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# Super Infection and Density Dependent Dynamics of an SI Type Epidemic Model for Two Strains Pathogen

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**Abstract**

Super infection has an impact on the disease dynamics for some infectious diseases. As a consequence, in this paper super infection of an SI type epidemic model has been studied for two strains pathogen. Moreover, the density dependent infectious rate is considered as the spread of infectious disease depends on the density of the population. It has shown the disease free equilibrium is locally asymptotically stable if $\Re_0^k < 1, k = 1, 2$ and unstable if either of $\Re_0^k > 1$. The global stability at disease free equilibrium has been shown using Lyapunov function. It has also been shown that under which conditions the boundary endemic steady states $E_1$ and $E_2$ are locally asymptotically stable and unstable. For coexistence endemic steady state, stability is shown with numerical simulation. In this paper, some numerical simulations have been done through Mat lab program.

**AMS subject classifications:** 34K20, 37B25, 92D30.

**Keywords:** Super infection, Epidemics, Intravenous drug user (IVDU), Strain1, Strain2, Lyapunov function, Jacobian Matrix & Next generation matrix.

## 1. Introduction

Infectious disease is a great issue throughout the world. According to UNAIDS/WHO, there are over 33 million people living with only HIV and AIDS worldwide. Super-infection is defined when a person gets infected by one strain of pathogen and after time gets infected with a second and different strain of pathogen.

Many authors have already studied epidemic models with two diseases such as AIDS and tuberculosis [2, 3] describes two strains of one disease present in the population, influenza or tuberculosis, for instance [4-6]. In realistic situation the super infection occurs mainly the case of sexually transmitted disease (STD) or intravenous drug user (IVDU) such as HIV/AIDS and gonorrhea [1]. Therefore, we propose an SI type epidemic model considering super infection, especially HIV for two strains. Many doctors and researchers [7, 8, 10] have already given the practical evidence that super infection occurred in HIV through laboratory test. In the 7th annual conference on February 2, 2000 on Retroviruses and Opportunistic Infections [10], Dr. Jonathan Angel presented documentation regarding a suspected infection of a super strain of HIV-1. This "super strain" is resistant to all current highly active anti –retroviral therapy (HAART) regimens, and leads to rapid advance of HIV disease in the infected person. Dr. Angel described a situation where one person (Patient A) with non-progressive HIV infection, was super-infected by a second person (Patient B) with an HIV infection containing several drug resistant mutations in their virus. He reported in his study, patient A met patient B in 1997 and they began having a relationship. This relationship included unprotected sexual intercourse since both of them were already HIV positive. During the course of their relationship, patient A whose HIV had been non-progressive for several years, suddenly experienced a rapid decline in CD4 counts and a dramatic increase in viral load. Following initiation of HAART, which included a protease inhibitor, patient A failed to respond to treatment. Blood samples were taken from both patients A and B. The samples were then compared to determine if patient A had been infected with a super-resistant strain of HIV from patient B. A phylogenetic analysis of the blood samples identified a positive correlation between the point mutations in the virus in both patient A and B, leading to the conclusion that patient A was very likely infected with a resistant strain of HIV-1 by patient B. Another evidence of dual HIV infection in humans appeared in 2002. A report in the Journal of Virology in August of that year strongly suggested super infection in two injection drug users (IDUs) from Thailand (one female, one male) [8].

There are several concerns if a person gets super-infection with two different strains of HIV, which apparently can occur sexually or through IVDU: (1) one virus may be a more virulent or potent virus than the other and this may make a person sicker and develop HIV-related symptoms; (2) viral load may increase and CD4 count might decline after becoming infected with a second virus; (3) one of the viruses may be a drug resistant virus and may prevent the patient from responding to HIV-treatment. For instance, a patient was infected with a multi-drug resistant virus (MDR) initially and later it appears super-infected with a wild-type virus [7]. Subsequent to initial HIV-infection the patient reported an unprotected sexual encounter after which his viral load increased. When the MDR will emerge, he will fail therapy. In our model, we assume that there is no permanent recovery by strain- 1 and strain -2 respectively but some immune response is established temporarily by both strains. For convenient, we assume in our model strain-1 is only super infected by strain-2. That means when an HIV infected individual infected by strain-1 then after treatment/vaccination he gets some temporary immune response and after some

times he super infected by strain-2. Because the HIV virus changes (mutates) so rapidly, it has created many different strains and will likely continue to create new ones. This complicates efforts to develop a vaccine for AIDS, because a vaccine to produce immunity to one strain may not protect against a different strain. We also assume that there is no strain specific disease caused death rate.

## 2. Mathematical Model

Let $S$, $I_1$, $I_2$ and $N$ denote the numbers of susceptible, infected with strain-1, infected with strain-2, and total population .Our model consists of a system of four differential equations.

$$\begin{aligned}
\frac{dS}{dt} &= \mu N - \beta_1 S\frac{I_1}{N} - \beta_2 S\frac{I_2}{N} - \delta S \\
\frac{dI_1}{dt} &= \beta_1 S\frac{I_1}{N} - \beta_{21} I_1\frac{I_2}{N} - \delta I_1 \\
\frac{dI_2}{dt} &= \beta_2 S\frac{I_2}{N} + \beta_{21} I_1\frac{I_2}{N} - \delta I_2 \\
\frac{dN}{dt} &= (\mu - \delta)N
\end{aligned} \tag{2.1}$$

With $S + I_1 + I_2 = N$

Here $\mu$ denote the birth rate of susceptible population and $\delta$ denote the death rate for each compartment. Here we consider the density dependent incidence rate. $\beta_1$ is the transmission coefficient for stain-1, $\beta_2$ is the transmission coefficient for strain- 2, and $\beta_{21}$ is the transmission coefficient for super infection of strain-1 population by strain-2.

