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Mathematical Model of Pneumonia and HIV/AIDS Co-Infection in the Presence of Protection

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Abstract

In this paper, a deterministic co-infection model incorporating protection from infection for both pneumonia infection and HIV/AIDS is considered. The model is shown to be positively invariant as well as bounded. Specifically we consider the case of maximum protection against pneumonia and the case of maximum protection against HIV/AIDS. In both cases, the endemic states are shown to exist provided the reproduction number for each case is greater than unity. Furthermore, by use of a suitable Lyapunov function, the endemic states have been shown to be globally asymptotically stable. Numerical sim-

ulations indicate that enhanced protection against a disease lowers the rate of infection or disease prevalence.

Mathematics Subject Classification: 35E21

Keywords: Reproduction number, Protection, Next Generation Matrix, Pneumonia, HIV/AIDS

1 Introduction

HIV/AIDS continues to be a major global public health issue, having claimed more than 39 million lives so far. Sub-saharan Africa accounts for almost 70% of the global new HIV infections,with 24.7 million people living with HIV by 2013[16]. As a result of the high HIV prevalence, opportunistic infections(OIs), such as pneumonia which are rare in people with healthy immune systems, are also in high prevalence giving rise to co-infections. A co-infection is the simultaneous infection of the same host with two or more different pathogens or different strains of the same pathogens, leading to coexistence of strains (pathogens) at population level. There are cases where a host who is HIV positive, is infected with pneumonia[8].

Pneumonia is a lung infection involving the lung alveoli (air sacs), caused by microbes, including bacteria, viruses, or fungi. The viruses which cause pneumonia include influenza A and B viruses; respiratory syncytial virus (RSV); and haemophilus parainfluenzae types 1, 2 and 3. Streptococcus pneumoniae is the most common cause of bacterial pneumonia. Haemophilus influenzae type b, group A streptococcus, and Mycobacterium tuberculosis (TB) are bacteria that also cause pneumonia. Pneumonia can be transmitted when airborne microbes from an infected individual are inhaled by an individual. Generally, the symptoms of pneumonia include: cough, difficult breathing, fever, muscle aches, loss of appetite and lethargy[6]. Pneumonia mortality in children is very high especially in the developing world, with an estimate of 5,500 deaths per day [15]. The challenge of scarce resources for treatment of pneumonia necessitates the implementation of programs aimed at preventing the infection. Protective measures against pneumonia may include but not limited to; vaccination, practising good hygiene, avoiding close contacts with sick people and limiting exposure to cigarette smoke[17].

Upon getting infected with HIV virus, the body's immune system becomes weak and with time, infected individuals progresses to the AIDS stage. Early treatment of HIV- positive people with antiretroviral drugs can lower the viral load set point while prolonging the life of the infected person[5]. Although therapeutic treatment strategies appear promising for retarding the progression of

HIV related diseases, prevention remains the most effective strategy against the HIV/AIDS epidemic[1]. Use of condoms has been studied numerously[7, 18]. Lately, public health campaigns against many infections are focusing on protection measures. HIV/AIDS protection may involve abstinence, being faithful, use of condoms, male circumcision among others. Co - infection mathematical models of HIV/AIDS and pneumonia are rare in literature yet the synergy between the two infections exist[8].

2 Model Formulation

We formulate a model in which the total human population at any time t denoted by N, is subdivide into subclasses, $S(t)$ the class of individuals susceptible to both pneumonia and HIV/AIDS infection, P_P, individuals who are protected against pneumonia. The protection is lost at the rate α_1. The class I_P consists of individuals who are infected with pneumonia at a rate λ_P. Treatment for pneumonia is assumed to be successful and is done at the rate ε. The class T_P consists of individuals who have recovered from pneumonia infection. Mortality occurs among pneumonia patients at the rate δ_P, while natural death is assumed to occur in all classes at the rate μ.

The class P_H consist of individuals who are protected against HIV/AIDS infection. For various forms of protection against HIV/AIDS, see for instance[18, 14]. This protection may be lost due to risky behaviour at the rate α_2. Since the modes of transmission for the two diseases are different and also for purposes of simplicity we do not assume dual protection for pneumonia and HIV/AIDS. The class I_H is made up of individuals who are asymptomatically infected with HIV/AIDS. This infection occurs at the rate λ_H. In the absence of intervention (therapy), individuals develop symptoms of HIV/AIDS and progress from the class I_H to the class I_A at the rate τ. Mortality occurs among HIV/AIDS patients at the rate δ_A.

Individuals in the class I_H can acquire pneumonia at the rate $\varphi_1\lambda_P$ and progress to the class I_{HP}, where φ_1 is a modification parameter accounting for the fact that individuals who have HIV virus are more susceptible to pneumonia infection than HIV negative individuals due to immunosuppression. Individuals in the class I_A can also acquire pneumonia at the rate $\varphi_2\lambda_P$, where $\varphi_2 > \varphi_1$ and progress to the class I_{AP}. In the absence of intervention (therapy), individuals in the class I_{HP} develop symptoms of HIV/AIDS and progress to the class I_{AP} at the rate $\varrho\tau$, where ϱ is a modification parameter. Upon effective pneumonia treatment, individuals in the class I_{HP} and I_{AP} move to the classes I_H and I_A respectively. Mortality occurs due to the dual infection of HIV/AIDS and pneumonia at a rate δ_{AP}.

