Static Behavioral Effects On Gonorrhea Transmission Dynamics

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Abstract

An SIS/SAS model of gonorrhea transmission in a men-seeking-men (MSM) system is presented in this paper to study the impact of education on the dynamics of gonorrhea prevalence. Education affects behaviors that may fall into two categories– prevention and disease status awareness. Stability conditions for the disease free equilibrium and endemic equilibrium are determined along with an analytic expression and nominal value for the basic reproductive number and the control reproductive number. We carry out a complete analysis of global dynamics. Moreover, a time-dependent sensitivity analysis of the system and a sensitivity analysis of the control reproductive number is performed.

1 Introduction

The task of disease eradication and prevention is undertaken at the level of society and of individuals. Economic epidemiology is concerned with the impact behavioral changes have on the dynamics of the disease within a population. The behavior of individuals in a population can be influenced by education about behaviors that limit the spread of disease. These behaviors may fall into two categories–prevention and treatment. Prevention is specific to the characteristics of each disease. For example, in vector-borne disease like dengue and malaria, simple knowledge of the types of environment that serve best as breeding grounds and which environments attract mosquitos can mitigate disease spread by non-pharmacological prevention measures such as bed-netting and eliminating stagnant water. Moreover, in the case of influenza, the spread of the virus may be contained through preventative actions such as social distancing, staying at home when sick, proper coughing etiquette and hand washing. These actions are often adopted by individuals only when it is clear to them that the virus is present in the population and that they are at risk for infection. It is important to note that educational efforts are not perfect largely for two reasons. The individual must decide whether or not to make use of their education of the disease and the education may not reach everyone who needs it.

With sexually transmitted diseases (STDs) such as gonorrhea, prevention education advocates safe sex practices, condom use for a larger proportion of the time and reduction in the number of sexual partners. Treatment awareness leads to frequent STD screening as a disease control method since individuals may be infected but have no knowledge of their own status. In particular, infected individuals may or may not show symptoms but still be equally infectious [4]. If an infected individual is asymptomatic, but relatively uneducated about the disease, they will not seek medical attention and thus their average infectious period is potentially much greater than that of a symptomatically infected individual. Typically, symptomatically infected individuals recover 14 days after the start of treatment which, due to the pain associated with the symptoms, usually begins a few days after gonorrhea is contracted [2].

Seminal mathematical work was done by Hethcote and York on heterosexual gonorrhea transmission [4]. There, a two-sex model of symptomatically infected, asymptomatically infected and susceptible populations with different activity levels was used. The focus was the effect that contract tracing and increased STD testing would have on the dynamics of the disease. It was found that contact tracing of infectees was the least effective while contact tracing of infectors was most effective. Here the infectors were identified as a core group, or highly sexually active subpopulation. Hethcote concluded that focusing on the core group's activities was key to controlling the spread of gonorrhea.

Li *et al.* specifically modeled STDs like gonorrhea using an SIS model with a multiple strains and varied reaction to infection [6]. It was found that sufficient heterogeneity of the female in the form of contact structure, immune response or activity level was necessary in order to have coexistence of the multiple strains. The conditions for the existence and stability of an endemic equilibrium with two strains were found. Coexistence demanded that one strain should be better at infecting one subpopulation while the other be better

at infecting another. It was concluded that each strain creates reservoirs in the population that it is less able to infect.

Although not specifically looking at gonorrhea, previous mathematical epidemiological studies by Kemper *et al.* have developed a general model for curable diseases with symptomatic or asymptomatic infection [5]. There an SIS/SAS model was developed to considered the impact of asymptomatic attacks. However, this model does not treat the different recovery time of asymptomatic infected individuals, nor does it account for the different proportion of contacts with symptomatically infected individuals that lead to symptomatic versus asymptomatic infection and vice-versa.

