Co-infection Model of HIV/AIDS-Pneumonia on the Effect of Treatment at Initial and Final Stages

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Abstract: *Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome and pneumonia co-infection is a combination of two infections. Here, an individual contracted with both Human Immunodeficiency Virus and Pneumococcal Carinii Pneumonia at the same time is involved. This paper focuses on the HIV/AIDS - pneumonia co-infection model which is shown to be positively bounded. In this model, we consider treatment for co-infection at both initial and final stages of development. The endemic states are considered to exist when the basic reproduction number for each disease is greater than one. The basic reproduction numbers are also used to see the impact of treating one disease on the co-infection. Numerical simulations indicate the effect of varying the treatment parameters on single disease and the co-infection dynamics. As we increase treatment rates, the infections decrease.*

Keywords: *CD4+ T-cells, HIV, AIDS, Pneumonia, Initial stage, Final stage, Stability, Co-infection, Sensitivity, Reproduction number.*

I. Introduction

1.1 The Epidemiology of HIV/AIDS

Human immunodeficiency virus is a virus which attacks and weakens the body immunity. If untreated, it continues to multiply into the host until it reaches the peak leading into a very serious disease called AIDS, the stage where the symptoms of the disease occur frequently (Mann and Tarantola, 1996). HIV is transmitted through direct contact of blood or other body fluids containing the virus through sexual intercourse, needle-sharing and mother to child during childbirth. HIV/AIDS infections increase faster in developing countries because of poverty and low education on prevention and transmission. Poverty causes people to engage into risk activities such as prostitution resulting into a higher rate of drug injection cases.

The human bodies have the immune system used to protect themselves against infectious diseases. The system involves a group of specialized cells and proteins that fights and eliminates pathogens (disease causing micro-organisms). The immune system has two mechanisms in operation which are; (a) Specific response. This is the immune system involving antibodies (proteins that recognize and destroy pathogens) in defence against infections. (b) Non-specific response. The defence against infections involves the basic system such as intact skin which prevents pathogens to get into blood.

HIV destroys the helper CD4+ T - cells responsible for stimulating other components of the immune response by secreting signals that activate B cells which synthesize and secrete antibodies (Janeway et al., 1997). The immune system can not get rid of HIV infection because of three major reasons; (a) The virus hides inside the cells which makes it difficult for antibodies to detect them. (b) The virus replicates very rapidly which is not proportional to the antibodies production. (c) The virus mutates faster than the production of antibodies to fight the original virus. (Alizon and Magnus, 2012).

Many of the individuals in Sub-Sahara region are unaware of their HIV status due to the symptoms taking long time to show up, leading to the increase in the number of infections in the population as well as lateness in treating the patient (Huang and Crothers, 2009). At the early (acute) stage of infection, the virus multiplies into the host exponentially because it takes one to two weeks for the body to create the antibodies for new intruders. During this stage, the infected individual does not show any symptoms apart from flu-like symptoms and it occurs within the first three to six weeks of infection (Clark et al., 1991). The second stage, virus decreases to a constant number due to the antibodies produced. This is known as the asymptomatic stage or clinical latency which takes about five to ten years. At this stage the virus continues to destroy the immune system by attacking the CD4+ T-cells (Shankarappa et al., 1999). This leads to a great loss of body immunity

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and great increase of the virus leading to the final stage known as AIDS In this stage, the host is easily attacked by the opportunistic disease

1.2 The Epidemiology of Pneumonia

Pneumonia is a deadly but treatable respiratory lung infectious disease caused by bacteria, fungi or virus. Pathogens attack the lungs causing accumulation of pus in the air holes called alveoli. This mucus makes the air passage to blood capillaries to be limited and through this the infected person finds it difficult to breathe. These pathogens are normally present in human throats or nose and attack the lungs when the body immunity is weak. The common bacterial type that causes pneumonia is *streptococcus pneumoniae* which affects the alveoli part (air passage) of the lung. The lungs become filled up with fluid which makes it difficult to absorb and exchange oxygen (Saravolatz et al., 1979). *Pneumocystis jirovecii* (PCP) is the common fungus responsible for pneumonia development in the hosts. The virus that causes pneumonia is known as *influenza A* and *B*, respiratory syncytial virus (RSV) (Lawi et al., 2013). Pneumonia is a density dependent transmission disease; its transmission depends on the population density. It is an airborne disease transmitted mainly through inhaling the air that contains pathogens. This occurs when the infected individual sneezes or coughs spreading the sputum into air. The individual who inhales such air is likely to be infected. Pneumonia pathogens are mostly the colonizing agents where patients infected do not show symptoms. This leads to more transmissions and late treatment (Nicholson et al., 1997).

Bacterial pneumonia can cause death if the patients do not get treatment or do not respond to the treatment. Pneumonia causes lack of oxygen into the blood system causing the damage of some body organs such as kidneys, the heart and the brain. Pneumonia can be treated using some antibiotics such as ampicillin and gentamycine which was common in 2010 to 2011 (WHO). Research shows that developing countries have the highest number of children infected with pneumonia especially those living in rural areas. Indoor pollution caused by cooking and heating with biomass fuels (firewood and dungs), living in crowded homes and parental smoking increase a child's susceptibility to pneumonia (Wardlaw et al., 2006). Globally, pneumonia is the leading cause of deaths for children under 5 years of age and adults aged 65 and older compared to HIV, malaria and measles combined. The infected individual is faced with severe breathing problem which leads to death (Gregory et al., 1979). Pneumonia can be prevented through vaccination. The Clinton Health Access Initiative (CHAI) has increased the access to vaccine and in case vaccine fails, Amoxicillin dispersible tablets (DT) are recommended for treatment.

1.3 Co-infection of HIV/AIDS and Pneumonia

Co-infection is more than one disease co-existing within a single host. HIV/AIDS and Pneumonia are among the diseases that infect a large number of individuals worldwide. This paper explains the rate of co-infection of HIV/AIDS and pneumonia on the effect of treatment. The aim is to see the effects of drugs to a patient with HIV-pneumonia co-infection and to a patient with AIDS-pneumonia cases. People with a weakened immune system such as those with HIV/AIDS are susceptible to diseases such as pneumonia. HIV/AIDS-pneumonia is the co-infection of two diseases responsible for loss of many lives. The individuals with T CD4+ T-cells less than 200 *cells/mm*^{3} are vulnerable to acquire pneumonia. The patient with the co-infection is observed to have some of the symptoms including dry cough, weakness and difficulty in breathing (Polaczek et al., 2014). If the body immune system is strong, pneumonia infection can be fought off. For HIV/AIDS victims, the opportunistic diseases are the ones causing very serious sickness and if not treated they cause death as well (Kalipeni et al., 2004). HIV/AIDS weakens or destroys body immune system giving room for other opportunistic diseases to easily attack the body. Pneumonia caused by the fungus called *pneumocystis jirovecii* is the most common opportunistic disease affecting individuals with HIV. Some of the symptoms of the co-infection include chronic cough, weight loss, breathlessness (dyspnoea), chest discomfort, wheezing, fever and general body weakness (Polaczek et al., 2014). When an individual is co-infected with pneumonia and HIV at acute and clinical latency stages is called the initial stage. The final stage of the co-infection of HIV and pneumonia involves AIDS and pneumonia

II.   Literature Review

Pneumonia is the most opportunistic disease observed in people with HIV/AIDS. It can be cured when treated. If not cured, it is the one common opportunistic disease causing the increased death rates of people with HIV/AIDS. Pneumonia posses a very serious threat to children and adults with a weakened immune system because it causes severe illness (Hirsch et al., 2001). This project develops and analyses the mathematical model of HIV/AIDS and pneumonia co-infection. The mathematical model is used as a tool for better understanding of the co-infection dynamics, studying the approximations, and effects of the parameters and predicting the behaviour of the problem in a specific period of time as well as showing the connectivity of theories and observations using the system of equations with state variables and parameters (Aris, 2012). Parameters are the

constants incorporated into the equations to express the fundamental quantities such as birth rates, transmission rates,recovery rates and death rates (Culshaw and Ruan, 2000).

Mathematical models on the co-infection of HIV/AIDS and pneumonia have been used in modelling the co-infection dynamics (Nthiiri et al., 2015; Onyinge and Ongati). According to Nthiiri(2015) maximum protection against the co-infection was discussed. The maximum protection against HIV/AIDS and the maximum protection against pneumonia was the main concern of their project. The model was formulated with nine compartments. The project found that when protection is high, the number of HIV/AIDS and pneumonia cases decrease. Onyinge and Ongati formulated the mathematical model for HIV/AIDS and pneumonia co-infection with treatment. Their model had a total number of six compartments and it was found that if the basic reproduction number of pneumonia becomes very small approaching zero, there is no new pneumonia infection which reduces the rate of AIDS progression. In this project we modify the model developed by (Onyinge and Ongati) by adding the treatment at the final stage of the co-infection. The co-infection of HIV/AIDS-pneumonia on the effects of treatment at initial and final stages has never been done before. We discuss the effects of treatment for the co-infected patient with HIV/AIDS and pneumonia at initial and final stages. The initial stage is the time of co-infection considering the acute and asymptomatic phases of HIV. We also discuss the effects of treatment on the final stage which occur during AIDS phase (Alizon and Magnus, 2012)

III. Methods: Basic Models Of HIV/AIDS

3.1 Model Formulation.

The model divides the human population into three groups: Susceptible group (S), HIV infected individuals with no clinical symptoms of AIDS (I) and those with AIDS (A). From Figure 3.1, we can understand the full picture of HIV transmission so that the treatment timing as well as preventive measures can be taken into account. The human population is assumed to be uniform and homogeneously mixing. We consider the total population as N(t)=S(t)+I(t)+A(t). It is assumed that individuals are recruited into the susceptible population through ageing and migration at a constant rate *b*. Figure 3.1 shows the HIV/AIDS compartments;
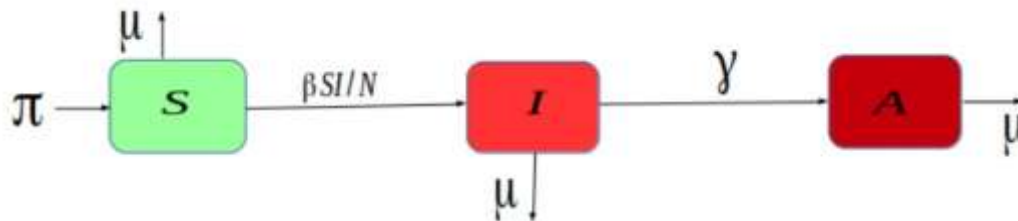

Figure 3.1: Schematic diagram showing HIV/AIDS dynamics

The parameters descriptions are indicated in the table below; Using Figure 3.1, we obtain the following system of deterministic linear differential equations; Table 3.1: Shows the parameters description as used in model formulation

Parameter	Description
μ	Natural death rate
π	Recruitment rate of susceptible individuals to HIV.
γ	Rate of HIV infectives developing into AIDS.
λ	Rate HIV infection of the susceptible individual.
β	Probability of acquiring HIV.

$$\frac{dS}{dt} = \pi - \beta \, \frac{SI}{N} - \mu S.$$

$$\frac{dI}{dt} = \beta \, \frac{SI}{N} - (\mu + \gamma)I.$$

$$\frac{dA}{dt} = \gamma I - \mu A.$$

3.1.2 Model Analysis.

For clear and better understanding of the disease dynamics within the population, the analysis of the model above is used.

