University of Alberta

Modeling the Effects of Carriers on the Transmission Dynamics of Infectious Diseases

by

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Abstract

In this thesis we examine and analyze an S-I_c-I-R epidemic model where S represents susceptibles, I_c carriers, I infectives, and R the removed class. This model can be applied to diseases that can be transmitted through carriers, which are individuals who carry the disease and can pass it on to other individuals, but who do not show any symptoms. Two such diseases are Hepatitis B and HIV/AIDS. In case of HIV we consider carriers to be those who are unaware of their infection. In the case of Hepatitis B, we examine a model that includes the removal of susceptibles from the population through a vaccine. We will find the disease-free and endemic equilibria (P_0 and P^* respectively) and prove their local and global stability in the feasible region depending on the reproductive number R_0.

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1 Introduction

In the cases of certain infectious diseases, there are individuals who are able to transmit their illness but who do not exhibit any symptoms. These individuals are called "carriers" and they play an important role in the transmission of certain infectious diseases. There are two types of carriers. The first type are *genetic carriers*, who carry the illness on their recessive genes. This type of carrier can only pass on their disease to their children and are not contagious. The second type - which are the focus of this article - are *infectious disease carriers*. Due to the fact that these individuals do not display any outward symptoms, it is more likely that they are unaware of their condition and therefore more likely that they will infect another person. For this reason, the analysis of carriers and their effect on epidemics is so vital to the study of infectious diseases.

One infamous example of a disease that spread through the unknowing actions of a carrier is found in the story of Typhoid Mary. Mary Mallon, or *Typhoid Mary* as she later became known as, was a cook hired by various households in the early 1900's. Legend has it that she was the first "healthy carrier" of typhoid fever in the United States. Typhoid fever can be spread through food and water sources, and from 1900 to 1907 Mary worked in 7 households where 22 people fell ill with the disease and one young girl died. In 1906, the Warren family that she was working for became suspicious when 6 of the 11 people in the house became ill with the fever and hired a health inspector to investigate. When the inspector approached Mary in 1907, she appeared healthy and was unwilling to cooperate. The fame of the case comes from the fact that she was tried, but alluded authorities until she was finally captured and forced into seclusion on a small island off of New York.

A modern disease that can be transmitted trough carriers is Hepatitis B. Hepatitis B is a viral liver infection that is spread through body fluids, mainly through sexual contact. Those who are carriers of the disease may not be aware and therefore may have more encounters leading to transmission.

Another such disease is HIV or AIDS. HIV has little or no symptoms, and those that do appear resemble symptoms of the flu. Since HIV is a sexually transmitted disease, carriers can be very dangerous as they would not alter their behaviour as would someone who was aware of their condition. HIV was once a top 10 cause of death in the world, but dropped off of the list due to better treatments and reduced transmission. Our analysis will show what effect reducing the amounts of carriers through greater awareness would have

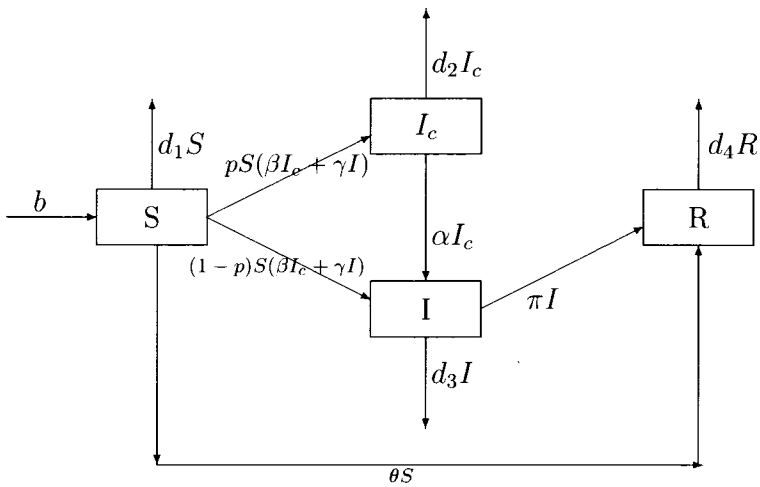
on overall AIDS occurrence. Of the 900,000 Americans who have the HIV virus, 1/4 (225,000) are unaware. AIDS also has an incidence rate of 0.01 percent. That means that HIV infects 3,333 new individuals per month, 769 per week, 109 per day, and 4 per hour. It also has an undiagnosed prevalence rate of 0.08 percent.

The transmission of infectious disease by those who are unaware that they have the disease themselves is a very serious hindrance to reducing transmission. This article will study and discuss the effects of carriers on the transmission of infectious diseases and determine values associated with the problems that these carriers can cause.

2 A General Model with Carriers

2.1 Model Formulation

In order to analyze the importance of carriers in disease dynamics, we first formulate an $S - I_c - I - R$ epidemic model where S represents susceptibles, I_c carriers, I infectives, and R the removed class. We take into account that susceptibles who come into contact with infected individuals or carriers can become either infected without symptoms (carriers) or infecteds who exhibit symptoms themselves by assuming that a proportion (p) of those in contact become carriers and $1 - p$ become infecteds.



The differential equations that govern our model are:

$$\begin{aligned} S' &= b - d_1 S - S(\beta I_c + \gamma I) - \theta S \\ I_c' &= pS(\beta I_c + \gamma I) - (d_2 + \alpha) I_c \\ I' &= (1-p)S(\beta I_c + \gamma I) - (d_3 + \pi) I + \alpha I_c \\ R' &= \pi I + \theta S - d_4 R \\ N' &= S' + I_c' + I' + R' = b - d_1 S - d_2 I_c - d_3 I - d_4 R. \end{aligned} \tag{1}$$

Here, the $'$ symbol represents the derivative with respect to time, t, and N represents the total population. Also, b is the birth rate, d_1, d_2, d_3, d_4 are the death rates of those in the susceptible, carrier, infected, and removed classes (respectively) who die of non disease-related causes, β is the rate that susceptibles come into contact with carriers, γ is the rate that susceptibles come into contact with infecteds, α is the rate at which individuals pass from the carrier class into the infecteds class, θ is the vaccination rate, and π is the disease-related removal rate.

We assume that those in the carrier class have a higher rate of transmission ($\beta > \gamma$) due to the fact that they are more likely to be unaware of their condition and therefore continue with their regular behaviours. In the case of HIV, I_c can be said to be those who are not aware of their infection versus those in the I class who are aware and more careful. Therefore in the case of HIV, most of the individuals would first enter the carrier class, and then move to the infected class.

2.2 Equilibria

From (1) we have that $S' \leq b-(d_1+\theta)S$, and therefore $\limsup_{t\to\infty} S \leq \frac{b}{(d_1+\theta)}$ along each solution to (1). Now from (1) we can see that

$$N' = b - d_1 S - d_2 I_c - d_3 I - d_4 R \leq b - \bar{d} N,$$

where $\bar{d} = min\{d_1, d_2, d_3, d_4\}$. Thus $\limsup_{t\to\infty} N \leq b/\bar{d}$. The equation for R can be omitted in our analysis as R does not appear in any of the other equations. This shows that the model can be studied in the feasible region

$$\Gamma = \{(S, I_c, I) \in \Re^3_+ \ : \ S \leq \frac{b}{(d_1+\theta)}, S + I_c + I \leq b/\bar{d}\}.$$

It can be verified that Γ is positively invariant with respect to (1).

The first step in our analysis is to find the equilibria (S^*, I_c^*, I^*) such that

$$0 = b - d_1 S^* - S^*(\beta I_c^* + \gamma I^*) - \theta S^*,$$

$$0 = pS^*(\beta I_c^* + \gamma I^*) - (d_2 + \alpha) I_c^*,$$

$$0 = (1-p)S^*(\beta I_c^* + \gamma I^*) - (d_3 + \pi) I^* + \alpha I_c^*.$$

There are two equilibria.