Individuals in the class I_P can acquire HIV/AIDS at a rate $\kappa\lambda_H$ and progress to the class I_{HP} where $0 < \kappa < 1$ is a modification parameter accounting for the fact that individuals infected with pneumonia, due to morbidity have reduced activity and are less susceptible.

The total population

$$N = S(t) + P_P(t) + P_H(t) + I_H(t) + I_{HP}(t) + I_P(t) + T_P(t) + I_A(t) + I_{AP}(t) \tag{1}$$

We define the rate at which individuals acquire pneumonia as

$$\lambda_P = \frac{\pi\theta[I_P + \phi_1 I_{HP} + \phi_2 I_{AP}]}{N}, \tag{2}$$

where π is the probability that one will acquire pneumonia upon contact with pneumonia infected individuals and θ is the contact rate with pneumonia infected individuals while $\phi_2 > \phi_1$ are modification parameters accounting for the assumed increased infectivity due to dual infection.

The rate at which individual acquire HIV/AIDS is defined as

$$\lambda_H = \frac{\beta C[I_H + \omega_1 I_{HP} + \omega_2 I_A + \omega_3 I_{AP}]}{N}, \tag{3}$$

where β is the probability that susceptible individuals acquire HIV upon contact with an HIV infected individual and C is the effective contact rate with HIV/AIDS infected individuals. $\omega_3 > \omega_2 > \omega_1$, are modification parameters showing the infectious rate per class.with the assumption that an individual who is asymptomatic with HIV/AIDS and has pneumonia is more infectious of HIV/AIDS than one asymptomatically infected with HIV/AIDS. Similarly AIDS individuals are more infectious than individuals asymptomatic with HIV/AIDS due to high viral load[9].

HIV/AIDS prevalence denoted by Φ will be

$$\Phi = \frac{\lambda_H}{\pi C}. \tag{4}$$

This study seeks to investigate the effect of protection for HIV/AIDS and pneumonia in the co-infection model. Let χ_P and χ_H denote the probability of success of protection against pneumonia and HIV/AIDS respectively. The modified forces of infection λ_P^p and λ_H^p become

$$\lambda_P^p = \lambda_P(1 - \chi_P) \tag{5}$$

and

$$\lambda_H^p = \lambda_H(1 - \chi_H) \tag{6}$$

From the above definitions, the resulting flow diagram for the co-infection is given below.

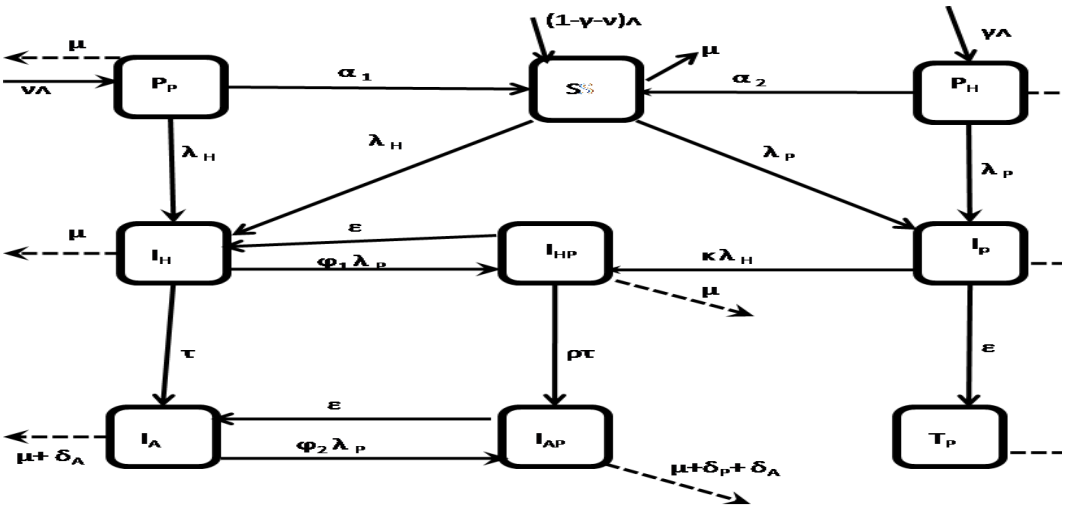

Figure 1: *The flow diagram for the pneumonia and HIV/AIDS co-infection*

The mathematical model defined by a system of differential equations based on the above flow diagramme is given by