As a case study in the effects of behavior change in the spread of a curable disease that confers no permanent immunity, we present an SIS/SAS model of a men-seeking-men (MSM) gonorrhea transmission that incorporates the effects of education listed. The influence of safe sex will be modeled exogenously with a weighted average on the effective contact rate that accounts for changing behavior with respect to condom use. Also disease status awareness will be modeled via an increase in infectious period for asymptomatic versus symptomatic individuals. Analysis is done describing the disease free and endemic dynamics as well as quantifying the sensitivity of these dynamics to the system's parameters and control reproductive number.

2 Basic Homosexual Gonorrhea Transmission Model

2.1 Mathematical Model

The population modeled is single sex with three homogeneous compartmental states available: susceptible individuals, S, symptomatically infected, I, and asymptomatically infected individuals, A. The model equations are:

$$\begin{aligned}
\frac{dS}{dt} &= \mu(N-S) - (\lambda_1 + \varepsilon\lambda_2)\beta S\frac{I+A}{N} + \alpha I + p\alpha A, \\
\frac{dI}{dt} &= (\lambda_1 + \varepsilon\lambda_2)\beta S\left(q_1\frac{I}{N} + (1-q_2)\frac{A}{N}\right) - (\mu+\alpha)I, \\
\frac{dA}{dt} &= (\lambda_1 + \varepsilon\lambda_2)\beta S\left((1-q_1)\frac{I}{N} + q_2\frac{A}{N}\right) - (\mu+p\alpha)A.
\end{aligned} \tag{1}$$

The model assumes a constant population size, N, with constant recruitment and removal, μ. The rate at which the susceptible population is lost to infection is $\beta S\frac{I+A}{N}$, where β is the effective contact rate. We introduce control into the system via λ_1, λ_2, and ε. The proportion of the population participating in non-safe sex is λ_1 and the proportion of the population participating in safe sex is λ_2. The effect of safe sex in preventing the transmission of gonorrhea is $(1-\varepsilon)$. Thus for the proportion of the time an individual participates in safe sex, λ_2 is multiplied by the reduction factor, ε. These parameters combine to become a total reduction factor on the force of infection, i.e. $(\lambda_1 + \varepsilon\lambda_2)\beta$. Individuals are recruited into the I class from the S class at a rate $(\lambda_1 + \varepsilon\lambda_2)\beta S\left(q_1\frac{I}{N} + (1-q_2)\frac{A}{N}\right)$, where q_1 and $(1-q_2)$ are the proportion of individuals that become symptomatically infected from contact with symptomatically infected individuals and asymptomatically infected individuals, respectively. Similarly individuals are recruited into the A class from the S class at a rate $(\lambda_1 + \varepsilon\lambda_2)\beta S\left((1-q_1)\frac{I}{N} + q_2\frac{A}{N}\right)$. Individuals from the I class reenter the susceptible class due to treatment at a rate α. Since individuals in the A class do not know they have gonorrhea the average duration of infection is longer. We represent this increase in infectious period via $p \in [0,1]$. Thus reentry into the susceptible population from the asymptomatic class occurs at the rate $p\alpha A$.

2.2 Parameter Estimation

To estimate the parameters in this model, several sources are used. Given that we are choosing to illustrate the impact of our model in college populations, μ is taken to equal $\frac{1}{4yr}$. The effective contact rate is the product of the number of risky contacts per year and the proportion these that lead to infection. Thus based on the information given by [9], $\beta = .5 \times 50$ contacts per year. We assume a highly active population with 50 risky contacts per year. To determine the amount of time spent practicing safe versus risky behavior, according to a study of an MSM population by Shlay et al [7], 25.6% of participants report consistent condom usage, thus $\lambda_2 = .256$ and $\lambda_1 = 1 - \lambda_2 = .744$. To determine ε we take into account that although condoms are 97% effective at preventing gonorrhea infection with