By making non dimensional, it is convenient to convert the system of differential equations (2.1) using the differential equation for $N$ and $s = \frac{S}{N}$, $i_1 = \frac{I_1}{N}$, $i_2 = \frac{I_2}{N}$, $\mu = \delta + \gamma$, where $\gamma$ is the net growth rate i.e. $\gamma = \mu - \delta$.

The system (2.1) becomes

$$\begin{aligned}
\frac{ds}{dt} &= \mu - \beta_1 s\, i_1 - \beta_2 s\, i_2 - \mu s \\
\frac{di_1}{dt} &= \beta_1 s\, i_1 - \beta_{21} i_1\, i_2 - (\delta + \gamma) i_1 \\
\frac{di_2}{dt} &= \beta_2 s\, i_2 + \beta_{21} i_1\, i_2 - (\delta + \gamma) i_2
\end{aligned} \tag{2.2}$$

with $s + i_1 + i_2 = 1$

A suitable domain

$$D = \{(s, i_1, i_2) : s \geq 0, i_1 \geq 0, i_2 \geq 0 \quad , s + i_1 + i_2 \leq 1\}$$

Here the domain *D* is positively invariant, because no solution paths leave through any boundary. Since paths can not live *D,* therefore solution exists for all positive time. Thus the model is mathematically and epidemiologically well posed. Now, through out this article we will analyze the model (2.2)

## 3. Equilibria and Basic reproduction number

The system (2.2) has several equilibria: when disease is absent from both strains called disease free equilibrium, when disease is present either one or both strain called endemic equilibrium. Before going to analyze the stability of equilibria of the system (2.2), we find the basic reproduction number. The system (2.2) has a disease free equilibrium $E_0 = (1, 0\ 0)$ . Taking the infected compartments to be $i_1$ and $i_2$, and using the idea from [12], we obtain from the model (2.2)

$$f = \begin{pmatrix} \beta_1 s i_1 \\ \beta_2 s i_2 \end{pmatrix} \quad , \qquad \vartheta = \begin{pmatrix} \beta_{21} i_1 i_2 + (\delta + \gamma) i_1 \\ -\beta_{21} i_1 i_2 + (\delta + \gamma) i_2 \end{pmatrix}$$

At disease free equilibrium,

$$F = \begin{pmatrix} \beta_1 & 0 \\ 0 & \beta_2 \end{pmatrix} \quad , \qquad V = \begin{pmatrix} \delta + \gamma & 0 \\ 0 & \delta + \gamma \end{pmatrix}$$

The matrix $F$ is non negative and is responsible for new infections, while the matrix $V$ is invertible and is referred to as the transmission matrix for the model (2.2)

$$\therefore V^{-1} = \begin{pmatrix} \frac{1}{\delta + \gamma} & 0 \\ 0 & \frac{1}{\delta + \gamma} \end{pmatrix}$$

Therefore, the next generation matrix is $K = F\ V^{-1} = \begin{pmatrix} \frac{\beta_1}{\delta + \gamma} & 0 \\ 0 & \frac{\beta_2}{\delta + \gamma} \end{pmatrix}$

Thus the next generation matrix $K$ has two positive eigen values. Using the next generation matrix approach [12], we obtain basic reproductive numbers associated with strain-1 and strain-2 are $\Re_0^1 = \frac{\beta_1}{\delta + \gamma}$ and $\Re_0^2 = \frac{\beta_2}{\delta + \gamma}$ respectively.

These numbers give the number of secondary infective cases of the disease produced by an individual infected with strain-1, strain-2 during his/her effective period when introduced in a population of susceptible.Consequently, the basic reproductive number associated with the model (2.2) is the maximum of the two strains, that is $\Re_0 = \max\{\Re_0^1, \Re_0^2\}$.Hence the system (2.2) has a disease free equilibrium if $\Re_0 = \max\{\Re_0^1, \Re_0^2\} < 1$, and endemic equilibrium if $\Re_0 = \max\{\Re_0^1, \Re_0^2\} > 1$.

## 4. Disease-free equilibrium and its stability analysis

In this section the analysis of the disease free equilibrium for the system (2.2) and its stability is performed. Consideration of stability of a disease- free steady state gives certain conditions under which disease will die out or stay in the population. We have the following theorem

**Theorem 4.1:** The disease- free equilibrium $E_0 = (1, 0, 0)$ is locally asymptotically stable if $\Re_0^k < 1$, $k = 1, 2$, and unstable if either of $\Re_0^k > 1$ .

**Proof:** To prove this Theorem, we have the follwing Lemma

**Lemma 4.1:** If $F$ is non negative and $V$ is a non singular $M$ matrix then $\Re_0 = \rho(FV^{-1}) < 1$ iff all the eigen values of $(F - V)$ have negative real parts (For Proof see, [12]).