$$\begin{aligned}
\frac{dS}{dt} &= (1-\nu-\gamma)\Lambda + \alpha_1 P_P + \alpha_2 P_H - (\mu + \lambda_H + \lambda_P)S \\
\frac{dP_P}{dt} &= \nu\Lambda - (\mu + \alpha_1 + \lambda_H)P_P \\
\frac{dP_H}{dt} &= \gamma\Lambda - (\alpha_2 + \mu + \lambda_P)P_H \\
\frac{dI_H}{dt} &= \lambda_H P_P + \lambda_H S + \varepsilon I_{HP} - (\varphi_1\lambda_P + \mu + \tau)I_H \\
\frac{dI_A}{dt} &= \tau I_H + \varepsilon I_{AP} - (\mu + \delta_A + \varphi_2\lambda_P)I_A \\
\frac{dI_P}{dt} &= \lambda_P P_H + \lambda_P S - (\mu + \kappa\lambda_H + \varepsilon + \delta_P)I_P \\
\frac{dT_P}{dt} &= \varepsilon I_P - \mu T_P \\
\frac{dI_{HP}}{dt} &= \varphi_1\lambda_P I_H + \kappa\lambda_H I_P - (\mu + \varrho + \delta_P + \varepsilon)I_{HP}
\end{aligned} \tag{7}$$

$$\frac{dI_{AP}}{dt} = \varrho\tau I_{HP} + \varphi_2\lambda_P I_A - (\mu + \delta_A + \delta_P + \delta_{AP} + \varepsilon)I_{AP}$$

where $\nu\Lambda$ is the constant recruitment rate into the class of individuals protected against pneumonia, $\gamma\Lambda$ is the constant recruitment rate into the class of individuals protected against HIV and $(1-\nu-\gamma)\Lambda$ is the constant recruitment rate into the class of susceptible individuals to both pneumonia and HIV virus.

3 Model Analysis

Based on the fact that the model deals with human population, all the state variables and parameters are assumed to be non-negative $\forall t \geq 0$. This model is studied in the feasible region Ω, where

$$\{S(t), P_H(t), P_P(t), I_H(t), I_{HP}(t), I_P(t), T_P(t), I_A(t), I_{AP}(t)\} \in \Omega \subset \mathbb{R}^9_+.$$

To show that our solutions are bounded in the set Ω, we take the time derivative of N from Equation (7), thus we have

$$\frac{dN}{dt} = \Lambda - \mu N - (\delta_P + \delta_A + \delta_{AP})I_{AP} - \delta_P I_{HP} - \delta_P)I_P - \delta_A I_A \tag{8}$$

thus

$$\frac{dN}{dt} \leq \Lambda - \mu N \tag{9}$$

which upon integration yields

$$0 \leq N \leq \frac{\Lambda}{\mu} + N(0)e^{-\mu(t)} \tag{10}$$

where $N(0)$ is the initial total population. Clearly as $t \to \infty$ we have

$$0 \leq N \leq \frac{\Lambda}{\mu}, \tag{11}$$

which shows that the set of solutions is bounded. With the assumption of positivity of solutions and having established that the same are bounded, the model Equation (7) is epidemiologically well posed in the region Ω

3.1 The case of maximum protection against pneumonia

In this case the probability of the success of protection is unity, That is $\chi_P = 1$. Assuming that there are no new pneumonia infections in the population, then

$\lambda_P = I_P = 0$. Therefore Equation (7) becomes

$$\begin{aligned}
\frac{dS}{dt} &= (1-\nu-\gamma)\Lambda + \alpha_2 P_H - (\mu+\lambda_H)S \\
\frac{dP_P}{dt} &= \nu\Lambda - (\mu+\lambda_H)P_P \\
\frac{dP_H}{dt} &= \gamma\Lambda - (\alpha_2+\mu)P_H \\
\frac{dI_H}{dt} &= \lambda_H P_P + \lambda_H S - (\mu+\tau)I_H \\
\frac{dI_A}{dt} &= \tau I_H - (\mu+\delta_A)I_A
\end{aligned} \tag{12}$$

The force of infectionλ_H^h Equation (6) is now defined as as

$$\lambda_H^h = \frac{C\beta(1-\chi_H)(I_H+\theta I_A)}{N} \tag{13}$$

The basic reproduction numberR_H, computed using the next generation matrix method approach[13] for Equation (12) is given by

$$R_H = \frac{C\beta(1-\chi_H)(\delta_A+\mu+\theta\chi_H)(\mu(1-\gamma)+\alpha_2)}{(\delta_A+\mu)(\mu+\tau)(\mu+\alpha_2)} \tag{14}$$

The endemic state is defined as

$$I_H^* = \frac{\Lambda(\delta_A+\mu)(R_H-1)}{(1-\chi_H)(\delta_A+\mu+\theta\tau)-\delta_A\tau}. \tag{15}$$

For an infection to be endemic in a population, $I_H^* > 0$. This inequality holds provided that $R_H > 1$ with $(1-\chi_H) > 0$ and $[(1-\chi_H)(\delta_A+\mu+\theta\tau)] > (\delta_A\tau)$