3.1.3 Positivity and Boundedness.

Assume that all parameters and state variables are positive for all t≥0 because the model deals with human population. This model is studied in the feasible region Ω, where $\{S(t), I(t), A(t)\} \in \Omega \subset R_+^3$.

To show that the solutions are bounded in the set Ω, we take the time derivative of *N*.

Let N(t) be the population present at the time t≥ 0. Thus N(t)=S(t)+I(t)+A(t)

$$\frac{dN}{dt} = \pi - \beta \ \frac{SI}{N} - \mu S + \beta \ \frac{SI}{N} - (\mu + \gamma)I + \gamma I - \mu A,$$
$$= \pi - \mu S - \mu I - \mu A,$$
$$= \pi - \mu(S + I + A),$$
$$\frac{dN}{dt} \leq \pi - \mu N.$$

Upon integration it leads to,

$$\int_0^\infty \frac{dN}{\pi - \mu N} \leq \int_0^\infty dt,$$
$$-\frac{1}{\mu}ln(\pi - \mu N) \leq t,$$
$$ln(\pi - \mu N) \geq -\mu t,$$
$$\pi - \mu N \geq e^{-\mu t},$$
$$\pi - e^{-\mu t} \geq \mu N,$$
$$\frac{\pi}{\mu} - \frac{1}{\mu}e^{-\mu t} \geq N.$$

As $t \to \infty$ we get; $0 \leq N \leq \frac{\pi}{\mu}$ which shows that all solutions of the system are positively bounded.

3.1.4 Equilibrium Points.

Here we work under the assumption that there is no infection in the population: Thus, $(I^0, A^0) = (0,0)$. Now,

$$\pi - \frac{\beta \ S^0 I^0}{N} - \mu S^0 = 0,$$
$$\pi - \mu S^0 = 0,$$
$$S^0 = \frac{\pi}{\mu}.$$

DFE point obtained is $E^0 = (\frac{\pi}{\mu}, 0, 0)$.

3.1.5 Basic Reproduction Number.

We find the basic reproduction number (R_0) as the number of secondary infections expected to be produced by a single primary infection in a population of susceptible class. Using the next generation method: We consider first the infective classes.

I and *A* thus *m* = 2.

Re-arranging the system starting with the infection classes;

$$\frac{dI}{dt} = \beta \ \frac{SI}{N} - (\mu + \gamma)I.$$
$$\frac{dA}{dt} = \gamma I - \mu A.$$
$$\frac{dS}{dt} = \pi - \beta \ \frac{SI}{N} - \mu S.$$

New infections are represented as matrix F:

$$\mathcal{F} = \begin{pmatrix} \beta \ \frac{SI}{N} \\ 0 \end{pmatrix}.$$

The rates of transfer of individuals from one compartment to another is indicated by matrix V:

$$\mathcal{V} = \begin{pmatrix} (\mu + \gamma)I \\ \mu A - \gamma I \end{pmatrix}.$$

Jacobian matrices for F at DFE is $JF_i E^0 = F_i$

$$F = \begin{pmatrix} \frac{\partial F_1}{\partial I} & \frac{\partial F_1}{\partial A} \\ \frac{\partial F_2}{\partial I} & \frac{\partial F_2}{\partial A} \end{pmatrix}$$

$$= \begin{pmatrix} \frac{\beta\ \pi}{\mu N} & 0 \\ 0 & 0 \end{pmatrix}.$$

For V at DFE is $JV_i E^0 = V_i$.

$$V = \begin{pmatrix} \frac{\partial V_1}{\partial I} & \frac{\partial V_1}{\partial A} \\ \frac{\partial V_2}{\partial I} & \frac{\partial V_2}{\partial A} \end{pmatrix}$$

$$= \begin{pmatrix} \mu + \gamma & 0 \\ -\gamma & \mu \end{pmatrix}.$$

We obtain the inverse using Sage;

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu+\gamma} & 0 \\ \frac{\gamma}{(\gamma+\mu)\mu} & \frac{1}{\mu} \end{pmatrix},$$

The product of FV^{-1} resulting to:

$$= \begin{pmatrix} \frac{\beta\ \pi}{N(\mu+\gamma)\mu} & 0 \\ 0 & 0 \end{pmatrix}.$$

The Eigenvalues of the product are $\left[\frac{\beta}{(\gamma\mu+\mu^2)N},\ 0 \right].$

The maximum absolute value of eigenvalues is the basic Reproduction ratio given by $R_0 = \frac{\beta}{(\gamma\mu+\mu^2)N}$.

3.1.6 Endemic Equilibrium Point.
Let
$$E^* = (S^*, I^*, A^*).$$
At Equilibrium
$$\frac{dS}{dt} = 0, \frac{dI}{dt} = 0, \frac{dA}{dt} = 0,$$
$$\pi - \frac{\beta\ S^*I^*}{N} - \mu S^* = 0$$
$$\frac{\beta\ S^*I^*}{N} - \mu I^* - \gamma I^* = 0, \qquad , (3.1.1)$$
$$\gamma I^* - \mu A^* = 0. \qquad (3.1.2)$$
$$I^* \left(\beta \frac{S^*}{N} - (\mu + \gamma) \right) = 0$$
$$I^* = 0 \text{ or } \left(\beta \frac{S^*}{N} - (\mu + \gamma) \right) = 0 \qquad . (3.1.3)$$
substituting Equation (3.1.3) in Equation (3.1.1), we get
$$S^* = \frac{N}{\beta}(\mu + \gamma)$$
$$(\mu + \gamma)I^* = \pi - \frac{N}{\beta} - \frac{N}{\beta}\mu(\mu + \gamma)$$
$$I^* = \frac{\pi}{\mu + \gamma} - \frac{\mu N}{\beta},$$
$$I^* = \frac{\beta\ \pi - \mu N(\mu + \gamma)}{\beta\ (\mu + \gamma)}.$$

From Equation (3.1.2),

$$A^* = \frac{\gamma}{\beta}\left(\beta\ \frac{\pi - \mu N(\mu + \gamma)}{\mu(\mu + \gamma)}\right).$$

The Endemic Equilibrium Point (EEP) is represented as

$$E^* = \left(\frac{N}{\beta}(\mu + \gamma), \beta\ \frac{\pi - \mu N(\mu + \gamma)\beta}{\beta\ (\mu + \gamma)}, \frac{\pi - \mu N(\mu + \gamma)}{\mu(\mu + \gamma)}\right)$$

.

3.1.7 Local Stability Analysis.

Expressing the Jacobian matrix through the derivatives of each equation with respect to each compartment value:

$$J(S, I, A) = \begin{pmatrix} \frac{\partial J_1}{\partial S} & \frac{\partial J_1}{\partial I} & \frac{\partial J_1}{\partial A} \\ \frac{\partial J_2}{\partial S} & \frac{\partial J_2}{\partial I} & \frac{\partial J_2}{\partial A} \\ \frac{\partial J_3}{\partial S} & \frac{\partial J_3}{\partial I} & \frac{\partial J_3}{\partial A} \end{pmatrix},$$

$$= \begin{pmatrix} \beta\frac{I}{N} - \mu & \beta\frac{S}{N} & 0 \\ \beta\frac{I}{N} & \beta\frac{S}{N} - (\mu + \gamma) & 0 \\ 0 & \gamma & -\mu \end{pmatrix}.$$

At disease-free,

$$J(S, I, A)E^0 = \begin{pmatrix} -\mu & \beta\frac{\pi}{\mu N} & 0 \\ 0 & \beta\frac{\pi}{\mu N} - (\mu + \gamma) & 0 \\ 0 & \gamma & -\mu \end{pmatrix}.$$

From Sage, the eigenvalues obtained are;

$$[\lambda_1, \lambda_2, \lambda_3] = \left[-\frac{(\gamma\mu + \mu^2)N - \beta}{\mu N}, -\mu, -\mu\right].$$

The Endemic equilibrium point stability we have:

$$\begin{pmatrix} \beta\frac{\pi - \mu N(\mu + \gamma)}{N(\mu + \gamma)} - \mu & \mu + \gamma & 0 \\ \beta\frac{\pi - \mu N(\mu + \gamma)}{N(\mu + \gamma)} & 0 & 0 \\ 0 & \gamma & -\mu \end{pmatrix}.$$

Using Sage, the eigenvalues obtained are,

$$\lambda_1 = -\frac{2(\gamma\mu + \mu^2)N - \beta\ + \sqrt{\pi\beta^2 - 4(\gamma^3\mu + 2\gamma^2\mu^2 + \gamma\mu^3)N^2 + 4(\beta\ \gamma^2 + \beta\ \gamma\mu)N}}{2N(\gamma + \mu)},$$

$$\lambda_2 = -\frac{2(\gamma\mu + \mu^2)N - \beta\ - \sqrt{\pi\beta^2 - 4(\gamma^3\mu + 2\gamma^2\mu^2 + \gamma\mu^3)N^2 + 4(\beta\ \gamma^2 + \beta\ \gamma\mu)N}}{2N(\gamma + \mu)},$$

$$\lambda_3 = -\mu.$$

Since all eigenvalues are negative, we have the locally asymptotically stable point

Figure 3.2 shows the dynamics of HIV/AIDS in human population using R program.

From Figure 3.1 we see that Initially as the number of susceptible population increases the HIV and AIDS are constant. After about five years, the number of HIV infections increases and reaches the peak after twenty years. After ten years, the individuals with AIDS increase. As the disease prevails in the population, the number of susceptible individuals decreases and becomes constant with infections after twenty years.
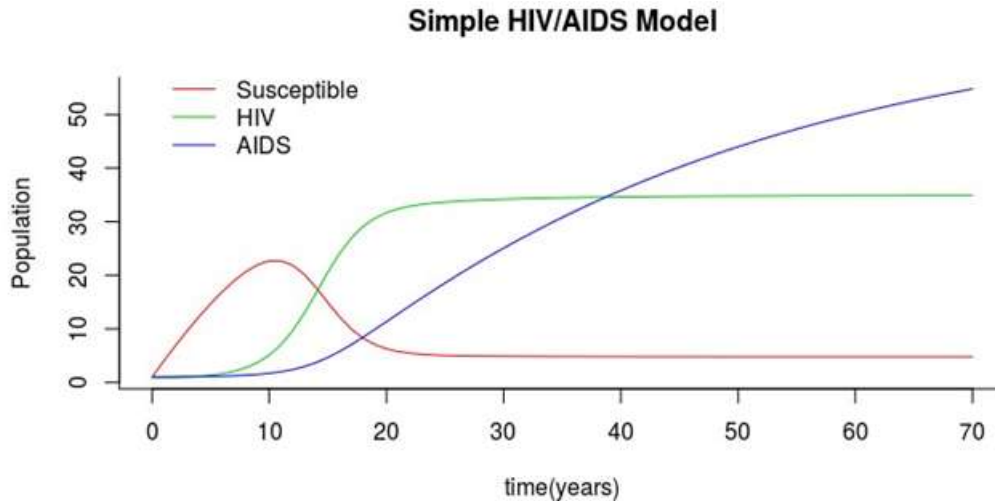
Simple HIV/AIDS Model

Figure 3.2: HIV/AIDS dynamics

A. 3.2 Basic Model of Pneumonia

Here we formulate the SIR model for pneumonia. We divide the population into Susceptible individuals (S), Infective individuals (I) and Recovered individuals (R). Recovered individuals are those who are being removed from the population, or have gained immunity.

