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The Effect of the HIV/AIDS Epidemic on the Population of Truck Drivers in South Africa and its Economic Impact

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Abstract

As of the end of 2001, an estimated 28,100,000 people in Sub-Saharan Africa (SSA) had been infected with the HIV virus, making up 70% of the world's population of HIV-infected people. Such an aggressive epidemic has not only impacted the SSA demography, but also the African economy and culture. The epidemic has begun to seriously damage the transport sector, where long distance truck drivers (TDs) are at an increased risk of infection due to their frequent contacts with commercial sex workers (CSWs). The spread of AIDS in the transport industry is especially significant to the SSA economy as truck drivers are largely responsible for transporting crops and supplies needed for daily subsistence. In this project we present four mathematical models that describe the interaction between the TDs and CSWs from various perspectives. From the analysis and simulation of these models we qualify the decrease in the TD population due to the HIV/AIDS epidemic and discuss its impact on the transportation industry and the SSA economy in general.

1 Introduction

HIV/AIDS is one of the leading causes of death in the world and is especially destructive in Africa. As of the end of 2001, an estimated 28,100,000 people in Sub-Saharan Africa (SSA) have been infected with the HIV virus, making up 70% of the world's population of HIV-infected people. The estimated number of adults and children newly infected with HIV in 2001 is a startling 3,400,000, and the number of adult and child deaths due to HIV/AIDS in 2001 is 2,300,000 [15]. In fact, the levels of infection are so high that the number of deaths in the next decades may result in population decline [24].

An epidemic of such magnitude has serious repercussions on the African society and economy. The AIDS epidemic is particularly damaging to the transportation sector, where long distance truck drivers (TDs) are at an increased risk of infection due to the migratory nature of their job and their prolonged absence from home. As a result, TDs are more likely to have sexual interactions with commercial sex workers (CSWs), who often provide them with affordable food and lodging during their journeys. The spread of AIDS is further exacerbated by the highly sexually active lifestyles of both the TDs and the prostitutes they visit. Many of the TDs and CSWs have multiple sexual partners (i.e. many of the TDs are involved in polygamous relationships), and very few of them use condoms. A number of studies show a high prevalence of AIDS among long-distance TDs in SSA (56%), and a corresponding high prevalence of the virus among CSWs and their clients (56%) [7, 8, 26, 22]. In some studies the prevalence rates in these high-risk groups were as high as 95% [26].

The spread of AIDS in the transport industry is especially significant to the SSA economy. The TDs are largely responsible for transporting a majority of the goods and supplies needed for daily subsistence. Badly

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affected areas are losing a large percentage of these valuable skilled drivers to the AIDS epidemic. As more experienced drivers are lost, it may become costly to hire and train new recruits. Moreover, the prevalence of AIDS among experienced TDs is higher than that among the less experienced drivers since these TDs generally have higher wages and can afford repeated visits with CSWs, making their chances of infection much greater [7, 8]. Once TDs have contracted HIV, their physical health diminishes, resulting in reduced efficiency for the transport industry as a whole.

In this study, we investigate the effect of the HIV/AIDS epidemic on the population of TDs and CSWs in SSA, and more specifically its impact on the transport industry economy. Both deterministic and stochastic models are used to study the evolution of HIV/AIDS in the population of TDs and CSWs. The first deterministic model considers a constant population of TDs; i.e., we assume that for every TD lost to the disease, a new susceptible TD is available to replace him. In the second model, the population of TDs changes as the epidemic progresses. Specifically, we assume that there exists a maximum number of men available to be hired as TDs. As the epidemic worsens, it is possible that the number of TDs lost due to HIV exceeds the number of TDs available for hiring. In the third model we consider a switch between the first two models. Since recovery from HIV/AIDS infection is not possible, we use SIR models to describe the population dynamics, where in this paper S denotes the class of susceptible individuals, I the class of infected inviduals, and R the class of individuals removed due to the disease. For these SIR models, we derive explicit formulas for the contact numbers and the disease-free and endemic equilibria. Finally, we consider a stochastic SIS model to project the number of TDs needed over time, to replace the removed TDs. Here we will assume that TDs are replaced after death, and we monitor the number of transitions from the I to the S compartments in order to determine the number of TDs that die from HIV/AIDS.

2 Deterministic Models for the Spread of AIDS in High Risk Groups

Deterministic models are often used to model the effects of epidemics within a population. We will use three deterministic models to observe the effects of HIV/AIDS in the TD population and the economic implications. To make analysis of the problem simpler, we will focus on the core group population or a small population of individuals who are extremely infectious and have a high probability of becoming infected. This concept, adopted in [21], is based on the assumption that the core group members have a significantly more active sexual lifestyle than the general population and are largely responsible for a disease explosion within the total population. Kribs-Zaleta [21] used an SIS model for transmission of sexually transmitted diseases from the core group population into the general population. In our models, we concentrate on the core groups and the transmission of HIV between them. In South Africa, AIDS is primarily transmitted through heterosexual activities so we will only consider male to female transmission and female to male transmission [24]. Thus we focus on the transmission of AIDS among the female CSW and male TD core populations. We must also take into consideration the removal rate due to AIDS since there is no possible chance of recovering and returning to the susceptible class. Therefore we will use a two-sex SIR model, resulting in a 6-dimensional system. In all of our models we consider constant population for CSWs. In the first model, we assume the population of males available for recruitment as TDs is large enough to supply a replacement when a TD is removed due to HIV/AIDS infection, yielding a constant population. In the second model, we assume the removal rate due to HIV/AIDS and other factors exceeds the recruitment rate and the desired population will begin to decrease, resulting in a nonconstant population. The third model is a hybrid model, in which the recruitment rate is a switching function designed to switch between the previous two models, when the population of infecteds reaches a critical value. In order to calculate the basic reproductive or contact number, R_0, we use a method from spectral theory called the *next generation operator approach*, which is reviewed in [9]. In order to analyze the stability of the steady states, we linearize the systems by determining the Jacobian and analyzing the eigenvalues.

In summary, we investigate the following deterministic models:

1. *Core-Group Model with Constant Population*: Assume there will always be enough TD recruits to replace the removed TDs, and that all newly recruited TDs are non-infected. This will yield a constant population of TDs.

2. *Core-Group Model with Constant Recruitment*: Assume that the TD recruitment rate is a specified function $\Lambda(t)$. This will yield a non-constant population of TDs since the rate of removal of TDs will exceed the rate of recruitment of TDs.

3. *Core-Group Model with Switching Function*: Assume the recruitment rate is the minimum of the number of TDs needed and $\Lambda(t)$.

2.1 Notation

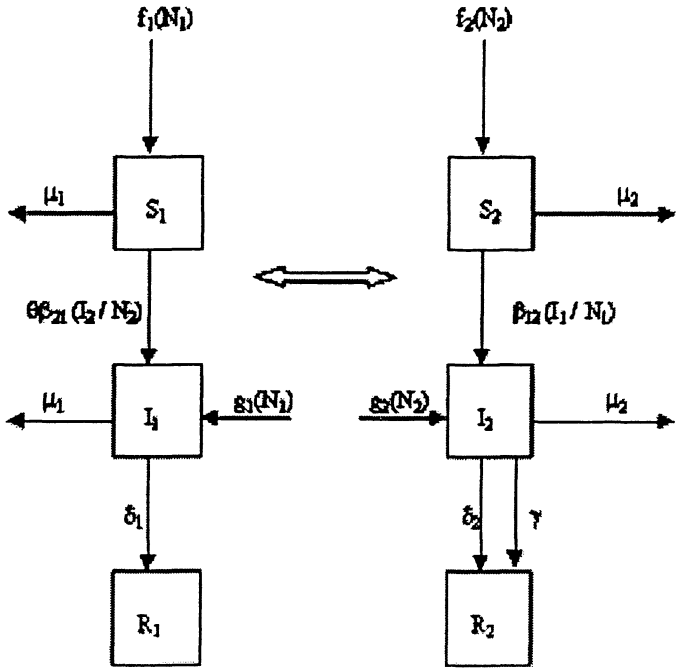

Figure 1: This figure represents the SIR core-group deterministic model.

Here all the parameters are nonnegative and an infected individual is considered infectious, so there is no latent period for the disease. Table 1 lists the notation used in this section.

The prevalence of the disease is defined as the number of cases of AIDS at a given time t, so it corresponds to $I_1(t) + I_2(t)$.

Let β_{ij} be the average number of adequate contacts to susceptibles in group j per infective from group i per year, i.e., the transmission or infection rate. Then the incidence, or the number of cases of AIDS transmission per year is $\beta_{ij}\frac{I_i}{N_i}S_j$. This is a mass action law since the proportion of new infections is proportional to the number of infectives in group i and the number of susceptibles in group j. The incidence $\beta_{12}\frac{I_1}{N_1}S_2$ may be interpreted as the number of cases in which a portion of infectious TDs ($\frac{I_1}{N_1}$) infects the class of susceptible CSWs (S_2), at a rate β_{12}.

We assume that the death of infected individuals due to AIDS occurs at a rate proportional to the size of the infected class, I_i, with proportionality constant δ_i. That is, δ_i is the annual death removal rate.

As TDs and CSWs may be removed from the susceptible and infective classes by other causes, let μ_i be the annual removal rate due to all non-AIDS related causes. Then the average working lifetime of an infected TD is $\frac{1}{\mu_1+\delta_1}$ years.

We suppose further that the CSWs may be screened for the disease and may subsequently be removed at a rate γ, proportional to the infected class I_2. Then the average active lifetime of an infected CSW is $\frac{1}{\mu_2+\delta_2+\gamma}$ years.

Recently, several organizations have worked with the African government to implement AIDS awareness and prevention campaigns, and have targeted long-distance TDs as one of their main outreach groups. Such

Parameter	Meaning
$S_1(t)$	the population of susceptible TDs
$I_1(t)$	the population of infected TDs
$R_1(t)$	the population of removed TDs
$N_1(t)$	the population of susceptible and infected TDs
$f_1(N_1)$	the recruitment rate of noninfected TDs
$g_1(N_1)$	the recruitment rate of infected TDs
μ_1	loss rate of TDs due to other factors besides AIDS
δ_1	loss rate of TDs due to AIDS
$\beta_{21} I_2/N_2$	the rate of infection of TDs by infected CSWs
θ	the reduction factor due to effectiveness of TD-targetted AIDS awareness and prevention campaigns
$S_2(t)$	the population of susceptible CSWs
$I_2(t)$	the population of infected CSWs
$R_2(t)$	the population of removed CSWs
$N_2(t)$	the population of susceptible and infected CSWs
$f_2(N_2)$	the rate of noninfected CSWs entering the truck stops
$g_2(N_2)$	the rate of infected CSWs entering the truck stops
μ_2	loss rate of CSWs due to other factors besides AIDS
δ_2	loss rate of CSWs due to AIDS
γ	removal rate of CSWs due to screening
$\beta_{12} I_1/N_1$	the rate of infection of CSWs by infected TDs

Table 1: Model parameters.

a campaign may involve the distribution of pamphlets and condoms, and the efficacy of the effort depends on how positively the TDs respond to the information and resources given to them. Several studies [7, 8, 26] show that despite their awareness of the threat of HIV, many TDs continue to have multiple sexual partners and often do not use condoms. The efficacy of the campaign is measured by the parameter θ, where $0 \leq \theta \leq 1$. When $\theta = 1$, the campaign is 0% efficient and when $\theta = 0$, the campaign is 100% efficient.

2.2 Deterministic Core-Group Model with Constant Population

2.2.1 Model Assumptions

In the following model, the number of TDs recruited/hired is the same as the number needed to compensate for all the TDs removed due to AIDS and other factors. Thus the recruitment rate of the TDs is $f_1(N_1) = \mu_1 N_1 + \delta_1 I_1$. The population of CSWs is constant so that the entrance rate of CSWs to the truck stops is $f_2(N_2) = \mu_2 N_2 + \delta_2 I_2 + \gamma I_2$. It will also be assumed that all newly recruited TDs and all entering CSWs are noninfected, so that $g_i(N_i) = 0$ for $i = 1, 2$.

