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A Mathematical Analysis of HIV/TB Co-Infection Model

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Abstract In this work we developed and analyzed a mathematical model of HIV/TB co infection. The model is a first order Ordinary Differential Equations, in which the human population is divided into six mutually- exclusive compartments namely; TB- Susceptible individuals (S), TB-Infected individuals (I), TB-Recovered individuals (R), HIV-Infected individuals (P₁), Co-Infected individuals (P₂) and individuals with AIDS (A). The equilibrium states were obtained and their stabilities were analyzed by using Bellman and Cooke's theorem. The result shows that the endemic equilibrium state is stable and the disease free equilibrium state will be stable if $(\beta_T c\Lambda)/\mu N < (a + \mu + d_T)$.

Keywords Tuberculosis, Human Immunodeficiency Virus, Co infection, Treatment, Equilibrium, Stability

1. Introduction

Tuberculosis (TB) is a bacterial disease caused by Mycobacterium tuberculosis (tubercle bacilli). Transmission of TB occurs by air borne infectious droplets [13]. It typically affects the lungs (pulmonary TB) but can affect other parts as well (extra pulmonary TB) [10]. The World Health Organization (WHO) declared TB in 1993 has a global emergency. Nigeria ranks 10th among the 22 high burden in the world [11]. TB is preventable and curable. A mathematical model for TB has been a useful tool in accessing the spread of the disease, as well as its control. See [3] for example.

HIV stands for Human Immunodeficiency Virus. HIV is a virus that gradually attacks the immune system and the immune system is our body's natural defence against illness. If a person becomes infected with HIV, he or she will find it hard to fight off infections and diseases. The virus destroys a type of white blood cell called T-helper cells and makes copies of it inside them. T-helper cells are also referred to as CD4 cells [16]. There is no cure or vaccine for AIDS [5]. However, antiretroviral (ART) treatment improves health, pro-longs life, and substantially reduces the risk of HIV transmission. In both high-income and low-income countries, the life expectancy of patients infected with HIV who have access to ART is now measured in decades, and might approach that of uninfected populations in patients who receive an high HIV treatment [5].

Co-infection of TB and HIV is when someone has both HIV and TB infections. When someone has HIV and TB, HIV infection accelerates the activation of tuberculosis and

tuberculosis increases the rate at which HIV infection develops into AIDS [9]. It is worthy of note that HIV infection and infection with TB bacteria are two different infections as established in [6].

Co-treatment of HIV related TB improves survival especially in patients with CD4 counts <50 ells/mm³ [8]. Globally, one-third of the 34 million people living with HIV are infected with TB [14]. In 2014, about 400 000 people who had both TB and HIV were estimated to have died, in addition to the 1.1 million people who died from TB alone and 800 000 deaths from HIV alone. So in 2014 more people died from TB than from HIV related infections [15].

[2] in his work, formulated a sex-structured model to capture the effect of complacency on the dynamics of HIV/AIDS but did not include how TB will affect the mix. But, recently a lot of great work has been done in the mathematical modelling of co infection of different pathogens though very little was done in the modelling of HIV-TB co infection. [13], [12], [6], [7] developed a mathematical model of HIV/TB co infection under TB treatment. Their work did not include anti-HIV treatment. [4] Developed a TB-HIV/AIDS co infection model and optimal control treatment. In this paper we incorporate all aspects of TB transmission dynamics as well HIV transmission dynamics to come up with a mathematical model of HIV/TB co-infection. This paper incorporate antiretroviral therapy for individuals infected with HIV. We investigate the effect of treatment on the TB in HIV/TB co infection and also investigate the effect of co treatment of HIV/TB co infection.

The paper is organized as follows. Section 1 is introduction. The model formulation, the positive invariant region, the positivity of solutions, equilibrium states and the basic reproduction number are obtained in Section 2. The stability analysis of the model and concluding remark are carried out in sections 3 and 4 respectively.

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2. Model Formulation

The model subdivides the human population into six mutually –exclusive compartments, Namely TB-susceptible individuals (S), TB-infected individuals who have active TB diseases and infectious (I), TB-recovered individuals (R), HIV-infected individuals (P_1), co-infected individuals (P_2) and full blown AIDS (A).