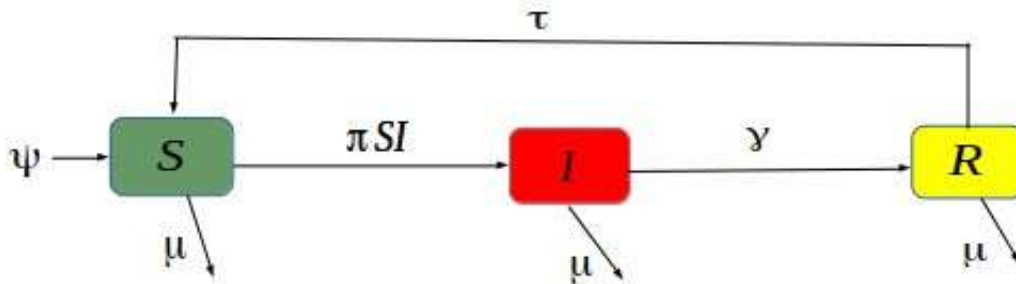Below is the schematic diagram for pneumonia compartments;

Figure 3.3: Schematic diagram showing simple pneumonia dynamics model

Table 3.2 indicates parameters in the pneumonia model;

Table 3.2: Parameters Description

Parameter	Description
ψ	Recruitment rate of susceptible individuals to pneumonia.
μ	Natural death rate.
γ	Rate at which an infected individual receives treatments.
τ	Recovery rate due to natural immunity.
π	Probability of acquiring pneumonia

The following is the system of ordinary differential equations represented by the schematic model above.

$$\frac{dS}{dt} = \psi - (\pi I + \mu)S + \tau R,$$

$$\frac{dI}{dt} = \pi SI - (\mu + \gamma)I,$$

$$\frac{dR}{dt} = \gamma I - (\mu + \tau)R.$$

3.2.1 Mathematical Analysis.
3.2.2 Positivity and Boundedness.

From the model we assume that all parameters are greater than zero. We need to see if the solutions of the system are bounded and positive with $t > 0$. Now, let $N(t)$ represent the population. Thus, $N(t) = S(t) + I(t) + R(t)$ also $dNdt = dSdt + dIdt + dRdt$.

$$\frac{dN}{dt} = \psi - \pi SI - \mu S + \tau R + \pi SI - \mu I - \gamma I + \gamma I - \mu R - \tau R,$$

$$\frac{dN}{dt} = \psi - \mu S - \mu I - \mu R,$$

$$= \psi - \mu(S + I + R),$$

$$= \psi - \mu N,$$

$$\geq N.$$

$$\frac{dN}{dt} \leq \psi - \mu N,$$

$$\frac{dN}{\psi - \mu N} \leq dt,$$

Integrating on both sides:

$$\int \frac{dN}{\psi - \mu N} \leq \int dt,$$

$$-\frac{1}{\mu}ln(\psi - \mu N) \leq t + c,$$

$$ln(\psi - \mu N) \leq -\mu t + c,$$

$$\psi - \mu N \leq N_0 e^{-\mu t},$$

$$N \leq e^{-\mu t},$$

Thus; $\quad 0 \leq N \leq e^{-\mu t}.$

This is proof for boundedness and positivity.

3.2.3 Disease - free Equilibrium Point.

We obtain the disease free equilibrium when there are no disease infections in the population. This means that $I = 0$. Thus $DFE = (S^0, I^0, R^0)$.

$$\psi - \mu S^0 + \tau R^0 = 0,$$

$$I^0 = 0, \quad \gamma(0) - (\mu + \tau)R^0 = 0$$

$$R^0 = 0,$$

$$S^0 = \frac{\psi}{\mu},$$

DFE obtained is $E^0 = (\frac{\psi}{\mu}, 0, 0)$.

3.2.4 Basic Reproduction Number for Disease - free Equilibrium.

From Next generation method, New infections from susceptible class is given by matrix F: $F = [\pi SI]$ and $V = [(\mu + \gamma)I]$.

$$F = \left[\frac{\partial F}{\partial t}\right]$$

$$V = \left[\frac{\partial V}{\partial t}\right].$$

$$F = [\pi S^0],$$

$$= \left[\frac{\pi \psi}{\mu}\right],$$

$$V = [\mu + \gamma],$$
$$V^{-1} = \left[\frac{1}{(\mu + \gamma)} \right].$$

$$FV^{-1} = \left[\frac{\pi\psi}{\mu} \right] \left[\frac{1}{(\mu + \gamma)} \right]$$
$$= \left[\frac{\pi\psi}{(\mu + \gamma)} \right] \quad ,$$

The Absolute value of the eigenvalue is itself, thus; $= \frac{\pi\psi}{(\mu + \gamma)}$.

The basic reproduction number is defined as the average number of infections that occur when one infective individual is introduced into a completely susceptible population. For our system $R_0 = \frac{\pi\psi}{(\mu + \gamma)}$ is the basic reproduction number.

3.2.5 Endemic Equilibrium Point.

For the equilibrium point we have $\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} =$ and $E^* = (S^*, I^*, R^*)$. From the system of deterministic differential equations,

$$\psi - \pi S^* I^* - \mu S^* + \tau R^* = 0, \ \pi S^* I^* - \mu I^* - \gamma I^* = 0, \ \gamma I^* - (\mu + \tau) R^*.$$

$$(\pi S^* - (\mu + \gamma)) I^* = 0,$$
$$\pi S^* - (\mu + \gamma) = 0$$
$$S^* = \frac{\mu + \gamma}{\pi}.$$

$I^* \ne 0$, and

Also $R^* = \frac{\gamma I^*}{\mu + \tau}$. Substituting S^* and R^* into first equation we get

$$\psi - \pi I^* \left[\frac{\mu + \gamma}{\pi} \right] - \mu \left[\frac{\mu + \gamma}{\pi} \right] + \tau \left[\frac{\gamma I^*}{\mu + \tau} \right] = 0,$$
$$\psi - \mu \left[\frac{\mu + \gamma}{\pi} \right] = \left(\pi \left[\frac{\mu + \gamma}{\pi} \right] - \left[\frac{\gamma\tau}{\mu + \tau} \right] \right) I^*$$
$$\frac{\pi\psi - \mu(\mu + \gamma)}{\pi} = \frac{((\mu + \gamma)(\mu + \tau) - \gamma\tau)}{\mu + \tau} I^*,$$
$$I^* = \frac{(\mu + \tau)(\pi\psi - \mu(\mu + \gamma))}{\pi((\mu + \gamma)(\mu + \tau) - \gamma\tau)}.$$

For R^* we get;

$$R^* = \frac{\gamma}{\pi} \left(\frac{\pi\psi - \mu(\mu + \gamma)}{(\mu + \gamma)(\mu + \tau) - \gamma\tau} \right).$$

3.2.6 Local Stability at Disease - free Equilibrium.

Finding the Jacobian matrix at DFE we get;

$$JE^0 = \begin{pmatrix} \frac{\partial F_1}{\partial S^0} & \frac{\partial F_1}{\partial I^0} & \frac{\partial F_1}{\partial R^0} \\ \frac{\partial F_2}{\partial S^0} & \frac{\partial F_2}{\partial I^0} & \frac{\partial F_2}{\partial R^0} \\ \frac{\partial F_3}{\partial S^0} & \frac{\partial F_3}{\partial R^0} & \frac{\partial F_3}{\partial R^0} \end{pmatrix},$$

$$JE^0 = \begin{pmatrix} -\mu & \frac{\psi\pi}{\mu} & \tau \\ 0 & \frac{\psi\pi}{\mu} - (\mu + \gamma) & 0 \\ 0 & \gamma & -(\mu + \tau) \end{pmatrix},$$

B. *Section 3.3. Co-infection Model of HIV/AIDS and Pneumonia*

From Sage, the eigenvalues are

$$\left[-\frac{\gamma\mu + \mu^2 - \pi\psi}{\mu}, -\mu - \tau, -\mu \right]$$

$$= \left[-\frac{\gamma\mu + \mu^2 - \pi\psi}{\mu}, -\mu - \tau, -\mu \right],$$

$$= \left[(\gamma + \mu)\left(\frac{\pi\psi}{(\mu\gamma + \mu^2)} - 1\right), -\mu - \tau, -\mu \right],$$

$$= [(\gamma + \mu)(R_0 - 1), -\mu - \tau, -\mu].$$

It is observed that all the eigenvalues are negative. This is possible when $R_0 < 1$. This implies that the disease free equilibrium point for pneumonia is locally asymptotically stable.

3.3 Co-infection Model of HIV/AIDS and Pneumonia

The total human population N is subdivided into seven compartments. The whole population is susceptible to both HIV/AIDS and Pneumonia denoted as (S) with the recruitment rate b. It is assumed that individuals enter the susceptible compartment through birth at a rate b and become infected with HIV (I_H) at the rate λ_H. After five to ten years, the infected class progress to AIDS I_A. The susceptible individual is infected with pneumonia I_P at the rate λ_P. T class contains individuals on treatment of pneumonia, HIV and the co-infections. An individual in the class I_H is infected with pneumonia, and an individual in the class I_P infected with HIV at the rates of infections ψ_1 and v respectively, progressing to the class I_{HP} which is a class of individuals with HIV and Pneumonia co-infection. An individual with AIDS can acquire pneumonia infections at the rate ψ_2 and proceeds into the class with co-infection of AIDS and Pneumonia (I_{AP}). All individuals are subject to a natural death at the rate μ

Table 3.3 indicates the summary of state variables;

The flow diagram in Figure 3.4 is used to express the diseases dynamics which assisted in obtaining our model.

State Variable	Description
S	Number of susceptible human individuals.
IH	Number of individuals infected with HIV.
IP	Number of individuals infected with pneumonia.
IA	Number of AIDS patients.
IHP	Individuals with both HIV and pneumonia infections.
IAP	Individuals with both AIDS and pneumonia.
T	Number of individuals on treatment.