Our model is given by the following system of equations:

$$\frac{dS_1}{dt} = \mu_1 N_1 + \delta_1 I_1 - \mu_1 S_1 - \theta \beta_{21} \frac{I_2}{N_2} S_1 \tag{1}$$

$$\frac{dI_1}{dt} = \theta \beta_{21} \frac{I_2}{N_2} S_1 - \mu_1 I_1 - \delta_1 I_1 \tag{2}$$

$$\frac{dR_1}{dt} = \delta_1 I_1 \tag{3}$$

$$\frac{dS_2}{dt} = \mu_2 N_2 + \delta_2 I_2 + \gamma I_2 - \mu_2 S_2 - \beta_{12} \frac{I_1}{N_1} S_2 \tag{4}$$

$$\frac{dI_2}{dt} = \beta_{12}\frac{I_1}{N_1}S_2 - \mu_2 I_2 - \delta_2 I_2 - \gamma I_2 \tag{5}$$

$$\frac{dR_2}{dt} = \delta_2 I_2 + \gamma I_2 \tag{6}$$

Since R_i is a function of I_i only, this system can be reduced to a four-dimensional system by ignoring equations (3) and (6). This is valid because R_i does not appear in any of the other equations, i.e., they are uncoupled. To further reduce the system, we will show $N_1 = S_1 + I_1$, $N_2 = S_2 + I_2$ are constant. Observe that:

$$\frac{dN_1}{dt} = \frac{dS_1}{dt} + \frac{dI_1}{dt} = \mu_1 N_1 - \mu_1 S_1 - \mu_1 I_1 = 0,$$

and

$$\frac{dN_2}{dt} = \frac{dS_2}{dt} + \frac{dI_2}{dt} = \mu_2 N_2 - \mu_2 S_2 - \mu_2 I_2 = 0,$$

since $N_i = S_i + I_i$, and using equations (1), (2), (4), and (5). Since $\frac{dN_i}{dt} = 0$, N_i is constant. As a result, we can eliminate S_i from the system by writing $S_i = N_i - I_i$, and thus we can reduce our system to two ordinary differential equations for I_1, I_2:

$$\frac{dI_1}{dt} = \theta\beta_{21}\frac{I_2}{N_2}(N_1 - I_1) - \mu_1 I_1 - \delta_1 I_1 \tag{7}$$

$$\frac{dI_2}{dt} = \beta_{12}\frac{I_1}{N_1}(N_2 - I_2) - \mu_2 I_2 - \delta_2 I_2 - \gamma I_2 \tag{8}$$

2.2.2 Disease-Free Equilibrium and Determination of R_0

Let $f = \frac{dI_1}{dt}$ and $g = \frac{dI_2}{dt}$. The disease-free equilibrium (DFE) is given by $(I_1^*, I_2^*) = (0,0)$. The following method for determining the first stage R_0, outlined in [9], involves the use of the next generation operator. We first determine that the Jacobian of the system is given by

$$J(I_1, I_2) = \begin{pmatrix} f_{I_1} & f_{I_2} \\ g_{I_1} & g_{I_2} \end{pmatrix} = \begin{pmatrix} -\theta\beta_{21}I_2/N_2 - (\mu_1 + \delta_1) & \theta\beta_{21}(N_1 - I_1)/N_2 \\ \beta_{12}(N_2 - I_2)/N_1 & -\beta_{12}I_1/N_1 - (\mu_2 + \delta_2 + \gamma) \end{pmatrix}, \tag{9}$$

where $f_{I_i} = \partial f/\partial I_i$ and $g_{I_i} = \partial g/\partial I_i$. Evaluating at the DFE, we get

$$J(0,0) = \begin{pmatrix} -(\mu_1 + \delta_1) & \theta\beta_{21}N_1/N_2 \\ \beta_{12}N_2/N_1 & -(\mu_2 + \delta_2 + \gamma) \end{pmatrix}.$$

As there are no susceptible or noninfectious infected populations, we can simply consider the subclass of infected populations, $Z = (I_1, I_2)$. We have

$$\frac{\partial Z}{\partial t} = (f, g),$$

and

$$\frac{\partial}{\partial t}\left(\frac{\partial Z}{\partial t}\right) = \frac{\partial}{\partial t}(f, g) = J(0,0).$$

Writing $J(0,0)$ as $M - D$, where M is a matrix with entries $m_{ij} \geq 0$ and D is a diagonal matrix with entries $d_{ii} > 0$, we get

$$M = \begin{pmatrix} 0 & \theta\beta_{21}N_1/N_2 \\ \beta_{12}N_2/N_1 & 0 \end{pmatrix}$$

and

$$D = \begin{pmatrix} \mu_1 + \delta_1 & 0 \\ 0 & \mu_2 + \delta_2 + \gamma \end{pmatrix}.$$

After solving the characteristic equation associated with MD^{-1},

$$\lambda^2 - \frac{\theta\beta_{12}\beta_{21}}{(\mu_1+\delta_1)(\mu_2+\delta_2+\gamma)} = 0$$

for the maximum eigenvalue, we get

$$R_0 = \sqrt{\frac{\theta\beta_{12}\beta_{21}}{(\mu_1+\delta_1)(\mu_2+\delta_2+\gamma)}}.$$

This represents the average number of people infected by one infective after the first stage of infection. As shown, R_0 is the square root of the product of the infection rates $\theta\beta_{21}$, β_{12} and the average lifetimes of the infected TDs and CSWs: $\frac{1}{\mu_1+\delta_1}$, $\frac{1}{\mu_2+\delta_2+\gamma}$. As $0 \leq \theta \leq 1$, then if R_0^* represents the average number of infectious contacts without an AIDS prevention campaign (i.e. $\theta = 1$) and R_0 is the average number of contacts with an AIDS campaign, then we see that $R_0 = \sqrt{\theta}R_0^*$, so that if the campaign is effective, it will reduce the contact number, and thereby reduce the chance that the disease will become endemic.

Next we determine the conditions under which the DFE is stable. The trace tr $J(0,0) = -(\mu_1+\delta_1) - (\mu_2+\delta_2+\gamma)$ is always negative, and the determinant is given by det $J(0,0) = (\mu_1+\delta_1)(\mu_2+\delta_2+\gamma) - \theta\beta_{12}\beta_{21}$. As the determinant must be positive in order for the DFE to be stable, the condition for stability is

$$(\mu_1+\delta_1)(\mu_2+\delta_2+\gamma) - \theta\beta_{12}\beta_{21} > 0,$$

or

$$\frac{\theta\beta_{12}\beta_{21}}{(\mu_1+\delta_1)(\mu_2+\delta_2+\gamma)} < 1. \tag{10}$$

The left-hand side of (10) represents the average number of secondary infections caused by one infective at the second stage of infection, or the second-stage basic reproductive/contact number, R_0^2. Effectively, this value is the average number of TDs infected, via sexual interaction with CSWs, by an infected TD. We can observe that the left-hand side of the equation $\frac{\theta\beta_{12}\beta_{21}}{(\mu_1+\delta_1)(\mu_2+\delta_2+\gamma)} = R_0^2$. From this we deduce that the condition for stability is $R_0^2 < 1$.

2.2.3 Endemic Equilibrium and Stability Analysis

Solving the the system $f = 0$, $g = 0$ for I_1 and I_2, we find (in addition to the DFE) the endemic equilibrium (EE), which is given by

$$(I_1^*, I_2^*) = \left(-\frac{N_1(\mu_1\mu_2+\mu_1\delta_2+\mu_1\gamma+\delta_1\mu_2+\delta_1\delta_2+\delta_1\gamma-\theta\beta_{21}\beta_{12})}{(\theta\beta_{21}+\mu_1+\delta_1)\beta_{12}}, \quad -\frac{N_2(\mu_1\mu_2+\mu_1\delta_2+\mu_1\gamma+\delta_1\mu_2+\delta_1\delta_2+\delta_1\gamma-\theta\beta_{21}\beta_{12})}{\theta\beta_{21}(\mu_2+\delta_2+\gamma+\beta_{12})} \right). \tag{11}$$

Substituting the EE into the Jacobian, we find that the trace is

$$\text{tr } J(EE) = -\frac{(\theta\beta_{21}+\mu_1+\delta_1)\beta_{12}}{\mu_2+\delta_2+\gamma+\beta_{12}} - \frac{\theta\beta_{21}(\mu_2+\delta_2+\gamma+\beta_{12})}{\theta\beta 21+\mu_1+\delta_1},$$

which is always negative. When we calculate the determinant, we find

$$\det J(EE) = \theta\beta_{21}\beta_{12} - (\mu_1+\delta_1)(\mu_2+\delta_2+\gamma),$$

which is positive when $R_0^2 > 1$.

Thus the basic reproductive number R_0, is a threshold indicator of whether or not the AIDS epidemic will become endemic. If $R_0 < 1$, the disease will eventually stabilize and die off, and if $R_0 > 1$, the epidemic will become endemic.

2.2.4 Global Stability of the DFE and EE

We next show that the local stability of both the DFE and the EE extend to global stability. The Poincaré-Bendixson Theorem (p. 155ff. in [4]) states that for a two-dimensional system of ODE's[1] any solution will either:

1. approach an equilibrium,
2. grow without bound, or
3. approach a periodic solution/orbit.

We first show that in our system, cases 2 and 3 do not occur. We are then left with case 1, which means that any solution will approach an equilibrium. However, since at any given time there is only one equilibrium that is locally stable (DFE if $R_0 < 1$ or EE if $R_0 > 1$), all solutions which fail 2 and 3 at a given time will approach the same equilibrium; thus the DFE and EE are globally stable in this case.

First we check that our system is well-posed [2] in the state space $D = \{(I_1, I_2) | \ 0 \leq I_i \leq N_i\}$. D is a rectangular region with vertices $(0,0), (N_1,0), (0,N_2), (N_1,N_2)$. In order to check for well-posedness, we analyze the points along the edges of the rectangle, which leads us to four cases:

CASE 1: POINTS LYING ON THE TOP OF THE RECTANGLE. Consider the points along the top horizontal edge of D. These points are of the form $P_{top} = (I_1, N_2)$, where $0 \leq I_1 \leq N_1$. In order to be well-posed, the solution must decrease in the vertical direction (in the direction of I_2). But we have that $I_2' = -(\mu_2 + \delta_2 + \gamma)N_2 < 0$, so I_2 decreases as needed. Physically, this means that when the number of infected CSWs is equal to the total population of CSWs, the infected CSW population will inevitably decrease because it cannot grow and exceed the total number of CSWs (N_2).

CASE 2: POINTS LYING ON THE RIGHT EDGE OF D. These points are of the form $P_{right} = (N_1, I_2)$, where I_2 lies between 0 and N_2. We have that $I_1' = -(\mu_1 + \delta_1)N_1 < 0$, so that I_1 decreases back into the state space. This means that when the number of infected TDs equals the total number of TDs, the infected TD population will decrease back into the state space.

CASE 3: POINTS LYING ON THE BOTTOM OF D. These points are of the form $P_{bot} = (I_1, 0)$. We have that $I_2' = \beta_{12} I_1 N_2 / N_1 > 0$, so that if there are no infected CSWs, the population of infected CSWs will increase due to AIDS transmission from the infected TDs.

CASE 4: POINTS LYING ON THE LEFT EDGE OF D. Here the points have the form $P_{left} = (0, I_2)$. We have that $I_1' = \theta \beta_{21} I_2 N_1 / N_2 > 0$. Thus, if there are no infected TDs, the population of infected TDs can only increase. These results make biological sense, and they confirm that the system is well-posed. The well-posedness of the model further implies that the solutions are bounded.

Next we show that there are no periodic solutions in our system. Let $f = \frac{dI_1}{dt}$ and $g = \frac{dI_2}{dt}$. Taking partial derivatives, we obtain $\frac{\partial f}{\partial I_1} = -\theta \beta_{21} I_2 / N_2$ and $\frac{\partial g}{\partial I_2} = -\beta_{12} I_1 / N_1 - (\mu_2 + \delta_2 + \gamma)$, so that the sum $\frac{\partial f}{\partial I_1} + \frac{\partial g}{\partial I_2}$ is strictly negative. By the Bendixson-Dulac Criterion (p. 159 in [4]), this shows there are no periodic orbits. It can be concluded that because the model is well-posed and has no periodic solutions, it must approach an equilibrium, and therefore the DFE and EE are globally asymptotically stable.

2.2.5 Numerical Analysis

Here we check numerically the above result that the solution starting from any arbitrary point will tend toward either the DFE or the EE depending on the choice of parameters. Using the parameter values $\beta_{12} = \beta_{21} = 0.2$, $\mu_i = \delta_i = 0.125$, $N_1 = N_2 = 100$ yields an $R_0 < 1$, so we would expect the solutions to approach the stable DFE. As shown in Figure 2 (left), the solutions of the system starting from initial points indeed go to the DFE (0,0).

When the parameters are adjusted so that $R_0 > 1$, we see that the solutions which begin from initial points will tend toward the EE (Figure 2, right). Here, the parameters used are $\beta_{12} = \beta_{21} = 0.7$, $\mu_i = \delta_i = 0.3$, $N_1 = N_2 = 100$, and the EE is approximately (14.28,14.28) as calculated from equation (11).

[1] The Poincaré-Bendixson Theorem also requires that the system be autonomous and that it satisfies additional continuity conditions, which we do not explicitly show here.