3.2 Local stability of the endemic equilibrium

The long term behaviour of Equation (12) can be deduced from its stability analysis. From Equation (12) $N = S + P_P + P_H + I_H + I_A$. We can study the first four equations of Equation (12) since $I_A = N - (S + P_P + P_H + I_H)$. Thus

$$\begin{aligned}
\frac{dS}{dt} &= (1-\nu-\gamma)\Lambda + \alpha_2 P_H - (\mu+\lambda_H^h)S \\
\frac{dP_P}{dt} &= \nu\Lambda - (\mu+\lambda_H^h)P_P \\
\frac{dP_H}{dt} &= \gamma\Lambda - (\alpha_2+\mu)P_H \\
\frac{dI_H}{dt} &= \lambda_H^h P_P + \lambda_H^h S - (\mu+\tau)I_H
\end{aligned} \tag{16}$$

The Jacobian of Equation (16) at the endemic state $E_1^*(S^*, P_P^*, P_H^*, I_H^*)$ is given by

$$J(E_1^*) = \begin{pmatrix} -(\mu+\lambda_H^h) & 0 & \alpha_2 & \frac{-C\beta S^*(1-\chi_H}{N} \\ 0 & -(\mu+\lambda_H^h) & 0 & \frac{-C\beta P_P^*(1-\chi_H}{N} \\ 0 & 0 & -(\alpha_2+\mu) & 0 \\ \lambda_H^h & \lambda_H^h & 0 & -(\mu+\tau) \end{pmatrix} \tag{17}$$

Clearly $-(\alpha_2+\mu)$ is an eigenvalue of Equation (17). The other eigenvalues can be obtained from the reduced matrix defined by

$$J(E_{12}^*) = \begin{pmatrix} -(\mu+\lambda_H^h) & 0 & \frac{-C\beta S^*(1-\chi_H)}{N} \\ 0 & -(\mu+\lambda_H^h) & \frac{-C\beta P_P^*(1-\chi_H)}{N} \\ \lambda_H^h & \lambda_H^h & -(\mu+\tau) \end{pmatrix} \tag{18}$$

The trace of Equation (18) is negative and its determinant is given by

$$\begin{aligned} detJ(E_{12}^*) = [\frac{I_H^*(1-\chi_H)\beta C}{N} + \mu][\frac{I_H^*(1-\chi_H)(S^*-P_P^*)\beta C}{N^2} \\ (I_H^*(\mu+\tau) - (1-\chi_H)\beta C + \mu + \tau) - \mu(\mu+\tau)] \end{aligned} \tag{19}$$

$detJ(E_{12}^*) > 0$ provided that

$$I_H^*(\mu+\tau) \geq ((1-\chi_H)\beta C) + \mu + \tau \tag{20}$$

and

$$[\frac{I_H^*(1-\chi_H)(S^*-P_P^*)\beta C}{N^2}(I_H^*(\mu+\tau) - (1-\chi_H)\beta C + \mu + \tau) \geq (\mu(\mu+\tau)) \tag{21}$$

Since the trace is negative and the determinant is positive provided that inequality (20) and inequality (21) hold, the eigenvalues of Equation (18) will have negative real parts. Therefore the endemic equilibrium $E_1^*(S^*, P_P^*, P_H^*, I_H^*)$ is locally asymptotically stable.

3.3 Global stability of the endemic equilibrium

The global stability of the equilibria are obtained by means of Lyapunov's direct method and LaSalle's invariance principle[4]. Consider the non-linear Lyapunov function

$$V_e : (S, P_P, P_H, I_H, I_A) \in \Omega \subset \mathbb{R}_+^5 : S, P_P, P_H, I_H, I_A > 0 \tag{22}$$

defined as

$$V_e : (S, P_P, P_H, I_H, I_A) = \lambda_H^h(S - S^* - S^* \log \frac{S}{S^*}) + \lambda_H^h(P_P - P_P^* - P_P^* \log \frac{P_P}{P_P^*}) + \lambda_H^h(P_H - P_H^* - P_H^* \log \frac{P_H}{P_H^*}) + \lambda_H^h(I_H - I_H^* - I_H^* \log \frac{I_H}{I_H^*}) + \lambda_H^h(I_A - I_A^* - I_A^* \log \frac{I_A}{I_A^*}) \tag{23}$$

where V_e is C^1 in the interior of the region Ω. E_1^* is the global minimum of V_e on Ω and $V_e : (S, P_P, P_H, I_H, I_A) = 0$. The time derivative of Equation(23) is given by

