at a rate $d_A A$ and due to natural death at a rate μA .

2.1 Assumptions for model

- i. All latent TB individuals are not infectious.
- ii. Individuals can only get TB through contact with infectious individuals (I_T and I_{TH}).
- iii. AIDS individuals are considered to transmit HIV.
- iv. Transmission is only from susceptible, treated and HIV class all other forms of transmission are not considered.
- v. individuals in the class (A) can either remain in this class or die.
- vi. Treated TB individuals can be infected with HIV after having contact with HIV/AIDS individuals.
- vii. Individuals would not completely recover from TB but would be exposed after having contact with infectious TB individuals.
- viii. Co-infected individuals becomes HIV infectious after being treated from TB.

The model diagram for HIV/AIDS and TB Co-infection is displayed in Figure 1.

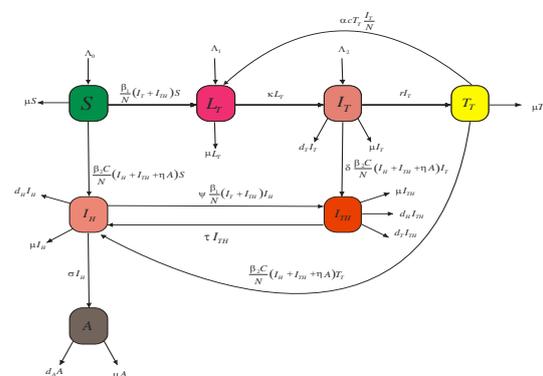


Figure 1. Schematic diagram for the model with HIV/AIDS and TB co-infection.

2.2 HIV/AIDS and TB model equations

$$\left. \begin{aligned} \frac{dS}{dt} &= \Lambda_0 - \frac{\beta_1}{N}(I_T + I_{TH})S - \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)S - \mu S \\ \frac{dL_T}{dt} &= \Lambda_1 + \frac{\beta_1}{N}(I_T + I_{TH})S + \alpha c T_T \frac{I_T}{N} - (\kappa + \mu)L_T \\ \frac{dI_T}{dt} &= \Lambda_2 + \kappa L_T - (d_T + r + \mu)I_T - \delta \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)I_T \\ \frac{dT_T}{dt} &= r I_T - \alpha c T_T \frac{I_T}{N} - \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)T_T - \mu T_T \\ \frac{dI_H}{dt} &= \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)S + \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)T_T - \psi \frac{\beta_2 C}{N}(I_T + I_{TH})I_H - (\sigma + d_H + \mu)I_H + \tau I_{TH} \\ \frac{dI_{TH}}{dt} &= \delta \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)I_T + \psi \frac{\beta_2 C}{N}(I_T + I_{TH})I_H - (\mu + \tau + d_T + d_H)I_{TH} \\ \frac{dA}{dt} &= \sigma I_H - \mu A - d_A A \end{aligned} \right\} \quad (1)$$

With initial conditions

$$S(0) = S_0, L_T(0) = L_{T_0}, I_T(0) = I_{T_0}, T_T(0) = T_{T_0}, I_H(0) = I_{H_0}, I_{TH}(0) = I_{TH_0}, A(0) = A_0. \quad (2)$$

3. Mathematical analysis

3.1 HIV/AIDS sub model

The HIV/AIDS sub model equations are obtained by letting $L_T = I_T = T_T = I_{TH} = 0$ in system (1) which gives.

$$\left. \begin{aligned} \frac{dS}{dt} &= \Lambda_0 - \frac{\beta_2 C}{N_H}(I_H + \eta A)S - \mu S \\ \frac{dI_H}{dt} &= \frac{\beta_2 C}{N_H}(I_H + \eta A)S - (\sigma + \mu + d_H)I_H \\ \frac{dA}{dt} &= \sigma I_H - \mu A - d_A A \end{aligned} \right\} \quad (3)$$

with initial conditions

$$S(0) = S_0, L_T(0) = L_{T_0}, I_T(0) = I_{T_0}, T_T(0) = T_{T_0}. \quad (4)$$

3.1.1 Disease-free equilibrium for HIV/AIDS sub model

At equilibrium

$$\frac{dS}{dt} = \frac{dI_H}{dt} = \frac{dA}{dt} = 0$$

The disease-free equilibrium point for HIV/AIDS sub model (2) is

$$E_{0H} = (S^0, I_H^0, A^0) = \left(\frac{\Lambda_0}{\mu}, 0, 0 \right) \quad (5)$$