[2] We call a system well-posed if it has the following properties: (1) if a solution exists, it is unique; (2) Under suitable restrictions on the data, there is a solution; (3) The solution depends continuously on data [16].

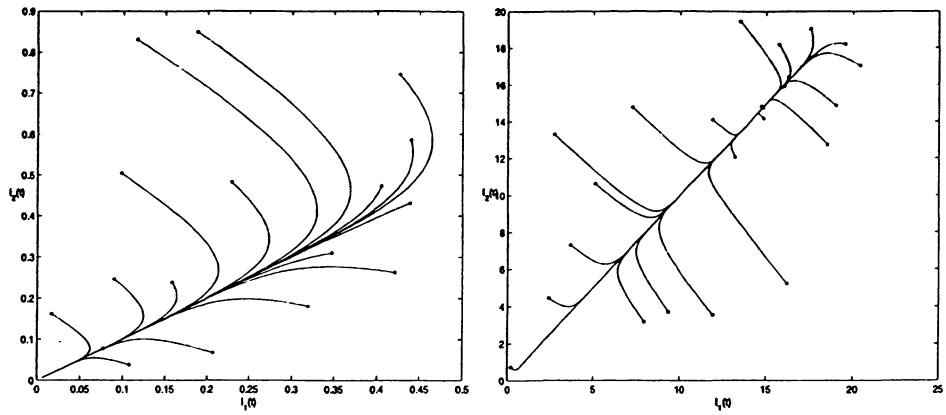

Figure 2: Left: Stability of the DFE. The dots denote the initial points that are used in calculating the solutions to the model system. As shown, all the solution curves converge to the DFE. Right: Stability of the EE. The EE is located at approximately (14.28,14.28).

2.3 Deterministic Core-Group Model with Constant Recruitment

2.3.1 Model Assumptions

Suppose now, the number of TDs removed due to AIDS and other factors exceeds the number of TDs recruited for each unit of time. We would like to observe the changes in the TD population when the availability of TDs is limited. In particular, suppose the recruitment rate $f_1(N_1)$ is a constant, Λ. As in the previous model, we assume that the population of CSWs is constant, and that all incoming TDs and CSWs are noninfected. Thus $f_2(N_2) = \mu_2 N_2 + \delta_2 I_2 + \gamma I_2$ and $g_i(N_i) = 0$ for $i = 1, 2$.

Now we are ready to formulate our model, which is described by the following system of equations:

$$\frac{dS_1}{dt} = \Lambda - \mu_1 S_1 - \theta\beta_{21}\frac{I_2}{N_2}S_1 \tag{12}$$

$$\frac{dI_1}{dt} = \theta\beta_{21}\frac{I_2}{N_2}S_1 - \mu_1 I_1 - \delta_1 I_1 \tag{13}$$

$$\frac{dR_1}{dt} = \delta_1 I_1 \tag{14}$$

$$\frac{dS_2}{dt} = \mu_2 N_2 + \delta_2 I_2 + \gamma I_2 - \mu_2 S_2 - \beta_{12}\frac{I_1}{N_1}S_2 \tag{15}$$

$$\frac{dI_2}{dt} = \beta_{12}\frac{I_1}{N_1}S_2 - \mu_2 I_2 - \delta_2 I_2 - \gamma I_2 \tag{16}$$

$$\frac{dR_2}{dt} = \delta_2 I_2 + \gamma I_2 \tag{17}$$

Since R_i is a function of I_i, this system can be reduced to a four-dimensional system. To further reduce the system, we set $N_2 = S_2 + I_2$ which is constant:

$$\frac{dN_2}{dt} = \frac{dS_2}{dt} + \frac{dI_2}{dt} = \mu_2 N_2 - \mu_2 S_2 - \mu_2 I_2 = 0.$$

Writing $N_1 = S_1 + I_1$ and $S_2 = N_2 - I_2$, the system can be reduced to a three-dimensional system:

$$\frac{dS_1}{dt} = \mu_1 S_0 - \mu_1 S_1 - \theta\beta_{21}\frac{I_2}{N_2}S_1 \tag{18}$$

$$\frac{dI_1}{dt} = \theta\beta_{21}\frac{I_2}{N_2}S_1 - (\mu_1 + \delta_1)I_1 \tag{19}$$

$$\frac{dI_2}{dt} = \beta_{12}\frac{I_1(N_2 - I_2)}{(S_1 + I_1)} - (\mu_2 + \delta_2 + \gamma)I_2 \tag{20}$$

where $S_0 = \Lambda/\mu_1$.

2.3.2 Disease-Free Equilibrium and Determination of R_0

Let $F = \frac{dS_1}{dt}$, $G = \frac{dI_1}{dt}$, and $H = \frac{dI_2}{dt}$. The DFE is given by $(S_1^*, I_1^*, I_2^*) = (S_0, 0, 0)$.

By applying the next generation operator approach outlined in the previous section, we determine R_0. Because there are no noninfectious infected populations, we consider the subclasses of susceptible populations, $X = (S_1)$, and infected populations, $Z = (I_1, I_2)$. We have

$$\frac{\partial Z}{\partial t} = (G, H),$$

and

$$\frac{\partial}{\partial t}\left(\frac{\partial Z}{\partial t}\right) = \frac{\partial}{\partial t}(G, H) = A.$$

where A is a 2×2 matrix given by:

$$A = \begin{pmatrix} F_{I_1} & F_{I_2} \\ G_{I_1} & G_{I_2} \end{pmatrix} = \begin{pmatrix} -\delta_1 - \mu_1 & (\theta\beta_{21}S_1)/(N_2) \\ \beta_{12}(N_2 - I_2)((1/(S_1 + I_1)) - (1/(S_1 + I_1)^2)) & -\beta_{12}(I_1/(S_1 + I_1)) - \mu_2 - \delta_2 - \gamma \end{pmatrix}. \tag{21}$$

Evaluating at the DFE, we obtain

$$A(S_0, 0, 0) = \begin{pmatrix} -\delta_1 - \mu_1 & (\theta\beta_{21}S_0)/N_2 \\ \beta_{12}/S_0 & -\mu_2 - \delta_2 - \gamma \end{pmatrix}.$$

Writing $A(S_0, 0, 0)$ as $M - D$, we obtain

$$M = \begin{pmatrix} 0 & (\theta\beta_{21}S_0)/N_2 \\ \beta_{12}/S_0 & 0 \end{pmatrix}$$

$$D = \begin{pmatrix} \delta_1 + \mu_1 & 0 \\ 0 & \mu_2 + \delta_2 + \gamma \end{pmatrix}.$$

After solving the characteristic equation of MD^{-1},

$$\lambda^2 - \frac{\theta\beta_{12}\beta_{21}}{(\mu_1 + \delta_1)(\mu_2 + \delta_2 + \gamma)} = 0$$

for the maximum eigenvalue, we obtain

$$R_0 = \sqrt{\frac{\theta\beta_{12}\beta_{21}}{(\mu_1 + \delta_1)(\mu_2 + \delta_2 + \gamma)}}.$$

Note that the contact number R_0 is the same as that of the constant population model in the previous section. A possible explanation for this similarity is that HIV's ability to invade the population depends

only on conditions affecting the relative growth and loss of infective classes, and not the size the of susceptible pool ($S_1 = N_1$ or S_0), which is the only difference between the two models.

Now we can determine the stability of the DFE by analyzing the Jacobian of the three-dimensional system (18), (19), and (20).

$$J = \begin{pmatrix} F_{S_1} & F_{I_1} & F_{I_2} \\ G_{S_1} & G_{I_1} & G_{I_2} \\ H_{S_1} & H_{I_1} & H_{I_2} \end{pmatrix} = \begin{pmatrix} -\mu_1 - \frac{\theta\beta_{21}I_2}{N_2} & 0 & \frac{-\theta\beta_{21}S_1}{N_2} \\ \frac{\theta\beta_{21}I_2}{N_2} & -(\mu_1+\delta_1) & \frac{\theta\beta_{21}S_1}{N_2} \\ \frac{-\beta_{12}I_1(N_2-I_1)}{(S_1+I_1)^2} & \frac{\beta_{12}(N_2-I_2)}{S_1+I_1} - \frac{\beta_{12}I_1}{S_1+I_1} - \frac{\beta_{12}I_1(N_2-I_1)}{(S_1+I_1)^2} & -(\mu_2+\delta_2+\gamma) \end{pmatrix}. \tag{22}$$

Evaluating the Jacobian at the DFE, we obtain

$$J(S_0,0,0) = \begin{pmatrix} -\mu_1 & 0 & \frac{-\theta\beta_{21}S_0}{N_2} \\ 0 & -(\mu_1+\delta_1) & \frac{\theta\beta_{21}S_0}{N_2} \\ 0 & \frac{\beta_{12}N_2}{S_0} & -(\mu_2+\delta_2+\gamma) \end{pmatrix}.$$

The associated characteristic polynomial is given by

$$\begin{aligned} \lambda^3 + \lambda^2(\mu_2+\delta_2+\gamma+2\mu_1+\delta_1) &+ \lambda((\mu_1+\delta_1)(\mu_2+\delta_2+\gamma) - \theta\beta_{12}\beta_{21} + \mu_1(\mu_2+\delta_2+\gamma+\mu_1+\delta_1)) \\ &+ \mu_1((\mu_1+\delta_1)(\mu_2+\delta_2+\gamma) - \theta\beta_{12}\beta_{21}). \end{aligned}$$

Let us denote the coefficients of the characteristic polynomial as

$$\begin{aligned} a_1 &:= \mu_2+\delta_2+\gamma+2\mu_1+\delta_1; \\ a_2 &:= (\mu_1+\delta_1)(\mu_2+\delta_2+\gamma) - \theta\beta_{12}\beta_{21} + \mu_1(\mu_2+\delta_2+\gamma+\mu_1+\delta_1); \\ a_3 &:= \mu_1((\mu_1+\delta_1)(\mu_2+\delta_2+\gamma) - \theta\beta_{12}\beta_{21}). \end{aligned}$$

To check for the stability of the DFE, we apply the Routh-Hurwitz criterion (p. 216 in [4]), which requires that $a_1 > 0$, $a_3 > 0$, and $a_1a_2 > a_3$. By inspection, a_1 is clearly positive. a_3 is positive exactly when $(\mu_1+\delta_1)(\mu_2+\delta_2+\gamma) - \theta\beta_{12}\beta_{21} > 0$, or when $R_0^2 < 1$. Finally, $a_1a_2 > a_3$ if and only if

$$(2\mu_1+\delta_1)(\mu_2+\mu_1+\gamma+\delta_2) - \theta\beta_{12}\beta_{21} > 0. \tag{23}$$

However, if $R_0^2 < 1$, then (23) holds since (23) is less restrictive than requiring $R_0^2 < 1$. Thus we see that the DFE is locally stable by the Routh-Hurwitz criterion when $R_0^2 < 1$.

2.3.3 Endemic Equilibrium and Stability Analysis

Substituting equations (18) and (19) into $N_1' = S_1' + I_1'$, we get

$$\frac{dI_1}{dt} = \theta\beta_{21}\frac{I_2}{N_2}(N_1 - I_1) - (\mu_1+\delta_1)I_1 \tag{24}$$

$$\frac{dN_1}{dt} = \mu_1 S_0 - \mu_1 N_1 - \delta_1 I_1 \tag{25}$$

In order to simplify our calculations, we rescale the system (24)-(25) by letting $x_1 = I_1/N_1$ and $x_2 = I_2/N_2$. Then

$$\frac{dI_1}{dt} = \theta\beta_{21}N_1(1-x_1)x_2 - (\mu_1+\delta_1)x_1N_1 \tag{26}$$

$$\frac{dN_1}{dt} = \mu_1 S_0 - (\mu_1 + \delta_1 x_1)N_1. \tag{27}$$

By the quotient rule,

$$x_1' = \frac{N_1 I_1' - I_1 N_1'}{N_1^2} = \theta\beta_{21}(1-x_1)x_2 - (\mu_1+\delta_1)x_1 - x_1(\frac{\mu_1 S_0}{N_1} - (\mu_1+\delta_1 x_1)) \tag{28}$$

and since N_2 is a constant, we use equation (20) to obtain

$$x_2' = \frac{I_2'}{N_2} = \beta_{12}x_1(1-x_2) - (\mu_2+\delta_2+\gamma)x_2. \tag{29}$$

Our system has now been rewritten as:

$$x_1' = \theta\beta_{21}(1-x_1)x_2 - (\mu_1+\delta_1)x_1 - x_1(\frac{\mu_1 S_0}{N_1} - (\mu_1+\delta_1 x_1)) \tag{30}$$

$$x_2' = \beta_{12}x_1(1-x_2) - (\mu_2+\delta_2+\gamma)x_2 \tag{31}$$

$$N_1' = \mu_1 S_0 - (\mu_1+\delta_1 x_1)N_1. \tag{32}$$

Solving the system $x_1' = 0$, $x_2' = 0$, and $N_1' = 0$ for the EE, we get

$$(x_1^*, x_2^*, N_1^*) = \left(\tfrac{\theta\beta_{21}\beta_{12} - (\mu_1+\delta_1)(\mu_2+\delta_2+\gamma)}{\theta\beta_{21}\beta_{12} + \beta_{12}(\mu_1+\delta_1)}, \ \tfrac{\beta_{12}x_1^*}{\mu_2+\delta_2+\gamma+\beta_{12}x_1^*}, \ \tfrac{\mu_1 S_0}{\mu_1+\delta_1 x_1^*} \right).$$

Observe that $x_2^* \in (0,1)$ and $x_1^* \in (0,1)$. Since $x_1^* > 0$, the EE exists only when $R_0^2 > 1$.