$$\begin{aligned} \frac{dV_e}{dt} = \dot{V_e} &= \lambda_H^h(1 - \frac{S^*}{S})\frac{dS}{dt} + \lambda_H^h(1 - \frac{P_P^*}{P_P})\frac{dP_P}{dt} + \lambda_H^h(1 - \frac{P_H^*}{P_H})\frac{dP_H}{dt} + \\ &\quad \lambda_H^h(1 - \frac{I_H^*}{I_H})\frac{dI_H}{dt} + \lambda_H^h(1 - \frac{I_A^*}{I_A})\frac{dI_A}{dt} \\ &= -\lambda_H^h(\frac{S - S^*}{S})[(\mu + \lambda_H^h)(S - S^*) + \alpha_2(P_H - P_H^*)] \\ &-\lambda_H^h(\frac{P_H - P_H^*}{P_H})[(\alpha_2 + \mu)(P_H - P_H^*)] - \lambda_H^h(\frac{P_P - P_P^*}{P_P})[(\mu + \lambda_H^h)(P_P - P_P^*)] \\ &\quad -\lambda_H^h(\frac{I_H - I_H^*}{I_H})[(\mu + \tau_2)(I_H - I_H^*)] - \lambda_H^h(\frac{I_A - I_A^*}{I_A})[(\mu + \delta_A)(I_A - I_A^*)] \end{aligned} \tag{24}$$

Hence $\dot{V_e} < 0$. We see that $\dot{V_e} = 0$ iff $S = S^*, P_H = P_H^*, P_P = P_P^*, I_H = I_H^*$ and $I_A = I_A^*$. Thus the largest compact invariant set in $\{S, P_H, P_P, I_H, I_A\} \in \Omega : \dot{V_e} = 0$ is the Singleton E_1^*, where E_1^* is the endemic equilibrium. Thus E_1^* is globally asymptotically stable in the interior of the region Ω.

3.4 Case of maximum protection against HIV/AIDS

In this case the probability of the success of protection is unity, i.e $\chi_H = 1$. Assuming that there are no new HIV/AIDS infections in the population, then $\lambda_H = I_H = I_A = 0$. Therefore Equation (7) becomes

$$\begin{aligned} \frac{dS}{dt} &= (1 - \nu - \gamma)\Lambda + \alpha_1 P_P - (\mu + \lambda_P)S \\ \frac{dP_P}{dt} &= \nu\Lambda - (\mu + \alpha_1)P_P \\ \frac{dP_H}{dt} &= \gamma\Lambda - (\mu + \lambda_P)P_H \end{aligned} \tag{25}$$

$$\begin{aligned}\frac{dI_P}{dt} &= \lambda_P P_H + \lambda_P S - (\mu + \varepsilon + \delta_P) I_P \\ \frac{dT_P}{dt} &= \varepsilon I_P - \mu T_P\end{aligned}$$

The force of infection Equation (5) is now defined as

$$\lambda_P^p = \frac{\pi\theta(1-\chi_P)I_P}{N} \tag{26}$$

while the basic reproduction number is given by

$$R_P = \frac{\pi\theta(1-\chi_P)(\mu(1-\nu)+\alpha_1)}{(\delta_P+\mu+\varepsilon)(\mu+\alpha_1)}. \tag{27}$$

For the infection to be endemic in a population $I_P^* > 0$. From Equation (25)

$$I_P^* = \frac{\Lambda(R_P - 1)}{(\pi\theta)(1-\chi_P) - \delta_P} > 0 \tag{28}$$

provided $R_P > 1$ with $\delta_P < \pi\theta(1-\chi_P)$.

3.5 Local stability of the endemic equilibrium

The total population N from Equation (25) is $N = S + P_P + P_H + I_P + T_P$ for which $T_P = N - S + P_P + P_H + I_P$. Thus we study the first four equations of Equation (25) at the endemic state $E_2^*(S^*, P_H^*, P_P^* I_P^*)$.

$$\begin{aligned}\frac{dS}{dt} &= (1-\nu-\gamma)\Lambda + \alpha_1 P_P - (\mu + \lambda_P^p) S \\ \frac{dP_P}{dt} &= \nu\Lambda - (\mu+\alpha_1) P_P \\ \frac{dP_H}{dt} &= \gamma\Lambda - (\mu + \lambda_P^p) P_H \\ \frac{dI_P}{dt} &= \lambda_P^p P_H + \lambda_P^p S - (\mu + \varepsilon + \delta_P) I_P\end{aligned} \tag{29}$$

The Jacobian of Equation (29) is given by

$$J(E_2^*) = \begin{pmatrix} -(\mu+\lambda_P^p) & \alpha_1) & 0 & \frac{-\pi\theta S^*(1-\chi_P^p)}{N} \\ 0 & -(\mu+\alpha_1 & 0 & 0 \\ 0 & 0 & -(\mu+\lambda_P^p) & \frac{-\pi\theta P_H^*(1-\chi_P)}{N} \\ \lambda_P^p & 0 & \lambda_P^p & -(\mu+\varepsilon+\delta_P) \end{pmatrix} \tag{30}$$