3.1.2 Basic reproduction number of HIV/AIDS sub model

We evaluated F and V , which are the Jacobian matrices of \hat{F} and \hat{V} , respectively at disease

free equilibrium E_{0H} following the approach in [16] to get

$$F = \begin{bmatrix} \beta_2 C & \eta \beta_2 C \\ 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} (\sigma + \mu + d_H) & 0 \\ -\sigma & (\mu + d_A) \end{bmatrix}$$

We then obtain the spectral radius of $\rho(FV^{-1})$, which is defined as the largest eigenvalue of FV^{-1} . Thus, the basic reproduction number for HIV/AIDS sub model, R_{0H} is

$$R_{0H} = \frac{\beta_2 C}{(\sigma + \mu + d_H)} + \frac{\eta \beta_2 C}{(\mu + d_A)(\sigma + \mu + d_H)} \quad (6)$$

3.1.3 Local stability of the disease free equilibrium for HIV/AIDS sub model

Theorem 1: The disease free equilibrium point E_{0H} of HIV/AIDS sub model is locally asymptotically stable (LAS) if $R_{0H} < 1$ and unstable if $R_{0H} > 1$.

Proof:

We let

$$\left. \begin{aligned} F_1 &= \Lambda_0 - \frac{\beta_2 C}{N_H}(I_H + \eta A)S - \mu S \\ F_2 &= \frac{\beta_2 C}{N_H}(I_H + \eta A)S - (\sigma + \mu + d_H)I_H \\ F_3 &= \sigma I_H - \mu A - d_A A \end{aligned} \right\} \quad (7)$$

The Jacobian of system (7) at disease free is

$$J(E_{0H}) = \begin{bmatrix} -\mu & \beta_2 C & -\eta \beta_2 C \\ 0 & -((\sigma + \mu + d_H) - \beta_2 C) & \eta \beta_2 C \\ 0 & \sigma & -(\mu + d_A) \end{bmatrix}$$

The eigen value of $J(E_{0H})$ is $\lambda_1 = -\mu_1$ and other two are solutions of

$$\begin{vmatrix} -((\sigma + \mu + d_H) - \beta_2 C) & \eta \beta_2 C \\ \sigma & -(\mu + d_A) \end{vmatrix} = 0 \quad (8)$$

From equation (8) we have

$$a_2\lambda^2 + a_1\lambda + a_0 = 0 \tag{9}$$

where

$$a_2 = 1; a_1 = ((\sigma + \mu + d_H) - \beta_2 C) + (\mu + d_A)$$

$$\text{and } a_0 = (1 - R_{0H}) \tag{10}$$

By Routh-Hurwitz criterion, all the eigenvalues of the polynomial have negative real parts when $R_{0H} < 1$, hence the disease-free equilibrium point is locally asymptotically stable.

3.1.4 Global stability of the disease-free equilibrium point for HIV/AIDS sub model

Theorem 2: If $R_0 \leq 0$, then the disease free equilibrium $(I_H, A) = (0, 0)$ of the system is globally asymptotically stable on Ω .

Proof: We construct the following Lasalle-Lyapunov function $V(I_H, A)$ on the positively invariant compact set Ω thus on Ω , $V(I_H, A)$ is continuous and non-negative.

Consider the infectious compartment of equation (3)

$$\left. \begin{aligned} \frac{dI_H}{dt} &= \frac{\beta_2 C}{N_H} (I_H + \eta A) S - (\sigma + \mu + d_H) I_H \\ \frac{dA}{dt} &= \sigma I_H - (\mu + d_A) A \end{aligned} \right\} \tag{11}$$

Equation (11) can be written as

$$\begin{bmatrix} \dot{I} \\ \dot{A} \end{bmatrix} = \begin{bmatrix} \beta_2 C + (\sigma + \mu + d_H) & \beta_2 C \eta \\ \sigma & -(\mu + d_A) \end{bmatrix} \begin{bmatrix} I_H \\ A \end{bmatrix} \tag{12}$$

Equation (12) can be written as $\dot{I} = A(I)$ where

$$A = \begin{bmatrix} \beta_2 C + (\sigma + \mu + d_H) & \beta_2 C \eta \\ \sigma & -(\mu + d_A) \end{bmatrix} \text{ and } I = \begin{bmatrix} I_H \\ A \end{bmatrix}$$

If we define $V^T = [\sigma, ((\sigma + \mu + d_H) - \beta_2 C)]$ then the derivative along the trajectories is given by $\dot{V} = V^T A(I)$ is

$$V^T A(I) = \begin{bmatrix} 0 \\ (\mu + d_A)(\sigma + \mu + d_H)(R_{0H} - 1) \end{bmatrix}$$

Which is strictly decreasing when $R_{0H} < 1$.