The disease-free equilibria occurs when there are no carriers or infecteds in the system: $P_0 = (\frac{b}{d_1+\theta}, 0, 0)$. An endemic equilibrium $P^* = (S^*, I_c^*, I^*)$ satisfies $S^*, I_c^*, I^* > 0$. From the equilibrium equations we obtain a unique P^* with

$$S^* = \frac{(d_3+\pi)(d_2+\alpha)}{pd_3\beta + (d_2+\alpha)\gamma + p(\pi\beta - d_2\gamma)}.$$

In order for P^* to exist in Γ, we arrive at the condition that $0 < S^* \leq \frac{b}{d_1+\theta}$. In other words, $\frac{b}{(d_1+\theta)S^*} \geq 1$. If $S^* > \frac{b}{d_1+\theta}$, I_c and I are negative, and not in our feasible region. Define

$$R_0 = \frac{1}{S^*}\frac{b}{d_1+\theta} = \frac{b}{d_1+\theta}\frac{pd_3\beta + (d_2+\alpha)\gamma + p(\pi\beta - d_2\gamma)}{(d_3+\pi)(d_2+\alpha)}.$$

Proposition 2.1 *If $R_0 \leq 1$ then P_0 is the only equilibrium in Γ; if $R_0 > 1$, then there are two equilibria, P_0 and the endemic equilibrium P^*.*

To continue our analysis, we will make use of the Jacobian matrix of our system of differential equations at a point $P = (S, I_c, I)$

$$J(P) = \begin{bmatrix} -d_1 - \beta I_c - \gamma I - \theta & -\beta S & -\gamma S \\ p(\beta I_c + \gamma I) & pS\beta - (d_2+\alpha) & pS\gamma \\ (1-p)(\beta I_c + \gamma I) & (1-p)S\beta + \alpha & (1-p)S\gamma - (d_3+\pi) \end{bmatrix}.$$

3 Mathematical Theory

3.1 Basic Reproductive Number

Rewrite R_0 in the following form:

$$R_0 = \Big[(1-p)\,\gamma\,\frac{1}{d_3+\pi} + p\Big(\beta\,\frac{1}{d_2+\alpha} + \frac{\alpha}{d_2+\alpha}\,\gamma\,\frac{1}{d_3+\pi}\Big)\Big]\frac{b}{d_1+\theta}.$$

We will explain that R_0 represents the average number of secondary infections caused by a single infective in an entirely susceptible population during its whole infectious period.

When a single infective is introduced into the population, with probability $1-p$ it is a non-carrier, hence makes γ effective contacts per unit time. This is multiplied by the average infectious period for non-carriers, $\frac{1}{d_3+\pi}$; with probability p the infective is a carrier, and hence makes β effective contacts per unit time during the average period $\frac{1}{d_2+\alpha}$ it remains a carrier. This number should be augmented by the number of infections caused by this infective after it becomes a non-carrier,with probability $\frac{\alpha}{d_2+\alpha}$, after it becomes a noncarrier, it infects $\gamma\frac{1}{d_3+\pi}$.

This explains the terms in the big square brackets. It is the per capita average number of secondary infections. This number multiplied by the number of susceptibles at the disease-free equilibrium, $\frac{b}{d_1+\theta}$, gives R_0.

The carriers in our system have a great effect on our R_0. The parameters α, d_2, and β all come from the carrier class, and all appear in the basic reproductive number. To decrease the negative impacts of the carriers to our population, we want to either reduce β (the incidence of carriers), or increase α (awareness of those carrying the disease). Doing either of these things or both, will reduce the value of R_0. This means that we can reduce the number of people getting infected by focusing our attention on carriers.

3.2 P_0: Local and Global Stability

To examine the local stability of the disease-free equilibrium P_0 we evaluate the Jacobian at $P_0 = (\frac{b}{d_1+\theta}, 0, 0)$

$$J(P_0) = \begin{bmatrix} -d_1-\theta & -\beta(\frac{b}{d_1+\theta}) & -\gamma(\frac{b}{d_1+\theta}) \\ 0 & p\beta(\frac{b}{d_1+\theta}) - (d_2+\alpha) & p\gamma(\frac{b}{d_1+\theta}) \\ 0 & (1-p)\beta(\frac{b}{d_1+\theta}) + \alpha & (1-p)\gamma(\frac{b}{d_1+\theta}) - (d_3+\pi) \end{bmatrix}.$$

Proposition 3.1 *P_0 is locally asymptotically stable if $R_0 < 1$.*

Proof. One eigenvalue of $J(P_0)$ is $\lambda_1 = -(d_1 + \theta) < 0$. The other two eigenvalues λ_2, λ_3 are eigenvalues of the 2×2 matrix

$$A = \begin{bmatrix} p\beta(\frac{b}{d_1+\theta}) - (d_2+\alpha) & p\gamma(\frac{b}{d_1+\theta}) \\ (1-p)\beta(\frac{b}{d_1+\theta}) + \alpha & (1-p)\gamma(\frac{b}{d_1+\theta}) - (d_3+\pi) \end{bmatrix}.$$

We want to show, when $R_0 < 1$, that the Routh-Hurwitz conditions hold, namely,

1. $trace(A) < 0$
2. $det(A) > 0$.

Simple calculations show that

$$tr(A) = (d_2+\alpha)[\frac{p\beta\frac{b}{d_1+\theta}}{d_2+\alpha} - 1] + (d_3+\pi)[\frac{(1-p)\gamma\frac{b}{d_1+\theta}}{d_3+\pi} - 1].$$

Using our assumption that $R_0 = \frac{b}{d_1+\theta}(\frac{p\beta}{(d_2+\alpha)} + \frac{p\alpha\gamma}{(d_2+\alpha)(d_3+\pi)} + \frac{(1-p)\gamma}{(d_3+\pi)}) < 1$ we have

$$\frac{p\beta\frac{b}{d_1+\theta}}{d_2+\alpha} < 1, \quad \text{and} \quad \frac{(1-\mathrm{p})\gamma\frac{\mathrm{b}}{\mathrm{d}_1+\theta}}{\mathrm{d}_3+\pi} < 1.$$

Therefore

$$[\frac{p\beta\frac{b}{d_1+\theta}}{d_2+\alpha} - 1] < 0, \quad \text{and} \quad [\frac{(1-\mathrm{p})\gamma\frac{\mathrm{b}}{\mathrm{d}_1+\theta}}{\mathrm{d}_3+\pi} - 1] < 0.$$

This shows that

$$tr(A) < 0.$$

Now we calculate

$$\begin{aligned} det(A) &= [p\beta(\frac{b}{d_1+\theta}) - (d_2+\alpha)][(1-p)\gamma(\frac{b}{d_1+\theta}) - (d_3+\pi)] \\ &\quad -[(1-p)\beta(\frac{b}{d_1+\theta}) + \alpha][p\gamma(\frac{b}{d_1+\theta})] \\ &= (d_2+\alpha)(d_3+\pi) - (d_3+\pi)p\beta(\frac{b}{d_1+\theta}) - d_2(1-p)\gamma(\frac{b}{d_1+\theta}) \\ &= (d_2+\alpha)(d_3+\pi)[1-R_0]. \end{aligned}$$

Therefore, $det(A) > 0$ if and only if $R_0 < 1$. This proves the proposition.

Theorem 3.1 *P_0 is globally asymptotically stable for $R_0 \leq 1$ and unstable if $R_0 > 1$.*

Proof. To prove global asymptotic stability of P_0 we use Lyapunov functions. Choose

$$L = (\frac{\beta}{d_2+\alpha} + \frac{\gamma\alpha}{(d_3+\pi)(d_2+\alpha)})I_c + (\frac{\gamma}{d_3+\pi})I > 0.$$

Then

$$\begin{aligned} L' &= (\frac{\beta}{d_2+\alpha} + \frac{\gamma\alpha}{(d_3+\pi)(d_2+\alpha)})I_c' + (\frac{\gamma}{d_3+\pi})I' \\ &= [(\frac{p\beta}{d_2+\alpha} + \frac{p\gamma\alpha}{(d_3+\pi)(d_2+\alpha)} + \frac{(1-p)\gamma}{d_3+\pi})]S(\beta I_c + \gamma I) - (\beta I_c + \gamma I) \\ &= [(\frac{p\beta}{d_2+\alpha} + \frac{p\gamma\alpha}{(d_3+\pi)(d_2+\alpha)} + \frac{(1-p)\gamma}{d_3+\pi})S - 1](\beta I_c + \gamma I). \\ &= [\frac{d_1+\theta}{b}R_0 S - 1](\beta I_c + \gamma I) \end{aligned}$$

Using $R_0 \leq 1$ and $S \leq \frac{b}{d_1+\theta}$,

$$L' \leq [R_0 - 1](\beta I_c + \gamma I) \leq 0.$$

So $L' \leq 0$ if $R_0 \leq 1$. Also, $L' = 0 \Leftrightarrow I_c = I = 0$ or $R_0 = 1$ and $S = \frac{b}{d_1+\theta} = S^*$. Therefore the largest compact invariant set in the closure of Γ, $(\bar{\Gamma})$ where $L' = 0$ for $R_0 \leq 1$ is the singleton $\{P_0\}$. By LaSalle's Invariance Principle, we see that all solutions in $\bar{\Gamma}$ converge to P_0. If $R_0 > 1$, then $L' > 0$ except when $I_c = I = 0$ in a small neighborhood of P_0. In this case, all solutions in $\bar{\Gamma}$ starting close enough to P_0 leave the neighbourhood of P_0 except those starting on the S axis, or in other words, when $S' = b - (d_1 + \theta)S$ and $S \to \frac{b}{d_1+\theta}$. Thus we have proven our proposition.