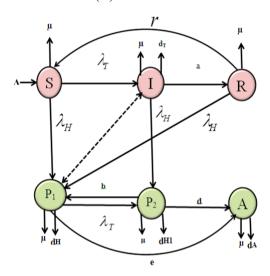


Figure 1. The diagram for the flow of the model

2.1. Basic Assumptions

- ➤ Individuals can only get TB through contact with TB infectious individuals (I and P₂).
- ➤ Individuals may become HIV infected only through contact with HIV infectious individuals (P₁ and P₂).
- The infectious I, both TB and HIV infectious P2 and AIDS individuals are considered too ill to remain sexually active and they are unable to transmit HIV through sexual activity.

$$W = N - I - P_2 - A = S + R + P_1$$
, is the active population.

To represent this mathematically, we have the following system of differential equations:

$$\frac{ds}{dt} = \Lambda + rR - (\lambda_T + \lambda_H + \mu)S \tag{2.1}$$

$$\frac{dI}{dt} = \lambda_T S - (\lambda_H + a + d_T + \mu)I \tag{2.2}$$

$$\frac{dR}{dt} = aI - (\lambda_H + r + \mu)R \tag{2.3}$$

$$\frac{dP_1}{dt} = (S + R)\lambda_H + bP_2 - (\lambda_T + e + d_H + \mu)P_1(2.4)$$

$$\frac{dP_2}{dt} = \lambda_H I + \lambda_T P_1 - (b + d + d_{H1} + \mu) P_2$$
 (2.5)

$$\frac{dA}{dt} = eP_1 + dP_2 - (d_A + \mu)A \tag{2.6}$$

Where $N=S+I+R+P_1+P_2+A$.

The force of infection λ_T , associated with TB infection is

$$\lambda_T = \beta_T C \left(\frac{I + P_2}{N} \right).$$

The force of infection λ_H , associated with HIV infection is

$$\lambda_H = \beta_H \delta \left(\frac{P_1 + P_2}{N} \right).$$

Table 1. Definition of State Variables and Parameters Used in the TB/HIV Model

Symbol	Definition
N	total population
S	number of susceptible (that is no infection)
I	number of persons with active TB
R	number of persons recovered from TB
P_1	number of persons with HIV infection
P_2	number of persons with both HIV and TB infection
A	number of persons with AIDS
W	number of active population
Λ	constant recruitment rate
$oldsymbol{eta}_{T}$	Probability of transmission of TB infection from an active to a susceptible per contact unit time
$oldsymbol{eta}_H$	Probability of transmission of HIV infection from an infected person to an uninfected person per contact unit time.
С	per capita contact rate for TB
δ	per capita contact rate for HIV
μ	Natural death rate
a	treatment rate of active TB individuals
b	treatment rate of infectious TB in HIV individuals
d	AIDS progression rate for individuals in P ₂
e	AIDS progression rate for individuals in P ₁
r	rate at which TB recovered individuals become susceptible to TB
d_{T}	active TB induced death rate
$d_{\rm H1}$	death rate due to both HIV and TB infection
d _H	HIV induced death rate
d_A	AIDS induced death rate

2.2. The Positive Invariant Region

The total population size is

$$N = S + I + R + P_{1} + P_{2} + A$$
 (2.7)

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} + \frac{dP_{1}}{dt} + \frac{dP_{2}}{dt} + \frac{dA}{dt}$$

$$\frac{dN}{dt} = \Lambda - \mu S - \mu I - \mu R - \mu P_{1} - \mu P_{2} - \mu A$$
 (2.8)

The positive invariant region can be obtained by using the following theorem.

 $-d_T-d_H-d_{H1}-dA$

Theorem 2.1

The solutions of the system of equations (2.1) to (2.6) are feasible for t < 0 if they enter the invariant region D.

Proof

Let $D = (S, I, R, P_1, P_2, A) \in \mathbb{R}^{6}_{+}$ be any solution of the system of equations (2.1) to (2.6) with non-zero initial conditions.