perfect use, many uses are imperfect due to slippage, breaking, etc. According to Shlay et al.[7] and Stone et al. [8], 16.6 - 17.3% usage failure of condoms in a MSM population. Taken these data together, $\varepsilon \approx .173$. According to [9] among men, symptoms typically appear within 2-5 days, $\frac{2}{365} - \frac{5}{365}$ years, following infection while treatment duration is 14 days, $\frac{14}{365}$ years, thus we take $\alpha \approx \frac{365}{18yr}$ and allow p to range within $[0, 1]$. The CDC suggests a highly active MSM population that an individual get tested once every 3 months, thus we will let $p = 1/6$ to measure the effect of this policy. Although there is limited contact tracing data, according to [9] 10% of infections are asymptomatic. A numerical investigation, using the nominal values for every other parameter, suggests that $q_1 = .98$ and $q_2 = .01$ in order to have the 90-10 split with the symptomatic and asymptomatic populations. In truth q_1 and q_2 are functions of the rest of the parameters. It is acknowledged here that these estimates are imperfect.

The intuitive behavior of the system shares a lot in common with the simple $S - I - S$ model. If we consider $J = I + A$ we may arrive at the following where $\omega := \lambda_1 + \varepsilon\lambda_2$

$$\begin{aligned} \frac{dJ}{dt} &= \frac{dI}{dt} + \frac{dA}{dt} \\ &= \omega\beta S\frac{J}{N} - \mu J - \alpha(I + pA). \end{aligned} \tag{2}$$

From this equation we can see that an important parameter that distinguishes this system is the presence of the p parameter. For $p \approx 1$ we should expect dynamics very similar to the $S - I - S$ model, redefining $\gamma := \omega\beta$.

3 Analysis

3.1 Global Stability Analysis

The main focus of this section is to prove Theorem (1). The discussion proving this will involve a treatment of the stability of the disease free equilibrium, conditions on the number of endemic equilibrium that may exist, and a preclusion of closed orbits which will make all stability arguments global.

Theorem 1. *System* (1) *has 2 fixed points: a disease free and an endemic equilibrium. The disease free equilibrium is globally stable when the control reproductive number is less than one and unstable when the control reproductive number is greater than one. The endemic equilibrium does not exist when the control reproductive number is less than one and is globally stable when the control reproductive number is greater than one.*

Since $S(t) + I(t) + A(t) = N$ we may eliminate one state variable for the purpose of analysis. We may also rescale in both state and time to reduce the overall system. Using $x(\tau) = \frac{I}{N}$, $y(\tau) = \frac{A}{N}$, and $\tau = t\omega\beta(1-x-y)$, where $\omega = \lambda_1 + \lambda_2\varepsilon$, we arrive at:

$$\begin{aligned} \frac{dx}{d\tau} &= q_1 x + (1-q_2)y - \frac{q_1 x}{\mathfrak{R}_{II}(1-x-y)}, \\ \frac{dy}{d\tau} &= (1-q_1)x + q_2 y - \frac{q_2 y}{\mathfrak{R}_{AA}(1-x-y)}, \end{aligned}$$

where $\mathfrak{R}_{II} = \frac{\beta\omega q_1}{\mu+\alpha}$ and $\mathfrak{R}_{AA} = \frac{\beta\omega q_2}{\mu+p\alpha}$. Since each state variable S, I and A are positive we have that $x + y \leq 1$. Thus the rescaling is positive invariant. There is a disease free equilibrium that always exists, $DFE := (x^*, y^*) = (0, 0)$, implying $S^* = N, I^* = 0$ and $A^* = 0$. To determine the local stability of the DFE the system is linearized about (x^*, y^*) resulting in the following:

$$\mathfrak{J}_{(x^*,y^*)} = \begin{pmatrix} q_1 - \frac{q_1}{\mathfrak{R}_{II}} & (1-q_2) \\ (1-q_1) & q_2 - \frac{q_2}{\mathfrak{R}_{AA}} \end{pmatrix} \tag{3}$$