Table 3.3: State Variables of the Co-infection Model

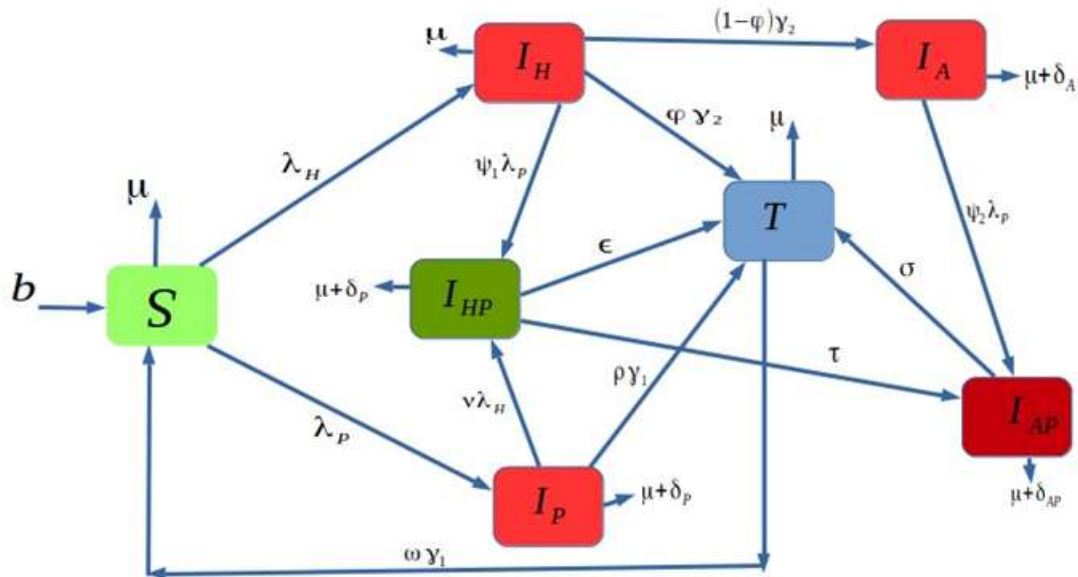Section 3.3. Co-infection Model of HIV/AIDS and Pneumonia

Figure 3.4: Flow diagram including HIV/AIDS and pneumonia co-infection

Parameter	Description
μ	Natural death rate
b	Recruitment rate of susceptible individuals to both HIV and Pneumonia
δA	AIDS induced mortality
δP	Pneumonia induced mortality
γ_1	Treatment rate for pneumonia infected individuals.
γ_2	Treatment rate for HIV infected individuals.
λ_p	Rate of pneumonia new-infection
λH	Rate of HIV new-infection
ψ_1	Modification parameter accounting that individuals with HIV are more susceptible to pneumonia.
ψ_2	Modification parameter accounting that individuals with AIDS are mostly susceptible to pneumonia.
ν	Modification parameter accounting that individuals with pneumonia are susceptible to HIV. Probability of acquiring HIV.
β	Probability of acquiring pneumonia.
π	The proportion of HIV infected individuals receiving treatment.
ϕ	Treatment rate for HIV-pneumonia co-infection.
ϵ	Progression rate.
τ	Proportion of pneumonia patients recovered after treatment.
ω	Contact rate with HIV infective.
c	Contact rate with pneumonia infective.
θ	Proportion of pneumonia infecteds receiving treatment.
ρ	Treatment rate for AIDS-pneumonia co-infection.
σ	

Table 3.4: Description of Parameters

3.3.1 The Model Assumptions.
(i) The population is not constant.
(ii) Individuals enter the Susceptible class through birth and recovered from pneumonia. There is no vertical transmission.
(iii) Recovered individuals from pneumonia do not acquire immunity.
(iv) No natural recovery from pneumonia infection.
(v) Not all HIV and AIDS infected individuals receive treatment.
(vi) Individuals who return to susceptible are those recovering from pneumonia after treatment.

Now we obtain the following system of deterministic ordinary differential equations;

$$\frac{dS}{dt} = b + \omega\gamma_1 T - (\mu + \lambda_H + \lambda_P)S,$$

$$\frac{dI_H}{dt} = \lambda_H S - (\mu + \psi_1\lambda_P + \gamma_2)I_H,$$

$$\frac{dI_P}{dt} = \lambda_P S - (\mu + \delta_P + \nu\lambda_H + \rho\gamma_1)I_P,$$

$$\frac{dI_A}{dt} = (1 - \phi)\gamma_2 I_H - (\mu + \delta_A + \psi_2\lambda_P)I_A,$$

$$\frac{dI_{HP}}{dt} = \psi_1\lambda_P I_H + \nu\lambda_H I_P - (\mu + \delta_P + \epsilon + \tau)I_{HP},$$

$$\frac{dI_{AP}}{dt} = \psi_2\lambda_P I_A + \tau I_{HP} - (\mu + \delta_{AP} + \sigma)I_{AP},$$

$$\frac{dT}{dt} = \phi\gamma_2 I_H + \rho\gamma_1 I_P + \epsilon I_{HP} + \sigma I_{AP} - (\mu + \omega\gamma_1)T. \qquad (3.3.1)$$

Let forces of infection be;

$$\lambda_P = \pi\theta I_P,$$

where, θ is the contact rate of pneumonia infectious individuals.

$$\lambda_H = \beta c I_H,$$

where, c is the HIV infectious individuals contact rate.

3.4 Mathematical Model Analysis
3.4.1 Positivity and Boundedness.
Here we assume that all parameters are positive because they represent the population of human beings. Then the model lies in R^7 where $S > 0$, $I_H \geq 0$, $I_P \geq 0$, $I_A \geq 0$, $I_{HP} \geq 0$, $I_{AP} \geq 0$ and $T \geq 0$. Since the initial values of the model are positive, we have to prove that the solutions are all positive and bounded.

3.4.2 Theorem. *The solutions of the system in R^7 for $t \geq 0$ are positively bounded if the initial values are positive.*
Proof. Let $N(t)$ represent the whole population at time t;
Thus,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI_H}{dt} + \frac{dI_P}{dt} + \frac{dI_A}{dt} + \frac{dI_{HP}}{dt} + \frac{dI_{AP}}{dt} + \frac{dT}{dt}.$$

$$\frac{dN}{dt} = b - \mu S - \lambda_P S - \lambda_H S + \omega\gamma_1 T + \lambda_H S - \mu I_H - \psi_1\lambda_P I_H - \gamma_2 I_H + \lambda_P S - \mu I_P - \delta_P I_P$$

$$-\nu\lambda_H I_P - \rho\gamma_1 I_P + (1 - \phi)\gamma_2 I_H - \mu I_A - \delta_A I_A - \psi_2\lambda_P I_A$$

$$+\psi_1\lambda_P I_H + \nu\lambda_H I_P - \mu I_{HP} - \delta_P I_{HP} - \epsilon I_{HP} - \tau I_{HP} + \psi_2\lambda_P I_A$$

$$+\tau I_{HP} - \mu I_{AP} - \delta_{AP} I_{AP} - \sigma I_{AP} + \rho\gamma_1 I_P + \epsilon I_{HP} + \phi\gamma_2 I_H + \sigma I_{AP} - \mu T - \delta_{AP} T - \omega\gamma_1 T,$$

$$= b - \mu(S + IH + IP + IA + IHP + IAP + T) - \delta PIP - \delta AIA - \delta PIHP - \delta APIAP, \leq b - \mu N.$$

Integrating on both sides;

$$\int \frac{dN}{b - \mu N} \leq \int dt,$$

$$-\frac{1}{\mu}ln(b - \mu N) \leq t,$$

$$ln(b - \mu N) \geq -\mu t,$$

$$b - \mu N \geq e^{-\mu t}$$

$$\frac{b - e^{-\mu t}}{\mu} \geq N.$$

As $t \to \infty$,

$$\frac{b}{\mu} \geq N$$

,
Thus

$$0 \leq N \leq \frac{b}{\mu},$$

Hence, all the solutions of the system are positive and bounded. $\square$

3.4.3 Equilibrium Points.

Equilibrium point is the specific point of the solutions of the model where the state variables satisfy the model. In this project we have to deal with two types of equilibrium points. Disease - free equilibrium point (DFE) denoted by $E^0 = (S^0, I_H{}^0, I_P{}^0, I_A{}^0, I_{HP}{}^0, I_{AP}{}^0, T^0)$ and endemic equilibrium point (EE) denoted by $E* = (S*, IH*, IP*, IA*, IHP*, IAP*, T*)$.

3.4.4 Disease - free Equilibrium Point.

At DFE point it is assumed that there is no disease in the population; ie $I_H = I_P = I_A = I_{HP} = IAP = 0$. From our model the DFE point is represented as ($S0, IH0, IP0, IA0, IHP0, IAP0, T0$).

Through solving the system and substituting the state variables we get,

$$b - (\mu + \lambda_H + \lambda_P)S^0 + \omega\gamma_1 T^0 = 0$$

$$\lambda_H S^0 = 0,$$

$$\lambda_P S^0 = 0,$$

$$(\mu + \omega\gamma_1)T^0 = 0,$$

$$b - \mu S^0 = 0,$$

$$S^0 = \frac{b}{\mu}.$$

Therefore, the disease free equilibrium point is given as $E^0 = (\frac{b}{\mu}, 0, 0, 0, 0, 0, 0)$.

3.4.5 Basic Reproduction Number.

The basic reproductive ratio is the average number of infections caused by a single infectious individual in a susceptible population (Heffernan et al., 2005).

For this project we get two reproductive ratios for the two diseases. This number is used by epidemiologists in judging whether the disease dies out or prevails (endemic). When $R_0 < 1$ means that each individual produces on average less than one new infection. Hence the disease dies out. When $R_0 > 1$ shows that the disease is endemic. The case where $R_0 = 1$ means that that there is one infectious who can infect one individual in a population.

In this project we use next generation to calculate the basic reproduction number.

Re-arranging the system model starting with the infection class, we have;

$$\frac{dI_H}{dt} = \lambda_H S - (\mu + \psi_1 \lambda_P + \gamma_2)I_H,$$

$$\frac{dI_P}{dt} = \lambda_P S - (\mu + \delta_P + \nu \lambda_H + \rho\gamma_1)I_P,$$

$$\frac{dI_A}{dt} = (1 - \phi)\gamma_2 I_H - (\mu + \delta_A + \psi_2 \lambda_P)I_A,$$

$$\frac{dI_{HP}}{dt} = \psi_1 \lambda_P I_H + \nu \lambda_H I_P - (\mu + \delta_P + \epsilon + \tau)I_{HP},$$

$$\frac{dI_{AP}}{dt} = \psi_2 \lambda_P I_A + \tau I_{HP} - (\mu + \delta_{AP} + \sigma)I_{AP},$$

$$\frac{dS}{dt} = b + \omega\gamma_1 T - (\mu + \lambda_H + \lambda_P)S,$$

$$\frac{dT}{dt} = \phi\gamma_2 I_H + \rho\gamma_1 I_P + \epsilon I_{HP} + \sigma I_{AP} - (\mu + \omega\gamma_1)T. \qquad (3.4.1)$$

From the model we have five classes of infection ($I_H, I_P, I_A, I_{HP}, I_{AP}$).

But only four classes with new infections are (I_H, I_P, I_{HP}, I_{AP}).

$$\mathcal{F} = \begin{pmatrix} \beta \ c\frac{b}{\mu}I_H \\ \pi\theta\frac{b}{\mu}I_P \\ 0 \\ \psi_1\pi\theta I_P I_H + \beta \ cI_H I_P \\ \psi_2\pi\theta I_P I_A \end{pmatrix},$$

$$V = \begin{pmatrix} (\mu + \psi_1\pi\theta I_P + \gamma_2)I_H \\ (\mu + \delta_P + \beta \ cI_H + \rho\gamma_1)I_P \\ (\mu + \delta_A + \psi_2\pi\theta I_P)I_A - (1 - \phi)\gamma_2 I_H \\ (\mu + \delta_P + \epsilon + \tau)I_{HP} \\ (\mu + \delta_{AP} + \sigma)I_{AP} - \tau I_{HP} \end{pmatrix}.$$