Thus, by Lasalle's invariant principle the disease-free equilibrium is globally symptomatically stable on Ω .

3.2 TB sub model

The TB sub model is obtained by letting $I_H = I_{TH} = A = 0$ in model (1) to get

$$\left. \begin{aligned} \frac{dS}{dt} &= \Lambda_0 - \frac{\beta_1 I_T}{N_T} S - \mu S \\ \frac{dL_T}{dt} &= \Lambda_1 + \frac{\beta_1 I_T}{N_T} S + \alpha c T_T \frac{I_T}{N_T} - (\kappa + \mu) L_T \\ \frac{dI_T}{dt} &= \Lambda_2 + \kappa L_T - (d_T + r + \mu) I_T \\ \frac{dT_T}{dt} &= r I_T - \alpha c T_T \frac{I_T}{N_T} - \mu T_T \end{aligned} \right\} \tag{13}$$

With initial conditions

$$S(0) = S_0, L_T(0) = L_{T_0}, I_T(0) = I_{T_0}, T_T(0) = T_{T_0}. \tag{14}$$

3.2.1 Disease free equilibrium point of TB sub model

At disease free equilibrium,

$$\frac{dS}{dt} = \frac{dL_T}{dt} = \frac{dI_T}{dt} = \frac{dT_T}{dt} = 0$$

The disease-free equilibrium point for TB sub model (12) is

$$E_{OT} = (S^0, L_T^0, I_T^0, T_T^0) = \left(\frac{\Lambda_0}{\mu}, 0, 0, 0 \right) \tag{15}$$

3.2.2 Basic reproduction number for TB

We evaluated F and V , which are the Jacobian matrices of \hat{F} and \hat{V} , respectively at disease free equilibrium E_{OT} to get

$$F = \begin{bmatrix} 0 & \beta_1 \frac{\Lambda_0}{\Lambda_V} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and}$$

$$V = \begin{bmatrix} (\kappa + \mu) & 0 & 0 \\ -\kappa & (d_T + r + \mu) & 0 \\ 0 & -r & \mu \end{bmatrix}$$

We obtain the spectral radius of $\rho(FV^{-1})$, which is defined as the largest eigenvalue of FV^{-1} . Thus the basic reproduction number for TB sub model, R_{0T} is

$$R_{0T} = \frac{\beta_1 \kappa \Lambda_0}{\Lambda_V (\kappa + \mu)(d_T + r + \mu)} \tag{16}$$

3.2.3 Local stability of disease-free equilibrium point for TB sub model

Theorem 3: The disease free equilibrium point E_{0T} of TB-only model is locally asymptotically stable (LAS) if $R_{0T} < 1$ and unstable if $R_{0T} > 1$.

Proof:

We let

$$\left. \begin{aligned} F_1 &= \Lambda_0 - \frac{\beta_1 I_T}{N_T} S - \mu S \\ F_2 &= \Lambda_1 + \frac{\beta_1 I_T}{N_T} S + \alpha c T_T \frac{I_T}{N_T} - (\kappa + \mu) L_T \\ F_3 &= \Lambda_2 + \kappa L_T - (d_T + r + \mu) I_T \\ F_4 &= r I_T - \alpha c T_T \frac{I_T}{N_T} - \mu I_T \end{aligned} \right\} \tag{17}$$

The Jacobian of system (17) at disease free is

$$J(E_{0T}) = \begin{bmatrix} -\mu & 0 & -\beta_1 \frac{\Lambda_0}{\Lambda_V} & 0 \\ 0 & -(\kappa + \mu) & \beta_1 \frac{\Lambda_0}{\Lambda_V} & 0 \\ 0 & \kappa & -(d_T + r + \mu) & 0 \\ 0 & 0 & r & -\mu \end{bmatrix}$$

The eigen value of $J(E_{0T})$ are $\lambda_1 = -\mu_1, \lambda_2 = -\mu_1$ and other two are solutions of

$$\begin{vmatrix} -(\kappa + \mu) & \beta_1 \frac{\Lambda_0}{\Lambda_V} \\ \kappa & -(d_T + r + \mu) \end{vmatrix} = 0 \tag{18}$$

From equation (18), we have

$$b_2 \lambda^2 + b_1 \lambda + b_0 = 0 \tag{19}$$

By Routh Hurwitz criteria all the values of $\lambda_i < 0$ for $i = 1, 2,$ and 3 when $R_{0T} < 1$, we conclude that disease free equilibrium for TB sub model is locally asymptotically stable.