Here, we observe that $F$ is non negative and $V$ is non singular $M$ matrix since off diagonal elements of $V$ are zero.

$$\therefore\ F - V = \begin{pmatrix} \beta_1 - (\delta + \gamma) & 0 \\ 0 & \beta_2 - (\delta + \gamma) \end{pmatrix}.$$

Eigen values of $(F - V)$ is $\det((F - V) - \lambda I) = 0$. i.e.

$$\begin{vmatrix} \beta_1 - (\delta + \gamma) - \lambda & 0 \\ 0 & \beta_2 - (\delta + \gamma) - \lambda \end{vmatrix} = 0$$

$$\therefore\ \lambda_1 = \beta_1 - (\delta + \gamma) = \beta_1 - \frac{\beta_1}{\Re_0^1} = \beta_1\left(1 - \frac{1}{\Re_0^1}\right)$$

$$\lambda_2 = \beta_2 - (\delta + \gamma) = \beta_2 - \frac{\beta_2}{\Re_0^2} = \beta_2\left(1 - \frac{1}{\Re_0^2}\right).$$

Hence by lemma 4.1, the eigen values of $(F - V)$ matrix are negative iff $1 - \frac{1}{\Re_0^1} < 0$ and $1 - \frac{1}{\Re_0^2} < 0$, i.e. $\Re_0^1 < 1$ and $\Re_0^2 < 1$ , because $\Re_0 = \max\{\Re_0^1, \Re_0^2\} < 1$.Therefore, the disease free equilibrium is locally asymptotically stable iff $\Re_0^k < 1$, $k = 1, 2$.On the other hand , the disease free equilibrium is unstable if, $1 - \frac{1}{\Re_0^1} > 0$ or $1 - \frac{1}{\Re_0^2} > 0$ that is $\Re_0^1 > 1$ or $\Re_0^2 > 1$. That means either any one of $\Re_0^k > 1$, $k = 1, 2$ because. $\Re_0 = \max\{\Re_0^1, \Re_0^2\} > 1$. This completes the proof of the theorem.

**Global stability of disease free Equilibrium**

Now we want to show that the disease free equilibrium is globally asymptotically stable. To prove this:

Consider the Lyapunov function

$$V(i_1(t), i_2(t)) = \frac{\beta_1\ \beta_2}{\Re_0^1\ \Re_0^2}\ i_1 + \frac{\beta_1\ \beta_2}{\Re_0^1\ \Re_0^2}\ i_2$$

Here, (i) $V(i_1(t), i_2(t)) = 0$, if $(i_1(t), i_2(t)) = (0,0)$ and

(ii) $V(i_1(t), i_2(t)) > 0$ if $i_1(t), i_2(t) > 0$.

Now we have to show that

(iii) $\frac{dV}{dt} \le 0$

$$\therefore \quad \frac{dV}{dt} = \frac{\partial V}{\partial i_1}\frac{di_1}{dt} + \frac{\partial V}{\partial i_2}\frac{di_2}{dt}$$

$$= \frac{\beta_1\ \beta_2}{\Re_0^1\ \Re_0^2}\ i_1 + \frac{\beta_1\ \beta_2}{\Re_0^1\ \Re_0^2}\ i_2$$

$$= \frac{\beta_1\ \beta_2}{\Re_0^1\ \Re_0^2}[\beta_1\, s\, i_1 - \beta_{21}\, i_1\, i_2 - (\delta+\gamma) i_1] + \frac{\beta_1\ \beta_2}{\Re_0^1\ \Re_0^2}[\beta_2\, s\, i_2 + \beta_{21} i_1\, i_2 - (\delta+\gamma) i_2]$$

$$= \frac{\beta_1\ \beta_2}{\Re_0^1\ \Re_0^2}\left[\beta_1\, s\, i_1 - \beta_{21}\, i_1\, i_2 - \frac{\beta_1}{\Re_0^1} i_1\right] + \frac{\beta_1\ \beta_2}{\Re_0^1\ \Re_0^2}\left[\beta_2\, s\, i_2 + \beta_{21}\, i_1\, i_2 - \frac{\beta_2}{\Re_0^2} i_2\right]$$

$$= \frac{\beta_1^2\ \beta_2}{\Re_0^1\ \Re_0^2}\, s\, i_1 - \frac{\beta_1\beta_2\ \beta_{21}}{\Re_0^1\ \Re_0^2} i_1\, i_2 - \frac{\beta_1^2\beta_2}{\Re_0^1\ \Re_0^1\Re_0^2} i_1 + \frac{\beta_1\beta_2^2}{\Re_0^1\ \Re_0^2}\, s\, i_2 +$$

$$\frac{\beta_1\beta_2\ \beta_{21}}{\Re_0^1\ \Re_0^2} i_1\ i_2 - \frac{\beta_1\beta_2^2}{\Re_0^1\ \Re_0^2\ \Re_0^2}\ i_2$$

$$= \frac{\beta_1^2\ \beta_2}{\Re_0^1\ \Re_0^2}\left(s - \frac{1}{\Re_0^1}\right) i_1 + \frac{\beta_1\ \beta_2^2}{\Re_0^1\ \Re_0^2}\left(s - \frac{1}{\Re_0^2}\right) i_2 \le 0$$

Since at disease free equilibrium $\Re_0^k < 1$, $k = 1, 2$. Equality holds only at the equilibrium point $E_0 = (1, 0, 0)$.Hence $V(i_1(t), i_2(t))$ is a strictly Lyapunov function. Therefore, disease free equilibrium is globally asymptotically stable in $D$.