3.3 P^*: Local and Global Stability

We study the local stability of P^* by examining the Jacobian matrix

$$J(P^*) = \begin{bmatrix} -d_1 - \theta - (\beta I_c^* + \gamma I^*) & -S^*\beta & -S^*\gamma \\ p(\beta I_c^* + \gamma I^*) & p\beta S^* - (d_2 + \alpha) & p\gamma S^* \\ (1-p)(\beta I_c^* + \gamma I^*) & (1-p)\beta S^* + \alpha & (1-p)\gamma S^* - (d_3 + \pi) \end{bmatrix}.$$

Proposition 3.2 *P^* is locally asymptotically stable if $R_0 > 1$.*

Proof. We want to show, when $R_0 < 1$, that the Routh-Hurwitz conditions hold, namely,

1. $trace(J) < 0$
2. $det(J) < 0$
3. $tr(J)(a_2) - det(J) > 0$, where a_2 is the sum of the determinants of the 2x2 principle minors of J.

We calculate that

$$tr(J) = -(d_1 + \theta) - (d_2 + \alpha) - (d_3 + \gamma) - (\beta I_c^* + \gamma I^*) + p\beta S^* + (1-p)\gamma S^*.$$

From the equilibrium equations, we have that

$$[p\beta S^* - (d_2 + \alpha)] = \frac{-1}{I_c^*}[p\gamma S^* I^*] < 0 \tag{2}$$

and

$$[(1-p)\gamma S^* - (d_3 + \pi)] = \frac{-1}{I^*}[(1-p)\beta S^* I_c^* + \alpha I_c^*] < 0. \tag{3}$$

Therefore

$$tr(J) = \frac{-1}{I_c^*}[p\gamma S^* I^*] - \frac{1}{I^*}[(1-p)\beta S^* I_c^* + \alpha I_c^*] - (\beta I_c^* + \gamma I^*) - (d_1 + \theta) < 0.$$

Straightforward calculation gives

$$det(J) = -C_1(d_1 + \theta + \beta I_c^* + \gamma I^*) + C_2\beta S^* - C_3\gamma S^*, \tag{4}$$

where

$$\begin{aligned} C_1 &= (p\beta S^* - (d_2+\alpha))((1-p)\gamma S^* - (d_3+\pi)) - p\gamma S^*\beta S^*(1-p) - \alpha\gamma p S^* \\ &= (d_2+\alpha)(d_3+\pi) - S^*[p\beta d_3 + p\beta\pi + (1-p)\gamma d_2 + \gamma\alpha] \\ &= 1 - S^*[\frac{p\beta d_3 + p\beta\pi + (1-p)\gamma d_2 + \gamma\alpha}{(d_2+\alpha)(d_3+\pi)}] \\ &= 1 - \frac{S^*}{S^*} = 0, \end{aligned} \tag{5}$$

$$\begin{aligned} C_2 &= (p\beta I_c^* + p\gamma I^*)((1-p)\gamma S^* - (d_3+\pi)) - p\gamma S^*(1-p)(\beta I_c^* + \gamma I^*) \\ &= -p(d_3+\pi)(\beta I_c^* + \gamma I^*) < 0, \end{aligned} \tag{6}$$

and

$$\begin{aligned} C_3 &= (p\beta I_c^* + p\gamma I^*)((1-p)\beta S^* - (d_2+\alpha)) \\ &\quad -[(1-p)(\beta I_c^* + \gamma I^*)](p\beta S^* - (d_2+\alpha)) \\ &= d_2(1-p)(\beta I_c^* + \gamma I^*) + \alpha(\beta I_c^* + \gamma I^*) > 0. \end{aligned} \tag{7}$$

It follows from (4)-(7) that

$$det(J) < 0.$$

We now find that

$$\begin{aligned} tr(J)(a_2) &= [-(d_1+\theta) - (\beta I_c^* + \gamma I^*) + (p\beta S^* - (d_2+\alpha)) + ((1-p)\gamma S^* \\ &\quad -(d_3+\pi))][(-(d_1+\theta) - (\beta I_c^* + \gamma I^*))(p\beta S^* - (d_2+\alpha)) \\ &\quad +p\beta S^*(\beta I_c^* + \gamma I^*) - ((d_1+\theta) + (\beta I_c^* + \gamma I^*))((1-p)\gamma S^* \\ &\quad -(d_3+\pi)) + (1-p)\gamma S^*(\beta I_c^* + \gamma I^*)]. \end{aligned}$$

Using (2) and (3) we can see that all of the terms in $tr(J)$ are negative and all of the terms in a_2 are positive. Also, we know that $det(J) < 0$ so $-det(J) > 0$.
It is simple to see that

$$\begin{aligned} tr(J)(a_2) - det(J) &= [-(d_1+\theta) - (\beta I_c^* + \gamma I^*)]a_2 + [p\beta S^* - (d_2+\alpha)] \\ &\quad [(-(d_1+\theta) - (\beta I_c^* + \gamma I^*))(p\beta S^* - (d_2+\alpha)) \\ &\quad + p\beta S^*(\beta I_c^* + \gamma I^*)] + [(1-p)\gamma S^* - (d_3+\pi)] \\ &\quad [-((d_1+\theta) + (\beta I_c^* + \gamma I^*))((1-p)\gamma S^* - (d_3+\pi)) \\ &\quad + (1-p)\gamma S^*(\beta I_c^* + \gamma I^*)] + D_1 + D_2 + D_3 + D_4, \end{aligned}$$

where:

$$\begin{aligned} D_1 &= [-((d_1+\theta) + (\beta I_c^* + \gamma I^*))((1-p)\gamma S^* - (d_3+\pi))][p\beta S^* - (d_2+\alpha)] \\ &= -p\gamma S^*((1-p)\beta S^* + \alpha)(d_1+\theta) - p\gamma S^*((1-p)\beta S^* + \alpha)(\beta I_c^* + \gamma I^*), \end{aligned}$$

$$\begin{aligned} D_2 &= [-((d_1+\theta) + (\beta I_c^* + \gamma I^*))((1-p)\gamma S^* - (d_3+\pi))][p\beta S^* - (d_2+\alpha)] \\ &= -(d_1+\theta)((1-p)\gamma S^* - (d_3+\pi))(p\beta S^* - (d_2+\alpha)) \\ &\quad -(\beta I_c^* + \gamma I^*)[p(1-p)\beta S^*\gamma S^* - p\beta S^*(d_3+\pi) \\ &\quad -(1-p)\gamma S^*(d_2+\alpha) + (d_3+\pi)(d_2+\alpha)] \\ &< -(\beta I_c^* + \gamma I^*)p(1-p)\gamma S^*\beta S^* - (\beta I_c^* + \gamma I^*)[-p\beta S^*(d_3+\pi) \\ &\quad -(1-p)\gamma S^*(d_2+\alpha) + (d_3+\pi)(d_2+\alpha)] \\ &= -p\beta S^*(1-p)\gamma S^*(\beta I_c^* + \gamma I^*) - (\beta I_c^* + \gamma I^*)(p\gamma\alpha S^*), \end{aligned}$$

$$D_3 = [((1-p)\gamma S^* - (d_3+\pi)][p\beta S^*(\beta I_c^* + \gamma I^*),$$

and

$$D_4 = [p\beta S^* - (d_2+\alpha)][(1-p)\gamma S^*(\beta I_c^* + \gamma I^*)].$$