3.2.4 Global stability of the disease-free equilibrium for TB sub model

Theorem 4: If $R_{0T} < 1$, then the disease free equilibrium $(L_T, I_T) = (0, 0)$ of the system is globally asymptotically stable on Ω .

Proof: We construct the following Lasalle-Lyapunov function $V(L_T, I_T)$ on the positively invariant compact set Ω thus on $\Omega, V(L_T, I_T)$ is continuous and non-negative.

Consider the infectious compartments of system (13)

$$\left. \begin{aligned} \frac{dL_T}{dt} &= \Lambda_1 + \frac{\beta_1 I_T}{N} S + \alpha c T_T \frac{I_T}{N} - (\kappa + \mu) L_T \\ \frac{dI_T}{dt} &= \Lambda_2 + \kappa L_T - (d_T + r + \mu) I_T \end{aligned} \right\} \tag{20}$$

The equation (20) can be written as

$$\begin{bmatrix} \dot{L}_T \\ \dot{I}_T \end{bmatrix} = \begin{bmatrix} -(\kappa + \mu) & \frac{\beta_1 \Lambda_0}{\Lambda_V} \\ \kappa & -(d_T + r + \mu) \end{bmatrix} \begin{bmatrix} L_T \\ I_T \end{bmatrix} \tag{21}$$

Equation (21) can be written as $\dot{I} = B(I)$, where

$$B = \begin{bmatrix} -(\kappa + \mu) & \frac{\beta_1 \Lambda_0}{\Lambda_V} \\ \kappa & -(d_T + r + \mu) \end{bmatrix} \text{ and } I = \begin{bmatrix} L_T \\ I_T \end{bmatrix}$$

If we define $V^T = [\kappa, (\kappa + \mu)]$ then the derivative

along the trajectories given by $\dot{V} = V^T A(I)$ is

$$V^T B(I) = \begin{bmatrix} 0 \\ (\kappa + \mu)(d_T + r + \mu)[R_{0T} - 1] \end{bmatrix} \tag{22}$$

Equation (22) is strictly decreasing when $R_{0T} < 1$. Thus, by Lasalle's invariant principles the disease free equilibrium is globally asymptotically stable on Ω .

3.3.1 Disease free equilibrium point for HIV/AIDS and TB co-infection model

The disease-free equilibrium state for the HIV/AIDS and TB co-infection model is

$$E_{OTH} = \left(\frac{\Lambda_0}{\mu}, 0, 0, 0, 0, 0, 0 \right) \tag{23}$$

3.3.2 Basic reproduction number for HIV/AIDS and TB co-infection

We evaluated F and V , which are the Jacobian matrices of \hat{F} and \hat{V} , respectively at disease free equilibrium E_{OTH} to get

$$F = \begin{bmatrix} 0 & \frac{\beta_1 \Lambda_0}{\Lambda_V} & 0 & 0 & \frac{\beta_1 \Lambda_0}{\Lambda_V} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta_2 C \Lambda_0}{\Lambda_V} & \frac{\beta_2 C \Lambda_0}{\Lambda_V} & \frac{\beta_2 C \eta \Lambda_0}{\Lambda_V} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

and $V = \begin{bmatrix} a_1 & 0 & 0 & 0 & 0 & 0 \\ -\kappa & a_2 & 0 & 0 & 0 & 0 \\ 0 & -r & a_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & a_4 & -\tau & 0 \\ 0 & 0 & 0 & 0 & a_5 & 0 \\ 0 & 0 & 0 & -\sigma & 0 & a_6 \end{bmatrix}$

where

$$a_1 = (\kappa + \mu); a_2 = (d_T + r + \mu); a_3 = \mu; a_4 = (\sigma + d_H + \mu); a_5 = (\mu + \tau + d_T + d_H); a_6 = (\mu + d_A)$$