The Jacobian matrices are;

$$JF_i E^0 = F = \begin{pmatrix} \frac{\partial F_1}{dI_H} & \frac{\partial F_1}{dI_P} & \frac{\partial F_1}{dI_A} & \frac{\partial F_1}{dI_{HP}} \\ \frac{\partial F_2}{dI_H} & \frac{\partial F_2}{dI_P} & \frac{\partial F_2}{dI_A} & \frac{\partial F_2}{dI_{HP}} \\ \frac{\partial F_3}{dI_H} & \frac{\partial F_3}{dI_P} & \frac{\partial F_3}{dI_A} & \frac{\partial F_3}{dI_{HP}} \\ \frac{\partial F_4}{dI_H} & \frac{\partial F_4}{dI_P} & \frac{\partial F_4}{dI_A} & \frac{\partial F_4}{dI_{HP}} \\ \frac{\partial F_5}{dI_H} & \frac{\partial F_5}{dI_P} & \frac{\partial F_5}{dI_A} & \frac{\partial F_5}{dI_{HP}} \end{pmatrix}$$

$$V = \begin{pmatrix} \beta \ \frac{cb}{\mu} \\ 0 & \frac{\pi\theta b}{\mu} \\ \\ \\ \end{pmatrix}$$

$$\begin{pmatrix} \frac{\partial F_1}{dI_{AP}} \\ \frac{\partial F_2}{dI_{AP}} \\ \frac{\partial F_3}{dI_{AP}} \\ \frac{\partial F_4}{dI_{AP}} \\ \frac{\partial F_5}{dI_{AP}} \end{pmatrix},$$

$$\begin{pmatrix} & 0 & 0 & 0 & 0 \\ & & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}.$$

For *V*

$$JV_iE^0 = V = \begin{pmatrix} \frac{\partial V_1}{\partial I_H} & \frac{\partial V_1}{\partial I_P} & \frac{\partial V_1}{\partial I_A} & \frac{\partial V_1}{\partial I_{HP}} & \frac{\partial V_1}{\partial I_{AP}} \\ \frac{\partial V_2}{\partial I_H} & \frac{\partial V_2}{\partial I_P} & \frac{\partial V_2}{\partial I_A} & \frac{\partial V_2}{\partial I_{HP}} & \frac{\partial V_2}{\partial I_{AP}} \\ \frac{\partial V_3}{\partial I_H} & \frac{\partial V_3}{\partial I_P} & \frac{\partial V_3}{\partial I_A} & \frac{\partial V_3}{\partial I_{HP}} & \frac{\partial V_3}{\partial I_{AP}} \\ \frac{\partial V_4}{\partial I_H} & \frac{\partial V_4}{\partial I_P} & \frac{\partial V_4}{\partial I_A} & \frac{\partial V_4}{\partial I_{HP}} & \frac{\partial V_4}{\partial I_{AP}} \\ \frac{\partial V_5}{\partial I_H} & \frac{\partial V_5}{\partial I_P} & \frac{\partial V_5}{\partial I_A} & \frac{\partial V_5}{\partial I_{HP}} & \frac{\partial V_5}{\partial I_{AP}} \end{pmatrix},$$

$$V = \begin{pmatrix} \gamma_2 + \mu & 0 & 0 & 0 & 0 \\ 0 & \delta_P + \rho\gamma_1 + \mu & 0 & 0 & 0 \\ \gamma_2\phi - \gamma_2 & 0 & \delta_A + \mu & 0 & 0 \\ 0 & 0 & 0 & \tau + \delta_P + \epsilon + \mu & 0 \\ 0 & 0 & 0 & -\tau & \delta_{AP} + \sigma + \mu \end{pmatrix},$$

From sage math program we obtain V^{-1}

$$V^{-1} = \begin{pmatrix} \frac{1}{\gamma_2+\mu} & 0 & 0 & 0 & 0 \\ 0 & \frac{1}{\gamma_1\rho+\delta_P+\mu} & 0 & 0 & 0 \\ -\frac{\gamma_2\phi-\gamma_2}{(\delta_A+\mu)(\gamma_2+\mu)} & 0 & \frac{1}{\delta_A+\mu} & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{\delta_P+\epsilon+\mu+\tau} & 0 \\ 0 & 0 & 0 & \frac{\tau}{(\delta_{AP}+\mu+\sigma)(\delta_P+\epsilon+\mu+\tau)} & \frac{1}{\delta_{AP}+\mu+\sigma} \end{pmatrix},$$

$$FV^{-1} = \begin{pmatrix} \frac{\beta\ c}{(\gamma_2+\mu)\mu} & 0 & 0 & 0 & 0 \\ 0 & \frac{b\pi\theta}{(\gamma_1\rho+\delta_P+\mu)\mu} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}.$$

The eigenvalues are

$$\left[\frac{b\pi\theta}{\gamma_1\mu\rho + \delta_P\mu + \mu^2}, \frac{\beta\ c}{\gamma_2\mu + \mu^2}, 0, 0, 0 \right].$$

Now, there are two reproduction numbers at disease free equilibrium, representing the two diseases. Where R_H represents the expected number of HIV/AIDS infections produced by one infectious individual. While R_P represents the average pneumonia infections produced by one infectious individual as given below,.

$$R_P = \frac{b\pi\theta}{\gamma_1\mu\rho + \delta_P\mu + \mu^2},$$

for pneumonia and

$$R_H = \frac{\beta\ c}{\gamma_2\mu + \mu^2},$$

for HIV.
Since the method used to find them is the same, we can compare the obtained basic reproduction numbers by finding the maximum basic reproduction number among the two. From sage program we obtain the basic reproduction number which is the maximum eigenvalue of the model to be, $R_P =$

$$\frac{b\pi\theta}{\gamma_1\mu\rho+\delta_P\mu+\mu^2}.$$

For the co-infection treatment, the interpretation of the above results can be used as follows in epidemiological sense. Since $R_P < 1$ implies that pneumonia infections die out. Since no pneumonia infections, the immunity of the body increases leading to the reduction of HIV progress into AIDS. So the treatment of pneumonia is highly recommended for the individuals living with both pneumonia and HIV.

3.4.6 Impact of HIV on Pneumonia Dynamics.
Since the reproduction number for the co-infection is indicated in terms of two reproduction numbers with respect to the two diseases, we have to analyse the co-infection by considering the impact of one disease on the other.

To see the impact of HIV in pneumonia dynamics we express R_P in terms of R_H. We express the parameter independent of both diseases as the subject. In this project we make μ the subject and after few manipulations we obtain the Equations (3.4.2) and (3.4.4).

$$R_P = \left[\frac{4R_H^2 b\pi\theta}{(\sqrt{(R_H\gamma_2)^2 + 4R_H \beta \, c} - R_H\gamma_2)(2R_H\rho\gamma_1 + 2R_H\delta_P + \sqrt{(R_H\gamma_2)^2 + 4R_H \beta \, c} - R_H\gamma_2)} \right].$$

(3.4.2)

Let,

$$x = \sqrt{(R_H\gamma_2)^2 + 4R_H \beta \, c},$$
$$y = 2R_H\rho\gamma_1 + 2R_H\delta_P - R_H\gamma_2$$
$$z = 2\rho\gamma_1 + 2\delta_P - \gamma_2.$$

,

Obtaining the partial derivative of R_P with respect to R_H we get

$$\frac{\partial R_P}{\partial R_H} = \left[\frac{4R_H^2 b\pi\theta(xz + 2\beta \, c + R_H\gamma_2)}{x(y+x)^2(R_H\gamma_2 - x)} + \frac{4R_H^2 b\pi\theta(x\gamma_2 - 2\beta \, c + R_H\gamma_2^2)}{x(y+x)(R_H\gamma_2 - x)^2} - \frac{8R_H b\pi\theta}{(y+x)(R_H\gamma_2 - x)} \right].$$

(3.4.3)

If Equation (3.4.3) is positive, implies that an increase in HIV cases results in an increase of pneumonia infections in the community, when Equation (3.4.3) equals to zero means that HIV has no impact on pneumonia infection and when Equation (3.4.3) is negative implies that HIV has a negative impact on pneumonia infections (Okosun and Smith, 2017).

To see the impact of pneumonia in HIV dynamics we express R_H in terms of R_P. We obtain the following expression.

Let,

$$x_1 = \sqrt{(R_P(\rho\gamma_1 + \delta_P))^2 + 4R_P b\pi\theta},$$
$$y_1 = 2R_P(\rho\gamma_1 + \delta_P) - 2R_P\gamma_2,$$
$$z_1 = \rho\gamma_1 + \delta_P - 2\gamma_2.$$

$$R_H = \left[\frac{4R_P^2 \beta \, c}{(x - R_P(\rho\gamma_1 + \delta_P))(2R_P\gamma_2 + x - R_P(\rho\gamma_1 + \delta_P))} \right].$$

(3.4.4)

Obtaining the partial derivative of R_H with respect to R_P we get

$$\frac{\partial R_H}{\partial R_P} = \left[\frac{4R_P^2 \beta \, c(R_P(\rho\gamma_1 + \delta_P)^2 - x_1 z_1 + 2b\pi\theta)}{x_1(y_1 - x_1)^2(R_P(\rho\gamma_1 + \delta_P) - x_1)} - \frac{4R_P^2 \beta \, c(x_1(\rho\gamma_1 + \delta_P) - R_P(\rho\gamma_1 + \delta_P)^2 + 2b\pi\theta)}{x_1(y_1 - x_1)(R_P(\rho\gamma_1 + \delta_P) - x_1)^2} \right.$$
$$\left. + \frac{8R_P \beta \, c}{(y_1 - x_1)(R_P(\rho\gamma_1) - x_1)} \right].$$

(3.4.5)

Whenever Equation (3.4.5) is positive, it shows that an increase in pneumonia cases in the co-infected group results in an increase of HIV cases in the community. If Equation (3.4.5) equals to zero, means that pneumonia has no impact on HIV. When Equation (3.4.5) is negative, means that the increase of pneumonia leads to decrease in HIV.

3.4.7 Impact of Pneumonia Treatment on HIV/AIDS-Pneumonia Co-infection.
Differentiating partially Equation (3.4.4) with respect to the parameter representing the rate at which pneumonia patients receive treatment. So we have,

$$\frac{\partial R_H}{\partial \gamma_1} = \left[\frac{4R_P^2 \beta \, c(R_P^2\rho(\rho\gamma_1 + \delta_P) - R_P\rho x_1)}{x_1(y_1 - x_1)(R_P(\rho\gamma_1 + \delta_P) - x_1)^2} + \frac{4R_P^2 \beta \, c(R_P^2\rho(\rho\gamma_1 + \delta_P) - R_P\rho x_1)}{x_1(y_1 - x_1)^2(R_P(\rho\gamma_1 + \delta_P) - x_1)} \right]$$

(3.4.6)

If Equation (3.4.6) is positive, then treatment of pneumonia will have a negative impact on the dynamics of pneumonia and HIV co-infection (Okosun and Smith, 2017). If Equation (3.4.6) is equal to zero, then treatment of pneumonia has no impact on HIV. When Equation (3.4.6) is negative, implies that the treatment of pneumonia will have a positive impact on the dynamics of HIV and pneumonia co-infection.

From the above results, we can summarise the analysis in the following lemma.

3.4.8 Lemma. Treatment of pneumonia only in the co-infection model, will have.

- A positive impact on the pneumonia and HIV co-infection if $\frac{\partial R_H}{\partial \gamma_1} < 0$.

- No impact on the pneumonia and HIV co-infection if $\frac{\partial R_H}{\partial \gamma_1} = 0$.

- A negative impact on the pneumonia and HIV co-infection if $\frac{\partial R_H}{\partial \gamma_1} > 0$.

Note: The same procedures can be followed when checking for the impact of HIV treatment on pneumonia.

3.4.9 Local Stability of Disease - free Equilibrium.

R_0 is also used in explaining the stability of the equilibrium points.