Assuming there is no disease equation (2.8) now becomes

$$\frac{dN}{dt} \le \Lambda - \mu N \tag{2.9}$$

$$\frac{dN}{dt} + \mu N \le \Lambda \tag{2.10}$$

The integrating factor for (2.10) is $(IF) = e^{\mu t}$

Multiply both sides of (2.10) by $e^{\mu t}$ gives

$$e^{\mu t} \frac{dN}{dt} + \mu N e^{\mu t} \le \Lambda e^{\mu t} \tag{2.11}$$

$$d(Ne^{\mu t}) \le \Lambda e^{\mu t} dt \tag{2.12}$$

Integrating both sides we have

$$N(t)e^{\mu t} = \frac{\Lambda e^{\mu t}}{\mu} + c \tag{2.13}$$

$$N(t) = \frac{\Lambda}{\mu} + ce^{-\mu t} \tag{2.14}$$

Applying the initial condition t = 0; $N_h(0) = N_0$

$$N_0 \le \frac{\Lambda}{\mu} + C \Rightarrow N_0 - \frac{\Lambda}{\mu} \le C \tag{2.15}$$

$$\Rightarrow N \le \frac{\Lambda}{u} + (N_0 - \frac{\Lambda}{u}) e^{-\mu t}$$
 (2.16)

Therefore, as $t \to \infty$ in (2.16) the human population N approaches $K = \frac{\Lambda}{\mu}$ (That is, $N \to K = \frac{\Lambda}{\mu}$) the parameter

 $K = \frac{\Lambda}{\mu}$ is called the carrying capacity.

Hence all feasible solution set of the human population of the model equations (2.1) to (2.6) enter the region

$$D = \begin{cases} (S, I, R, P_1, P_2, A) \in R^6 : S > 0, I \ge 0, R \ge 0, \\ P_1 \ge 0, P_2 \ge 0, A \ge 0, N \le \frac{\Lambda}{N} \end{cases}$$

Therefore, the region D is positively-invariant (that is the solution is positive for all times, (t) and equations (2.1) to (2.6) are epidemiologically meaningful and mathematically well-posed in the domain D. Hence in this model it is sufficient to consider the dynamics of flow generated by the model (2.1) to (2.6). In addition, the existence, uniqueness, and continuation of results hold for the system.

2.3. Positivity of Solutions

Lemma 2.1

Let the initial data be

$${S(0) > 0, (I(0), R(0), P_1(0), P_2(0), A \ge 0)} \in D$$

Then the solution set $\{S, I, R, P_1, P_2, A\}(t)$ of the system of equations (2.1) to (2.6) is positive for all t > 0

Proof

From the first equation (2.6), we have

$$\frac{dS}{dt} = \Lambda + rR - (\lambda_T + \lambda_H + \mu)S \ge -(\lambda_T + \lambda_H + \mu)S \quad (2.17)$$

$$\frac{dS}{dt} \ge -(\lambda_T + \lambda_H + \mu)S \tag{2.18}$$

Separating the variables and integrating both sides we have

$$\frac{dS}{S} \ge -(\lambda_T + \lambda_H + \mu)dt \tag{2.19}$$

$$InS \ge -(\lambda_T + \lambda_H + \mu)t + c \qquad (2.20)$$

$$S(t) = e^{-(\lambda_T + \lambda_H + \mu)t + C}$$
 (2.21)

$$S(t) \ge Ke^{-(\lambda_T + \lambda_H + \mu)t}$$
 Where $K = e^C$

Using the initial condition $t = 0 \implies S(0) \ge K$

Therefore, $S(t) \ge S(0)e^{-(\lambda_T + \lambda_H + \mu)t} \ge 0$

From equation (3.2)

$$\frac{dI}{dt} = \lambda_T S - (\lambda_H + a + d_T + \mu)I \ge -(\lambda_H + a + d_T + \mu)I \quad (2.22)$$

$$\frac{dI}{dt} \ge -(\lambda_H + a + d_T + \mu)I \tag{2.23}$$

$$\frac{dI}{I} \ge -(\lambda_H + a + d_T + \mu)dt \tag{2.24}$$

$$InI \ge -(\lambda_H + a + d_T + \mu)t + c \tag{2.25}$$

$$I(t) \ge Ke^{-(\lambda_H + a + d_T + \mu)t} \tag{2.26}$$

Applying the initial condition $t = 0 \implies I(0) \ge K$

is

Therefore,

$$I(t) \ge I(0)e^{-(\lambda_H + a + d_T + \mu)t}$$
 (2.27)

Similarly, it can be verified that the rest of the equations are positive for all t > 0 since $e^{\omega} > 0$ for all $\omega \in R$.