The Jacobian of the system is given by

$$J = \begin{pmatrix} -(\mu_1+\delta_1 x_1^*) & -\delta_1 N_1^* & 0 \\ x_1^*\mu_1 S_0/N_1^{*2} & -(\mu_1+\delta_1+\theta\beta_{21}x_2^*) & \theta\beta_{21}(1-x_1^*) \\ 0 & \beta_{12}(1-x_2^*) & -(\mu_2+\delta_2+\gamma+\beta_{12}x_1^*) \end{pmatrix}.$$

After evaluating the Jacobian at the EE, we get that the characteristic polynomial is:

$$\begin{aligned} \lambda^3 \ &+ \ \lambda^2(\beta_{12}(\theta\beta_{21}+\mu_1+\delta_1)^2 + \theta\beta_{21}(\mu_2+\delta_2+\gamma+\beta_{12})^2 - \alpha) \\ &+ \ \lambda(\theta\beta_{12}\beta_{21} - (\mu_1+\delta_1)(\mu_2+\delta_2+\gamma) + \frac{(\theta\beta_{12}\beta_{21} + (\mu_1+\delta_1)(\mu_2+\delta_2+\gamma))\delta_1}{\beta_{12}(\theta\beta_{21}+\mu_1+\delta_1)^2} \\ &- \ \alpha(\beta_{12}(\theta\beta_{21}+\mu_1+\delta_1)^2 + \theta\beta_{21}(\mu_2+\delta_2+\gamma+\beta_{12})^2) \\ &+ \ \frac{(\theta\beta_{12}\beta_{21} + (\mu_1+\delta_1)(\mu_2+\delta_2+\gamma))\delta_1\theta\beta_{21}(\mu_2+\delta_2+\gamma+\beta_{12})}{\beta_{12}(\theta\beta_{21}+\mu_1+\delta_1)^3} - \alpha(\theta\beta_{12}\beta_{21} - (\mu_1+\delta_1)(\mu_2+\delta_2+\gamma)), \end{aligned}$$

where

$$\alpha = \frac{-\mu_1\beta_{12}(\theta\beta_{21}+\mu_1+\delta_1) - \delta_1\theta\beta_{12}\beta_{21} + (\delta_1\mu_1+\delta_1^2)(\mu_2+\delta_2+\gamma)}{\beta_{12}(\theta\beta_{21}+\mu_1+\delta_1)}.$$

As for the DFE, we apply the Routh-Hurwitz criterion to determine the stability of the EE. Letting a_1, a_2, and a_3 be the coefficients of λ^2, λ, and the constant coefficient respectively, we require that $a_1 > 0$, $a_3 > 0$, and $a_1 a_2 > a_3$. By observation, $a_1 > 0$ when $\alpha < 0$, or

$$\delta_1(\mu_1+\delta_1)(\mu_2+\delta_2+\gamma)(1-R_0^2) < \mu_1\beta_{12}(\theta\beta_{21}+\mu_1+\delta_1), \tag{33}$$

which is guaranteed to be true when $R_0 > 1$ since then the left hand side of (33) is negative and the right hand side of (33) is positive. Similarly, $a_3 > 0$ when $\alpha < 0$ and when

$$\theta\beta_{12}\beta_{21} - (\mu_1+\delta_1)(\mu_2+\delta_2+\gamma) > 0 \Leftrightarrow R_0^2 > 1.$$

It is clear that $a_1 a_2 > a_3$. Hence, the EE is stable when $R_0 > 1$.

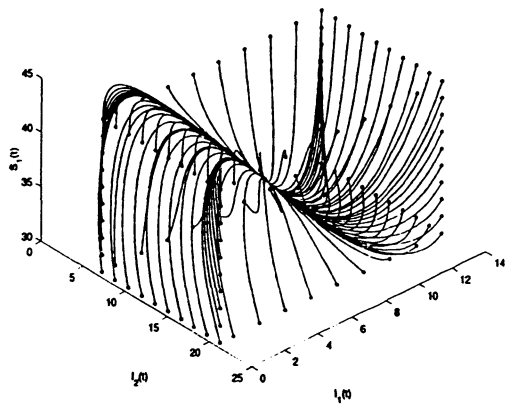

Figure 3: A neighborhood of the EE contained in the basin of attraction. Using the parameters $\beta_{ij} = 0.7$, $\mu_i = \delta_i = 0.3$, $S_0 = 50$, $N_2 = 100$, the coordinates of the EE are $(I_1^*, I_2^*, S_1^*) = (6.25, 14.2857, 37.50)$. These parameters yield an $R_0 = 1.3611$. In the figure, a cube of 120 uniformly distributed points (10 points on each edge of the cube) are used as initial values for the model, and the model system is numerically solved. The faces of the cube are a distance 7 away from the EE. The solutions starting from these initial points all converge to the EE.

2.3.4 Towards the Global Stability of the DFE and EE

Since this model is a three-dimensional system, we cannot apply the Poincaré-Bendixson criterion as we did in the previous model. Instead, we use a numerical approach in which we generate uniformly distributed points on the edges of a cube surrounding the equilibrium point, and use those points as initial values in order to obtain numerical solutions to the model system of differential equations. After running several numerical computations with increasing sizes of the cube, we found that all the solutions approach the equilibrium point. Moreover, we observed that the solutions tend toward a surface, as shown in Figure 3. In order to determine global stability, we would require that the solutions all approach the equilibrium point as the box expands to infinity. This procedure is computationally impossible, however, with sufficient time, it is possible to strongly support numerically the global stability and even find the basins of attraction for the equilibria (i.e. the regions in which the equilibria are stable). From our numerical computations, it appears that the DFE and EE are globally stable.

It is also possible to determine if the DFE and EE are globally stable using an analytical approach such as finding a Lyapunov function, however, we do not attempt to find such a function in this study. The reader is directed to p. 224 in [5] for more about this method.

2.3.5 Numerical Analysis

The DFE is given by $(S_0, 0, 0)$. Figure 4 (left) shows the projection of the solutions to the system onto the I_1I_2- plane. It is clear from the graph that all the solutions which start from the initial points shown approach the DFE $(I_1^*, I_2^*) = (0, 0)$. The parameters used in this computation are: $\beta_{12} = \beta_{21} = 0.2$, $\mu_i = \delta_i = 0.125$, $S_0 = 50$, $N_1 = N_2 = 100$, which yields an $R_0 < 1$.

For the endemic equilibrium, we use the following parameters in order to satisfy the condition that $R_0 > 1$: $\beta_{12} = \beta_{21} = 0.7$, $\mu_i = \delta_i = 0.3$, $S_0 = 50$, $N_1 = N_2 = 100$. As shown in Figure 4 (right), all the solutions which start at the randomly selected initial points approach the EE = (6.25, 14.2857, 37.5), from this set of parameters, as we would expect.

The information we obtain from this analysis allows us to generate bifurcation diagrams for the model. Figure 5 is the set of bifurcation diagrams depicting a transcritical bifurcation at $R_0 = 1$ (or at the critical bifurcation parameter $\beta_{21} = (\mu_1 + \delta_1)(\mu_2 + \delta_2 + \gamma)/\theta\beta_{12}$). When $R_0 < 1 \Leftrightarrow \beta_{21} > (\mu_1 + \delta_1)(\mu_2 + \delta_2 + \gamma)/\theta\beta_{12}$, the DFE is stable and the EE is unstable, but when $R_0 > 1$, the stability of the equilibria switches.

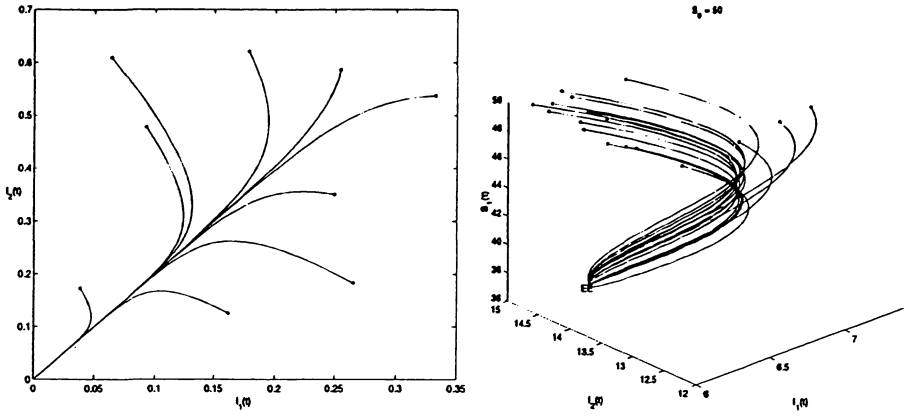

Figure 4: Left: Stability of the DFE. Right: Stability of the EE.

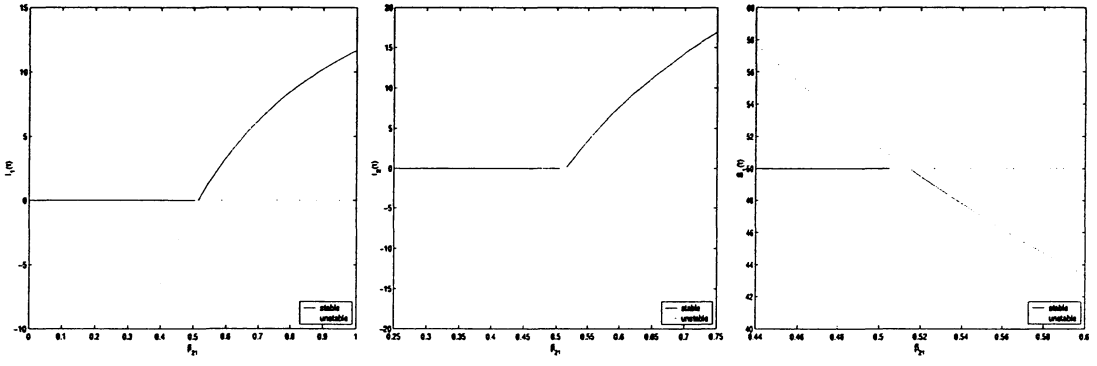

Figure 5: Transcritical Bifurcation: the two equilibria collide at the bifurcation point ($\beta_{21} \approx 0.514$) and exchange stability. That is, the stable DFE becomes unstable, and the unstable EE becomes stable. The figures, from left to right, are graphs of I_1, I_2, and S_1 as functions of the bifurcation parameter β_{21} respectively.

2.4 Parameter Estimation

Here we review studies and data on the parameters for the deterministic models.

Contact rates. The rate of infectious contact, β_{ij}, depends on the total number of contacts per person per year, and the probability of transmission per contact. We assume that the rate of infectious contact from CSWs to TDs, β_{21}, is equal to the rate of infectious contact from the TDs to the CSWs, β_{12}. According to [29], the per-contact probability of female to male transmission ranges from $0.0003 - 0.0060$, and the per-contact probability of male to female transmission ranges from $0.0005 - 0.0080$. Here we assume that TDs and CSWs are both considered high risk groups of equal degree of risk; thus we take the probabilities of transmission to be the upper bound of these ranges, 0.0080. Now, since TDs make about 2 trips per month [7, 8], they have a total of 24 trips per year. Again, suppose that the TDs contact the CSWs the same number of times the CSWs contact the TDs so that their contact rates are equal: $\beta_{12} = \beta_{21} = 24 * 0.008 = 0.192$.

Natural loss rates. We let the natural loss rate be the reciprocal of the average remaining lifetime of a healthy TD or CSW (without AIDS). Assuming that the life expectancy of a healthy South African individual is 62 years (CITE), the average age of a TD is 37 [26], and the average age of a CSW is 25 [26], we deduce that the average remaining lifetimes of a healthy TD and CSW are $\mu_1^{-1} = 62 - 37 = 25$ and $\mu_2^{-1} = 62 - 25 = 37$ respectively. This means their natural loss rates (i.e. loss/death due to other causes besides HIV/AIDS) are given by $\mu_1 = 1/25 = 0.04$ and $\mu_2 = 1/37 = 0.0276$.