## 5. Endemic equilibria and its stability

In this section, we analyze endemic equilibria for the system (2.2). The non zero steady –states can be present if there is only infected by strain-1, only strain-2 or both strains. When only one strain is present we call it boundary endemic equilibrium point and when both strains are present we call it coexistence endemic equilibrium point. But the expression for coexistence endemic steady state of the model (2.2) can not be obtained analytically; therefore stability analysis of coexistence endemic steady state is shown by numerical simulation using Mat lab. The model (2.2) has two boundary endemic equilibrium states which are

$E_1 = \left( \frac{1}{\Re_0^1}, \frac{\mu}{\beta_1}(\Re_0^1 - 1), 0 \right)$ [Infected only by strain-1 but not strain-2] and

$E_2 = \left( \frac{1}{\Re_0^2}, 0, \frac{\mu}{\beta_2}(\Re_0^2 - 1) \right)$ [Infected only by strain-2 but not strain-1]

The Jacobian Matrix of the system (2.2) is

$$J = \begin{pmatrix} -\beta_1 i_1 - \beta_2 i_2 - \mu & -\beta_1 s & -\beta_2 s \\ \beta_1 i_1 & \beta_1 s - \beta_{21} i_2 - (\delta + \gamma) & -\beta_{21} i_1 \\ \beta_2 i_2 & \beta_{21} i_2 & \beta_2 s + \beta_{21} i_1 - (\delta + \gamma) \end{pmatrix} \quad (5.1)$$

Now we are trying to find the stability analysis of the system (2.2) at $E_1 = \left( \frac{1}{\Re_0^1}, \frac{\mu}{\beta_1}(\Re_0^1 - 1), 0 \right)$ and $E_2 = \left( \frac{1}{\Re_0^2}, 0, \frac{\mu}{\beta_2}(\Re_0^2 - 1) \right)$.

At $E_1 = \left( \frac{1}{\Re_0^1}, \frac{\mu}{\beta_1}(\Re_0^1 - 1), 0 \right)$, (5.1) becomes

$$\begin{pmatrix} -\beta_1 \frac{\mu}{\beta_1}(\Re_0^1 - 1) - \mu & -\frac{\beta_1}{\Re_0^1} & -\frac{\beta_2}{\Re_0^1} \\ \beta_1 \frac{\mu}{\beta_1}(\Re_0^1 - 1) & 0 & -\beta_{21} \frac{\mu}{\beta_1}(\Re_0^1 - 1) \\ 0 & 0 & \frac{\beta_2}{\Re_0^1} + \beta_{21} \frac{\mu}{\beta_1}(\Re_0^1 - 1) - \frac{\beta_1}{\Re_0^1} \end{pmatrix}$$

The eigen values of $J_{E_1}$ at the equilibrium point $E_1$ is $\det(J_{E_1} - \lambda I) = 0$, i.e.

$$\det \begin{pmatrix} -\mu \Re_0^1 - \lambda & -\frac{\beta_1}{\Re_0^1} & -\frac{\beta_2}{\Re_0^1} \\ \mu(\Re_0^1 - 1) & -\lambda & -\beta_{21} \frac{\mu}{\beta_1}(\Re_0^1 - 1) \\ 0 & 0 & \frac{1}{\Re_0^1}(\beta_2 - \beta_1) + \beta_{21} \frac{\mu}{\beta_1}(\Re_0^1 - 1) - \lambda \end{pmatrix} = 0$$

$$\Rightarrow \left\{ \frac{1}{\Re_0^1}(\beta_2 - \beta_1) + \beta_{21} \frac{\mu}{\beta_1}(\Re_0^1 - 1) - \lambda \right\} \begin{vmatrix} -\mu \Re_0^1 - \lambda & -\frac{\beta_1}{\Re_0^1} \\ \mu(\Re_0^1 - 1) & -\lambda \end{vmatrix} = 0$$

$$\Rightarrow \left\{ \frac{1}{\Re_0^1}(\beta_2 - \beta_1) + \beta_{21} \frac{\mu}{\beta_1}(\Re_0^1 - 1) - \lambda \right\} \left\{ \lambda^2 + \mu \Re_0^1 \lambda + \frac{\mu \beta_1}{\Re_0^1}(\Re_0^1 - 1) \right\} = 0$$

$$\therefore \lambda = \frac{1}{\Re_0^1}(\beta_2 - \beta_1) + \beta_{21} \frac{\mu}{\beta_1}(\Re_0^1 - 1)$$

$$\Rightarrow \lambda_1 = \frac{1}{\Re_0^1}(\beta_2 - \beta_1) + \beta_{21} \frac{\mu}{\beta_1}(\Re_0^1 - 1) < 0, \text{ if}$$

$\beta_1 > \beta_2$ i.e. $(\beta_2 - \beta_1) < 0$ and $\left|\frac{1}{\Re_0^1}(\beta_2 - \beta_1)\right| > \left|\beta_{21}\frac{\mu}{\beta_1}(\Re_0^1 - 1)\right|$

Again,

$$\lambda^2 + \mu\Re_0^1\lambda + \frac{\mu\beta_1}{\Re_0^1}(\Re_0^1 - 1) = 0$$

$$\therefore \lambda_{2,3} = \frac{-\mu\Re_0^1 \pm \sqrt{(\mu\Re_0^1)^2 - 4\mu\beta_1\left(1 - \frac{1}{\Re_0^1}\right)}}{2}$$