2.4. Equilibrium States

2.4.1. The Disease Free Equilibrium State of the Model

The equilibrium state with the absence of infection is known as Disease Free Equilibrium or Zero Equilibrium.

Therefore the disease-free equilibrium state of the model

 $(x_1, x_2, x_3, x_4, x_5, x_6) = \left(\frac{\Lambda}{2}, 0, 0, 0, 0, 0, 0\right)$

2.4.2. The Endemic Equilibrium State of the Model

The equilibrium state of the model is;

$$A = ((\lambda_H + a + d_T + \mu)(\lambda_H + r + \mu)(\lambda_H + \lambda_T + \mu) - ar\lambda_T)$$

$$((b + d + \mu + d_{H1})(\lambda_T + e + d + d_H) - b\lambda_T)$$
Where
$$B = ((\lambda_H + a + d_T + \mu)(\lambda_H + r + \mu)(\lambda_H + \lambda_T + \mu) - ar\lambda_T)$$

$$((b + d + \mu + d_{H1})(\lambda_T + e + d + d_H) - ar\lambda_T)$$

$$C = ((\lambda_H + a + d_T + \mu)(\lambda_H + r + \mu)(\lambda_H + \lambda_T + \mu) - ar\lambda_T)$$

2.5. Basic Reproduction Number R₀

$$F = \begin{bmatrix} \lambda_T S \\ (S+R)\lambda_H + bP_2 \\ \lambda_T P_1 + \lambda_H I \\ eP_1 + dP_2 \end{bmatrix}$$
(2.28)

$$V = \begin{bmatrix} (\lambda_H + a + d_T + \mu)I \\ (\lambda_T + e + d_H + \mu)P_1 \\ (b + d + \mu + d_{H1})P_2 \\ (\mu + dA)A \end{bmatrix}$$
(2.29)

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

$$\begin{pmatrix}
\frac{\beta_{T}c\Lambda}{\mu N(a+d_{T}+\mu)} & 0 & \frac{\beta_{T}c\Lambda}{\mu N(b+d+\mu+d_{H1})} & 0 \\
0 & -\frac{\beta_{H}\delta\Lambda}{\mu N(e+d_{H}+\mu)} & \frac{\beta_{H}\delta\Lambda}{\mu N(b+d+\mu+d_{H1})} + b & 0 \\
0 & 0 & 0 & 0 \\
0 & -\frac{e}{e+d_{H}+\mu} & \frac{d}{b+d+\mu+d_{H1}} & 0
\end{pmatrix}$$
(2.30)

Therefore, the basic reproduction number $R_0 = \rho(FV^{-1})$ = spectra radius of FV^{-1}

$$\Rightarrow R_0 = \frac{\beta_T c\Lambda}{\mu N(a + d_T + \mu)} \tag{2.31}$$

3. Stability Analysis of the Model

3.1. Stability Analysis of Disease Free Equilibrium (DFE)

Jacobean matrix of the system of equations at disease-free equilibrium is

$$J\left(\frac{\Lambda}{\mu},0,0,0,0,0,0\right) = \begin{bmatrix} -\mu & \frac{\beta_{T}c\Lambda}{\mu N} & r & \frac{\beta_{H}\delta\Lambda}{\mu N} & -\left(\frac{\beta_{T}c\Lambda}{\mu N} + \frac{\beta_{H}\delta\Lambda}{\mu N}\right) & 0 \\ 0 & \frac{\beta_{T}c\Lambda}{\mu N} - (a + \mu + d_{T}) & 0 & 0 & \frac{\beta_{T}c\Lambda}{\mu N} & 0 \\ 0 & a & -(r + \mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\frac{\beta_{H}\delta\Lambda}{\mu N} - (e + \mu + d_{H}) & \frac{\Lambda\beta_{H}\delta}{\mu N} + b & 0 \\ 0 & 0 & 0 & 0 & -(b + d + \mu + d_{H1}) & 0 \\ 0 & 0 & 0 & e & d & -(dA + \mu) \end{bmatrix}$$
The characteristic equation is