Clearly $-(\mu+\alpha_1)$ is an eigenvalue of Equation (30). To obtain the other eigenvalues, we analyze the system

$$J(E_{21}^*) = \begin{pmatrix} -(\mu+\lambda_P^p) & 0 & \frac{-\pi\theta S^*(1-\chi_P)}{N} \\ 0 & -(\mu+\lambda_P^p) & \frac{-\pi\theta P_H^*(1-\chi_P)}{N} \\ \lambda_P^p & \lambda_P^p & -(\mu+\varepsilon+\delta_P) \end{pmatrix} \tag{31}$$

$trJ(E_{21}^*) < 0$ and

$$detJ(E_{21}^*) = \frac{1}{N^2}[\frac{I_P^*(1-\chi_P)\pi\theta}{N}+\mu][(1-\chi_P)^2\pi^2\theta^2 I_P^*(P_H^*+S^*) + N(I_P^*(1-\chi_P)\pi\theta+N\mu)(\varepsilon+\mu+\tau)] \tag{32}$$

$detJ(E_{21}^*)$ is positive since $(1-\chi_P)>0$. Therefore the endemic equilibrium $E_2^*(S^*, P_H^*, P_P^* I_P^*)$ is locally asymptotically

3.6 Global stability of the endemic equilibrium

Consider the non-linear Lyapunov function

$$V : (S, P_P, P_H, I_P, T_P) \in \Omega \subset \mathbb{R}_+^5 : S, P_P, P_H, I_P, T_P > 0 \tag{33}$$

defined as

$$V : (S, P_P, P_H, I_P, T_P) = \lambda_P^p(S - S^* - S^* \log\frac{S}{S^*}) + \lambda_P^p(P_P - P_P^* - P_P \log\frac{P_P}{P_P^*}) + \lambda_P^p(P_H - P_H^* - P_H^* \log\frac{P_H}{P_H^*}) + \lambda_P(I_P - I_P^* - I_P^* \log\frac{I_P}{I_P^*}) + \lambda_P^p(T_P - T_P^* - T_P^* \log\frac{T_P}{T_P^*}) \tag{34}$$

where V is C^1 in the interior of the region Ω. E_2^* is the global minimum of V on Ω and $V : (S, P_P, P_H, I_P, T_P) = 0$. The time derivative of Equation (34) is given by

$$\frac{dV}{dt} = \dot{V} = \lambda_P^p(1-\frac{S^*}{S})\frac{dS}{dt} + \lambda_P^p(1-\frac{P_P^*}{P_P})\frac{dP_P}{dt} + \lambda_P^p(1-\frac{P_H^*}{P_H})\frac{dP_H}{dt} + \lambda_P^p(1-\frac{I_P^*}{I_P})\frac{dI_P}{dt} + \lambda_P^p(1-\frac{T_P^*}{T_P})\frac{dT_P}{dt} \tag{35}$$

With the derivatives of S, P_P, P_H, I_P, T_P defined in Equation (25) and by using

$(1-\nu-\gamma)\Lambda = -\alpha_1 P_P^* + (\mu+\lambda_P^p)S^*, \nu\Lambda - (\mu+\alpha_1)P_P^*, \gamma\Lambda - (\mu+\lambda_P^p)P_H^*, \lambda_P^p(P_H + S) = (\mu+\varepsilon+\delta_P)I_P^*$, and $\varepsilon T_P = \mu T_P$ into Equation (35) we obtain

$$\begin{aligned}\dot{V} = -\lambda_P^p(\frac{S-S^*}{S})[(\mu+\lambda_P^p)(S-S^*) + \alpha_1(P_P - P_P^*)] \\ -\lambda_P^p(\frac{P_P - P_P^*}{P_P})[(\alpha_1+\mu)(P_P - P_P^*)] - \lambda_P^p(\frac{P_H - P_H^*}{P_H})[(\mu+\lambda_P^p)(P_H - P_H^*)] \\ -\lambda_P^p(\frac{I_P - I_P^*}{I_P})[(\mu+\varepsilon+\delta_P)(I_L - I_L^*)] - \lambda_P^p(\frac{T_P - T_P^*}{T_P})[\mu(T_P - T_P^*)]\end{aligned} \tag{36}$$

hence $\dot{V} < 0$. Thus E_2^* is globally asymptotically stable in the interior of the region Ω.

3.7 NUMERICAL SIMULATIONS

To graphically illustrate the effect of protection on the dynamics of infection, numerical simulations are carried out.