Using linearisation in showing that the disease free equilibrium point is asymptotically stable. We obtain the Jacobian matrix through differentiating each equation with respect to each state variable. For simplicity, let

$$z_1 = (\mu + \lambda_H + \lambda_P),$$
$$z_2 = (\mu + \psi_1 \lambda_P + \gamma_2),$$
$$z_3 = (\mu + \delta_P + \nu \lambda_H + \rho \gamma_1)$$
$$z_4 = (\mu + \delta_A + \psi_2 \lambda_P),$$
$$z_5 = (\mu + \epsilon + \delta_P + \tau),$$
$$z_6 = (\mu + \delta_{AP} + \sigma).$$

At disease free equilibrium point we obtain the following Jacobian matrix;

$$J(E) = \begin{pmatrix} -z_1 & 0 & 0 & 0 & 0 & 0 & \omega\gamma_1 \\ \lambda_H & -z_2 & 0 & 0 & 0 & 0 & 0 \\ \lambda_P & 0 & z_3 & 0 & 0 & 0 & 0 \\ 0 & (1-\phi)\gamma_2 & 0 & -z_4 & 0 & 0 & 0 \\ 0 & \psi_1\lambda_P & \nu\lambda_H & 0 & -z_5 & 0 & 0 \\ 0 & 0 & 0 & \psi_2\lambda_P & \tau & -z_6 & 0 \\ 0 & \phi\gamma_2 & \rho\gamma_1 & 0 & \epsilon & \sigma & -(\mu+\omega\gamma_1) \end{pmatrix}.$$

At disease free equilibrium point we obtain,

$$\begin{pmatrix} -\mu & -\frac{\beta\ c}{\mu} & -\frac{b\pi\theta}{\mu} & 0 & 0 & 0 & \omega\gamma_1 \\ 0 & \frac{\beta\ c}{\mu}-\gamma_2-\mu & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{b\pi\theta}{\mu}-\delta_P-\rho\gamma_1-\mu & 0 & 0 & 0 & 0 \\ 0 & -\gamma_2(\phi-1) & 0 & -\delta_A-\mu & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\epsilon-\delta_P-\tau-\mu & 0 & 0 \\ 0 & 0 & 0 & 0 & \tau & -\delta_{AP}-\sigma-\mu & 0 \\ 0 & \phi\gamma_2 & \rho\gamma_1 & 0 & \epsilon & \sigma & -\omega\gamma_1-\mu \end{pmatrix}$$

.

Using Sage, the following are the eigenvalues obtained;

$$\left(-\frac{\gamma_1\mu\rho - b\pi\theta + \delta_P\mu + \mu^2}{\mu}, \frac{\beta\ c - \gamma_2\mu - \mu^2}{\mu}, -\delta_P-\epsilon-\mu-\tau, -\delta_{AP}-\mu-\sigma, -\gamma_1\omega-\mu, -\delta_A-\mu, -\mu\right)$$

.

$$= [-((\rho\gamma_1+\delta_P)+\mu)(-R_P+1), -(\gamma_2+\mu)(-R_H+1), -\delta_P-\epsilon-\mu-\tau, -\delta_{AP}-\mu-\sigma, -\gamma_1\omega-\mu, -\delta_A]$$

.

3.4.10 Theorem. *The disease free equilibrium point in R^7 is said to be locally asymptotically stable if $R_0 < 1$ and asymptotically unstable if $R_0 > 1$.*

Proof. We know that from definition, when the disease free equilibrium is locally asymptotically stable, then it must contain all real negative eigenvalues. For the eigenvalues $-(\gamma_2 + \mu)(-R_H + 1)$ and $-((\rho\gamma_1+\delta_P)+\mu)(-R_P+1))$ to be negative; R_H and R_P should be less than one respectively. $R_H < 1$ and $R_P < 1$. Hence proved Eigenvalues are negatives indicating that the equilibrium point is locally asymptotically stable.

□

3.4.11 Endemic Equilibrium Point.

The endemic equilibrium point (*EEP*) is obtained through the assumption that $\frac{dS}{dt} = \frac{dI_H}{dt} = \frac{dI_P}{dt} = \frac{dI_A}{dt} = \frac{dI_{HP}}{dt} = \frac{dI_{AP}}{dt} = \frac{dT}{dt} = 0$.

$$b - (\mu + \lambda_H + \lambda_P)S + \omega\gamma_1 T = 0,$$
$$\lambda_H S - (\mu + \psi_1\lambda_P + \gamma_2)I_H = 0,$$
$$\lambda_P S - (\mu + \delta_P + \nu\lambda_H + \rho\gamma_1)I_P = 0,$$
$$(1 - \phi)\gamma_2 I_H - (\mu + \delta_A + \psi_2\lambda_P)I_A = 0$$
$$\psi_1\lambda_P I_H + \nu\lambda_H I_P - (\mu + \delta_P + \epsilon + \tau)I_{HP} = 0,$$
$$\psi_2\lambda_P I_A + \tau I_{HP} - (\mu + \delta_{AP} + \sigma)I_{AP} = 0,$$
$$\phi\gamma_2 I_H + \rho\gamma_1 I_P + \epsilon I_{HP} + \sigma I_{AP} - (\mu + \omega\gamma_1)T = 0,$$

Now $(S^*, I_H^*, I_P^*, I_A^*, I_{HP}^*, I_{AP}^*, R_P^*)$ is the endemic equilibrium point.
For simplicity, let $(b+\omega\gamma_1 T^*) = a$, $(\mu+\lambda_H+\lambda_P) = B$, $(\mu+\psi_1\lambda_P+\gamma_2) = c$, $(\mu+\delta_P+\nu\lambda_H+\rho\gamma_1) = $
$(1-\phi)\gamma_2 = e$, $(\mu+\delta_A+\psi_2\lambda_P) = f$, $(\mu+\delta_P+\epsilon+\tau) = g$, $(\mu+\delta_A+\psi_2\lambda_P) = h$.

$$S^* = \frac{a}{B},$$
$$I_H^* = \frac{a\lambda_H}{Bc},$$
$$I_P^* = \frac{a\lambda_P}{Bd},$$
$$I_A^* = \frac{ae\lambda_H}{Bcf},$$
$$I_{HP}^* = \frac{ad\psi_1\lambda_P\lambda_H + ac\nu\lambda_P\lambda_H}{Bcdg},$$
$$I_{AP}^* = \frac{aedg\psi_2\lambda_P\lambda_H + ad\tau\psi_1\lambda_P\lambda_H + ac\nu\lambda_P\lambda_H}{Bcdfhg},$$
$$T^* = \frac{a-b}{\omega\gamma_1}.$$

After substituting the variables we see that the endemic equilibrium point is very long and complicated. We have therefore decided to use numerical simulation of the co-infection dynamics considering when $R_0 < 1$ and when $R_0 > 1$.
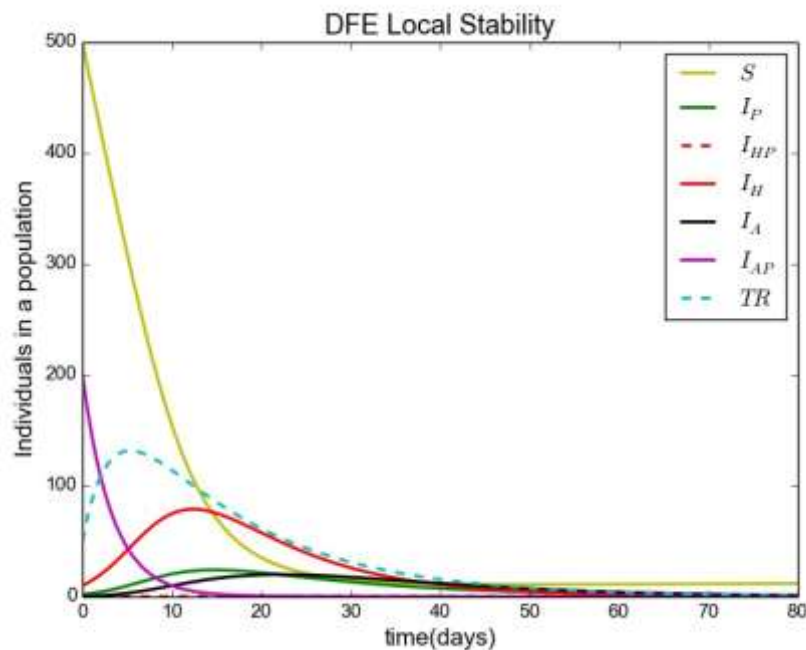

Figure 3.5: Graphical representation of a disease free equilibrium point stability

In Figure 3.5 we observe that all the solutions converge towards the equilibrium point. This was obtained when $R_P = 0.145$ and $R_H = 0.143$. At disease free equilibrium point, all infection solutions converge to zero while the susceptible population decreases and then remains constant. The susceptible population remain constant because HIV can not be cured. The virus can be suppressed to progress to AIDS stage by using ART. The figure was plotted using python program under consideration of the basic reproduction numbers being less than a unit. This indicates that the disease free equilibrium point is locally asymptotically stable.

Figure 3.6 was plotted using the values $R_P = 1.193$ and $R_H = 1.914$ which shows the stability of the endemic equilibrium point.

We observe that in the long run the convergence of the solutions is observed at the values greater than 400 days. The plot shows that the endemic equilibrium point is locally asymptotically stable.
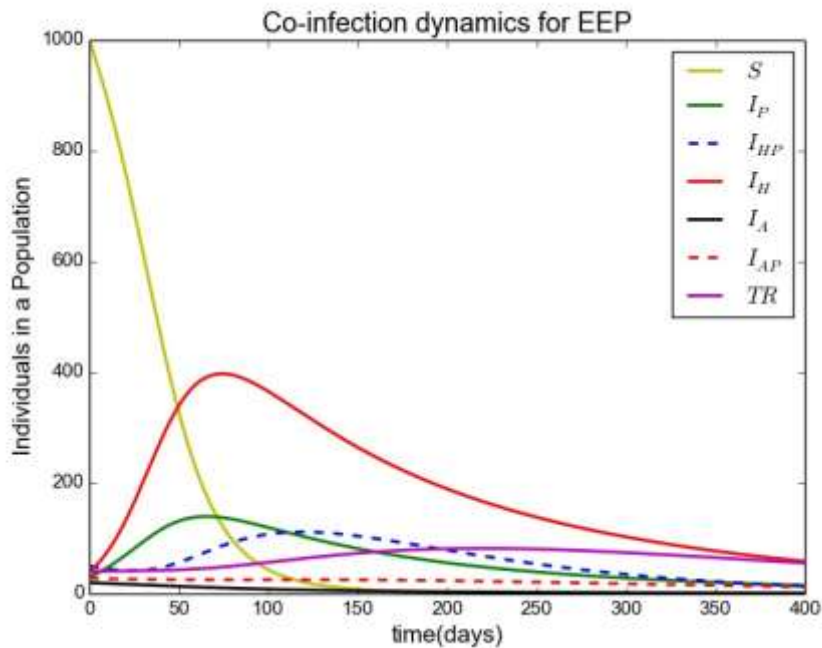

Figure 3.6: Local stability of the endemic equilibrium point.

3.5 Sensitivity Analysis
3.5.1 Sensitivity Analysis of a Single Disease.
We perform sensitivity analysis to determine the influence of parameters to the state variables. This method is used to adjust the key parameters basing on the results needed. We consider the most influential parameter to the R_0.

Let α_i be the parameter R_0 depends on and $H_{\alpha_i}^{R_0}$ be the sensitive index of α_i, then; $H_{\alpha_i}^{R_0} = \frac{\partial R_0}{\partial \alpha_i} \times \frac{\alpha_i}{R_0}\Big|_{\alpha_i value}$.