Since $\Re_0^1 > 1$, therefore two eigen values are negative. We have the following Theorem

**Theorem 5.1:** The boundary endemic steady state $E_1$ is locally asymptotically stable if

$\beta_1 > \beta_2$ and $\left|\frac{1}{\Re_0^1}(\beta_2 - \beta_1)\right| > \left|\beta_{21}\frac{\mu}{\beta_1}(\Re_0^1 - 1)\right|$, and unstable if $\beta_1 < \beta_2$

At $E_2 = \left(\frac{1}{\Re_0^2},\ 0,\ \frac{\mu}{\beta_2}(\Re_0^2 - 1)\right)$ , (5.1) becomes

$$\begin{pmatrix} -\beta_2\frac{\mu}{\beta_2}(\Re_0^2 - 1) - \mu & -\frac{\beta_1}{\Re_0^2} & -\frac{\beta_2}{\Re_0^2} \\ 0 & \frac{\beta_1}{\Re_0^2} - \beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1) - \frac{\beta_2}{\Re_0^2} & 0 \\ \beta_2\frac{\mu}{\beta_2}(\Re_0^2 - 1) & \beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1) & 0 \end{pmatrix}$$

The eigen values of $J_{E_2}$ at the equilibrium point $E_2$ is $\det(J_{E_2} - \lambda I) = 0$, i.e.

$$\det\begin{pmatrix} -\mu\Re_0^2 - \lambda & -\frac{\beta_1}{\Re_0^2} & -\frac{\beta_2}{\Re_0^2} \\ 0 & \frac{\beta_1}{\Re_0^2} - \beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1) - \frac{\beta_2}{\Re_0^2} - \lambda & 0 \\ \mu(\Re_0^2 - 1) & \beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1) & -\lambda \end{pmatrix} = 0$$

$$\Rightarrow \left\{\frac{1}{\Re_0^2}(\beta_1 - \beta_2) - \beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1) - \lambda\right\}\begin{vmatrix} -\mu\Re_0^2 - \lambda & -\frac{\beta_2}{\Re_0^2} \\ \mu(\Re_0^2 - 1) & -\lambda \end{vmatrix} = 0$$

$$\Rightarrow \left\{\frac{1}{\Re_0^2}(\beta_1 - \beta_2) - \beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1) - \lambda\right\}\left\{\lambda^2 + \mu\Re_0^2\lambda + \frac{\mu\beta_2}{\Re_0^2}(\Re_0^2 - 1)\right\} = 0$$

$$\therefore \lambda = \frac{1}{\Re_0^2}(\beta_1 - \beta_2) - \beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1)$$

$$\Rightarrow \lambda_1 = \frac{1}{\Re_0^2}(\beta_1 - \beta_2) - \beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1) < 0$$, for the following cases

(i) $\beta_2 > \beta_1$

(ii) $(\beta_1 - \beta_2) > 0$ and $\left|\frac{1}{\Re_0^2}(\beta_1 - \beta_2)\right| < \left|\beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1)\right|$

Again,

$$\lambda^2 + \mu\Re_0^2\lambda + \frac{\mu\beta_2}{\Re_0^2}(\Re_0^2 - 1) = 0$$

$$\therefore \lambda_{2,3} = \frac{-\mu\Re_0^2 \pm \sqrt{(\mu\Re_0^2)^2 - 4\mu\beta_2\left(1 - \frac{1}{\Re_0^2}\right)}}{2}$$

Since $\Re_0^2 > 1$, therefore two eigen values are negative.

**Theorem 5.2:** The boundary endemic steady state $E_2$ is locally asymptotically stable for the following cases

(i) $\beta_2 > \beta_1$

(ii) $(\beta_1 - \beta_2) > 0$ and $\left|\frac{1}{\Re_0^2}(\beta_1 - \beta_2)\right| < \left|\beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1)\right|$ ,

and unstable if

$(\beta_1 - \beta_2) > 0$ , and $\left|\frac{1}{\Re_0^2}(\beta_1 - \beta_2)\right| > \left|\beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1)\right|$

## 6. Result and Discussion

In section 4 & 5, some results have been shown analytically for disease free and boundary endemic steady states. In section 6, some numerical simulations have been done for these steady states. Moreover the expression for coexistence endemic steady state of the model (2.2) can not be solved analytically (coexistence steady state can be solved using Maple software); therefore stability analysis of coexistence endemic steady state is shown here by numerical simulation for different parameter values. It has shown analytically the disease- free equilibrium $E_0 = (1, 0, 0)$ is locally asymptotically stable if $\Re_0^k < 1$, $k = 1, 2$, and unstable if either of $\Re_0^k > 1$ , and disease still exists. Numerical simulation also shows when $\Re_0^k < 1$ then disease dies out and equilibrium state is stable (fig: 6.1).

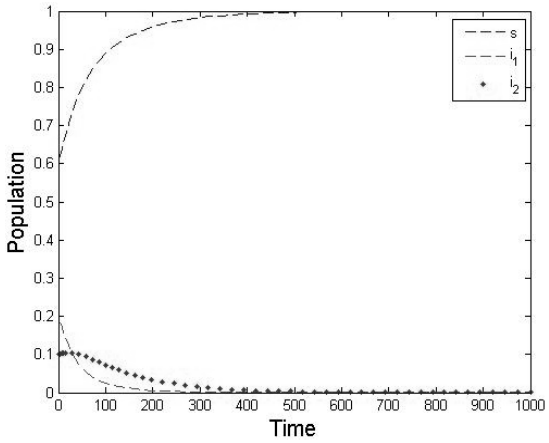


**Figure 6.1:** Parameter values are $\mu = 0.02, \beta_1 = .010, \beta_2 = 0.012, \beta_{21} = 0.09,\ \delta = 0.0005,$ $s(0) = .60$ , $i_1(0) = 0.2$, $i_2(0) = 0.1$ , $t = [0\ , 1000]$ .