AIDS-related death rates. Once an individual is infected with HIV, it is reported that the mean duration of infection ranges from $8.6-19$ years, although several studies offer inconsistent results about this parameter [18]. Here we assume that since health care in South Africa is often poor or lacking in quality, the average remaining working life of an infected TD or CSW is relatively short. So we take $\delta_i^{-1} = 8.6$ years, which translates to an AIDS-related death rate of $\delta_i = 1/8.6 = 0.116$ years^{-1} for $i = 1, 2$.

Initial population of TDs. We take the total initial population of TDs to be the size of the workforce in the transport industry as reported in [12], or $N_1(0) = \tilde{N}_1 = 239,000$. Note that often an assistant will accompany a TD on a long-distance trip, and the assistant is very likely to be involved in sexual relationships with CSWs. For simplicity, we take the number of workers in the transport industry (which consists of TDs, assistants and packers) to be the total number of TDs.

Initial population of CSWs. According to [10], at a typical South African truck stop, approximately 300 prostitutes service about 1000 men. So we take the initial population of the CSWs to be $N_2(0) = (300/1000) * N_1(0) = 0.3 * 239,000 = 71,700$.

Table 2 summarizes the parameters used in the model simulations.

Parameter	Value
contact rate from CSWs to TDs, β_{21}	0.192
contact rate from TDs to CSWs, β_{12}	0.192
natural loss rate of TDs, μ_1	0.04
natural loss rate of CSWs, μ_2	0.0276
loss rate of TDs due to AIDS, δ_1	0.116
loss rate of CSWs due to AIDS, δ_2	0.116
max number of TDs available for recruitment, Λ	15000
initial population of infected TDs, $I_1(0)$	2500
initial population of susceptible TDs, $S_1(0)$	236500
initial total population of TDs, $N_1(0) = \tilde{N}_1$	239000
initial population of infected CSWs, $I_2(0)$	1000
initial total population of CSWs, $N_2(0)$	71700

Table 2: Parameters used in the switching model simulation.

2.5 Deterministic Core-Group Model with Switching Function

Assume that at some point in time, the recruitment of TDs will reach a maximum value. Let $f_1(N_1) = \min\{\Lambda, \mu_1 N_1 + \delta_1 I_1\}$. We will henceforth call the model "Model 1" when $f_1 = \mu_1 N_1 + \delta_1 I_1$, and call the model "Model 2" when $f_1 = \Lambda$.

Suppose Λ, μ_1, $\tilde{N}_1$, and δ_1 are constants. We define Λ to be the maximum number of males willing to work as TDs per year, $\tilde{N}_1$ to be the initial (desired) number of TDs before the introduction of the disease, $N_1(t)$ to be the number of TDs at time t, and μ_1, δ_1 as before. Notice that in the presence of the disease, the number of TDs at time t, $N_1(t)$, will never exceed $\tilde{N}_1$ so that $N_1(t) \leq \tilde{N}_1$. The switch is continuous (i.e. there are no jumps) because f_1 switches exactly when the two values Λ and $\mu_1 N_1 + \delta_1 I_1$ are equal.

In Model 1, we have $N_1' = \mu_1 \tilde{N}_1 - \mu_1 N_1(t) = \mu_1(\tilde{N}_1 - N_1)$. So if $N_1 < \tilde{N}_1$, then $N_1' > 0$, or the total population of TDS, N_1, will increase. N_1 will increase until is approaches $\tilde{N}_1$.

In Model 2, $N_1' = \Lambda - (\mu_1 + \delta_1 x_1) N_1$, which has a unique equilibrium value: $\frac{\Lambda}{\mu_1 + \delta_1 x_1}$. The more infected TDs there are, the lower the equilibrium value becomes.

In a broader sense, Model 1 can be interpreted as the model when the supply of TDs equals the demand of TDs. In Model 2, the presence of the HIV virus causes a decrease in the number of TDs, since the recruitment rate remains constant while the rate at which TDs are lost increases.

2.6 Model Simulations

Note: $R_0^2 = 2.44$

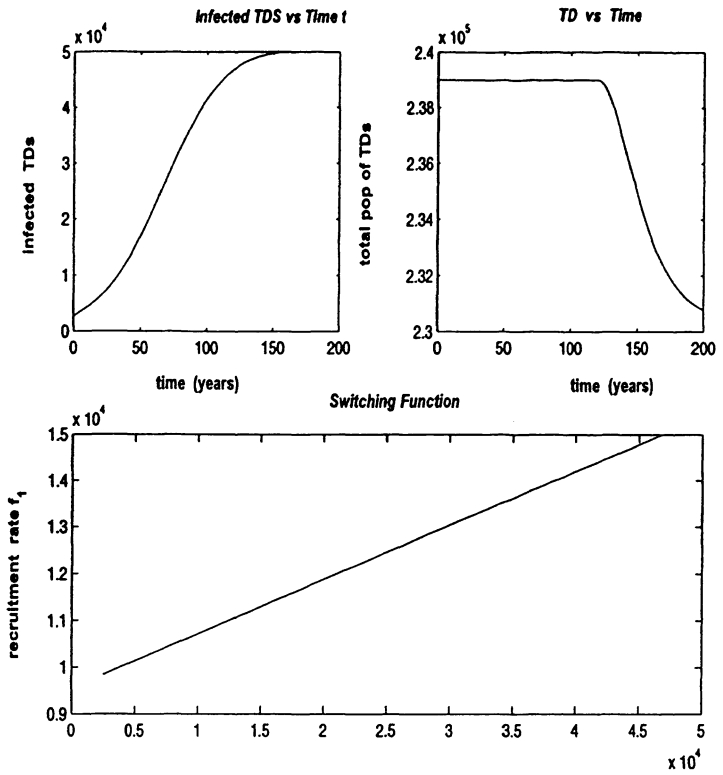

Figure 6: In this simulation, we assume that the maximum number of men available,Λ, is 15,000.

Simulation 1

In Figure 6, the graph of $I_1(t)$ vs. t depicts the evolution of the population of infected TDs as a function of time. The switch from Model 1 to Model 2 occurs when $I_1(t) = 46,900$, then $I_1(t)$ stablizes at the endemic equilibrium (EE) $I_1^* = 49,732$ after appoximately 150 years. The graph of $\tilde{N}_1$ vs. t represents the total desired population of TDs over time t. This graph illustrates the switch from Model 1 to Model 2 more clearly. The desired population of truck drivers $\tilde{N}_1$, remains constant for approximately 120 years and then begins to decrease towards the EE $N_1^* = 230,000$. The third figure depicts the switching function. When the recruitment rate reaches its threshold of 15,000 people, it switches to Model 2.

Simulation 2

In Figure 7, $I_1(t)$ reaches the EE about 30 years earlier than in the previous simulation at $I_1^* = 33,155$. After 3,790 TDs are infected, which takes place after 7.2 years, the switch from Model 1 to Model 2 occurs. The graph $\tilde{N}_1$ vs. t displays $\tilde{N}_1$ decreasing to the EE at $N_1^* = 153,857$ faster than in the previous simulation, which is a difference of approximately 75,000 individuals. The switch also occurs much earlier, as shown in the third figure.

3 Stochastic Model

We attempt to analyze the future impact of the HIV epidemic in the transportation sector mainly through its effect on TDs. First we need a model for the progression of the disease in this group. The underlying mode of transmission is complex since it involves a specific pattern of AIDS transmission that is dependent on the road structure and the availability of sex workers along the routes. These factors are neglected in the model, which assumes random mixing. Although we previously considered two-sex models, we will now make some simplifications that will allow us to keep track of infected TDs alone. That is, we consider that

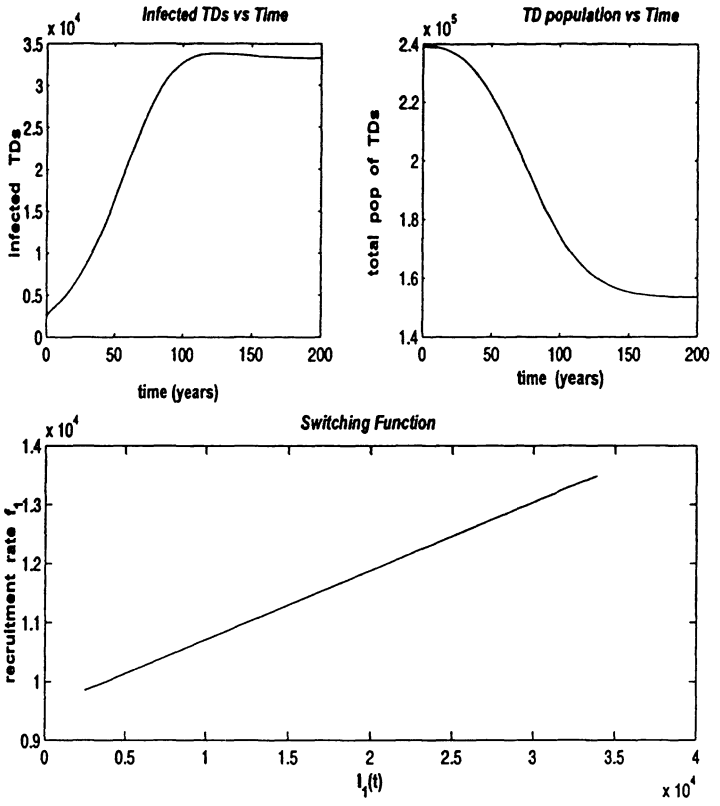

Figure 7: In this simulation, we assume that the maximum number of men available, Λ, is 10,000.

the source of an infection of a TD is another TD, although in reality the infection occurs trough the CSWs. In order to do this, we approximate the contact number to be equal to the value of R_0^2. R_0^2 represents the average number of secondary infections caused by an infected TD on other TDs.

The simplest model to use would be an SIR model with recruitment, and the effect of the AIDS epidemic on the TDs at some time t would be the number of TDs who died from the disease at that time $R(t)$, but we now take a slightly different approach, and concentrate our efforts in analyzing the potential difficulties in the recruitment of new TDs. That is, in our model, every TD who died from the disease is immediately replaced by a new, susceptible TD, and the disease continues to evolve. The total number of replacements accumulated at an arbitrary time t corresponds to $R(t)$, but then our model has the same properties as an SIS model since every removal is an instantaneous addition to the number of susceptibles. By keeping track of the number of transitions (deaths) from state I to S we are then able to account for the total number of removals at a particular time t, and avoid the complications of higher dimensional systems.

3.1 Results of the Stochastic Model

Figure 8 is a schematic representation of the stochastic model. As before, let $S(t)$, $I(t)$ denote the classes of susceptible and infected TDs respectively. Let β be the number of contacts per year, δ be the mortality rate due to AIDS, and $N(t)$ be the total number of TDs. Then the equations which describe the model are:

$$\frac{dS}{dt} = -\beta\frac{I}{N}S + \delta I \tag{34}$$

$$\frac{dI}{dt} = \beta\frac{I}{N}S - \delta I. \tag{35}$$

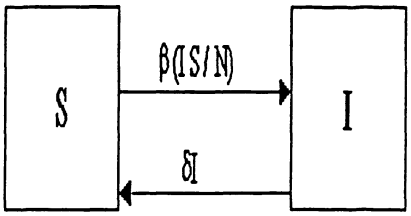

Figure 8: This figure depicts the SIS stochastic model.

Assuming a constant total population of TDs, N, we can rewrite the system as

$$\frac{dI}{dt} = \beta \frac{I(N-I)}{N} - \delta I, \tag{36}$$

that is, we get a form of the logistic equation. Unfortunately, the expected or average value of the number of infectives at time t is not given by a logistic expression. However, in order to illustrate our approach we will assume that

$$I(t) = \frac{I_0 N(\beta - \delta)}{\beta I_0 + ((\beta + \delta)N - \beta I_0)e^{-(\beta-\delta)t}}, \tag{37}$$

where $I(0) = I_0$. This sufficiently approximates the expected number of infectives at time t. An equilibrium is achieved when the number of susceptibles becoming infected equals the number of infecteds recovering, or when $\beta IS/N = \delta I \Rightarrow I = N - \delta N/\beta$.

Our goal is to illustrate a method for answering the following question: given a fixed time horizon t^*, how many TDs had died by time t^* (when there will be an expected number of infecteds equal to $I(t^*)$)? This is equivalent in our model formulation to counting the number of recoveries, or I to S transitions. In this way, we wil be able to find the expected number of TD deaths at time t^* necessary to achieve $I(t^*)$ infecteds.