Table 3.5 shows the $H_{\alpha_i}^{R_H}$ where i stands for each parameter in R_H. The absolute value of the index varies directly to its impact on R_H. This table is obtained in consideration to $R_H > 1$ that when the diseases prevail in the population. In this project we consider $R_H = \frac{\beta c}{\gamma_2 \mu + \mu^2} = 26.31$

we get the indices as shown in Table 3.5 . The natural death parameter has the highest impact on the basic reproduction number of HIV by having the largest absolute value number (1.189). HIV treatment rate parameter is the least sensitive with magnitude of 0.8108 meaning that it has least impact on the HIV reproductive number.

Table 3.5: Sensitivity analysis for R_H

Parameter	Descriptions	Index$_H$
μ	Natural death rate	-1.1892
b	Recruitment rate	1
β	Probability of acquiring HIV	1
c	Contact rate with HIV infective	1
γ_2	HIV treatment rate	-0.8108

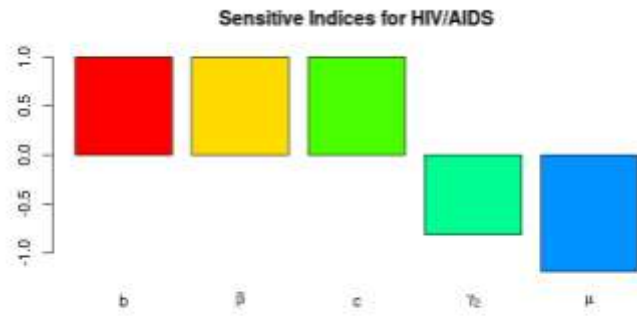The results displayed using Table 3.5 are plotted on Figure 3.7.

Figure 3.7: A bar chart to express the sensitivity indices.

Table 3.6 indicates the indices of sensitive parameters of the pneumonia reproduction number arranged from the most to the least sensitive parameter.

Table 3.6: Sensitivity analysis for R_P

Parameter		Descriptions	Index$_P$
μ		Natural death rate	-1.168
b		Recruitment rate	1
π		Probability of acquiring pneumonia	1
θ		Contact rate with pneumonia infective	1
	δP	Death due to pneumonia	-0.820
γ_1		Pneumonia treatment rate	-0.011
ρ		Proportion of pneumonia infected receiving treatments	-0.011

From Table 3.5 and Table 3.6, we see the sensitivity indices of each parameter on R_H and R_P respectively. The impact of the parameter on R_0 is determined by the magnitude of the index. Positive or negative sign of an index shows the positive impact of α_i on R_0.
Table 3.6 results can be expressed in a bar graph (Figure 3.8) for more clarification of the parameters explaining the pneumonia dynamics.
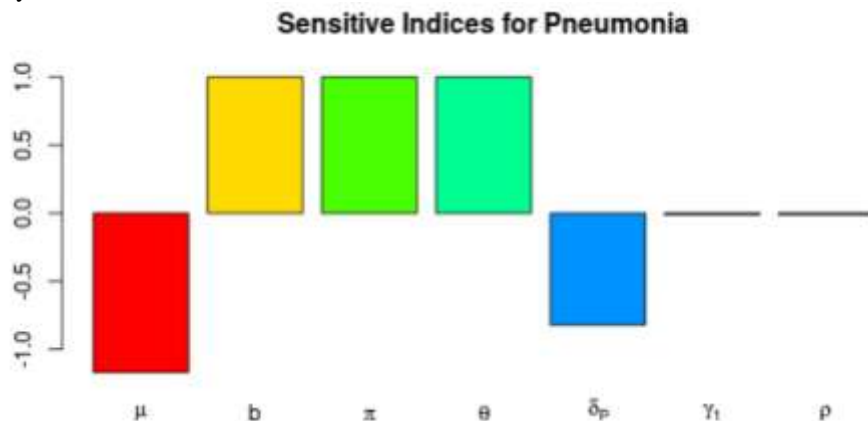

Figure 3.8: Sensitivity indices of the parameters associated in the basic reproduction number of pneumonia

3.5.2 Sensitivity Index when R_H is in Terms of R_P.

We find the sensitivity indices of R_H in each of the nine different parameters. We evaluate the sensitivity indices of these parameters using parameter values as obtained from literature reviews. Here we consider the sensitivity indices when $R_H < 1$ and when $R_H > 1$. We use the sensitivity index formula
Equation 3.5.1;

$$H_{\alpha_i}^{R_H} = \frac{\partial R_H}{\partial \alpha_i} \times \frac{\alpha_i}{R_H}\Big|_{\alpha_i value}. \quad (3.5.1)$$

Table 3.7: Shows sensitivity analysis of HIV basic reproduction number expressed in terms of R_P

Parameter	Description			Sensitivity index $R_P < 1$	Sensitivity index if-index if $R_P > 1$
θ	contact rate with pneumonia infective			-1.01898	-1.0031743
	Probability of acquiring HIV			1.0000	1.0000
c	probability of acquiring pneumonia	-1.01898	-1.0031743	1.0000	1.0000
δP	pneumonia induced deaths			0.359516	0.967606
γ_2	rate of HIV treatment			-0.326418	-0.974348
γ_1	rate of pneumonia treatments			0.004864	0.013091
ρ	Proportion of individuals with pneumonia receiving treatment			0.004864	0.013091
b	Recruitment rate			-0.01898	-0.0031743

The Table 3.7 shows the parameters, arranged from the most sensitive to the least. For $R_P < 1$ and $R_P > 1$, the most sensitive parameters are at the top of the index columns. The indices range between -1.5 and 1.5. The parameters with highest impact on R_0 are π, θ, β and c with indices of -1.0189 and 1. We observe that increasing (or decreasing) these rates by 10% increases (or reduces) R_H by $10\% \alpha_i$. The increase (or decrease) of HIV treatment rate by 10% decreases (or increases) R_H by 3.2%.

3.5.3 Sensitivity Index when R_P is in Terms of R_H.
Similarly, we derive the sensitivity of R_P indicated in Table 3.8. The sensitivity indices of R_P is obtained through the use of the given formula Equation 3.5.2.

$$H_{\alpha_i}^{R_P} = \frac{\partial R_P}{\partial \alpha_i} \times \frac{\alpha_i}{R_P}\Big|_{\alpha_i value}. \quad (3.5.2)$$

Table 3.8: Shows

Parameter	Description			Sensitivity index if $R_H < 1$	Sensitivity index if $R_H > 1$
θ	contact rate with pneumonia infective			1.0000	1.0000
	Probability of acquiring HIV			-0.9917756	1.0000
c	probability of acquiring pneumonia	1.0000	1.0000	-0.99177568	1.0000
δP	pneumonia induced deaths			-0.133968	-0.82178273
γ_2	rate of HIV treatment			0.1193327	0.7988916
b	Recruitment rate			-0.01898	0.01700465
γ_1	rate of pneumonia treatments			-0.0018125	-0.0111182
ρ	Proportion of individuals with pneumonia receiving treatment			-0.00181251906	-0.0111182

The sensitivity analysis of pneumonia basic reproduction number expressed in terms of R_H

Table 3.8 shows the parameters, arranged from the most sensitive to lowest sensitive. For $R_H < 1$ and $R_H > 1$, the most sensitive parameters are at the top of the index columns. For $R_H > 1$ the indices range between -1.5 and 1.5. The parameters with highest impact on π, θ, β and c with indices of 1. We observe that increasing (or decreasing) these rates by 10% increases (or reduces) R_H by $10\%\alpha_i$. The increase (or decrease) of HIV treatment rate by 10% decreases (or increases) R_H by 10%.

4.1 Effects of Treatment

Table 4.1. Parameter estimates of the model obtained from different literature

Parameter	Description	Units	Value	Source
b	Recruitment rate	Per day	0.0087	(Nthiiri et al., 2015)
γ_1	Rate of treatment of pneumonia infections	per day	0.02	Assumed
γ_2	Rate of progress of HIV infected to treatment		0.03	(Mukandavire and Garira, 2006)
λ_p	Rate of Pneumonia new infection	per day	0.025	(Onyinge and Ongati)
λH	Rate of HIV new infection	per day	0.054	Assumed
ψ_1	Rate of pneumonia infection to HIV patient	per	0.055	(Onyinge and Ongati)
ψ_2	Rate of pneumonia infection to AIDS patient	0.18	per day	Assumed
v	Rate of HIV infection to pneumonia patient	per day	0.009	(Onyinge and Ongati)
	Probability of acquiring HIV	per day	0.11	Assumed
	Probability of acquiring pneumonia	per day	0.012	Assumed
	Contact rate with HIV infective	per day	0.08	(Nthiiri et al., 2015)
θ	Contact rate with Pneumonia infective	per day	0.08	Nthiiri et al. (2015)
ρ	Proportion of pneumonia treatments	per day	0.023	(Onyinge and Ongati)
σ	Rate of treatment of AIDS-Pneumonia individuals	per day	0.0023	Assumed
φ	Proportion of HIV treatments	per day	0.0011	Assumed
	Rate of treatments of HIV-Pneumonia infected	per day	0.2	(Bhunu et al., 2009)
ω	Pneumonia recovery rate	per day	0.0021	Assumed
μ	Natural death rate	per day	0.02	(Mukandavire and Garira, 2006)
δP	Pneumonia induced death rate	per day	0.034	(Nthiiri et al., 2015)
δA	induced AIDS death rate	per day	0.0023	Assumed
$\delta_a P$	AIDS and pneumonia induced death rate	per day	0.004	Assumed

Table 4.1:

Numerical simulations to show the effects of treatment on the infected populations. We show how variations of treatment rates affect individuals infected with HIV, pneumonia, HIVpneumonia co-infection and AIDS-pneumonia co-infection. We use Python program and the assumed parameter values from table Table 4.1. Parameter estimates of the model obtained from different literature. The parameters given in this table are estimates of some of the parameters obtained from the literature in Table 3.4

4.2 Effects of Treatment on the Infected Population

Antiretroviral therapy (ART) is used to suppress the HIV virus and stop the progression to AIDS stage. WHO recommends the immediate use of ART soon after diagnosis especially at the early stage in order to prevent the onward transmission (Wardlaw et al., 2006). For pneumonia, antibiotics are used to treat bacterial pneumonia. The cough medicines are used to calm the coughs. Some medicines to reduce fever are applied to individuals infected with pneumonia.

In this subsection we report the effect of treatment when applied to 10% of the population. The observation shows how treatment affect the human populations with HIV only or pneumonia only, or with the combination of HIV and pneumonia which in this project we term as initial stage of co-infection, or the final stage of co-infection which consists of AIDS and pneumonia.

This is done in order to address the results found to the medical practitioners and public healthy institutions on how to treat and control the co-infection of HIV/AIDS and pneumonia.