From the parameter values, $\Re_0^1 = 0.5 < 1$ and $\Re_0^2 = 0.6 < 1$. It has been observed from fig.6.1, initially $i_1(0) = 0.2$, $i_2(0) = 0.1$ and $i_1$ and $i_2$ goes to zero when time increase. Therefore there is no population in infective class and disease eventually dies out and disease free equilibrium is stable.

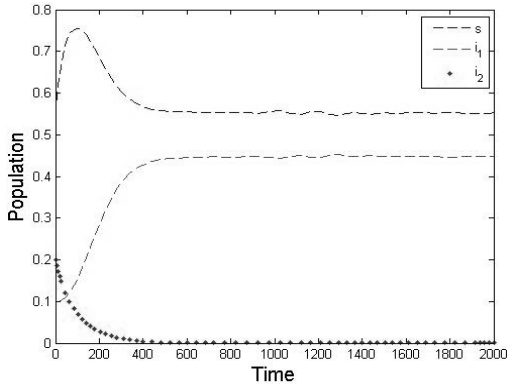


**Figure 6.2:** Parameter values are $\mu = 0.02, \beta_1 = .0362, \beta_2 = 0.012, \beta_{21} = 0.009,\ \delta = 0.0005,$ $s(0) = .56$ , $i_1(0) = 0.1$, $i_2(0) = 0.2$ , $t = [0\ , 2000]$ .

From the parameter values, $\Re_0^1 = 1.81 > 1$ and $\Re_0^2 = 0.6 < 1$.It has been observed from fig.6.2, initially $i_1(0) = 0.1$, $i_2(0) = 0.2$ and $i_2$ goes to zero when time increase but $i_1$ still exists in the population. Therefore, there is some population in infective class for $i_1$ , so disease does not die out completely and this steady state is unstable.

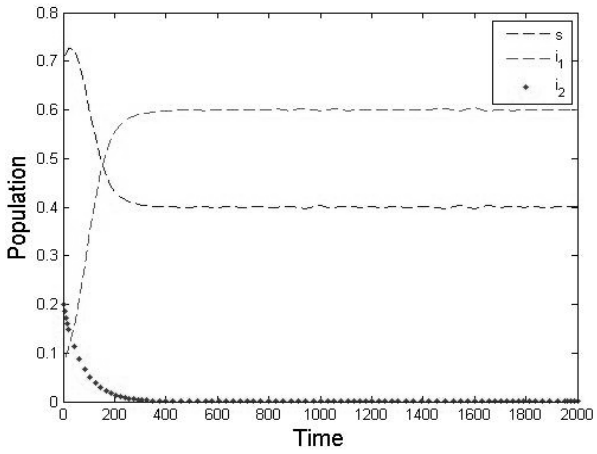


**Figure 6.3:** Parameter values are $\mu = 0.02, \beta_1 = .05, \beta_2 = 0.009, \beta_{21} = 0.004, \delta = 0.006$, $s(0) = .70$ , $i_1(0) = 0.08$, $i_2(0) = 0.2$ , $t = [0 , 2000]$ .

Here $\Re_0^1 = 2.5 > 1$, $\Re_0^2 = 0.45 < 1$, $\beta_1 > \beta_2$ and initially $i_1(0) = 0.08, i_2(0) = 0.2$. It has been observed from fig.6.3, $i_2$ goes to zero when time increase but $i_1$ still exists in the population, even though initially $i_2 > i_1$. Therefore, boundary endemic steady state $E_1$ is locally asymptotically stable.

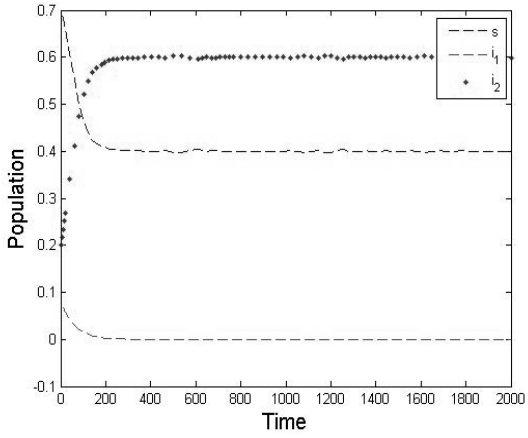


**Figure 6.4:** Parameter values are $\mu = 0.02, \beta_1 = .009, \beta_2 = 0.05, \beta_{21} = 0.004, \delta = 0.006$, $s(0) = .70$ , $i_1(0) = 0.08$, $i_2(0) = 0.2$ , $t = [0 , 2000]$ .