After making these substitutions, we arrive at:

$$
\begin{aligned}
& tr(J)(a_2) - det(J) \\
& < [-(d_1+\theta) - (\beta I_c^* + \gamma I^*)]a_2 + [p\beta S^* - (d_2+\alpha)] \\
& [-((d_1+\theta) + (\beta I_c^* + \gamma I^*))(p\beta S^* - (d_2+\alpha)) + p\beta S^*(\beta I_c^* + \gamma I^*)] \\
& +[(1-p)\gamma S^* - (d_3+\pi)][-((d_1+\theta) + (\beta I_c^* + \gamma I^*)) \\
& ((1-p)\gamma S^* - (d_3+\pi)) + (1-p)\gamma S^*(\beta I_c^* + \gamma I^*)] \\
& -p\gamma S^*(d_1+\theta)((1-p)\beta S^* + \alpha) - p\gamma\alpha S^*(\beta I_c^* + \gamma I^*) \\
& = -[(d_1+\theta) + (\beta I_c^* + \gamma I^*)]a_2 \\
& -[\frac{p\gamma S^* I^*}{I_c^*}][((d_1+\theta) + (\beta I_c^* + \gamma I^*))(\frac{p\gamma S^* I^*}{I_c^*}) + p\beta S^*(\beta I_c^* + \gamma I^*)] \\
& -[\frac{(1-p)\beta S^* I_c^*}{I^*}][((d_1+\theta) + (\beta I_c^* + \gamma I^*))\frac{(1-p)\beta S^* I_c^*}{I^*} + (1-p)\gamma S^* \\
& (\beta I_c^* + \gamma I^*)] - p\gamma S^*(d_1+\theta)((1-p)\beta S^* + \alpha) - p\gamma\alpha S^*(\beta I_c^* + \gamma I^*).
\end{aligned}
$$

All of the term in the determinant have been canceled, and the remaining terms are all, in fact, negative. This proves the proposition.

Theorem 3.2 *P^* is globally asymptotically stable if $R_0 > 1$.*

Proof. To study the global stability of the endemic equilibrium, we again make use of a Lyapunov function, this time of the form:

$$V = x_1(S - S^* \ln S) + x_2(I_c - I_c^* \ln I_c) + x_3(I - I^* \ln I), \tag{8}$$

and

$$
\begin{aligned}
V' &= x_1(S' - \frac{S^*}{S}S') + x_2(I_c' - \frac{I_c^*}{I_c}I_c') + x_3(I' - \frac{I^*}{I}I') \\
&= x_1(b - (d_1+\theta)S - (\beta I_c + \gamma I)S - bS^*/S + (d_1+\theta)S^* + (\beta I_c + \gamma I)S^*) \\
&+x_2((1-p)(\beta I_c + \gamma I)S - (d_2+\alpha)I_c - (1-p)\frac{\beta I_c S I_c^*}{I_c} - (1-p)\frac{\gamma I S I_c^*}{I_c} \\
&+(d_2+\alpha)I_c^*) + x_3(p(\beta I_c + \gamma I)S - \alpha I_c - (d_3+\pi)I - p\frac{\beta I_c S I^*}{I}
\end{aligned}
$$

$$
\begin{aligned}
&-p\frac{\gamma ISI^*}{I} - \frac{\alpha I_c I^*}{I} + (d_3+\pi)I*) \\
&= x_1((d_1+\theta)S^* + (\beta I_c^* + \gamma I^*)S^* - (d_1+\theta)S - (\beta I_c + \gamma I)S \\
&+(d_1+\theta)S^* + (\beta I_c^* + \gamma I^*)S^* - \frac{((d_1+\theta)S^* + (\beta I_c^* + \gamma I^*)S^*)S^*}{S}) \\
&+x_2((1-p)(\beta I_c + \gamma I)S - (d_2+\alpha)I_c - (1-p)\beta SI_c^* - (1-p)\frac{\gamma ISI_c^*}{I_c} \\
&+(d_2+\alpha)I_c^*) + x_3(p(\beta I_c + \gamma I)S - \alpha I_c - (d_3+\pi)I - p\frac{\beta I_c SI^*}{I} \\
&-p\gamma SI^* - \frac{\alpha I_c I^*}{I} + (d_3+\pi)I*) \\
&= x_1((d_1+\theta)S^*(2 - \frac{S^*}{S} - \frac{S}{S^*}) + (\beta I_c^* + \gamma I^*)S^* + (\beta I_c + \gamma I)(S^* - S) \\
&-\frac{(\beta I_c^* + \gamma I^*)S^{*2}}{S}) + x_2((1-p)(\beta I_c + \gamma I)S - (d_2+\alpha)I_c \\
&-(1-p)\beta SI_c^* - (1-p)\frac{\gamma ISI_c^*}{I_c} + (d_2+\alpha)I_c^*) + x_3(p(\beta I_c + \gamma I)S - \alpha I_c \\
&-(d_3+\pi)I - p\frac{\beta I_c SI^*}{I} - p\gamma SI^* - \frac{\alpha I_c I^*}{I} + (d_3+\pi)I*) \\
&= x_1((d_1+\theta)S^*(2 - \frac{S^*}{S} - \frac{S}{S^*}) \\
&+[x_1(\beta I_c^* + \gamma I^*)S^* + x_2(d_2+\alpha)I_c^* + x_3(d_3+\pi)I*] \\
&+[-x_1\frac{(\beta I_c^* + \gamma I^*)S^{*2}}{S} - x_2(1-p)\beta SI_c^* - x_2(1-p)\frac{\gamma ISI_c^*}{I_c} \\
&-x_3p\frac{\beta I_c SI^*}{I} - x_3p\gamma SI^* - x_3\frac{\alpha I_c I^*}{I}]
\end{aligned}
$$

after applying the identity $b = d_1S^* + \theta S^* + \beta I_c^* S^* + \gamma I^* S^*$. In order to cancel the nonlinear terms, x_1, x_2, and x_3 are positive constants determined by

$$-x_1 + x_2(1-p) + x_3p = 0,$$

$$x_1\gamma S^* - x_3(d_3+\pi) = 0,$$

$$x_1\beta S^* - x_2(d_2+\alpha) + x_3\alpha = 0.$$

So we find that

$$x_1 = 1, \tag{9}$$

$$x_2 = \frac{(d_3+\pi)\beta S^* + \gamma\alpha S^*}{(d_2+\alpha)(d_3+\pi)}, \tag{10}$$

$$x_3 = \frac{\gamma S^*}{(d_3+\pi)}. \tag{11}$$

Now we can rearrange V' into $V_1 + V_2 + V_3$ where

$$V_1 = (d_1+\theta)S^*(2 - \frac{S}{S^*} - \frac{S^*}{S}),$$

$$V_2 = x_1(\beta S^* I_c^* + \gamma I^* S^*) + x_2(d_2+\alpha)I_c^* + x_3(d_3+\pi)I^*,$$

and

$$\begin{aligned} V_3 \;&= -\frac{x_1(\beta I_c^* S^* + \gamma I^* S^*)S^*}{S} - x_2(1-p)\beta S I_c^* - x_3 p\gamma S I^* \\ &\quad - \frac{x_2(1-p)\gamma I S I_c^*}{I_c} - \frac{x_3 p\beta S I_c I^*}{I} - \frac{x_3\alpha I_c I^*}{I}. \end{aligned}$$

$V_1 < 0$ by $x + \frac{1}{x} \geq 2$ for $x > 0$ so we are left to show that $V_2 + V_3 < 0$. We begin by examining V_2 and re-write it using the values for x_1, x_2, and x_3 and the identities

$$x_2(1-p) + px_3 = 1, \tag{12}$$

$$(pd_2+\alpha)I_c^* = (1-p)(d_3+\pi)I^*. \tag{13}$$

So

$$V_2 = 2px_3\gamma I^* S^* + 2(1-p)x_2\beta I_c^* S^* + 4px_3\beta I_c^* S^* + \frac{3(1-p)\alpha}{(pd_2+\alpha)}\gamma I^* S^*.$$