The characteristic equation is

$$(J-I\lambda) = \begin{bmatrix} -\mu - \lambda & \frac{\beta_T c \Lambda}{\mu N} & r & \frac{\beta_H \delta \Lambda}{\mu N} & -\left(\frac{\beta_T c \Lambda}{\mu N} + \frac{\beta_H \delta \Lambda}{\mu N}\right) & 0 \\ 0 & \frac{\beta_T c \Lambda}{\mu N} - (a + \mu + d_T) - \lambda & 0 & 0 & \frac{\beta_T c \Lambda}{\mu N} & 0 \\ 0 & a & -(r + \mu) - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & -\frac{\beta_H \delta \Lambda}{\mu N} - (e + \mu + d_H) - \lambda & \frac{\Lambda \beta_H \delta}{\mu N} + b & 0 \\ 0 & 0 & 0 & 0 & -(b + d + \mu + d_{H1}) - \lambda & 0 \\ 0 & 0 & 0 & e & d & -(dA + \mu) - \lambda \end{bmatrix}$$

Therefore,

$$\lambda_{1} = -\mu \text{ or } \lambda_{2} = \frac{\beta_{T}c\Lambda}{\mu N} - \left(a + \mu + d_{T}\right) \text{ or } \lambda_{3} = -\left(r + \mu\right)$$
or
$$\lambda_{4} = -\frac{\beta_{T}c\Lambda}{\mu N} - \left(e + \mu + d_{H}\right) \text{ or } \lambda_{5} = -\left(b + d + \mu + d_{H1}\right) \text{ or } \lambda_{6} = -\left(dA + \mu\right)$$
(3.1)

$$\lambda_1, \lambda_3, \lambda_4, \lambda_5, \lambda_6 < 0 \quad \text{and} \quad \lambda_2 < 0 \quad \text{if and only if} \quad \frac{\beta_T c \Lambda}{\mu N} < \left(a + \mu + d_T\right) \quad \text{and} \quad \lambda_2 > 0 \quad \text{if and only if} \quad \frac{\beta_T c \Lambda}{\mu N} > \left(a + \mu + d_T\right)$$

Hence, the DFE is stable if $\frac{\beta_T c \Lambda}{\mu N} < (a + \mu + d_T)$ and otherwise unstable.

3.2. Stability Analysis of Endemic Equilibrium State (EE)

The characteristic equation is obtained from the Jacobi a determinant with the eigenvalues λ

Where

$$\begin{split} \beta_{1} &= \frac{\beta_{T}c(x_{2} + x_{5})}{N} + \frac{\beta_{H}\delta(x_{2} + x_{5})}{N} + \mu \,, \quad \beta_{2} = \frac{\beta_{T}cx_{1}}{N} \,, \quad \beta_{3} = \frac{\beta_{H}\delta x_{1}}{N} \,, \\ \beta_{4} &= \frac{\beta_{T}cx_{1}}{N} + \frac{\beta_{H}\delta x_{1}}{N} \,, \quad \beta_{5} = \frac{\beta_{T}c(x_{2} + x_{5})}{N} \,, \quad \beta_{6} = \frac{\beta_{T}cx_{1}}{N} - (\frac{\beta_{H}\delta(x_{4} + x_{5})}{N} + a + \mu + d_{T}) \,, \\ \beta_{7} &= \frac{\beta_{H}\delta x_{2}}{N} \,, \quad \beta_{8} = \frac{\beta_{T}cx_{1}}{N} - \frac{\beta_{H}\delta x_{2}}{N} \,, \quad \beta_{9} = \frac{\beta_{H}\delta(x_{4} + x_{5})}{N} + r + \mu \,, \quad \beta_{10} = \frac{\beta_{H}\delta x_{3}}{N} \,, \\ \beta_{11} &= \frac{\beta_{H}\delta x_{3}}{N} \,, \quad \beta_{12} = \frac{\beta_{H}\delta(x_{4} + x_{5})}{N} \,, \quad \beta_{13} = \frac{\beta_{T}cx_{4}}{N} \,, \quad \beta_{15} = \frac{\beta_{H}\delta(x_{4} + x_{5})}{N} \,, \\ \beta_{16} &= (x_{1} + x_{2}) \frac{\beta_{H}\delta}{N} - (\frac{\beta_{T}c(x_{2} + x_{5})}{N} + e + \mu + d_{H}) \,, \quad x_{17} = (x_{1} + x_{2}) \frac{\beta_{H}\delta}{N} + b - \frac{\beta_{T}cx_{4}}{N} \,, \\ \beta_{18} &= \frac{\beta_{T}cx_{4}}{N} + \frac{\beta_{H}\delta(x_{4} + x_{5})}{N} \,, \quad \beta_{19} = \frac{\beta_{T}c(x_{2} + x_{5})}{N} + \frac{\beta_{H}\delta x_{2}}{N} \,, \end{split}$$

$$\beta_{20} = \frac{\beta_T c x_4}{N} + \frac{\beta_H \delta x_2}{N} - (b + d + \mu + d_{H1}).$$