Here $\Re_0^1 = 0.45 < 1$, $\Re_0^2 = 2.5 > 1$, $\beta_1 < \beta_2$ and initially $i_1(0) = 0.08, i_2(0) = 0.2$ . It has been observed from fig.6.4, $i_1$ goes to zero when time increase but $i_2$ still exists in the population, even though all the parameter values in fig. 6.4 are same as

fig. 6.3 except $\beta_1$ and $\beta_2$ .Therefore, boundary endemic steady state $E_1$ is unstable when $\beta_1 < \beta_2$ .

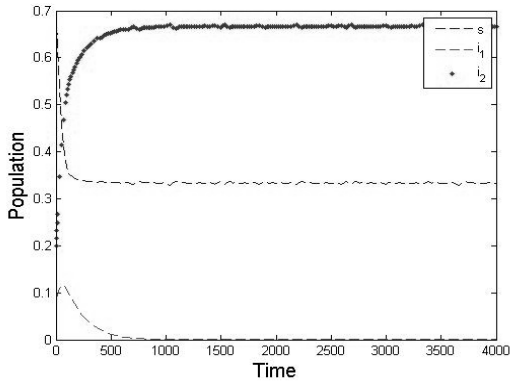


**Figure 6.5:** Parameter values are $\mu = 0.02, \beta_1 = .05, \beta_2 = 0.06, \beta_{21} = 0.004, \delta = 0.0006$, $s(0) = .70$ , $i_1(0) = 0.08$, $i_2(0) = 0.2$ , $t = [0\ ,\ 4000]$ .

From parameter values $\Re_0^1 = 2.5 > 1$, $\Re_0^2 = 3.0 > 1$, $\beta_2 > \beta_1$ and initially $i_1(0) = 0.08$, $i_2(0) = 0.2$ . It has been observed from fig.6.5, $i_1$ goes to zero when time increase but $i_2$ still exists in the population and it maintains an equilibrium state. Therefore, boundary endemic steady state $E_2$ is locally asymptotically stable.

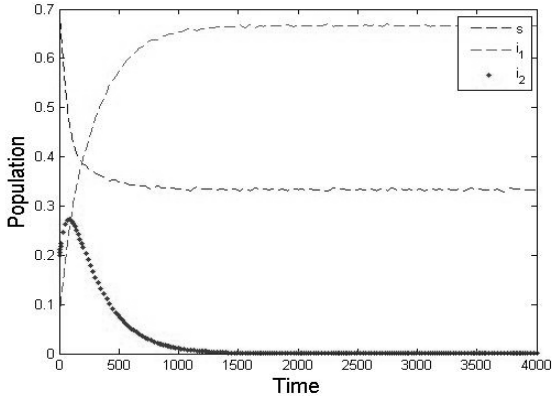


**Figure 6.6:** Parameter values are $\mu = 0.02, \beta_1 = .06, \beta_2 = 0.04, \beta_{21} = 0.004, \delta = 0.0006$ $s(0) = .70$ , $i_1(0) = 0.08$, $i_2(0) = 0.2$ , $t = [0\ ,\ 4000]$ .

From parameter values $\Re_0^1 = 3.0 > 1$, $\Re_0^2 = 2.0 > 1$, $\beta_1 > \beta_2$ and initially $i_1(0) = 0.08$, $i_2(0) = 0.2$ . It has been observed from fig.6.6, $i_2$ goes to zero when

time increase but $i_1$ still exists in the population, even though all the parameter values in fig. 6.6 are same as fig. 6.5 except $\beta_1$ and $\beta_2$ .Therefore, boundary endemic steady state $E_2$ is unstable when $\beta_2 < \beta_1$.

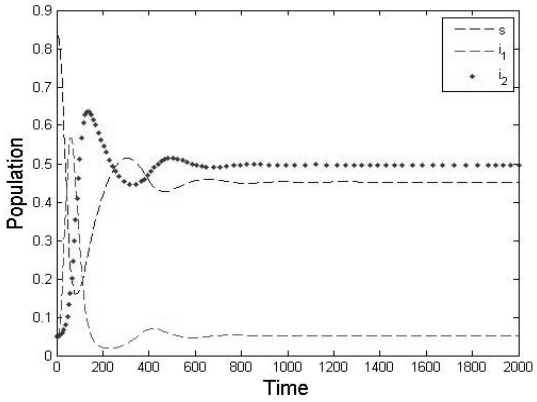


**Figure 6.7:** Parameter values are $\mu = 0.01, \beta_1 = .11, \beta_2 = 0.013, \beta_{21} = 0.08, \delta = 0.006$ $s(0) = .85$ , $i_1(0) = 0.03$, $i_2(0) = 0.05$ , $t = [0\ , 2000]$.

From parameter values $\Re_0^1 = 11 > 1$, $\Re_0^2 = 1.3 > 1$ and initially $i_1(0) = 0.03$, $i_2(0) = 0.05$. It has been observed from fig.6.7, $i_1$ and $i_2$ do not go to zero when time increase but $i_1$ and still $i_2$ exists in the population. Therefore, coexistence endemic steady state is stable.

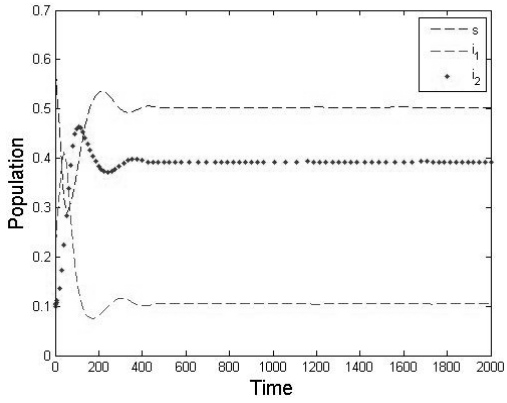


**Figure 6.8:** Parameter values are $\mu = 0.02, \beta_1 = .11, \beta_2 = 0.021, \beta_{21} = 0.09, \delta = 0.0005,$ $s(0) = .60$ , $i_1(0) = 0.2$, $i_2(0) = 0.1$ , $t = [0\ , 2000]$.