Hence, the reproduction number for the HIV/AIDS and TB co-infection is

$$R_{OTH} = \max(R_{0H}, R_{0T}) \tag{24}$$

Where

$$R_{0H} = \frac{\beta_2 C \Lambda_0}{\Lambda_V (\sigma + \mu + d_H)} + \frac{\eta \beta_2 C \Lambda_0}{\Lambda_V (\mu + d_A) (\sigma + \mu + d_H)}$$

$$R_{0T} = \frac{\beta_1 \kappa \Lambda_0}{\Lambda_V (\kappa + \mu) (d_T + r + \mu)}$$

This correspond to the reproduction number for HIV/AIDS sub model

$$R_{0H} = \frac{\beta_2 C}{(\sigma + \mu + d_H)} + \frac{\eta \beta_2 C}{(\mu + d_A) (\sigma + \mu + d_H)}$$

3.3.3 Local stability of the disease-free equilibrium point for HIV/AIDS and TB co-infection

Theorem 5: The disease free equilibrium E_{OTH} point of full model is locally asymptotically stable unstable if $R_{OTH} > 1$

(LAS) if $R_{OTH} < 1$ and

Proof:

We let

$$\left. \begin{aligned} F_1 &= \Lambda_0 - \frac{\beta_1}{N} (I_T + I_{TH}) S - \frac{\beta_2 C}{N} (I_H + I_{TH} + \eta A) S - \mu S \\ F_2 &= \Lambda_1 + \frac{\beta_1}{N} (I_T + I_{TH}) S + acT_T \frac{I_T}{N} - (\kappa + \mu) I_T \\ F_3 &= \Lambda_2 + \kappa L_T - (d_T + r + \mu) I_T - \delta \frac{\beta_2 C}{N} (I_H + I_{TH} + \eta A) I_T \\ F_4 &= r I_T - acT_T \frac{I_T}{N} - \frac{\beta_2 C}{N} (I_H + I_{TH} + \eta A) T_T - \mu T_T \\ F_5 &= \frac{\beta_2 C}{N} (I_H + I_{TH} + \eta A) S + \frac{\beta_2 C}{N} (I_H + I_{TH} + \eta A) T_T - \psi \frac{\beta_1}{N} (I_T + I_{TH}) I_H - (\sigma + d_H + \mu) I_H + \tau I_{TH} \\ F_6 &= \delta \frac{\beta_2 C}{N} (I_H + I_{TH} + \eta A) I_T + \psi \frac{\beta_1}{N} (I_T + I_{TH}) I_H - (\mu + \tau + d_T + d_H) I_{TH} \\ F_7 &= \sigma I_H - \mu A - d_A A \end{aligned} \right\} \tag{25}$$

The Jacobian of system (25) at disease free is

$$J(E_{OTH}) = \begin{bmatrix} -a_3 & 0 & -\beta_1 \frac{\Lambda_0}{\Lambda_V} & 0 & -\beta_2 \frac{\Lambda_0}{\Lambda_V} & -(\beta_1 + \beta_2 C) \frac{\Lambda_0}{\Lambda_V} & 0 \\ 0 & -a_1 & \beta_1 \frac{\Lambda_0}{\Lambda_V} & 0 & 0 & \beta_1 \frac{\Lambda_0}{\Lambda_V} & 0 \\ 0 & \kappa & -a_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & r & -a_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\left(a_4 - \beta_2 \frac{\Lambda_0}{\Lambda_V}\right) & \beta_2 \frac{\Lambda_0}{\Lambda_V} + \tau & \beta_2 C \frac{\Lambda_0}{\Lambda_V} \\ 0 & 0 & 0 & 0 & 0 & -a_5 & 0 \\ 0 & 0 & 0 & 0 & \sigma & 0 & -a_6 \end{bmatrix} \tag{26}$$

Based Routh-Hurwitz criterion, it has been verified that all the eigenvalues of the polynomial of (26) have negative real parts, since all the values of $\lambda_i < 0$, for $i = 1, 2, 3, 4, 5, 6, 7$ when $R_{OTH} < 1$.

We conclude that the disease-free equilibrium point for the HIV/AIDS and TB co-infection model is locally asymptotically stable.

3.3.4 Global stability of disease-free equilibrium point HIV/AIDS and TB co-infection

Theorem 6: The disease-free equilibrium point E_{0TH} of HIV/AIDS and TB co-infection is globally asymptotically stable if $R_{0TH} < 1$ and the two conditions of Castillo-Chavez [18] are satisfied.