Next we shall apply the result of Bellman and Cooke's (1963) to (3.2) to establish the stability or otherwise of the model we have;

$$F'(0) = 0$$
 (3.3)

$$F(0) = -\begin{bmatrix} (dA + \mu)((((-\beta_{11}\beta_{2} - a\beta_{4})\beta_{15} - r(-\beta_{11}\beta_{13} + a\beta_{17}))\beta_{19} \\ -\beta_{18}((\beta_{10}\beta_{4} - \beta_{11}\beta_{3})\beta_{5} - \beta_{12}(\beta_{6}\beta_{3} + \beta_{2}\beta_{7}))\beta_{20} \\ +((-\beta_{8}\beta_{13} - \beta_{17}\beta_{6})\beta_{19} - (\beta_{17}\beta_{7} + \beta_{8}\beta_{16})\beta_{18})\beta_{1} \\ +((\beta_{4}\beta_{13} + \beta_{17}\beta_{2})\beta_{19} - \beta_{18}((\beta_{3}\beta_{17} - \beta_{4}\beta_{16}))\beta_{5} \\ -((\beta_{2}\beta_{8} - \beta_{4}\beta_{6})\beta_{19} - (\beta_{7}\beta_{4} + \beta_{8}\beta_{3})\beta_{18})\beta_{12})\beta_{9} \\ +(\beta_{15}(-\beta_{6}\beta_{10} + a\beta_{7})\beta_{1} + ((\beta_{10}\beta_{2} + a\beta_{3})\beta_{15} \\ +r(a\beta_{16} - \beta_{10}\beta_{13}))\beta_{5} + \beta_{12}r(-\beta_{6}\beta_{10} + a\beta_{7}))\beta_{20} \\ +((\beta_{11}\beta_{6} + a\beta_{8})\beta_{19} + \beta_{18}(\beta_{10}\beta_{8} + \beta_{11}\beta_{13}))\beta_{1}\beta_{15} \\ +((\beta_{11}\beta_{6} + a\beta_{8})\beta_{19} + \beta_{18}(\beta_{10}\beta_{8} + \beta_{11}\beta_{7}))\beta_{12}r \end{bmatrix}$$