We also rewrite V_3 into the form:

$$
\begin{aligned}
V_3 &= [-x_2(1-p)\beta I_c^* S - \frac{(1-p)x_2\beta S^{*2} I_c^*}{S}] \\
&+[-x_3 p\gamma S I^* - \frac{px_3\gamma I^* S^{*2}}{S}] \\
&+[-y\frac{x_2(1-p)\gamma I S I_c^*}{I_c} - \frac{x_3\alpha I_c I^*}{I} - y\frac{(1-p)x_2\gamma I^* S^{*2}}{S}] \\
&+[-(1-y)\frac{x_2(1-p)\gamma I S I_c^*}{I_c} - \frac{x_3 p\beta I_c S I^*}{I} - (1-y)\frac{(1-p)x_2\gamma I^* S^{*2}}{S} \\
&-\frac{px_3\beta S^{*2} I_c^*}{S}],
\end{aligned}
$$

where

$$y = \frac{(1-p)\alpha}{(pd_2+\alpha)(1-p)x_2}$$

and

$$1 - y = \frac{(1-p)p\beta S^*}{(pd_2+\alpha)(1-p)x_2}.$$

Now let $V_3 = V_a + V_b + V_c + V_d$ accordingly. We can come up with an inequality for each part of V_3:

$$
\begin{aligned}
V_a &= -x_2(1-p)\beta I_c^* S - \frac{(1-p)x_2\beta S^{*2} I_c*}{S} \\
&\leq -2\sqrt{(x_2(1-p))^2(\beta I_c^* S^*)^2} \\
&= -2(1-p)x_2\beta I_c^* S^*,
\end{aligned}
$$

$$
\begin{aligned}
V_b &= -x_3 p\gamma S I^* - \frac{px_3\gamma I^* S^{*2}}{S} \\
&\leq -2\sqrt{(x_3 p)^2(\gamma I^* S^*)^2} \\
&= -2px_3\gamma I^* S^*,
\end{aligned}
$$

$$\begin{aligned} V_c &= -y\frac{x_2(1-p)\gamma I S I_c^*}{I_c} - \frac{x_3\alpha I_c I^*}{I} - y\frac{(1-p)x_2\gamma I^* S^{*2}}{S} \\ &\leq -3[(x_2(1-p))^2 x_3\alpha I_c^* y^2(\gamma I^* S^*)^2]^{\frac{1}{3}} \\ &= -\frac{3(1-p)\alpha}{(pd_2+\alpha)}\gamma I^* S^*, \end{aligned}$$

and

$$\begin{aligned} V_d &= -(1-y)\frac{x_2(1-p)\gamma I S I_c^*}{I_c} - \frac{x_3 p\beta I_c S I^*}{I} \\ &\quad -(1-y)\frac{(1-p)x_2\gamma I^* S^{*2}}{S} - \frac{px_3\beta S^{*2} I_c^*}{S} \\ &\leq -4[(x_2(1-p))^2(px_3)^2(1-y)^2\gamma I^* S^* 2\beta I_c^* S^{*2}\gamma\beta I^* I_c^*]^{\frac{1}{4}} \\ &= -4px_3\beta I_c^* S^*, \end{aligned}$$

so that $V_3 \leq -2(1-p)x_2\beta I_c^* S^* - 2px_3\gamma I^* S^* - 4px_3\beta I_c^* S^* - \frac{3(1-p)\alpha}{(pd_2+\alpha)}\gamma I^* S^*$. Therefore we have shown that $V_2 + V_3 < 0$ and indeed $V' \leq 0$. In fact $V' = 0 \Leftrightarrow (S, I_c, I) = (S^*, I_c^*, I^*)$.
This tells us that for for parameter choices where $R_0 > 1$ we will always reach the endemic equilibrium.

4 Discussion of Hepatitis B

4.1 Biology of the Disease

Hepatitis B is a liver disease caused by the HBV virus of the Hepadnavirus family. It is transmitted by body fluids through sexual contact, the sharing of infected needles, or from mother to infant. Hepatitis B's symptoms include jaundice, abdominal pain, nausea, fatigue and joint pain. However, 30 percent of people with the disease do not show any of these symptoms. These people are the carriers.

Individuals with the Hepatitis B virus are given the carrier status if they have the surface antigen HBsAg present for over 6 months, but have normal transaminase, are negative for Hepatitis e antigens, and have undetectable HBV-DNA levels. The course of the disease in these "healthy" carriers is generally considered benign, meaning that the clinically significant liver complications and liver disease do not develop. However, these people are more likely to be unaware of their condition due to this lack of effect and transmit to others. In those who do show symptoms, HBV is the cause of up to 80 percent of liver cancer cases. Also, since a majority of patients are asymptomatic at the early stages, by the time they seek medical attention, only 10-20 percent of patients have the prospect of recovery.

Hepatitis B is now an endemic in China and parts of both Asia and Africa. In fact, one third of the world either has been, or is now infected with Hepatitis B. There are now over 1.25 million chronic (meaning those who can not be cured) Americans of which 20-30 percent have had the disease since childhood. The highest rate of disease contraction is in those individuals who are 20-49 years old. In China (which has the world's highest Hepatitis B rate), 1 in 10 people die from from the virus, which translates to around 1/2 million people per year. Those who do not die are at more than 100 times higher risk for developing cirrhosis and liver cancer.

Hepatitis B is a retrovirus, which means that it synthesizes DNA from RNA through reverse transcription (as with HIV). Reverse transcription is the process of converting DNA to RNA. The virus has an inner core and an outer envelope made of the protein HBsAg, which is known as a "surface antigen". It replicates within infected liver cells called "hepatocytes". The virus attaches itself to a liver cell membrane, after which it is transported into the liver cell. Once inside, the virus's core releases its contents of DNA and DNA polymerase into the liver cell nucleus. The liver cell now begins to produce (via RNA) copies of the Hepatitis B DNA and releases these copies from the membrane into the blood to infect other liver cells. The incubation of the virus is anywhere from 6 to 25 weeks.

There is a vaccine for Hepatitis B. It has been made from recombinant technology since 1982 and since then many countries have been vaccinating infants and health care workers. The vaccine, which is made from the surface antigen HBsAg, is synthesized from common baker's yeast (Saccharomyces cerevisiae).

Routine vaccination has caused a great decline in the amount of children infected, from 260,000 in the 1980's to approximately 60,000 in 2004. This

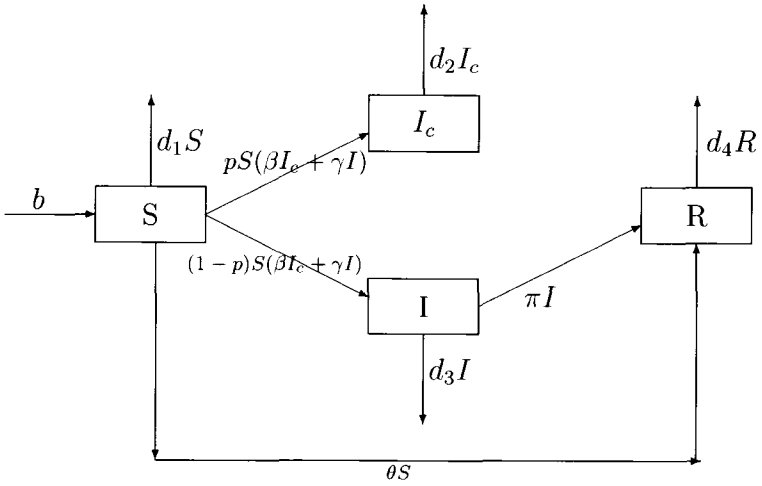
is immensely important as there may be many long term effects without vaccination. For example, 90 percent of of those infected as infants, 30 percent of children infected from 1 to 5 years old, and 6 percent of those infected after 5 years old become chronically infected, and 15-25 percent of those will die from the disease.

Hepatitis B is a serious disease with many far-reaching consequences all over the world. However, with a proper vaccination schedule, the disease may one day be completely eradicated.