Proof:

We have from theorem 6 that E_{0TH} is locally asymptotically stable if $R_{0TH} < 1$. Now consider

$$\left. \begin{aligned} \frac{dS}{dt} &= F(S, 0) = \begin{pmatrix} \Lambda_0 - \mu S \\ -\mu I_T \end{pmatrix} \\ \frac{dI}{dt} &= G(S, I) \\ G(S, 0) &= 0 \end{aligned} \right\} \quad (27)$$

Where

$$G(S, I) = \begin{pmatrix} \Lambda_1 + \frac{\beta_1}{N}(I_T + I_{TH})S + \alpha c T_T \frac{I_T}{N} - (\kappa + \mu)I_T \\ \Lambda_2 + \kappa I_T - (d_T + r + \mu)I_T - \delta \frac{\beta_2 C}{N}(I_H + I_{TH})I_T \\ \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)S + \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)T_T - \psi \frac{\beta_1}{N}(I_T + I_{TH})I_H - (\sigma + d_H + \mu)I_H + \tau I_{TH} \\ \delta \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)I_T + \psi \frac{\beta_1}{N}(I_T + I_{TH})I_H - (\mu + \tau + d_T + d_H)I_{TH} \\ \sigma I_H - \mu A - d_A A \end{pmatrix} \quad (28)$$

Given

$$G(S, I) = AI - \hat{G}(S, I) \quad (29)$$

Where

$$A = \begin{pmatrix} -a_1 & \beta_1 & 0 & \beta_1 & 0 \\ \kappa & -a_2 & 0 & 0 & 0 \\ 0 & 0 & \beta_2 C - (\sigma + d_H + \mu) & \beta_2 C + \tau & \beta_2 C \eta \\ 0 & 0 & 0 & -a_5 & 0 \\ 0 & 0 & \sigma & 0 & -a_6 \end{pmatrix}$$

$$\text{and } I = \begin{pmatrix} I_T \\ I_T \\ I_H \\ I_{TH} \\ A \end{pmatrix}$$

$$\hat{G}(S, I) = AI - G(S, I)$$

$$\hat{G}(S, I) = \begin{pmatrix} \hat{G}_1(S, I) \\ \hat{G}_2(S, I) \\ \hat{G}_3(S, I) \\ \hat{G}_4(S, I) \\ G_5(S, I) \end{pmatrix} = \begin{pmatrix} \frac{\beta_1}{N}(I_T + I_{TH})\left(1 - \frac{S}{N}\right) + \alpha c T_T \frac{I_T}{N} - \Lambda_1 \\ \delta \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)I_T - \Lambda_2 \\ \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)\left(1 - \frac{S - T_T}{N}\right) + \psi \frac{\beta_1}{N}(I_T + I_{TH})I_H \\ -\delta \frac{\beta_2 C}{N}(I_H + I_{TH} + \eta A)I_T - \psi \frac{\beta_1}{N}(I_T + I_{TH})I_H \\ 0 \end{pmatrix} \quad (30)$$

From equation (30), we have the condition H_2 in [21] not satisfied since $\hat{G}_4(S, I) < 0$. And therefore we can conclude that E_{0TH} may not be globally asymptotically stable for $R_{0TH} < 1$. This complete the proof.

4. Numerical Simulations

We performed some numerical experiments using ode45 function from MATLAB R2012a to generate figures for our model. The initial condition for each plot and parameter values are presented in table 1. Figures 2 to 9 are generated from numerical simulations carried out on the model equations.

Table 1. Variables and Parameters values used for numerical simulations.

Variables /Parameter	Values	Reference
β_1	0.035	[9]
β_2	0.075	[9]
μ	0.01430	[2]
σ	0.23	Assumed
η	1.02	Assumed
δ	1.2	[9]
ψ	1.05	[9]
c	3	[2]
τ	0.32	Assumed
C	0.4	Assumed
d_T	0.3500	[2]
d_H	0.2	[9]
d_A	0.333	[9]
α	0.2	[9]
r	1	[9]
κ	0.5	[2]
Λ_0	2000	[2]
Λ_1	500	[2]
Λ_2	500	[2]
$S(t)$	100000	[2]