$$(3.4)$$

$$G(0) = 0 \tag{3.5}$$

$$G'(0) = \left[-(dA + \mu)(((-\beta_{16} - \beta_6)\beta_1 + \beta_5\beta_2 + \beta_3\beta_{12} + \beta_{16}\beta_6 - a\beta_7\beta_{13})\beta_{20} \right. \\ + (-\beta_{16}\beta_6 + \beta_{19}\beta_{17} + \beta_7\beta_{13} + \beta_{18}\beta_8)\beta_1 + (\beta_{13}\beta_3 - \beta_4\beta_{18} + \beta_{16}\beta_2)\beta_5 \\ + (-\beta_{13}\beta_8 - \beta_4\beta_{12} - \beta_6\beta_{17})\beta_{19} + \beta_{12}\beta_6\beta_3 + \beta_{12}\beta_2\beta_7 - (\beta_{17}\beta_7 + \beta_8\beta_{16})\beta_{18})\beta_9 \\ + ((\beta_{16}\beta_6 + \beta_{10}\beta_{15} - \beta_7\beta_{13})\beta_1 + (-\beta_{16}\beta_2 - \beta_3\beta_{13} - ar)\beta_5 + (-\beta_3\beta_{12} - \beta_{10}\beta_{15})\beta_6 \\ + (\beta_{10}r - \beta_2\beta_7)\beta_{12} + a\beta_7\beta_{15})\beta_{20} + ((-\beta_{17}\beta_6 - \beta_{15}\beta_{11} - \beta_8\beta_{13})\beta_{19} + \beta_6\beta_{10}\beta_{15} \\ + (-\beta_{17}\beta_7 - \beta_8\beta_{16})\beta_{18} - a\beta_7\beta_{15})\beta_1 + ((\beta_4\beta_{13} + \beta_{17}\beta_2)\beta_{19} + \beta_6\beta_{10}\beta_{15} \\ + (-\beta_{17}\beta_7 - \beta_8\beta_{16})\beta_{18} - a\beta_7\beta_{15})\beta_1 + ((\beta_{13}\beta_4 + \beta_{17}\beta_2)\beta_{19} \\ + (\beta_4\beta_{16} - \beta_3\beta_{17} - \beta_{11}r)\beta_{18} + (-\beta_{10}\beta_2 - a\beta_3)\beta_{15} - r(a\beta_{16} - \beta_{10}\beta_{13}))\beta_5 \\ + ((\beta_4\beta_{12} + \beta_{13}\beta_{11})\beta_6 + (-\beta_8\beta_2 - \beta_{11}r)\beta_{12} + a\beta_8\beta_{15})\beta_{19} + \beta_{12}\beta_{16}\beta_{10}r \\ + ((\beta_7\beta_4 + \beta_3\beta_8)\beta_{18} - a\beta_7r)\beta_{12} + \beta_{18}\beta_{15}(\beta_{10}\beta_8 + \beta_{11}\beta_7)) \\ - (((-\beta_{11}\beta_2 - a\beta_4)\beta_{15} - r(-\beta_{11}\beta_{13} + a\beta_{17}))\beta_{19} - \beta_{18}((\beta_{10}\beta_4 - \beta_3\beta_{11})\beta_{15} \\ + r(\beta_{10}\beta_{17} - \beta_{11}\beta_{16})))\beta_5 - (((\beta_6\beta_{16} - \beta_7\beta_{13})\beta_1 + (-\beta_2\beta_{16} - \beta_3\beta_{13})\beta_5 \\ - \beta_{12}(\beta_3\beta_6 + \beta_7\beta_2))\beta_{20} + ((-\beta_{13}\beta_8 - \beta_{17}\beta_6)\beta_{19} - (\beta_7\beta_{17} + \beta_8\beta_{16})\beta_{18})\beta_1 \\ + ((\beta_4\beta_{13} + \beta_{17}\beta_2)\beta_{19} - \beta_{18}(\beta_3\beta_{17} - \beta_4\beta_{16})\beta_5 - ((\beta_2\beta_8 - \beta_4\beta_6)\beta_{19} \\ - (\beta_7\beta_4 + \beta_3\beta_8)\beta_{18})\beta_{12})\beta_9 - (\beta_{15}(-\beta_6\beta_{10} + a\beta_7))\beta_{20} - ((\beta_{11}\beta_6 + a\beta_8)\beta_{19} \\ + \beta_{18}(\beta_{10}\beta_8 + \beta_{11}\beta_7))\beta_{15} - ((\beta_{11}\beta_6 + a\beta_8)\beta_{19} + \beta_{18}(\beta_{10}\beta_8 + \beta_{11}\beta_7))\beta_{12}r \right]$$

Since
$$F'(0) = 0$$
, $G(0) = 0$, $F(0) \neq 0$ and $G'(0) \neq 0$
Hence $F(0)G'(0) - F'(0)G(0) \neq 0$ (3.7)

Therefore, the non –zero equilibrium state is stable.

4. Conclusions

In this study, we developed and analyzed a mathematical model of HIV/TB co infection. The model subdivides the human population into six compartments namely; Susceptible TB individuals S, TB-infectious individuals I, TB-recovered individuals R, HIV-infected individuals P_1 , co-infected individuals P_2 and individuals with full blown AIDS A.

The model solution was found to be positive (establishing the fact that human population cannot be negative) for all time t > 0 provided that the initial data set is positive.

The stability analysis of the Disease Free Equilibrium State (DFE) of the model shows that it will be stable if $\frac{\beta_T c \Lambda}{\mu N} < (a + \mu + d_T)$ that is the population is sustainable.

The analysis of the Endemic Equilibrium State (EES) shows that it is stable. This is in conformity with the real life scenario. That is, whenever HIV is present, the patient may likely be infected with TB if proper and timely care is not given. Also early detection of HIV and TB cases and provision of early treatment can help to control the disease.

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