The characteristic polynomial of the above Jacobian is:

$$\lambda^2 - \left[q_1 + q_2 - \frac{q_1}{\mathfrak{R}_{II}} - \frac{q_2}{\mathfrak{R}_{AA}}\right]\lambda + \left[\left(q_1 - \frac{q_1}{\mathfrak{R}_{II}}\right)\left(q_2 - \frac{q_2}{\mathfrak{R}_{AA}}\right) - (1-q_1)(1-q_2)\right], \tag{4}$$

which is in the form $\lambda^2 - b\lambda + c$. It may be shown that conditions for the determinant of the jacobian to be positive, $c > 0$, are identical to $\mathfrak{R}_E$, the basic control number, being less than one. First we use the next generation operator to compute the number of secondary infections a typical infectious individual creates in a completely susceptible population [3]. Here it is important to note that each element in Equation (5) is a reproductive factor. These reproductive numbers each have a different biological significance and give rise to the control reproductive number $\mathfrak{R}_E$. The reproductive number $\mathfrak{R}_{II}$ is the number of secondary

symptomatic infections caused by a symptomatically infected individual in a completely susceptible population. Likewise, $\mathfrak{R}_{AA}$ is the number asymptomatic secondary infections caused by an asymptomatically infected individual. The reproductive number $\mathfrak{R}_{AI}$ is the number of symptomatic secondary infections caused by an asymptomatically infected individual, and $\mathfrak{R}_{IA}$ is the reverse. The quantity $\mathfrak{R}_E$ incorporates p, λ_1, λ_2, and ε and measures the ability of the infection to spread in an environment that practices educated sexual behavior. We find that $\mathfrak{R}_E$ is given by the spectral radius, or dominant eigenvalue, of

$$\begin{bmatrix} \frac{\beta\omega q_1}{\alpha+\mu} & \frac{\beta\omega(1-q_2)}{\mu+p\alpha} \\ \frac{\beta\omega(1-q_1)}{\alpha+\mu} & \frac{\beta\omega q_2}{\mu+p\alpha} \end{bmatrix} = \begin{bmatrix} \mathfrak{R}_{II} & \mathfrak{R}_{AI} \\ \mathfrak{R}_{IA} & \mathfrak{R}_{AA} \end{bmatrix}. \tag{5}$$

The spectral radius of Equation (5) gives the following

$$\mathfrak{R}_E = \frac{\mathfrak{R}_{II} + \mathfrak{R}_{AA} + \sqrt{(\mathfrak{R}_{II} - \mathfrak{R}_{AA})^2 + 4\mathfrak{R}_{IA}\mathfrak{R}_{AI}}}{2}. \tag{6}$$

Corollary 1. *The condition for $\mathfrak{R}_E < 1$ is identical to that for $c > 0$.*

Proof. We begin with the condition for $\mathfrak{R}_E < 1$:

$$\begin{aligned} \frac{\mathfrak{R}_{II} + \mathfrak{R}_{AA} + \sqrt{(\mathfrak{R}_{II} - \mathfrak{R}_{AA})^2 + 4\mathfrak{R}_{IA}\mathfrak{R}_{AI}}}{2} &< 1, \\ \sqrt{(\mathfrak{R}_{II} - \mathfrak{R}_{AA})^2 + 4\mathfrak{R}_{IA}\mathfrak{R}_{AI}} &< \left(2 - (\mathfrak{R}_{II} + \mathfrak{R}_{AA})\right)^2, \\ \mathfrak{R}_{II}^2 - 2\mathfrak{R}_{II}\mathfrak{R}_{AA} + \mathfrak{R}_{AA}^2 + 4\mathfrak{R}_{IA}\mathfrak{R}_{AI} &< 4 - 4(\mathfrak{R}_{II} + \mathfrak{R}_{AA}) + (\mathfrak{R}_{II} + \mathfrak{R}_{AA})^2, \\ -4\mathfrak{R}_{II}\mathfrak{R}_{AA} + 4\mathfrak{R}_{IA}\mathfrak{R}_{AI} + 4(\mathfrak{R}_{II} + \mathfrak{R}_{AA}) &< 4, \\ \mathfrak{R}_{II} + \mathfrak{R}_{AA} + \mathfrak{R}_{II}\mathfrak{R}_{AA}\left(\frac{1 - q_1 - q_2}{q_1 q_2}\right) &< 1. \end{aligned}$$