In order to elucidate the problem, consider an example of a person walking up a flight of stairs. At each step, the person can either go up or step down. Let P_d be the probability of going down one step and $1 - P_d$ be the probability of going up one step. The question we wish to address is: how many backward steps will the person make before going up? To answer this question, suppose the person is at step k. There is a random variable, defined as the total number of backward steps the person must make before ascending to step $k + 1$. Then associated with this random variable is T_k, which we define to be the expected number of backward steps necessary to get to step $k + 1$. Using first step analysis [28], we obtain the following expression for T_k:

$$T_k = 0(1 - P_d) + (1 + T_{k-1} + T_k)P_d, \tag{38}$$

where $T_1 = 0$, i.e. we assume that at the first step, the person can only go forward. In equation (38), we see that the person can either go forward with probability $(1 - P_d)$ (which corresponds to the first term on the right-hand side), or he can go backward to step $k - 1$, in which case the expected number of backward steps T_k, is increased by 1 plus the expected numbers of backward steps he needs to get to steps k and $k + 1$.

Using recursion, we obtain the following expression for T_k:

$$T_k = \sum_{i=1}^{k-1} \left(\frac{P_d}{1 - P_d} \right)^i. \tag{39}$$

This expression is valid when P_d is a constant. However, in dealing with AIDS infections, the probability of a person dying is not necessarily constant. In fact, we have that the probability of becoming infected and of being removed are, respectively,

$$P_{inf} = \frac{\beta I(N-I)/N}{\beta I(N-I)/N + \delta I} \tag{40}$$

$$P_{rem} = \frac{\delta I}{\beta I(N-I)/N + \delta I} = \frac{\mu}{\beta(N-I)/N + \delta}. \tag{41}$$

As shown above, the probability of being removed is dependent on the number of infecteds I. We can view the problem in terms of the stair-walking problem, but with P_{inf} corresponding to the probability of a person going up a step, and P_{rem} corresponding to the probability of the person descends a step. Now we can see that the expected number of deaths that must occur before one new infected is added to the TD population is

$$T_k = \frac{P_k(1 + T_{k-1})}{1 - P_k} \tag{42}$$

where

$$P_k = \frac{\delta}{\beta(N-I)/N + \delta} = \frac{\delta N}{\beta} \frac{1}{(N-I)}.$$

Substituting the above expression into equation (42), we get

$$T_k = \sum_{i=1}^{k-1} \left(\frac{\delta N}{\beta}\right)^i \prod_{j=0}^{i-1} \frac{1}{(N-(k-j))}. \tag{43}$$

3.2 Parameter Estimation

Here we analyze studies and data on the parameters for the stochastic model.

Contact rate and average lifetime of a TD. The rate of infectious contact, β, depends on the total number of contacts per TD and CSW per unit of time, and the probability of female-male as well as male-female transmission per contact. We found that on average, the number of contacts per TDs per month is 2 [7], and that for every ten TDs there are three CSWs available per month [12]. This implies that the contact per CSW is ten thirds that of a TD. As mentioned in section 2.4, the per-contact probability of female to male transmission range is from 0.0003-0.0050, and the per-contact probability of male to female transmission range is from 0.0005-0.007. We also assume that TDs and CSWs are groups at high risk of infection, thus we take the probability of infection to be the upper bound of these ranges. The total number of contacts per TD per year is estimated to be 24. It has been estimated that the average duration of an infection, δ^{-1}, is 8.6 years [18]. Thus each infectious TD has a total of 24 x 8.6=206.4 infectious contacts in a lifetime. On the other hand, the total number of contacts per CSW per year is estimated to be ten thirds that of a TD, that is, (10 x 206.4)/3=688. As the female-male and the male-female transmissions are .005 and .007 respectively, the total number of effective contacts during an infectious life is 206. 4 x 0.007 x 688 x 0.005 = 4.97. Consequently, the effective number of contacts per TD per year is β=4.97/8.6.

Population of TDs. We have an estimated initial population of TDs, N, of 239,000 [12], but for modeling purposes we rounded it up to 250,000. We suppose that the initial number of infective TDs is 10% of the total population of truck drivers, that is, $I_0 = 25{,}000$.

3.3 Results of the Stochastic Model

Figure 9 shows the average behavior of the stochastic model using the parameters discussed in the previous section for the next five years[3]. The continuous line represents the average number of TDs infected, given by the logistic equation (37). The dotted line represents the accumulated expected number of deaths among TDs, which is given by our expression (43). At time zero we observe there are 25,000 TDs infected and no accumulated deaths. Within five years the expected number of infected TDs increases to 117,830 whereas the total accumulated deaths reaches 68,980. Forty-two percent of those deaths occurred after the fourth year and approximately fifty percent occurred after 3.3 years.

[3]Equation (43) is cumbersome and requires a large amount of computational resources for N = 250,000 and a forecast exceeding five years.

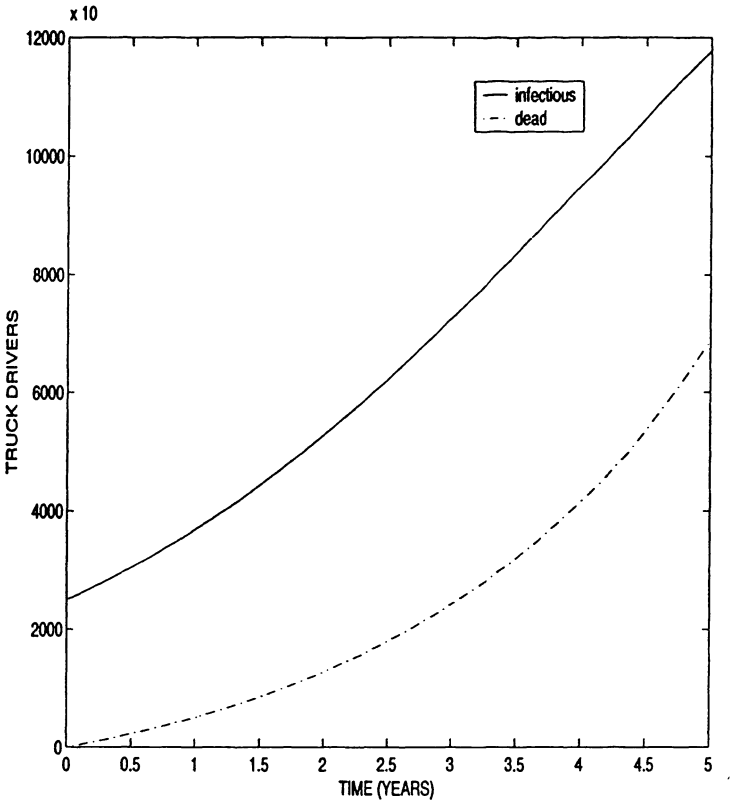

Figure 9: This figure illustrates a forecast of the behavior of the stochastic model.

4 Conclusion

4.1 Analysis of Economic Implications: Switching Model

Our main concern is with the spread of AIDS in the tranport industry and the resulting economic implications. We assume that in South Africa, economic health is directly related to the TD population in that a stable non-zero TD population implies a healthy economy. During our simulations, we analyzed different values for Λ and observed the impact of N_1^*. The more men available for recruitment yields a relatively high equilibrium value for N_1^*, and a large enough Λ results in $N_1^* > \tilde{N}_1$, indicating a sufficient population of TDs. On the other hand, a low Λ value results in a considerably lower equilibrium value and a crucial decrease in the TD population. This suggests that the value of Λ is significant to the stability of the TD population and the fortitude of the economy. It is predicted that 432,000,000 goods and supplies were transported by the trucking industry in 1999 [29]. When $\tilde{N}_1 = 239,000$, approximately 1,812 metric tons of goods and supplies are transported per TD. As N_1 decreases, the amount of goods transported decreases, posing a major threat on the economic status of the continent. Sufficient food and medical supplies may not be effectively distributed throughout SSA, which may result in starvation and a decrease in adequate medical attention in many small towns and communities, as well as bring about financial hardship in the transport industry.

4.2 Analysis of Economic Implications: Stochastic Model

We observe that in five years approximately 68,980 new TDs will be needed to replace the deceased TDs. This required level of replacement seems unattainable. It is often difficult to quantify the economic cost of the loss of skilled professionals such as doctors, teachers, nurses, etc. However, in the case of TDs we can assume that if a fraction f of TDs can be replaced, the economic cost would be $(1 - f) * C$, where C is

the total value of goods transported by TDs. South African authorities should be aware of this potential problem developing within a short period of time. It may be advisable to increase the current efforts of prevention among TDs and CSWs so that economic losses are less harmful. This would be the most likely scenario if the epidemic maintains its current development.

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6 Appendix

6.1 Switching Model

Below is the MATLAB code for the switching model. As shown below, we use the Runge-Kutta method to numerically integrate the system of equations.

```
function [tvals,xvals]=rkm(t0,tf,x0)
%
% rkm.m
%
% RUNGA-KUTTA METHOD FOR NUMERICALLY INTEGRATING ODES
%
% [t,x]=rkm(ti,tf,[I10;I20;N10]);
%
% ti = initial time
% tf = final time
% I10 = initial pop of infected TDs
% I20 = initial pop of infected CSWs
% S10 = initial pop of susceptible TDs
%
% SAMPLE RUN ON COMMAND PROMPT
% [t,x]=rkm(0,150,[2500;1000;236500]);
% plot I1 vs. t
% plot(t,x(1,:));
% plot N1 vs. t
% plot(t, x(1,:)+x(3,:));
% plot the switching function
% f = min (L, u1*N1+d1*I1)
% f=min(15000,.04*239000+.116*x(1,:));
% plot(x(1,:),f);

global b21 b12 n2 u1 u2 d1 d2 L n1
b21=.192;     % contact rate from CSWs to TDs = 24*.008
b12=.192;     % contact rate from TDs to CSWs
n2=71700;      % total pop of CSWs = .3*n1
u1=.04;       % loss rate of TDs due to causes other than AIDS = 1/(62-37)
u2=.0276;      % loss rate of CSWs due to causes other than AIDS = 1/(62-25)
d1=.116;       % death rate of TDs due to AIDS = 1/8.6
d2=.116;       % death rate of CSWs due to AIDS = 1/8.6
L=20000;       % max # of males available to be hired as TDs
n1=239000;      % total pop of TDs

h=.01;
n=round((tf-t0)/h);
x=x0;
t=t0;

tvals=t0;
xvals=x0;

fc=feval('derivs1',t,x);

for j=1:n                    % integrate
    if x(1) <(L-u1*n1)/d1    % model 1
```

```
        (L-u1*n1)/d1
        [t,x,fc]=RKstep('derivs1',t,x,fc,h);
        xvals=[xvals x];
        tvals=[tvals t];
        t
    else                    % model 2
        [t,x,fc]=RKstep('derivs2',t,x,fc,h);
        xvals=[xvals x];
        tvals=[tvals t];
    end
end
%*******************************************************************************
function [tnew,ynew,fnew]=RKstep(fname,tc,yc,fc,h)
%yc=ic;

k1 = h*fc;
k2 = h*feval(fname,tc+(h/2),yc+(k1/2));
k3 = h*feval(fname,tc+(h/2),yc+(k2/2));
k4 = h*feval(fname,tc+h,yc+k3);
ynew = yc +(k1 + 2*k2 + 2*k3 +k4)/6;

tnew = tc+h;
fnew = feval(fname,tnew,ynew);
%*******************************************************************************
% MODEL 1
function dx=derivs1(t,x)

global b21 b12 n2 u1 u2 d1 d2 L n1

dx=[b21*x(2)*(n1-x(1))/n2-u1*x(1)-d1*x(1); b12*x(1)*(n2-x(2))/n1-(u2+d2)*x(2);u1*n1+d1*x(1)-u1*(n1-x(1)
%*******************************************************************************
% MODEL 2
function dx=derivs2(t,x)

global b21 b12 n2 u1 u2 d1 d2 L

dx=[b21*x(2)*x(3)/n2-u1*x(1)-d1*x(1); b12*x(1)*(n2-x(2))/(x(3)+x(1))-(u2+d2)*x(2);L-u1*x(3)-b21*x(2)*x(

%*******************************************************************************
%Code for Simulations

[t,x]=rkm(0,200,[2500;1000;236500]);
subplot(221),plot(t,x(1,:),'b');
hold on
xlabel('time (years)');
ylabel('infected TDs');
hold off
subplot(222),plot(t,x(1,:)+x(3,:),'m');
hold on
xlabel('time (years)');
ylabel('total pop of TDs');
hold off
f=min(15000,.04*239000+.116*x(1,:));
subplot(212),plot(x(1,:),f);
```

```
hold on
title('Switching Function');
xlabel('I_1(t)');
ylabel('recruitment rate f_1');
hold off
```