Here $\Re_0^1 = 5.5 > 1$, $\Re_0^2 = 1.05 > 1$ and initially $i_1(0) = 0.2$, $i_2(0) = 0.1$ .It has been observed from fig.6.8, for different parameter values $i_1$ and $i_2$ do not go to zero when time increase but $i_1$ and still $i_2$ exists in the population. Therefore, coexistence endemic steady state is stable. Hence, it has been observed from fig 6.7 and 6.8 that for different parameter values coexistence endemic state exist and stable.

## 7. Conclusion

Numerical simulations have been performed in section 6 and it has been observed that super infection has a great impact on the result of numerical simulations (i.e. dynamics of the disease of HIV), because it has changed the dynamics of the disease due to the change of the value of super infection transmission coefficient $\beta_{21}$. Therefore, from the analytical results in section 4 & 5, and numerical simulations in section 6, the following conclusion may be drawn:

1. Disease free equilibrium is locally asymptotically stable and globally asymptotically stable if $\Re_0^k < 1$, $k = 1, 2$, and unstable if either of $\Re_0^k > 1$ .

2. The boundary endemic steady state $E_1$ is locally asymptotically stable if

$\beta_1 > \beta_2$ and $\left|\frac{1}{\Re_0^1}(\beta_2 - \beta_1)\right| > \left|\beta_{21}\frac{\mu}{\beta_1}(\Re_0^1 - 1)\right|$, and unstable if $\beta_1 < \beta_2$.

3. The boundary endemic steady state $E_2$ is locally asymptotically stable for the following cases:

(i) $\beta_2 > \beta_1$

(ii) $(\beta_1 - \beta_2) > 0$ and $\left|\frac{1}{\Re_0^2}(\beta_1 - \beta_2)\right| < \left|\beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1)\right|$ ,

and unstable if

$(\beta_1 - \beta_2) > 0$ , and $\left|\frac{1}{\Re_0^2}(\beta_1 - \beta_2)\right| > \left|\beta_{21}\frac{\mu}{\beta_2}(\Re_0^2 - 1)\right|$

4. From fig 6.7 and 6.8, it has been observed that for different parameter values, the coexistence endemic steady state exists and stable.

## References


[1] Kault, D. A.,1992,"Modelling the effects of AIDS on gonorrhea epidemiology," Math. Comput. Model., 16 , pp. 3-14.

[2] Massad, E., Burattini, M. N., and Coutinho, F. A. B., et al., 1993, "Modelling the interaction between AIDS and tuberculosis," Math. Comput. Model., 17, pp. 7-21.

[3] Kirschner, D., 1999, "Dynamics of co- infection with M. tuberculosis and HIV-1," Theor. Pop. Biol., 55, pp. 94-109.

[4] Castillo-Chavez, C., and Feng, Z., 1997, "To treat or not to treat: the case of tuberculosis," J. Math. Biol., 35**,** pp. 629-656.

[5] Lin, J., Andreasen, V., and Levin, S. A., 1999, "Dynamics of influenza A drift: the linear three-strain model," Math. Biosci. , 162, pp.33-51.

[6] Nuno, M., Feng, Z., Martcheva, M., and Castillo-Chavez, C., 2005, "Dynamics of two strain influenza with isolation and partial cross immunity," SIAM J. APPL. MATH. , Vol. 65( 3), pp. 964–982.

[7] Levin, J., 2003, "Super-infection and Co- infection," 12th international HIV drug resistance workshop, June 10-14, Los Cabos, Mexico. http://www.natap.org/2003/resistance/day2.htm

[8] Marcus, J. L., McConnell, J. J., Robert, M.A., Grant, M., MD, MPH, 2005, "HIV super infection vs dual initial infection: what clinicians and patients should know," Medscape HIV/AIDS. , 11(1) .

[9] http://www.medscape.com/viewarticle/504811

[10] Rong, L., Gilchrist, M., Feng, Z., and Perelson, A., 2007, "Modeling within-host HIV-1 dynamics and the evolution of drug resistance: Trade-offs between viral enzyme function and drug susceptibility," J. Theoretical Biology, 247 , pp. 804-818.

[11] Angel, J. B., Kravcik, S. , Balaskas, E. , Yen, P. , Badley, A.D., Cameron, D.W., and Hu., Y.W, 2000, "Documentation of HIV-1 super infection and acceleration of disease Progression ," Ottawa Hosp. and Canadian Blood Services, Ottawa, Canada. http://www.thebody.com/content/treat/art1936.html

[12] Rong, L., Feng, Z., and Perelson, A., 2007, "Emergence of HIV-1 drug resistance during antiretroviral treatment," Bulletin of Mathematical Biology,69,pp.2027-2060.

[13] Brauer, F., Driessche, P.V., and Watmough, J., Eds. 1945/2008, "Mathematical Epidemiology, Mathematical Biosciences Subseries," Springer Berlin.