β

π

c

4.3 Parameters Simulations

Considering the Figure 4.1, Figure 4.2, Figure 4.3 and Figure 4.4 we see that; Figure 4.1 presents the graph of variation of treatment rate on human population infected with HIV against time. We see that as the treatment rates increases the number of HIV infections decreases. This means that ART reduces the viral load in the HIV-positive individuals and lowers the risk of HIV transmission to HIV-negative individuals.
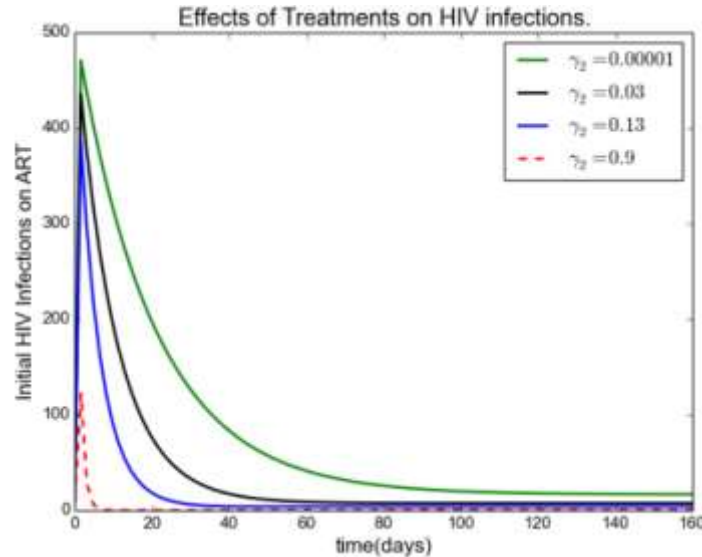

Figure 4.1: The population of HIV infected individuals response to treatment.

From Figure 4.2 we see the decrease of pneumonia cases after each increase of treatment rate. The duration of pneumonia therapy takes a minimum of five days for the non-drug resistant individual to be cured. We vary treatment rates because the initial therapy may not be effective against the identified pathogens depending on the body immune. So it may take more time for pneumonia to be cured.
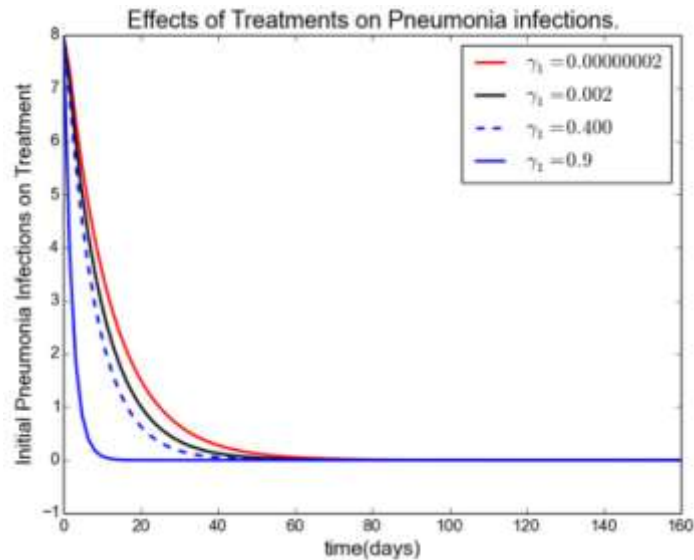

Figure 4.2: Variation of pneumonia treatment rates effects on a number of individuals infected with pneumonia only

Considering Figure 4.3 which is the initial stage of the co-infection we see that initially the infections increases with treatments. After five to seven days the number of infections decreases. This is when both ART and pneumonia antibiotics are applied simultaneously. This shows that increasing treatment rate, the number of individuals progress for HIV and pneumonia co-infection decrease.
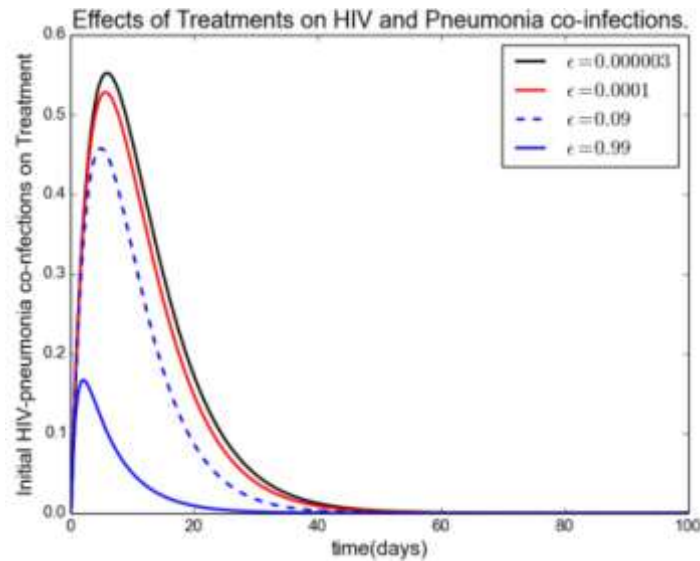
Figure 4.3: Effect of varying treatment rate on individuals with HIV and Pneumonia co-infection. Here it shows when both diseases are treated together simultaneously

It can be interpreted that, people recover as treatment rate increases hence decrease of the number of infected individuals. The decrease of the curves being close and the big slope indicates that when combined treatment is used on the co-infection, becomes more effective than treating one disease independently.

At the final stage of co-infection represented by Figure 4.4 we see that after ten days the number of AIDS-pneumonia co-infections becomes very low when treatment rate is very high. Since the interval of ten days is the same interval for pneumonia to be cured, it leads to the co-infection status to disappear in the population. This shows that as we increase treatment rate for the co-infection, the number of co-infected patients decrease. The slopes of the curves in Figure 4.4 are smaller compared to those in Figure 4.3 indicating that patients respond to the variation of treatment rates at the co-infection of AIDS and pneumonia compared to that of HIV and pneumonia. It is advised to treat pneumonia first at this stage then after the individual has improved the body immunity, ART follows.
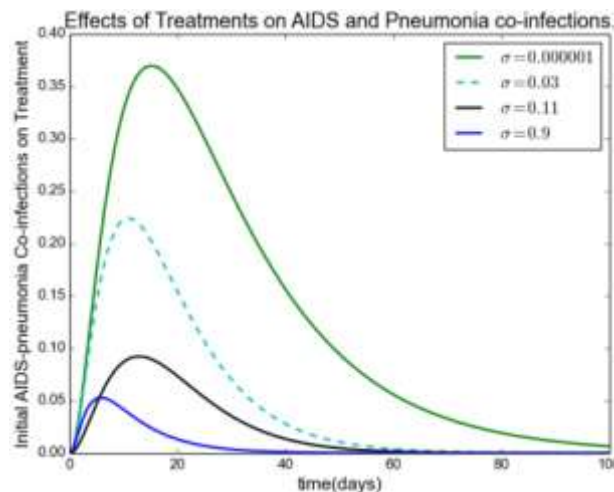

Figure 4.4: The effect of varying treatment rate on individuals with AIDS and Pneumonia co-infection.

IV. Discussion

This project aims at developing the mathematical co-infection model of HIV/AIDS and pneumonia. It's foundation based on extending the model in (Onyinge and Ongati) by including the compartment of AIDS and pneumonia co-infection on treatment.

We used the next generation matrix method to calculate the reproduction number. We obtain two reproduction numbers for the co-infection where R_P and R_H represents basic reproduction numbers for pneumonia and HIV respectively. We observed that when $R_0 < 1$ the disease free equilibrium point is locally asymptotically stable. When $R_0 > 1$ the disease is at endemic point, then it is advised to start treatment to the infected people and emphasise the control strategies because the disease persists. We plotted Figure 3.5 using

python program from the parameters giving $R_P = 0.145$ and $R_H = 0.143$ which is the stability of disease free equilibrium. The plot maintains the same shape even after twice the initial values. Figure 3.6 also was plotted from the parameters contained in $R_P = 26.31$ and $R_H = 1.91$ which indicates that the disease prevail in the population as time increases. This is the locally stable endemic equilibrium point because the solutions converge towards the same point as time increases.

Sensitivity analysis was done to determine the most influential parameter. Each parameter of the two basic reproduction numbers were computed using Sage program. From Table 3.6 we see that the most sensitive parameters for $R_P < 1$ are probability of acquiring pneumonia, contact with pneumonia infective, probability of acquiring HIV and HIV contact rate ($-1.0189, -1.089, 1$ and 1 respectively). Since the sensitivity index = -1.01898 implies that when the probability of acquiring pneumonia increases(decreases) by 10% it decreases(increases) R_H by 10.2%. This is the same for the pneumonia contact rate. Increasing(decreasing) HIV contact rate and probability of acquiring HIV by 10% decreases(increases) R_H by 10%. For $R_P > 1$ the most sensitive parameters are probability of acquiring pneumonia, contact with pneumonia infective, probability of acquiring HIV, HIV contact rate, pneumonia induced deaths and rate of HIV treatment ($-1.0031, -1.0031, 1, 1, 0.9676$ and -0.9743 respectively). Since the sensitivity index is $R_P = 3$ implying that the increase(decrease) of probability of acquiring pneumonia and contact rate with pneumonia infective by 10% decreases(increases) R_H by 10.03% and increasing(decreasing) pneumonia induced death rate by 10% increases(decreases) R_H by 9.67%. It is clear that R_H is sensitive to changes in R_P. It means that the sensitivity of R_H to parameters variation depends on R_P. Pneumonia basic reproduction number R_P indicates that the most sensitive parameter is μ. From Table 3.6 we see that increasing(decreasing) the contact rate with pneumonia infective increases(decreases) R_P by 10%. 10% Increasing(decreasing) pneumonia treatment rate decreases(increases) R_P by 0.11%. Pneumonia induced death parameter was observed to be more sensitive than others meaning that pneumonia treatment and health care should be improved within the population.

The impact of one disease on the other is explained by expressing the basic reproduction numbers in terms of each other. If expression Equation 3.4.5 is positive implies that as HIV infections increases results into pneumonia infections also to increases in the population. When Equation 3.4.5 equals to zero means that HIV has no effect on pneumonia. When Equation 3.4.5 is negative, shows that HIV increase with decreasing pneumonia cases in the population. The conditions are similar when R_H is expressed in terms of R_P.

V.   Conclusion

From Table 3.7 and Table 3.8 we can see that R_P is not sensitive to any variation in the HIV reproduction number. This is observed on the same indices displayed when $R_0 < 1$ and $R_0 > 1$. Considering the effects of treatments of the co-infection using the impact of one disease into another. If Equation 3.4.6 is positive, then the treatment of HIV will have negative impact on HIV/AIDS pneumonia co-infection. If it is negative, shows that HIV will have the positive impact on the coinfection. When Equation 3.4.6 equals to zero, the treatment has no impact on the co-infection. R_0 of pneumonia is seen to be greater than that of HIV meaning that pneumonia as a density dependency transmitted disease should be treated so as to increase the body immune. Also the reproduction number can be used to observe the impact of pneumonia to HIV patients (Okosun and Smith, 2017). Mathematical model was developed in order to understand the HIV/AIDS and Pneumonia co-infection in order to improve the treatments and control of the diseases. The disease free equilibrium is shown to be locally asymptotically stable. It was seen that whenever the rate of treatment of diseases is increased, the number of infections decreases. The government and other health institutions can use these results in distributing the right medications at right time considering the stages of the diseases. The basic reproduction number was used to state the stability of the model or to conclude whether the disease prevail or dies out. The contact rates and treatment rates were the high sensitive parameters where by decreasing these factors lead to the faster decrease of disease transmission. As shown under the impact of pneumonia on HIV/AIDS, it has been observed that a single individual with pneumonia can infect a larger number of individuals from the susceptible population compared to the impact caused by a HIV infected individual. So effective treatment of pneumonia can increase the lifespan of the co-infected individual. Pneumonia induced death was seen to be more sensitive in the population. R_P is not sensitive to any variation in R_H. This is shown on the same index when $R_0 < 1$ and $R_0 > 0$.

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