If we the consider the condition for $c > 0$:

$$\begin{aligned} \left(q_1 - \frac{q_1}{\mathfrak{R}_{II}}\right)\left(q_2 - \frac{q_2}{\mathfrak{R}_{AA}}\right) - (1 - q_1)(1 - q_2) &> 0, \\ q_1 q_2(1 - \mathfrak{R}_{II} - \mathfrak{R}_{AA}) - \mathfrak{R}_{II}\mathfrak{R}_{AA}(1 - q_1 - q_2) &> 0, \\ 1 - \mathfrak{R}_{II} - \mathfrak{R}_{AA} &> \mathfrak{R}_{II}\mathfrak{R}_{AA}\frac{1 - q_1 - q_2}{q_1 q_2}, \\ \mathfrak{R}_{II} + \mathfrak{R}_{AA} + \mathfrak{R}_{II}\mathfrak{R}_{AA}\left(\frac{1 - q_1 - q_2}{q_1 q_2}\right) &< 1, \end{aligned} \tag{7}$$

then we see Equation (7) identical to the condition for $\mathfrak{R}_E < 1$. □

Thus we have a somewhat easier condition for stability and may define

$$\hat{\mathfrak{R}}_E = \mathfrak{R}_{II} + \mathfrak{R}_{AA} + \mathfrak{R}_{II}\mathfrak{R}_{AA}\left(\frac{1-q_1-q_2}{q_1q_2}\right).$$

If $\mathfrak{R}_E > 1$ then the DFE is unstable, but we haven't discussed how many equilibrium may exist. Consider $z = x + y$. If $\frac{dz}{d\tau} = 0$ and $\frac{dy}{d\tau} = 0$ then we would be at a fixed point for System (3). Solving $\frac{dz}{d\tau} = 0$ we get an expression for y in terms of z by noting $x = z - y$. Plugging this expression into $\frac{dy}{d\tau}$ and solving the new expression for zero we get $zf(z) = 0$, where $f(z) = z^2 - Bz + C$,

$$B = 2 + \frac{q_1q_2\left(\frac{1}{\mathfrak{R}_{AA}} + \frac{1}{\mathfrak{R}_{II}}\right)}{1-q_1-q_2},$$

and

$$C = \frac{(1-q_1)(1-q_2) - q_1q_2\left(1-\frac{1}{\mathfrak{R}_{II}}\right)\left(1-\frac{1}{\mathfrak{R}_{AA}}\right)}{1-q_1-q_2} = \frac{c}{-(1-q_1-q_1)},$$

where c is from the characteristic polynomial, (4). If $z = 0$ then $x = y = 0$, the DFE. The interest thus lies in where $f(z) = 0$. A relationship between C and $\mathfrak{R}_E$ can be made using the existing relationship found in Corollary (1).

Corollary 2. *If $q_1 + q_2 < 1$ then $\mathfrak{R}_E < 1 \leftrightarrow C < 0$ and $\mathfrak{R}_E > 1 \leftrightarrow C > 0$. If $q_1 + q_2 > 1$ then $\mathfrak{R}_E < 1 \leftrightarrow C > 0$ and $\mathfrak{R}_E > 1 \leftrightarrow C < 0$.*

Proof. If $q_1 + q_2 < 1$ then C and c have differing sign and thus if $c > 0$ then $C < 0$ and their relationships to $\mathfrak{R}_E$ are the opposite of one another. If $q_1 + q_2 > 1$ then C and c have the same sign and their relationships to $\mathfrak{R}_E$ are identical. □