4.2 Model Study

To model Hepatitis B, we can use the model that we have already formulated, with some minor adjustments. Since in the case of Hepatitis B those who are carriers do not ever advance to the symptomatic stages of the disease (and therefore do not die of their infection), we can assume that the parameter $\alpha = 0$.

Our new model looks like:



The differential equations that govern our model are:

$$\begin{aligned} S' &= b - d_1 S - S(\beta I_c - \gamma I) - \theta S \\ I_c' &= pS(\beta I_c + \gamma I) - d_2 I_c \end{aligned}$$

$$\begin{aligned} I' &= (1-p)S(\beta I_c + \gamma I) - (d_3 + \pi)I \\ R' &= \pi I + \theta S - d_4 R \\ N' &= S' + I_c' + I' + R' = b - d_1 S - d_2 I_c - d_3 I - d_4 R. \end{aligned} \tag{14}$$

Our study of this adjusted model remains similar to that of the original model. In this case we have the two equilibria:

$$P_0 = (\tfrac{b}{d_1+\theta}, 0, 0),$$

and the endemic equilibria (the point where there are both infected people and susceptible individuals in the population)

$$P^* = (S^*, I_c^*, I^*),$$

$$S^* = \tfrac{d_2(d_3+\pi)}{pd_3\beta + d_2\gamma + p(\pi\beta - d_2\gamma)}.$$

We arrive at the same condition for P^* to exist (ie, for $S^*, I_c^*, I^* \geq 0$), that is, $0 < S^* \leq \frac{b}{d_1+\theta}$. In other words, $\frac{b}{(d_1+\theta)S^*} \geq 1$. This gives us a similar value for the reproductive number of the sysem,

$$R_0 = \frac{1}{S^*}\frac{b}{d_1+\theta} = \frac{b}{d_1+\theta}\left(\frac{pd_3\beta + d_2\gamma + p(\pi\beta - d_2\gamma)}{d_2(d_3+\pi)}\right).$$

4.3 Mathematical Results

Since those who are carriers of the Hepatitis B virus continue to not experience the symptoms of their disease, we can set $\alpha = 0$ in our analysis of the general model and arrive at the following results.

Theorem 4.1 *For the Hepatitis B model, P_0 is globally asymptotically stable for $R_0 < 1$, and P^* is globally asymptotically stable for $R_0 > 1$.*

4.4 Biological Interpretations

Carriers of the Hepatitis B disease pose a risk to themselves, their contacts, blood banking, and transplantation services. It can not be detected with routine blood tests, the Hepatitis B tests must be asked for specifically, which is all the more reason to focus on the prevention aspects of transmission.

A study done in the Plzen region of the Czech region from 1997-2004 examined the transmission of Hepatitis B. 939 HBV-positive people were observed, 63.4 percent of which were asymptomatic carriers. 18.2 percent of the 829 family and sexual contacts of the study group tested positive for HBV, with the highest percent being among siblings and parents (36.5 and 27.3 percent respectively). This study emphasizes the importance of immunizing household contacts of chronic Hepatitis B carriers which has the potential to significantly reduce the spread of the disease.

Another transmission method that can be eliminated with proper vaccination is the transmission from mother to baby via breast feeding. Studies have shown that with appropriate vaccination of the babies, breast feeding by HBV positive mothers poses no additional risk for virus transmission.

In the case of Hepatitis B, carriers are hard to identify due to their lack of symptoms. If the disease were to be treated, it would be treated in those who exhibit the HBV virus. This means that as most carriers would not know that they carry the infection, they would not be treated and would continue to spread the disease. Though vaccination is important in the case of any communicable disease, it is perhaps the most important parameter for health officials in this case because of the lack of effect that treatment or quarantine would have on the carrier class. θ is crucial since it takes susceptibles directly into the removed class so that they can not become infected even if they come into contact with someone carrying the Hepatitis B disease. For a disease such as Hepatitis B with carriers and a developed immunization, routine vaccination would be more effective then treatment. The presence of symptom-free carriers means that stopping individuals from becoming infected rather then treating them after infection would be one of the only ways to stop the spread on Hepatitis B.

If we can raise the value of θ through more vaccinations or more effective vaccination schedules (vaccinating those who are most likely to come in contact with the disease), then we reduce the value of R_0. This would have an effect of increasing the region where P_0 is stable and therefore would increase the chances of the population being able to reach and sustain a disease-free

equilibrium.

4.5 Numerical Simulations

Using mathematical software we can analyze the dynamics of our system visually. Studies have shown that the incidence rate for Hepatitis B in the USA is approximately 1 in 1,359 or 0.07 percent. So for our case we will take $\beta = 0.0009$ and $\gamma = 0.0005$ (their average is 0.0007). We can now choose our remaining parameters: $b = 3$, $d_1 = 0.002$, $d_2 = 0.002$, $d_3 = 0.004$, $\pi = 0.01$, $p = 0.5$ and initial conditions: $S[0] = 50$, $I_c[0] = 1$, $I[0] = 10$, and then solve the system (14)numerically.

To examine the effects of vaccination rates, we will first take $\theta = 0.25$. In this case, $R_0 = 2.89116 > 1$. Our previous analysis tells us to expect to reach an endemic equilibrium of $(S^*, I_c^*, I^*) = (4.11765, 490.588, 70.084)$.

If instead we raise θ from 0.25 to 0.75, we observe that without changing any of the other parameters we now have a reproductive number $R_0 = 0.968845 < 1$ and a new equilibrium of $(S^*, I_c^*, I^*) = (3.98936, 0, 0)$.

When we plot the progression of these systems from our numerical simulations we observe the same results.

Shown here are the trajectories for $\theta = 0.25$ (the higher curves) and $\theta = 0.75$ (the lower curves).

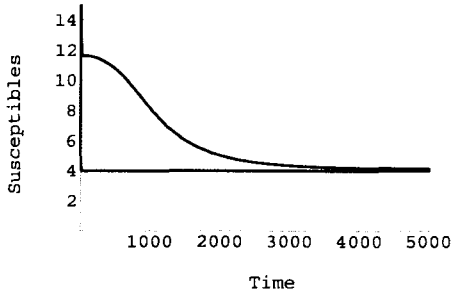


Figure 1: S^*

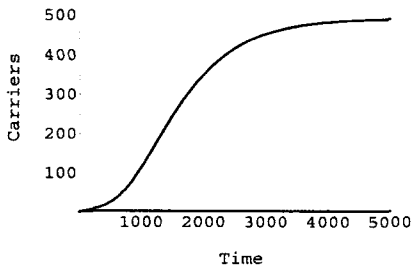


Figure 2: I_c^*

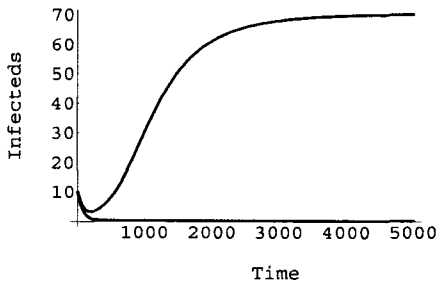


Figure 3: I^*

In fact we can calculate a threshold value for θ where the population will move from the endemic to disease-free equilibrium. If

$$\theta > \frac{b}{S^*} - d_1 \Rightarrow R_0 < 1,$$

$$\theta < \frac{b}{S^*} - d_1 \Rightarrow R_0 > 1.$$

Using our parameters, this threshold value is $\theta = 0.726571$.

Therefore, if we could use real-world data to calculate the threshold value, by increasing the vaccination rate past this value, we could eliminate Hepatitis B.

5 Discussion of HIV

5.1 Biology of the Disease

HIV (Human Immunodeficiency Virus) which becomes AIDS (Acquired Immunodeficiency Syndrome) is one of the top killers today. It has killed over 25 million people since it was first recognized, making it one of the most destructive pandemics in recorded history. As of yet, there is no vaccine for HIV because it uses the body's own genetic material to make copies of itself. There is also no cure for HIV, however, there is a fairly recent antiretroviral treatment called AZT (azidothymidine) that reduces progress of the virus to the AIDS stage. As with Hepatitis B, HIV is a "retrovirus", which means its genetic material is RNA (vs DNA) so it depends on its host to replicate. Also as with Hepatitis B, HIV is transmitted through body fluids, and it is therefore very important for those carrying the disease to be aware of it so that they can refrain from "risky" behaviours that may infect others.