Table 3.7.1: *Parameter values used in simulation*

Parameter description	Symbol	Value	Source
Natural death rate	μ	$7.0 \times 10^{-3} days^{-1}$	[3]
Recruitment rate	Λ	$8.7 \times 10^{-3} days^{-1}$	[3]
Rate of recruitment into HIV protected class	χ	6.7×10^{-3}	Estimated
Rate of recruitment into TB protected class	ϖ	1.2×10^{-3}	Estimated
Loss of protection against HIV/AIDS	α_2	1.0×10^{-4}	Estimated
Death due to HIV/AIDS	δ_A	$2.3 \times 10^{-4} days^{-1}$	[10]
Rate of progression to AIDS stage	τ_2	$1.25 \times 10^{-1} days^{-1}$	[12]
Probability of acquiring HIV/AIDS	β	$1.1 days^{-2}$	[2]
Protection against HIV/AIDS	χ_H	8.0×10^{-1}	Estimated
Contact rate with HIV/AIDS infectives	C	8.0×10^{-1}	Estimated
Rate of recruitment into pneumonia protected class	ϖ	5.97×10^{-1}	[11]
Loss of protection against pneumonia	α_2	5.0×10^{-3}	Estimated
Death due to pneumonia	δ_A	$3.4 \times 10^{-2} days^{-1}$	[11]
Probability of acquiring pneumonia	π	1.1×10^{-9}	Estimated
Protection against pneumonia	χ_P	9.0×10^{-1}	Estimated
Contact rate with pneumonia infective	θ	8.0×10^{-1}	Estimated

3.7.1 The effect of varying the protection term on HIV/AIDS infections

Numerical simulations were carried out to investigate the effect of protection on HIV/AIDS and pneumonia prevalence. The following graphs were obtained for a given set on initial conditions and parameter values in Table 3.7.1.

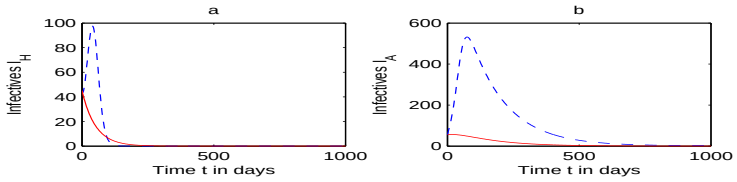

Figure 1: *Simulation of Equation (12) showing the evolution of HIV/AIDS against time*
Broken line: $\pi = 1.1 \times 10^{-2}, \chi_H = 1.0 \times 10^{-4}$
Continuous line:$\pi = 1.1 \times 10^{-10}, \chi_H = 8.0 \times 10^{-1}$

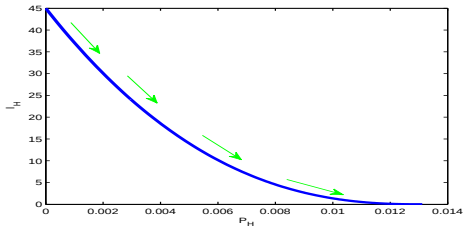

Figure 2: *The graph* I_H *against* P_H

3.7.2 The effect of varying the protection term on pneumonia infection against time

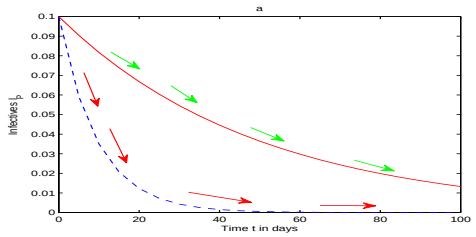

Figure 3: *Simulation of Equation (25) showing the evolution of pneumonia*
Continuous line: $\beta = 1.1 \times 10^{-3}, \chi_P = 6.0 \times 10^{-3}$
Broken line: $\beta = 1.1 \times 10^{-9}, \chi_P = 9.0 \times 10^{-1}$

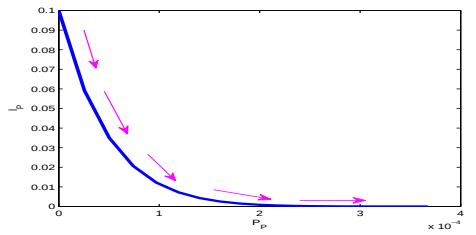

Figure 4: *The graph* I_P *against* P_P

3.8 Discussion

From Figure 1(a), we observe that, with a low protection rate the probability of infection is high and therefore the number of infectives I_H rises sharply in a short span before drastically dropping. This sharp drop may be attributed to the depletion of susceptibles or the susceptibles embracing protective measures. From the same figure a very high protection rate with a low probability of transmission results in reduced disease prevalence. From Figure 1(b), a low protection rate results in increased I_A cases. This is because it has been medically proven that increased viral load for HIV persons leads to a faster rate of progression to the symptomatic stage. On the contrary, when the protection rate is high then I_A cases are low. From Figure 2, we observe that the number of I_H infections reduces with increased protection.

From Figure 3, a high rate of protection against pneumonia leads to a low probability of disease transmission and a low protection rate leads to a high disease prevalence. Figure 4 shows that the number of I_P infectives reduces with increased protection.

In order to reduce the number of new HIV/AIDS and pneumonia infections, and reduce their impact on individual, families and communities, there is need to employ strategies such as increasing the public awareness drive to behaviour change and encourage openness, increasing access to voluntary HIV testing and counselling, promoting increased condom use to reduce the spread of HIV infection, improving access to antiretroviral drugs (ARV's) for people living with AIDS, practising proper hygiene, eating a balanced diet and vaccination in the case of pneumonia. These strategies will help in reducing the economic burden that are borne by a country in giving care and treating the infected individuals. As evidenced from these results, it is indeed true that prevention is better than cure. These results are consistent with results such as by[1].