6.2 Stochastic Model

Below is the MATLAB code for the stochastic simulation.

```
function A = m41(N,beta,delta,T,i0,howmany)
% N = TOTAL POP. SIZE
% beta = CONTACT RATE
% delta = recovery rate (death rate)
% T = stopping time
% i0 = initial number of infected
% howmany = number of simulations
% m4(250000,4.97/8.6,1/(8.6),1*5,2500,1)
hold on
for h = 1 : howmany
infected = 0;
A = zeros(10000,3);   % allocating memory (runs faster !!!)
% for i = 1 : howmany   % keeping track of the simulation
row = 1;
I = i0;
current_time = 0;
current_state = [0 I 0];  % [time = 0, i0 initial infected]
dead = 0;
pack
integral = 0;
while current_time < T & I > 0
    A(row,:) = current_state;
    infrate = beta*I*(N-I)/N;
    remrate = delta*I;
    totrate = infrate+remrate;
    pinf = infrate/totrate;
    next_ev = -log(rand)/totrate;      % time to next event
    integral = integral + next_ev*I;
    if rand < pinf
        I = I + 1;
    else
        I = I - 1;
        dead = dead +1;
    end;
    row = row + 1;
    current_time = current_time + next_ev;
        if row > 950
        A = [A; zeros(1000,3)];     % adding more rows
    end;
        current_state = [current_time I dead];
%     pause
 end    % {of while}
A=A(1:row-1,:);
result(h)=A(end,2);
```

```
deaths(h)=A(end,3);
%plotting starts here
xa = A(:,1);ya = A(:,2);
za = A(:,3);
%subplot(2,1,1)
%plot(t,f,'k--')
%hold off
integral2(h)=integral;
h
end;
%result
%subplot(2,1,2)
%hist(result)
%This part calculates the expected number of dead people
Tk(1)=0;
proye(1,:)=[1 0];
for k = 2 : N
    pd = N*delta/(delta+beta*(N-k));
    Tk(k) = pd*(1+Tk(k-1))/(1-pd);
    proye(k,:)=[k Tk(k)];
end;
clc
proye=[proye cumsum(proye(:,2))];
% logistic growth
%f = N*(beta-delta)*exp(t*beta)./(beta*exp(t*beta)-exp(t*delta)*(beta-N*beta+N*delta));
g=(beta-delta);
t = .01:.01:T;
f = ((beta-delta)*i0*N*exp(beta*t))./(exp(beta*t)*i0*beta-exp(delta*t)*(i0*beta+N*(delta-beta)));
f2 = ceil(f);
f=f';f2=f2';
est = proye(f2-1,3);
plot(xa,ya,'k-',t,f,'k-.',xa,za,'b--',t,est,'r:')
legend('infectious','forecast','dead','forecast')
hold off
[infected/howmany f2(end)]
mean(integral2)
mean(deaths)
```

MATLAB codes for Numerical Analysis of AIDS in Africa Project

Directory:

```
m1dfe_vf - equations for Model 1
m2dfe_vf - equations for Model 2
m2ee_vf - equations for the rescaled Model 2
ee2 - calculates the EE for Model 2 when given parameters p
dfe2 - calculates the EE for Model 2 when given parameters p
stab1 - illustrates stability of DFE for Model 1
stab2 - illustrates stability of EE for Model 1
stab3 - illustrates stability of DFE for Model 2
stab4 - illustrates stability of EE for Model 2
demo - script that runs the stability demos for Models 1 and 2
box_demo4 - illustrates the (global) stability of Model 2 EE
box3 - generates a cube with points on the edges of it
bifX1 - draws bifurcation diagram for Model 2, 1st coordinate (I1)
bifX2 - draws bifurcation diagrams for Model 2, all 3 coordinates
bifX3 - improved version of bifX2 that is more efficient b/c it saves info in arrays then plots

*****************************************************************************************

function  Xdot  = m1dfe_vf(t, V, p)

% function  Xdot  = m1dfe_vf(t, V, p)
%
% Model 1
%
% Input:  (t, V, p)
%    t -- scalar (ignored)
%    Y -- phase-space vector [x; y]
%    p -- parameter vector [b12; b21; u1; u2; d1; d2; n1; n2 ]
%
% Output: 2-vector [x_dot; y_dot]
%

b12 = p(1);
b21 = p(2);
u1 = p(3);
u2 = p(4);
d1 = p(5);
d2 = p(6);
n1 = p(7);
n2 = p(8);
I1 = V(1);
I2 = V(2);

Xdot(1,1) = (b21*I2*(n1-I1))/n2 - (u1 + d1)*I1;
Xdot(2,1) = (b12*I1*(n2-I2))/n1 - (u2 + d2)*I2;

*****************************************************************************************

function  Xdot  = m2dfe_vf(t, V, p)

% function  Xdot  = m2dfe_vf(t, V, p)
%
% Model 2
%
% Input:  (t, V, p)
%    t -- scalar (ignored)
%    Y -- phase-space vector [x; y; z]
%    p -- parameter vector [b12; b21; u1; u2; d1; d2; n2; S0]
%
% Output: 3-vector [x_dot; y_dot; z_dot]

b12 = p(1);
b21 = p(2);
u1 = p(3);
u2 = p(4);
d1 = p(5);
```

```
d2 = p(6);
n2 = p(7);
S0 = p(8);
I1 = V(1);
I2 = V(2);
S1 = V(3);

if S1+I1 == 0
    error('S1+I1=0');
end

Xdot(1,1) = (b21*I2*S1)/n2 - u1*I1 - d1*I1;
Xdot(2,1) = (b12*I1*(n2-I2))/(S1+I1) - (u2+d2)*I2;
Xdot(3,1) =  u1*S0 - u1*S1 - (b21*I2*S1)/n2;
```

****

```
function  Xdot  = m2ee_vf(t, V, p)
% Model 2 - EE
%
% Input:  (t, V, p)
%    t -- scalar (ignored)
%    Y -- phase-space vector [x; y; z]
%    p -- parameter vector [b12; b21; u1; u2; d1; d2; n2; S0]
%
% Output: 3-vector [x_dot; y_dot; z_dot]

b12 = p(1);
b21 = p(2);
u1 = p(3);
u2 = p(4);
d1 = p(5);
d2 = p(6);
n2 = p(7);
S0 = p(8);
x1 = V(1);
x2 = V(2);
N1 = V(3);

Xdot(1,1) = b21*(1-x1)*x2 - (u1+d1)*x1 - x1*(u1*S0/N1 - (u1+d1*x1));
Xdot(2,1) = b12*x1*(1-x2)-(u2+d2)*x2;
Xdot(3,1) = u1*S0 - (u1+d1*x1)*N1;
```