HIV works by causing an individual's immune system to fail, leaving them open to all kinds of infections. It floats through the body as a free virus and within infected immune cells. HIV works on the white blood cells. In particular, helper-T (CD4+T) cells whose function is to recognize the virus and trigger immune response, and macrophages, who are large white blood cells that engulf and destroy invaders.

HIV cells attach to CD4+T cells and inject their RNA. This viral RNA then turns to viral DNA copies through the reverse transcriptase enzyme after which the infected cell can produce up to 10,000 new HIV copies before it is destroyed. The immune system then weakens as the number of CD4+T cells available becomes smaller and smaller.

In the United States approximately 1 million people have HIV/AIDS and 40,000 more become infected each year. The CDC says that 1/4 of the people in the USA with HIV are unaware that they are infected. Awareness lets individuals reduce the chance of infection themselves or transmitting the infection by taking measures such as practicing safe sex or using clean, unshared needles and it is therefore very important that awareness is increased. This can be done through regular testing of all those people who have reason to believe that they may have been exposed to the virus. Less than 1 percent of the sexually active African urban population has been tested for HIV, and that number is even lower for the rural areas. This is a major contributing factor to the growing AIDS epidemic in Africa and other underdeveloped

countries.

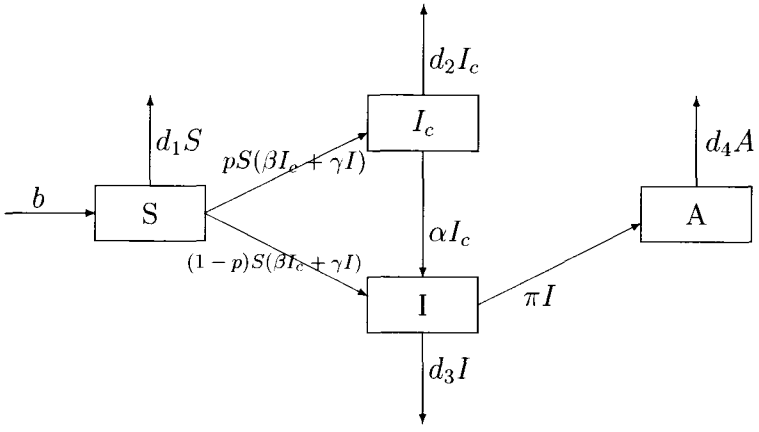
The HIV-1 test uses enzyme-linked immunosorbent assay (ELISA) to detect the antibodies that build up in those who are infected. This test, as well as many other blood tests detect HIV within 4 weeks of contraction, but it can take up to 3 to 6 months for the HIV antibodies to reach detectable levels. Most health officials recommend waiting 6 months before getting tested for greatest accuracy.

So, with an increase in awareness and testing, we may be able to slow the spread of HIV and eventually eliminate the disease altogether.

5.2 Model Study

To model HIV we can again use our original model with some changes. Since in the case of HIV there is no vaccine, we can assume that the parameter $\theta = 0$. Also, in this case, we assume that those in the carrier stage are those who are unaware that they are infected, and therefore would have a higher rate of transmission then those in the infected class. α is now the rate at which individuals become aware of their condition. The final point to notice in the case of HIV is that everyone who is infected eventually moves to the AIDS stage and dies of the disease, therefore π is now the rate of change from the HIV stage to the AIDS stage (the removed class, which we now label A).

Our HIV model looks like:



The differential equations that govern our model are now:

$$\begin{aligned} S' &= b - d_1 S - S(\beta I_c + \gamma I) \\ I_c' &= pS(\beta I_c + \gamma I) - (d_2 + \alpha) I_c \\ I' &= (1-p)S(\beta I_c + \gamma I) - (d_3 + \pi) I + \alpha I_c \\ A' &= \pi I - d_4 A \\ N' &= S' + I_c' + I' + A' = b - d_1 S - d_2 I_c - d_3 I - d_4 A. \end{aligned} \tag{15}$$

Our study of this adjusted model remains similar to that of the original model. We have $P_0 = (b/d_1, 0, 0)$, and

$$P^* = (S^*, I_c^*, I^*),$$

$$S^* = \frac{(d_3+\pi)(d_2+\alpha)}{pd_3\beta + (d_2+\alpha)\gamma + p(\pi\beta - d_2\gamma)}.$$

The reproductive number of the system is

$$R_0 = \frac{1}{S^*}\frac{b}{d_1} = \frac{b}{d_1}\left(\frac{pd_3\beta + d_2\gamma + p(\pi\beta - d_2\gamma)}{(d_2+\alpha)(d_3+\pi)}\right).$$

5.3 Mathematical Results

Our analysis is again the same, but we now let $\theta = 0$ since there is currently no known vaccine for HIV/AIDS.

Theorem 5.1 *For the HIV model, P_0 is again globally asymptotically stable for $R_0 < 1$, and P^* is globally asymptotically stable for $R_0 > 1$.*

5.4 Biological Interpretations

Many studies have been done that emphasize the lack of and importance of HIV testing around the world today. One study done in Italy observed 1,985 individuals in the population. 73.2 percent of them reported the Ministry of Health as their primary source of HIV information whereas 76.7 percent said that their main source was TV/radio. This gives health officials an idea as to the best methods for educating the public. The study also asked participants to state what they thought their risk of infection was. 45.7

percent reported that they felt they were at low risk, and only 6.9 percent felt they were high-risk. However, the study found that in actuality, 40 percent were participating in high-risk behaviours. This shows the gap in the public's awareness of HIV risks and their own actions.

Another study examined voluntary HIV testing patterns in Europe. It reported that between 49 and 89.3 percent of those who admitted to participating in high-risk behaviour never sought testing for HIV. This number is quite large considering the test is easily accessible and in most areas, free. It was also shown that the likelihood for testing was higher for those 24-39 years old than for younger individuals presenting the same types of behaviour. Among the Swiss, it was found that a higher level of education was positively correlated with a higher probability of testing. In America, the mean duration between acquiring HIV and initial primary care is 8.1 years (versus 2.5 years for those who acknowledged awareness). These parties could have had many high risk contacts during this period of time without even being aware that they were endangering others. Over 1/3 of HIV infected patients were unaware that they were even at risk for the disease. In fact, heterosexual intercourse as a risk was the most statistically significant factor for personal unawareness of risk. The general public today is poorly informed. If they were aware of the risks they were running, they would be much more likely to be tested, they would receive care faster, and they would transmit HIV less.

Today, the USA allocates more than 900 million dollars annually to the cause of HIV prevention. It has been shown that the odds of having been tested increase with the increase of CDC (Centre for Disease Control) funding per state. Estimates show that CDC HIV prevention funding translated into over 12.8 million more individuals being tested between 1998 and 2003 than if all states had received funding equal to the lowest funding received by a state. Unawareness is particularly high among young black adults, Hispanics, and gay men. This goes to show that with the proper investment in education for awareness, more people will be tested for HIV and therefore less people will be transmitting it unknowingly.

In the case of HIV, the parameter α is important because the rate of transmission of carriers (β) is higher than the rate of transmission of infecteds (γ) due to the fact that they are unaware of their condition. If we can reduce the number of carriers (ie, increase α so more carriers move into the infected stage), then the overall number of susceptible people becoming infected would be lower. So in this case, increasing α through an increase in awareness is

the key. By doing this, we would be decreasing the incidence of carriers (βI_c) and increasing the incidence of infecteds (γI), and therefore lowering overall incidence ($\beta I_c + \gamma I$).

5.5 Numerical Simulations

Using mathematical software we can analyze the dynamics of our system visually. It has been shown that the annual incidence for HIV in America is 1 in 6,800 or 0.01 percent. To take this value into account, we will choose $\beta = 0.00015$ and $\gamma = 0.00005$. We now choose our remaining parameters, taking into account that most people who are infected will first be unaware and therefore move into the carrier compartment after being infected: $b = 3$, $d_1 = 0.002$, $d_2 = 0.002$, $d_3 = 0.004$, $\pi = 0.01$, $p = 0.9$ and initial conditions: $S[0] = 50$, $I_c[0] = 1$, $I[0] = 10$, and then solve the system of o.d.e.'s numerically.