The equation $f(z)$ is a quadratic and thus may have 0, 1, or 2 roots in $(0, 1)$. The relative signs of $f(0)$ and $f(1)$ will allow us to determine under what conditions $f(z)$ has a particulat number of roots in the unit interval. Consider $f(0) = C$ and $f(1) = 1 + C - B$. We already have that the sign of C may be viewed as being dependent on the magnitude of $\mathfrak{R}_E$. We also have that

$$1 + C - B = -\frac{q_1q_2}{1-q_1-q_2}\frac{1}{\mathfrak{R}_{II}\mathfrak{R}_{AA}},$$

whose sign depends on $1-q_1-q_2$. Thus in order to study the zeros of $f(z) = z^2-Bz+C$ we must consider all 4 combinations of the sum of q_1 and q_2 with the magnitude of the control reproductive number.

Case 1. $\mathfrak{R}_E < 1$ *&* $q_1 + q_2 < 1$

In this situation $f(0) = C < 0$ and $f(1) = 1 + C - B < 0$. Thus there are no zeros of $f(z) \in (0, 1)$.

Case 2. $\mathfrak{R}_E > 1$ *&* $q_1 + q_2 < 1$

Here we have that $f(0) > 0$ and $f(1) < 0$. Thus there is a single root for $f(z) \in (0, 1)$.

Case 3. $\mathfrak{R}_E < 1$ *&* $q_1 + q_2 > 1$

In the most difficult situation we have that $f(0) > 0$ and $f(1) > 0$. The quadratic having exactly two roots occurs when

1. $\frac{B}{2} \in (0, 1)$, and
2. $B^2 - 4C > 0$.

In order for $B > 0$ we require that

$$\frac{\mathfrak{R}_{II} + \mathfrak{R}_{AA}}{\mathfrak{R}_{II}\mathfrak{R}_{AA}} < \frac{2(q_1 + q_2 - 1)}{q_1 q_2}.$$

The second condition yields

$$\frac{(\mathfrak{R}_{II} + \mathfrak{R}_{AA})^2}{\mathfrak{R}_{II}\mathfrak{R}_{AA}} > \frac{4(q_1 + q_2 - 1)}{q_1 q_2}. \tag{8}$$

These two conditions are contradictory. To illustrate the contradiction we invoke Equation (6) to get that $\mathfrak{R}_E < 1 \implies \mathfrak{R}_{II} + \mathfrak{R}_{AA} < 2$. Rearranging Equation (8) results in

$$\frac{\mathfrak{R}_{II} + \mathfrak{R}_{AA}}{\mathfrak{R}_{II}\mathfrak{R}_{AA}} > \frac{4}{\mathfrak{R}_{II} + \mathfrak{R}_{AA}} \frac{(q_1 + q_2 - 1)}{q_1 q_2}, \tag{9}$$

but $\dfrac{4}{\mathfrak{R}_{II} + \mathfrak{R}_{AA}} > 2$ which results in the contradiction. Thus there are no zeros of $f(z) \in (0, 1)$.

Case 4. $\mathfrak{R}_E > 1$ *&* $q_1 + q_2 > 1$

In this situation $f(0) > 0$ and $f(1) < 0$. Thus there is a single root for $f(z) \in (0, 1)$.

Combining the arguments gives us that if $\mathfrak{R}_E < 1$ then the only solution for our system is the DFE, and when $\mathfrak{R}_E > 1$ the two solutions are the unstable DFE and a single endemic equilibrium, EE. The entire above argument is valid only if $1 - q_1 - q_2 \neq 0$. When $1 - q_1 - q_2 = 0$, the system exhibits a single EE,

$$(x, y) = \left(\frac{\mathfrak{R}_{II}(\mathfrak{R}_{II} + \mathfrak{R}_{AA} - 1)}{(\mathfrak{R}_{II} + \mathfrak{R}_{AA})^2}, \frac{\mathfrak{R}_{AA}(\mathfrak{R}_{II} + \mathfrak{R}_{AA} - 1)}{(\mathfrak{R}_{II} + \mathfrak{R}_{AA})^2} \right),$$

which is only valid if $\mathfrak{R}_{II} + \mathfrak{R}_{AA} - 1 \geq 0$. We wish to make assertions about the stability of the EE without having to do a linearization around the fixed point which is very term intensive. We may disprove the existence of closed orbits in the plane and thus assert that when the EE exists, $\mathfrak{R}_E > 1$, it is stable.