$L_T(t)$	3000	[2]
$I_T(t)$	200	[2]
$T_T(t)$	150	[2]
$I_H(t)$	260	[9]
$I_{TH}(t)$	100	[9]
$A(t)$	40	Assumed
$N(t)$	103750	Assumed

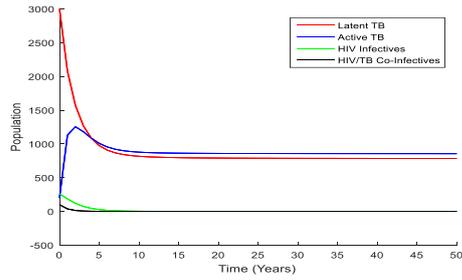


Figure 2. Showing the numerical simulation for the Total Population of Latent TB, Active TB, HIV-infectives and co-infected population.

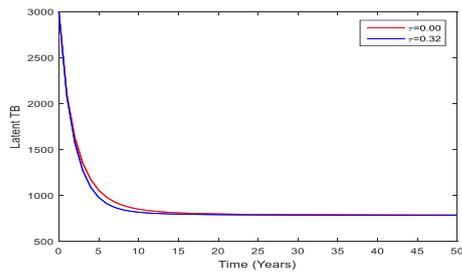


Figure 3. Showing the effect of TB treatment on Latent TB individuals. The following parameter values were used: $\tau = (0.00, 0.32)$.

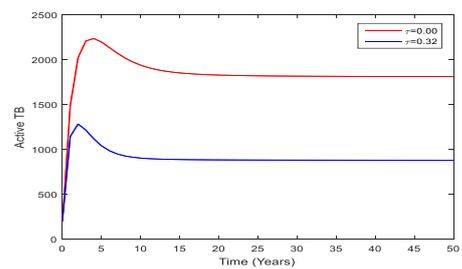


Figure 4. Showing the effect of TB treatment on Active TB individuals. The following parameter values were used: $\tau = (0.00, 0.32)$.

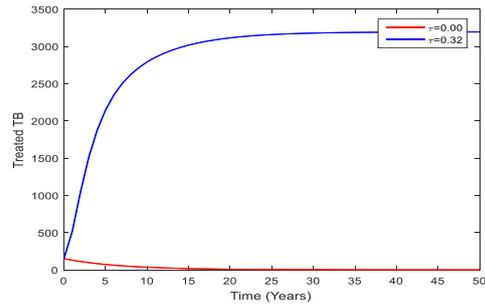


Figure 5. Showing the effect of TB treatment on Treated TB individuals. The following parameter values were used: $\tau = (0.00, 0.32)$.

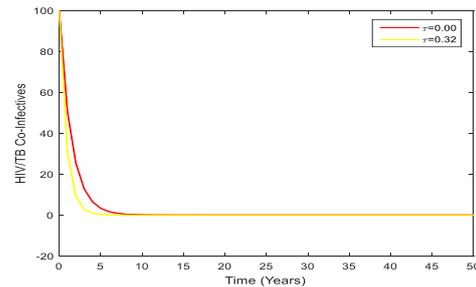


Figure 6. Showing the effect of treatment on HIV/TB co-infected individuals. The following parameter values were used: $\tau = (0.00, 0.32)$.

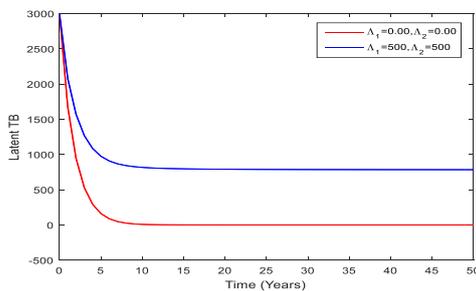


Figure 7. Showing the effect of immigration on Latent TB individuals. We set: $\Lambda_1 = 0.00, 500; \Lambda_2 = 0.00, 500$.

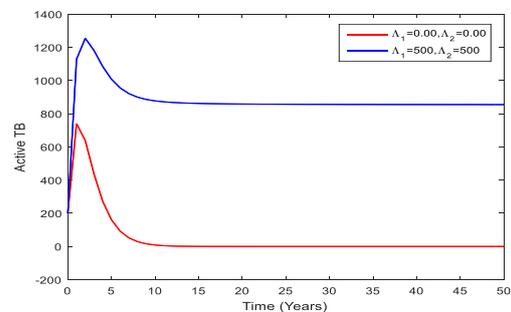


Figure 8. Showing the effect of immigration on Active TB individuals. We set $\Lambda_1 = 0.00, 500; \Lambda_2 = 0.00, 500$.