We now want to analyze the effects of an increase in awareness (ie. α).

We begin with $\alpha = 0.25$ (1/4 people are aware that they are HIV positive). Our equilibrium values are $(S^*, I_c^*, I^*) = (245, 8.96429, 178.005)$. If we raise α to $\alpha = 0.75$ (3/4 people are aware), we have a new equilibrium $(S^*, I_c^*, I^*) = (267.208, 2.95083, 175.692)$. In both cases, $R_0 > 1$ (= 6.12245 and 5.6136 respectively).

Since increasing α to 100 percent (ie. $\alpha = 1$ leaves $R_0 = 5.54962 > 1$) we can not change from endemic to disease-free equilibrium solely through changing α with these parameter values. However, if $R_0 > 1$ we can see that increasing α will have the effect of lowering the final number of carriers and infected people in the population.

Here we show our results with $\alpha = 0.25$ and $\alpha = 0.75$ followed by a parametric plot of both trajectories. In the case of the susceptibles, $\alpha = 0.25$ is the lower curve, and for carriers and infecteds it is the higher curve.

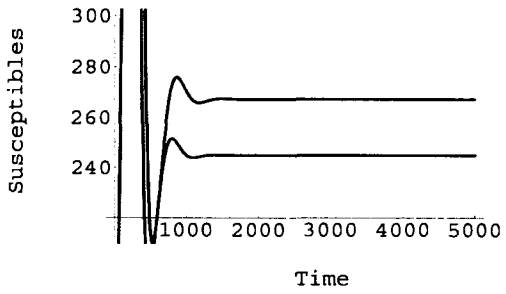


Figure 4: S^*

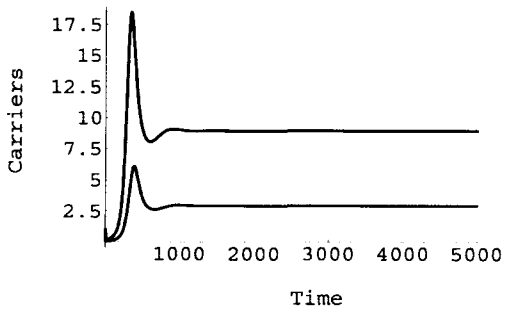


Figure 5: I_c^*

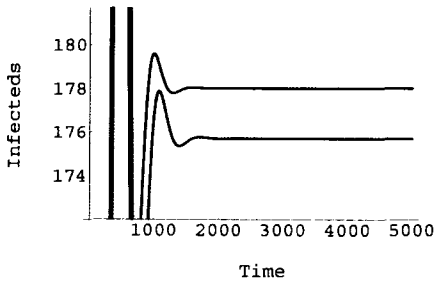


Figure 6: I^*

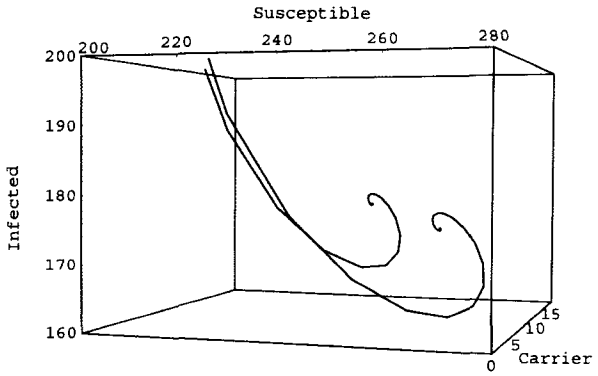


Figure 7: Equilibria

If we have $R_0 < 1$, increasing the value of α will still have a beneficial result. It will cause the population to reach the disease-free equilibrium in shorter time. For example, if we reduce transmission rates to $\beta = 0.000015$ and $\gamma = 0.000005$ (so overall incidence is 0.001 percent) we have $R_0 = 0.612245 < 1$ with $\alpha = 0.25$, $R_0 = 0.56136$ with $\alpha = 0.75$, and $(S^*, I_c^*, I^*) = (1500, 0, 0)$.

Again we have $\alpha = 0.25$ and $\alpha = 0.75$.

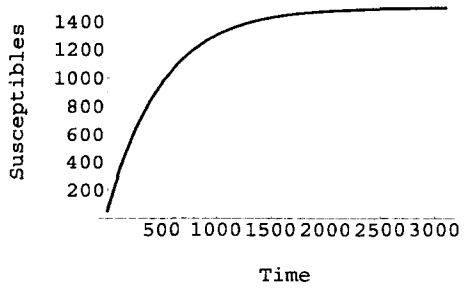


Figure 8: S^*

The trajectories are so similar that it is difficult to see the distinction in the graph for susceptibles. However, the difference can be seen in the graphs for the infected people and carriers, where the curve for $\alpha = 0.25$ is higher in both cases.

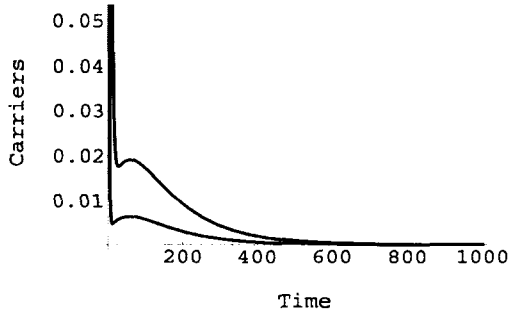


Figure 9: I_c^*

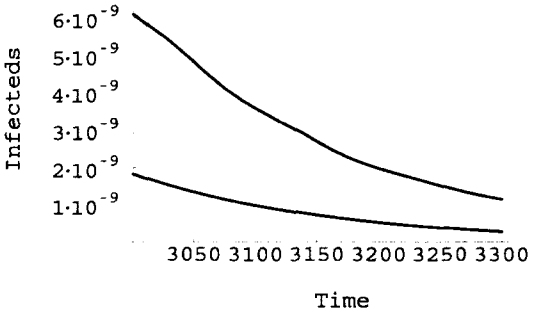


Figure 10: I^*

So it is clear that increasing the awareness of those with HIV through education and testing will have a positive effect on the population regardless of initial conditions and the value of R_0.

6 Conclusion

During our study and analysis of the ordinary differential equations formulated throughout this thesis, we have seen the negative effects that carriers

of an infectious disease can have on a population. Because the incidence rate of the carriers β is higher than that of the non-carrier infected individuals γ, the carriers increase the harm done by a disease by increasing the number of people infected.

Because they do not exhibit any symptoms, it is likely that carriers are in many cases unaware that they are infected. Thusly, the probability that they would participate in such "risky" behaviours that could infect others is higher than in those who know that they carry the disease. We have proven using Lyapunov functions that the disease-free equilibrium is globally stable for $R_0 < 1$ and that the endemic equilibrium is globally stable for $R_0 > 1$. Thus R_0, the reproductive number of the system, is crucial in determining the fate of an infectious disease.

In the case of diseases that have vaccines (such as Hepatitis B) the reproductive number can be directly manipulated through the change of the vaccination rate. We have seen that by increasing the rate (θ) just past its threshold value, we can reduce R_0 and possibly even eliminate the disease from the entire population.

For diseases where awareness is key (such as HIV), we have shown that moving individuals from the carrier (unaware) class to the infected class through testing can have quite an impact on the population. From lowering the number of equilibrium carriers and infecteds (in a system where $R_0 > 1$) to causing the disease to be eradicated sooner (in systems where $R_0 < 1$).

Current methods of disease control include attacking the source (through treatments of cases and carriers, as well as notification of cases and awareness), interrupting transmission (by personal hygiene and safe behaviour), and protecting susceptible individuals (immunization/vaccination). In addition to these things, it is important to identify which social groups are vulnerable to these diseases in order to implement successful prevention techniques. It has been said that:

> Current debates on a direction for public health in the 21st century indicate the need to move away from the belief that the key to health is the elimination of disease to a concept of public health based on the creation/production of health.[12]

It is clear that with proper techniques used by health officials, diseases with carriers can be managed effectively through either vaccinations or routine testing. In any case, education of the general public is elemental in

reducing the negative impacts on the population by carriers of infectious diseases.

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