Corollary 3. *System* (1) *has no closed orbits.*

Proof: By Dulac's criterion, if there exists a function $\varphi \in C^1$ such that $\frac{\partial(\varphi\dot{x})}{\partial x} + \frac{\partial(\varphi\dot{y})}{\partial y} \neq 0$, then the planar system $\dot{x}, \dot{y}$ has no closed orbits. Let $\varphi = \frac{1}{xy}$. Then:

$$\begin{aligned}
\frac{\partial(\varphi\dot{x})}{\partial x} &= \frac{\partial}{\partial x}\left[\frac{q_1}{y} + \frac{(1-q_2)}{x} - \frac{q_1}{\mathfrak{R}_{II}(1-x-y)y}\right] \\
&= -\frac{(1-q_2)}{x^2} - \frac{q_1}{\mathfrak{R}_{II}(1-x-y)^2 y} \\
\frac{\partial(\varphi\dot{y})}{\partial y} &= \frac{\partial}{\partial y}\left[\frac{(1-q_1)}{y} + \frac{q_2}{x} - \frac{q_2}{\mathfrak{R}_{AA}(1-x-y)x}\right] \\
&= \frac{-(1-q_1)}{y^2} - \frac{q_2}{\mathfrak{R}_{AA}(1-x-y)^2 x}.
\end{aligned}$$

Since $q_1, q_2 \in [0,1]$ and $\mathfrak{R}_{AA}, \mathfrak{R}_{II} > 0$ the sum $\frac{\partial(\varphi\dot{x})}{\partial x} + \frac{\partial(\varphi\dot{y})}{\partial y}$ is always negative. Thus there are no closed orbits for System (3). Since the dynamics are identical for System (1) we have precluded limit cycles in it and have shown what was intended. □

3.2 Sensitivity of Gonorrhea Transmission Model

In a perfect world, public health initiatives would be simple, multifaceted, and have great effects on the dynamics of a disease. However, this is not always the case and moreover, economic costs have to be considered in determining which interventions to support. An important question is whether the size of the population is important to the dynamics of the disease and intervention. In order to address these concerns, in this section with we examine the sensitivity of the system to changes in the parameter values. We take two approaches, first the time-dependent sensitivity of the original system to changes in parameter values. In addition, as stated in Section 3, the existence and nominal value of the endemic equilibrium depends on the control reproductive number. Furthermore, since we are interested in the effect education has on disease transmission dynamics, the sensitivity of $\mathfrak{R}_E$ is determined.

3.2.1 Sensitivity of $S(t)$

In order to discuss the importance of individual parameters one must investigate how their value affects the state variables over time. We will consider the concept of elasticity. Formally, one may define elasticity of a function $X(t;\theta)$ with respect to a parameter $\theta_i \in \theta$ as

$$E_{\theta_i}(t) = \frac{\theta_i}{X(t;\theta)} \frac{\partial X(t;\theta)}{\partial \theta_i}.$$

This measures the ratio of a percent change in a parameter to that of the function. This gives us a unit-less and scaled method with which to compare each parameter's affect on the solution $S(t)$. However, we do not have a closed form for $S(t)$ and thus we must make an approximation.