****

```
function x = ee2(p)

% Endemic equilibrium for Model 2
%
% INPUT - p = vector of parameter values.


b12 = p(1);
b21 = p(2);
u1 = p(3);
u2 = p(4);
d1 = p(5);
d2 = p(6);
n2 = p(7);
S0 = p(8);

top1 = b21*b12-(u1+d1)*(u2+d2);
bot1 = b21*b12 + b12*(u1+d1);

x1 = top1 / bot1;

x2 = b12 * x1 / (u2 + d2 + b12 * x1);

n1 = u1 * S0 / (u1 + d1 * x1);

I1 = n1 * x1;
```

```
I2 = n2 * x2;
S1 = n1 - I1;


x = [I1; I2; S1];

*****************************************************************************************

function x = dfe2(p)

b12 = p(1);
b21 = p(2);
u1 = p(3);
u2 = p(4);
d1 = p(5);
d2 = p(6);
n2 = p(7);
S0 = p(8);

x = [0; 0; S0];

*****************************************************************************************

% stability_demo.m
% stab1 - model 1, disease-free equilibrium

N1 = 100;
N2 = 100;

it = integro;
it = set(it,'F','m1dfe_vf');

b12 = .2;
b21 = .2;
u1 = .125;
u2 = .125;
d1 = .125;
d2 = .125;

R0 = b12*b21/(u1+d1)/(u2+d2)

p1 = [b12;b21;u1;u2;d1;d2;N1;N2];
it = set(it,'p', p1);

it=set(it, 't',[0 50]);

while 1

    try
        [x,y] = ginput(1);
        if x > N1 | x < 0 | y > N2 | y < 0
            disp('done')
            return
        end

        it = set(it, 'Y0', [x;y]);
        it = Run(it);
        plot2(it,'-'); hold on;
        plot2(it,[1,2], 1,'b.', 'markersize', 10);

        it = rmpoints(it);

    catch

        disp('Interrupted');
        return
    end
end

*****************************************************************************************
```

```
% stability_demo.m
% stab2 - model 1, endemic equilibrium

N1 = 100;
N2 = 100;

it = integro;
it = set(it,'F','m1dfe_vf');

b12 = .7;
b21 = .7;
u1 = .3;
u2 = .3;
d1 = .3;
d2 = .3;

R0 = b12*b21/(u1+d1)/(u2+d2)
p2 = [b12;b21;u1;u2;d1;d2;N1;N2];
it = set(it,'p', p2);

it=set(it, 't',[0 50]);

while 1

    try
        [x,y] = ginput(1);
        if x > N1 | x < 0 | y > N2 | y < 0
            disp('done')
            return
        end

        it = set(it, 'Y0', [x;y]);
        it = Run(it);
        plot2(it,'-'); hold on;
        plot2(it,[1,2], 1,'b.', 'markersize', 10);

        it = rmpoints(it);

    catch

        disp(lasterr);
        return
    end
end

*****************************************************************************************

% stability_demo.m
% stab3 - model 2, disease-free equilibrium

N1 = 100;
N2 = 100;

it = integro;
it = set(it,'F','m2dfe_vf');

b12 = .2;
b21 = .2;
u1 = .125;
u2 = .125;
d1 = .125;
d2 = .125;
S0 = 50;

R0 = b12*b21/(u1+d1)/(u2+d2)

p1 = [b12;b21;u1;u2;d1;d2;N2;S0];
it = set(it,'p', p1);

it=set(it, 't',[0 80]);
```

```
done = 0;
while ~done

    try
        [x,y] = ginput(1);
        if x > N1 | x < 0 | y > N2 | y < 0
            disp('done');
            done = 1;
            return
        end

        it = rmpoints(it);

        it = set(it, 'Y0', [x;y;S0]);
        it = Run(it);
        plot2(it,'-'); hold on;
        plot2(it,[1,2], 1,'b.', 'markersize', 10);

    catch

        disp(lasterr);
        return
    end
end

****************************************************************************************

% stability_demo.m
% stab4 - model 2, endemic equilibrium

N1 = 100;
N2 = 100;

it = integro;
it = set(it,'F','m2dfe_vf');

b12 = .7;
b21 = .7;
u1 = .3;
u2 = .3; % 125;
d1 = .3; % 125;
d2 = .3; %
S0 = 30;

R0 = b12*b21/(u1+d1)/(u2+d2)

p4 = [b12;b21;u1;u2;d1;d2;N2;S0];


ee = ee2(p4);
figure(1)
plot(ee(1), ee(2), 'm*'); hold on;

figure(2)
plot3(ee(1), ee(2), ee(3), 'm*'); hold on;
figure(1);


it = set(it,'p', p4);

it0 = set(it, 't',[0 80]);

done = 0;
while ~done

    try
        figure(1)
        [x,y] = ginput(1);
        xlim = get(gca,'xlim');
        ylim = get(gca,'ylim');
        if x > xlim(2) | x < xlim(1) | y > ylim(2) | y < ylim(1)
```

```
            disp('done');
            done = 1;
            zoom on
            return
        end

        it = it0;

        it = set(it, 'Y0', [x;y;S0]);
        it = Run(it)
        after_state = status(it)

        figure(1);
        plot2(it,'-'); hold on;
        plot2(it,[1,2], 1,'g.', 'markersize', 10);

        figure(2)
        plot3(it, '-'); hold on;
        plot3(it,[1,2,3], 1,'g.', 'markersize', 10);

    catch

        disp(lasterr);
        return
    end
end

*****************************************************************************************

function demo(k, axkey)

% function demo(k, axkey)
%
% Script that runs the demos for models 1 and 2, for the DFE and EE
%
% INPUT - (k, axkey)
% k = case number
% axkey = specifies an axis

if nargin<2
    axkey=0;
end

if axkey
    clf,                % clear function
    axis([0 100 0 100]) % set the axes
    hold on,
    zoom reset,
end

switch k
case 1
    stab1   % Model 1, DFE
case 2
    stab2   % Model 1, EE
case 3
    stab3   % Model 2, DFE
case 4
    stab4   % Model 2, EE
otherwise
    error('No stab function defined for this n');
end

*****************************************************************************************

function box_demo4(d, npts);

% function box_demo4(d, npts);
%
% Shows the stability of the EE in model 2 by taking initial points on the edges of a cube and
% computing solutions of the system using these initial values. The solutions tend toward the EE,
```

```
% indicating that the EE is stable. Returns 2 figures; fig 1 is a projection of I1 and I2
% coordinates of the solution on a 2D plane and fig 2 is the 3D graph of the cube and the solns.
%
% INPUTS - (d, npts)
% d - distance face-to-center of box (ee)
% npts - number of pts per edge

% if no 1st parameter is indicated, set d=1
if nargin<1
    d = 1;
end
% if no 2nd parameter is indicated, set npts = 2
if nargin<2
    npts = 2;
end

if npts<2
    disp('warning: N increased to 2');
    npts=2;
end

N1 = 100;
N2 = 100;

it = integro;
it = set(it,'F','m2dfe_vf');

b12 = .7;
b21 = .7;
u1 = .3;
u2 = .3;
d1 = .3;
d2 = .3;
S0 = 50;

R0 = b12*b21/(u1+d1)/(u2+d2)

p4 = [b12;b21;u1;u2;d1;d2;N2;S0];

ee = ee2(p4);
figure(1)
cla; % clear axes
plot(ee(1), ee(2), 'm*'); hold on;

boxpts = box3(ee, d, npts);

figure(2)
cla;
plot3(ee(1), ee(2), ee(3), 'm*'); hold on;
figure(1);

it = set(it,'p', p4);
it = setopt(it, 'verbose', 'off');

it0 = set(it, 't',[0 50]);

[rowN, colN] = size(boxpts);

for k = 1:rowN

    x = boxpts(k,1);
    y = boxpts(k,2);
    z = boxpts(k,3);

    it = it0;

    it = set(it, 'Y0', [x;y;z]);
    it = Run(it);

    its(k) = it;
end
```

```
after_state = status(it);

figure(1);
for k = 1:rowN
    it = its(k);
    plot2(it,'-'); hold on;
    plot2(it,[1,2], 1,'g.', 'markersize', 10);
end

figure(2)
for k = 1:rowN
    it = its(k);
    plot3(it, '-'); hold on;
    plot3(it,[1,2,3], 1,'g.', 'markersize', 10);
end

figure(2)
TriDTool;

*****************************************************************************************

function b = box3(p,d,N)

% function b = box3(p,d,N)
%
% This function creates a cube around the point p, with horizontal and
% vertical distances d away from p.
%
% INPUT - (p,d,N)
% p = (x0, y0, z0)
% d = scalar distance
% N = number of points along each edge of the cube
% OUTPUT - b, a 12*Nx3 matrix containing 12*N points along the edges of the box
%

% Generate N equally spaced points between x0-d and x0+d
X = linspace(p(1)-d,p(1)+d,N);
% Generate N equally spaced points between points y0-d and y0+d
Y = linspace(p(2)-d,p(2)+d,N);
% Generate N equally spaced points between points y0-d and y0+d
Z = linspace(p(3)-d,p(3)+d,N);

for i=1:N
    % front face
    ftx(i) = X(i);
    fty(i) = p(2)+d;
    ftz(i) = p(3)+d;
    frx(i) = p(1)+d;
    fry(i) = Y(i);
    frz(i) = p(3)+d;
    fbx(i) = X(i);
    fby(i) = p(2)-d;
    fbz(i) = p(3)+d;
    flx(i) = p(1)-d;
    fly(i) = Y(i);
    flz(i) = p(3)+d;
    % back face
    btx(i) = X(i);
    bty(i) = p(2)+d;
    btz(i) = p(3)-d;
    brx(i) = p(1)+d;
    bry(i) = Y(i);
    brz(i) = p(3)-d;
    bbx(i) = X(i);
    bby(i) = p(2)-d;
    bbz(i) = p(3)-d;
    blx(i) = p(1)-d;
    bly(i) = Y(i);
    blz(i) = p(3)-d;
    % right face
```

```
    rtx(i) = p(1)+d;
    rty(i) = p(2)+d;
    rtz(i) = Z(i);
    rbx(i) = p(1)+d;
    rby(i) = p(2)-d;
    rbz(i) = Z(i);
    % left face
    ltx(i) = p(1)-d;
    lty(i) = p(2)+d;
    ltz(i) = Z(i);
    lbx(i) = p(1)-d;
    lby(i) = p(2)-d;
    lbz(i) = Z(i);

end

bx = [ftx, frx, fbx, flx, btx, brx, bbx, blx, rtx, rbx, ltx, lbx]';
by = [fty, fry, fby, fly, bty, bry, bby, bly, rty, rby, lty, lby]';
bz = [ftz, frz, fbz, flz, btz, brz, bbz, blz, rtz, rbz, ltz, lbz]';

b = [bx(:),by(:),bz(:)];

j = b < 0;
b(j) = 0;

% plot3(b(:,1),b(:,2),b(:,3),'m*');

*****************************************************************************************

% bifX1.m
%
% Draws the bifurcation diagram for model 2. Computes R0. If R0<1, the DFE is stable
% and the EE is unstable. If R0>1, the DFE becomes unstable and the EE becomes stable.
% Thus, we see that the system has a transcritical bifurcation.

N2 = 100;

b12 = .7;
b21 = .7;
u1 = .3;
u2 = .3;
d1 = .3;
d2 = .3;
S0 = 50;

p4 = [b12;b21;u1;u2;d1;d2;N2;S0];

% create a vector of 100 equally spaced points between 1e-6 and 1 to use a the varying values of
b21.
% the reason we do not use 0 = b21 is to avoid division by zero.
BB = linspace(1e-6, 1, 100);

SEE = [];
UEE = [];
SDF = [];
UDF = [];
for k=1:length(BB)

    p4(2) = BB(k);

    % compute endemic equil
    ee = ee2(p4);

    % plot I1 vs b21 (b21==BB, varying)
    if ee(1) > 0
        SEE = [SEE; BB(k), ee(1)];
        % plot(BB(k), ee(1), 'b.', 'markersize', 10); hold on % stable EE
    else
        UEE = [UEE; BB(k), ee(1)];
        % plot(BB(k), ee(1), 'bo', 'markersize', 5); hold on % unstable EE
    end
```

```
    % compute if Ro < 1 ==> stability of dfe (0,0,S0)
    if b12 * BB(k) < (u1 + d1)*(u2 + d2)

        SDF = [SDF; BB(k), 0];

    else
        UDF = [UDF; BB(k), 0];
    end
end

% now do plots

plot(SEE(:,1), SEE(:,2), 'b-'); hold on % stable EE

plot(UEE(:,1), UEE(:,2), 'b:'); hold on % UNstable EE

plot(SDF(:,1), SDF(:,2), 'b-'); hold on % stable EE

plot(UDF(:,1), UDF(:,2), 'b:'); hold on % UNstable EE

axis([0 1 -10 15]);
legend('stable','unstable',4);
xlabel('\beta_{21}');
ylabel('I_1(t)');

*****************************************************************************************

% bifX2.m
%
% Draws the bifurcation diagrams for model 2. Computes R0. If R0<1, the DFE is stable
% and the EE is unstable. If R0>1, the DFE becomes unstable and the EE becomes stable.
% Thus, we see that the system has a transcritical bifurcation.

N2 = 100;

b12 = .7;
b21 = .7;
u1 = .3;
u2 = .3;
d1 = .3;
d2 = .3;
S0 = 50;

p4 = [b12;b21;u1;u2;d1;d2;N2;S0];

BB = linspace(1e-6, 1, 100);

for k=1:length(BB)

    p4(2) = BB(k);

    % compute endemic equil
    ee = ee2(p4);

    figure(1)  % plot I1 vs b21 (b21==BB, varying)

    % compute if Ro < 1 ==> stability of dfe (0,0,S0)
    if b12 * BB(k) > (u1 + d1)*(u2 + d2)

        plot(BB(k), ee(1), 'b-'); hold on
        plot(BB(k), 0, 'b:'); hold on

    else

        plot(BB(k), ee(1), 'b:'); hold on

        plot(BB(k), 0, 'b-'); hold on

    end
    legend('stable', 'unstable',4);
```

```
    figure(2) % plot I2 vs. b21
    %------------------------------
    % compute if Ro < 1 ==> stability of dfe (0,0,S0)
    if b12 * BB(k) > (u1 + d1)*(u2 + d2)


        plot(BB(k), ee(2), 'b-'); hold on
        plot(BB(k), 0, 'b:'); hold on


    else

        plot(BB(k), ee(2), 'b:'); hold on

        plot(BB(k), 0, 'b-'); hold on

    end
    legend('stable', 'unstable',4);

    figure(3)  % plot S1 vs. b21
    %------------------------------
    % compute if Ro < 1 ==> stability of dfe (0,0,S0)
    if b12 * BB(k) > (u1 + d1)*(u2 + d2)


        plot(BB(k), ee(3), 'b-'); hold on
        plot(BB(k), S0, 'b:'); hold on

    else

        plot(BB(k), ee(3), 'b:'); hold on

        plot(BB(k), S0, 'b-'); hold on

    end
    legend('stable', 'unstable',4);

end

*****************************************************************************************

% bifX3.m
%
% Draws the bifurcation diagram for model 2. Computes R0. If R0<1, the DFE is stable
% and the EE is unstable. If R0>1, the DFE becomes unstable and the EE becomes stable.
% Thus, we see that the system has a transcritical bifurcation.

N2 = 100;

b12 = .7;
b21 = .7;
u1 = .3;
u2 = .3;
d1 = .3;
d2 = .3;
S0 = 50;

p4 = [b12;b21;u1;u2;d1;d2;N2;S0];

% create a vector of 100 equally spaced points between 1e-6 and 1 to use a the varying values of
b21.
% the reason we do not use 0 = b21 is to avoid division by zero.
BB = linspace(1e-6, 1, 100);

SEE1 = [];
UEE1 = [];
SDF1 = [];
UDF1 = [];
SEE2 = [];
UEE2 = [];
```

```
SDF2 = [];
UDF2 = [];
SEE3 = [];
UEE3 = [];
SDF3 = [];
UDF3 = [];
for k=1:length(BB)

    p4(2) = BB(k);

    % compute endemic equil
    ee = ee2(p4);

    % plot I1 vs b21 (b21==BB, varying)
%     if ee(1) > 0
%         SEE = [SEE; BB(k), ee(1)];
%
%     else
%         UEE = [UEE; BB(k), ee(1)];
%
%     end
%
    % compute if Ro < 1 ==> stability of dfe (0,0,S0)
    if b12 * BB(k) < (u1 + d1)*(u2 + d2)

        SDF1 = [SDF1; BB(k), 0];
        UEE1 = [UEE1; BB(k), ee(1)];
        SDF2 = [SDF2; BB(k), 0];
        UEE2 = [UEE2; BB(k), ee(2)];
        SDF3 = [SDF3; BB(k), S0];
        UEE3 = [UEE3; BB(k), ee(3)];

    else
        UDF1 = [UDF1; BB(k), 0];
        SEE1 = [SEE1; BB(k), ee(1)];
        UDF2 = [UDF2; BB(k), 0];
        SEE2 = [SEE2; BB(k), ee(2)];
        UDF3 = [UDF3; BB(k), S0];
        SEE3 = [SEE3; BB(k), ee(3)];

    end
end

% now do plots

% I1 vs. b21

figure(1)
plot(SEE1(:,1), SEE1(:,2), 'b-'); hold on % stable EE

plot(UEE1(:,1), UEE1(:,2), 'b:'); hold on % UNstable EE

plot(SDF1(:,1), SDF1(:,2), 'b-'); hold on % stable EE

plot(UDF1(:,1), UDF1(:,2), 'b:'); hold on % UNstable EE

axis([0 1 -10 15]);
legend('stable','unstable',4);
xlabel('\beta_{21}');
ylabel('I_1(t)');

% I2 vs. b21

figure(2)
plot(SEE2(:,1), SEE2(:,2), 'b-'); hold on % stable EE

plot(UEE2(:,1), UEE2(:,2), 'b:'); hold on % UNstable EE

plot(SDF2(:,1), SDF2(:,2), 'b-'); hold on % stable EE

plot(UDF2(:,1), UDF2(:,2), 'b:'); hold on % UNstable EE
```

```
axis([.25 .75 -20 20]);
legend('stable','unstable',4);
xlabel('\beta_{21}');
ylabel('I_2(t)');

% S1 vs. b21

figure(3)
plot(SEE3(:,1), SEE3(:,2), 'b-'); hold on % stable EE

plot(UEE3(:,1), UEE3(:,2), 'b:'); hold on % UNstable EE

plot(SDF3(:,1), SDF3(:,2), 'b-'); hold on % stable EE

plot(UDF3(:,1), UDF3(:,2), 'b:'); hold on % UNstable EE

axis([.44 .6 40 60]);
legend('stable','unstable',4);
xlabel('\beta_{21}');
ylabel('S_1(t)');
```