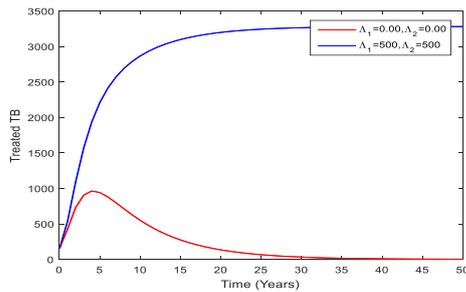


Figure 9. Showing the effect of immigration on Treated TB individuals.

We set:
 $\Lambda_1 = 0.00, 500$; $\Lambda_2 = 0.00, 500$.

5. Discussion of Results

We obtained the disease free and endemic equilibrium point for the HIV/AIDS sub-model, TB sub-model and HIV/AIDS and TB co-infected model. We also obtained their reproduction number, R_{OT} , R_{OH} and R_{OTH} using the next generation matrix. We established their local stability of the disease free equilibrium using the Routh-Hurwitz condition and found that they are locally asymptotically stable if $R_{OT} < 1$, $R_{OH} < 1$ and $R_{OTH} < 1$ which mean the disease can be eradicated and unstable if $R_{OT} > 1$, $R_{OH} > 1$ and $R_{OTH} > 1$ which mean the disease cannot be eradicated. Global stability of the disease free equilibrium using the Lasalle-Lyapunov and Castillo-Chavez conditions was carried out and found to be globally asymptotically stable except for the HIV/AIDS and TB co-infection which may not be globally asymptotically state because second condition of Castillo-Chavez conditions failed. Numerical simulations carried out shows the effects of TB treatment and immigration of TB individuals into different classes of the population.

The discussion of our numerical simulation is categorized into three subsections namely: Individuals subpopulation for HIV/AIDS and TB, combined HIV/AIDS and TB population and the effects of Latent TB, Active TB, HIV infective and co-infection population on the dynamics of the model.

5.1 Numerical simulations for the combined subpopulation of HIV/AIDS and TB

In Figure 2 the Active TB increase rapidly in few years and began to decrease to asymptotically low level due to TB treatment while the latent TB,

HIV and co-infected population decrease to asymptotically low level due to treatment of TB and HIV.

5.2 Effects of treatment and immigration on the dynamic of the model

Figure 3 and 6 shows the effect of treatment on the Latent and co-infected population, it is observed that the populations in these classes continue decreasing from treatment rate 0 to 0.32 as a result of effective TB treatment. Figure 4 depicts that when treatment rate is 0.00 there was no significant change in the Active TB population but as treatment rate increases to 0.32 the Active TB decreases to asymptotic low level. Figure 15 shows that when the treatment is 0 there was no change in the treated TB population but as treatment rate increases to 0.32 the treated TB population decrease drastically to zero. Figure 7 shows the effect of immigration on latent TB class which increases as the populations of the immigrants increases from 0.00 to 500. Also, in figure 8 shows the effect of immigration into active TB population which has no any change when the immigration rate 0.00, but when the immigration rate increase to 500 the active TB population rise up rapidly and start dropping again this happened as a result of coming in of latent or infectious TB into the active TB population. Figure 9 shows that treated TB population increases and dropped to zero level when the immigration rate 0.00 because there is no any latent or infectious TB individuals coming into the population, but the treated TB population also increases rapidly and dropped to asymptotic low level when the immigration rate increase to 500 as a result of coming in of latent or infectious TB into the treated population.

6. Conclusion

This study presents a mathematical model for HIV/AIDS and TB co-infection. The model subdivides the human population into seven compartments namely; Susceptible individuals S , Latent TB individuals L_T , TB infectious individuals I_T , Treated TB individuals T_T , HIV infected individuals I_H , co-infected individuals I_{TH} , and AIDS individuals A . The analytical studies were carried out which revealed that the disease free equilibrium for HIV/AIDS sub-model, TB sub-model and co-infected model are locally

asymptotically stable if $R_0 < 0$. We obtained Endemic equilibrium and Global stability of disease free equilibrium. The numerical simulation carried out shows that treatment of TB increase the human population. The two diseases also exhibit synergetic relation where the infection of one disease speeds the progression of the other.

Conflict of interest

The authors declare no conflict of interest.

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