3.9 Conclusion

In this work, we formulated a model for the co-infection of HIV/AIDS with pneumonia incorporating protection. To investigate the effect of protection, two cases were considered, namely the case of maximum protection against HIV/AIDS and the case of maximum protection against pneumonia. The existence of the endemic equilibrium for the two cases was established and the stability of the same was analysed. In both cases the endemic equilibrium is found to be globally asymptotically stable. This implies that at peak times of the reoccurrence of the diseases, the levels of infections are manageable with minimal interventions. From the numerical simulations, we observe that protection against a disease has the effect of reducing the disease prevalence.

Protection from HIV/AIDS infection may include measures such as use of condoms, male circumcision among others, while protection from pneumonia infection may involve vaccination, adequate nutrition, good hygiene and addressing environmental factors such as air pollution. The model developed and analyzed in this research does dot take into account the specific contributions of individual protective measures. Instead, protection had been used in a general sense. Models which incorporates specific protective measures may be considered for further research. This will guide policy makers on which particular protective measures to implement and emphasize as a means of reducing disease prevalence rates.

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References

[1] B. Nannyonga, J.Y.T. Mugisha and L.S. Luboobi, The Role of HIV Positive Immigrants and Dual Protection in a Co-Infection of Malaria and HIV/AIDS, *Applied Mathematical Sciences*, **5** (2011), no. 59, 2919 - 2942.

[2] C.P. Bhunu, W. Garira, G. Magombedze, Mathematical analysis of a two strain HIV/AIDS model with antiretroviral treatment, *Acta Biotheoretica*, **57** (2009), no. 3, 361 - 381. http://dx.doi.org/10.1007/s10441-009-9080-2

[3] C.I.A World Factbook, (2014).

[4] C.V. De Leon, Constructions of Lyapunov Functions for Classics SIS, SIR and SIRS epidemic model with variable population size, *Unite Academica de Mathematicas, UniversidadnAutonoma de Guerrere, Mexico Facultad de Estudios Superiores Zarogoza, UNAM, Mexico*, (2009).

[5] D.D. Ho, Time to hit HIV, early and hard, *New Engl. J. Med.*, **333** (1995), 450-451. http://dx.doi.org/10.1056/nejm199508173330710

[6] G.O. Lawi, J.Y.T. Mugisha and N. Omolo-Ongati, Modeling Co-infection of Paediatric Malaria and Pneumonia, *Int. Journal of Math. Analysis*, **7** (2013), no. 9, 413 - 424.

[7] D. Greenhalgh, M. Doyle, F. Lewis, A mathematical treatment of AIDS and condom use, *IMA J. Math. Med. Biol.*, **18** (2001), no. 3 225 - 262. http://dx.doi.org/10.1093/imammb18.3.225

[8] J.G. Castro, G. Manzi, L. Espinoza, M. Campos and C. Boulanger, Concurrent PCP and TB pneumonia in HIV infected patients, *Scandinavian Journal of Infectious Diseases*, **39** (2007), 1054 - 1058. http://dx.doi.org/10.1080/00365540701472056

[9] L.J. Abu-Raddad and J.G. Kublin, Dual infection with HIV and malaria fuels the spread of both diseases in sub-saharan Africa, *Science*, **314** (2006), 1603 - 1606. http://dx.doi.org/10.1126/science.1132338

[10] L.M. Arriola and J.M. Hyman, Being Sensitive to Uncertainity, *Computing Science and Engineering*, **9** (2007), 10 - 20. http://dx.doi.org/10.1109/mcse.2007.27

[11] National Survey: 2006 Annual Summary Report.

[12] N. Malunguza, S. Mushayabasa, C. Chiyaka, Z. Mukandavire, Modelling the effects of condom use and antiretroviral therapy in controlling HIV/AIDS among heterosexuals, homosexuals and bi sexuals, *Computational and Mathematical Methods in Medicine*, **11** (2010), 201 - 222. http://dx.doi.org/10.1080/17486700903325167

[13] O. Diekmann, J.A.P. Heesterbeek, J.A.J. Metz, On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogenious populations, *Journal of Mathematical Biology*, **28** (1990), 365 - 382. http://dx.doi.org/10.1007/bf00178324

[14] UNESCO/UNAIDS research project (2003). A cultural Approach to HIV/AIDS Prevention and cure.

[15] United Nations Childrens Fund (UNICEF), 2008. Tracking progress in maternal, newborn and child survival: The 2008 report. Received: September, 2012.

[16] WHO, fact sheet no. 360, updated November 2014.

[17] WHO report sheet no. 331, November 2014.

[18] Z. Mukandavire and W. Garira, Sex-structured HIV/AIDS model to analyse the effects of condom use with application to Zimbabwe, *J. Math. Biol.*, **54** (2007), no. 5, 669 - 699. http://dx.doi.org/10.1007/s00285-006-0063-5

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