In general consider $\frac{dX}{dt} = f(t, X; \theta)$ where θ is a parameter vector. Now consider the vector of nominal parameter values, $\hat{\theta}$, and a small perturbation, Δ_i, of the i^{th} element, $\hat{\theta}_i$, and call this new parameter vector $\hat{\theta}^i$. If what we are interested in is $\frac{\partial X}{\partial \theta^i}$ near the nominal value then we could do the following. Numerically find the solutions $X(t;\hat{\theta})$ and $X(t;\hat{\theta}^i)$ then take the difference quotient to arrive at

$$\frac{\partial X}{\partial \theta_i}(t) \approx \frac{X(t;\hat{\theta}^i) - X(t;\hat{\theta})}{\Delta_i}. \tag{10}$$

We may use this approximation in our computation of the elasticity of $S(t)$ with respect to each parameter.

The sensitivity of the susceptible class overtime with respect to the nominal parameters is presented in Figure 1. Considering the parameters that affect the susceptible population in a positive manner, q_1 seems to induce the most sensitivity. Unfortunately, there appears to be no way to directly control the proportion of infected individuals who develop symptoms, so manipulation of this powerful parameter becomes useless as a control measure.

The following results, while intuitive, offer useful insights into disease control. Investigating the sensitivity of α, the shorter period of time an individual is infectious then the

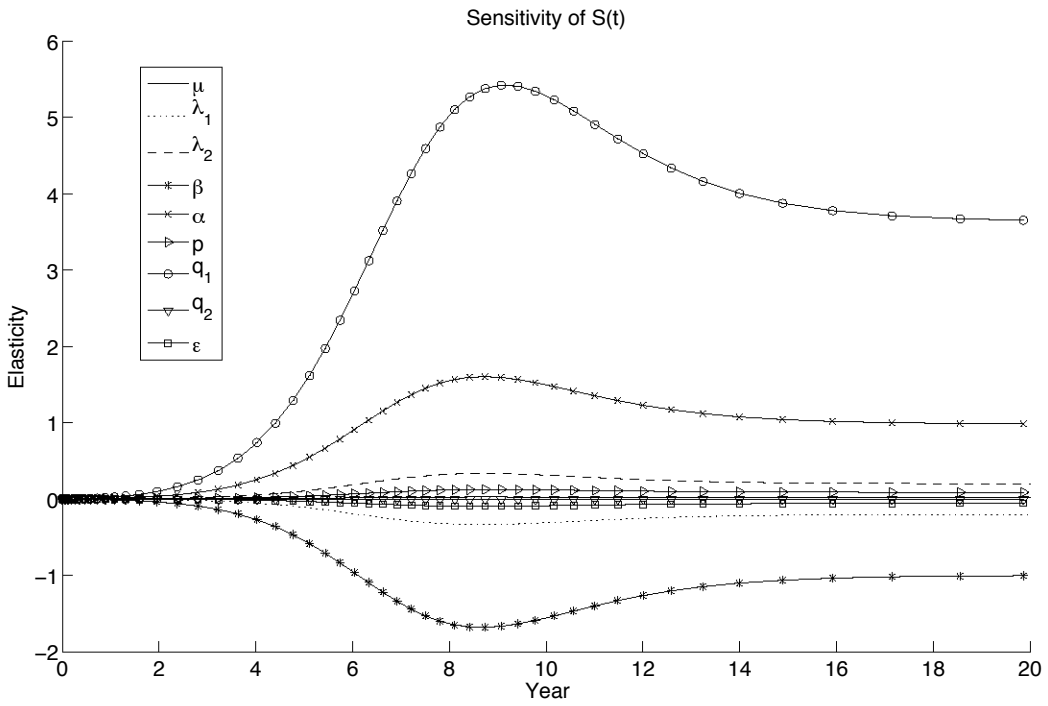

Figure 1: Sensitivity of the susceptible population with respect to each model parameter using the nominal values $(\mu, \lambda_1, \lambda_2, \beta, \alpha, p, q_1, q_2, \varepsilon) = (\frac{1}{4}, .744, .256, 25, \frac{365}{18}, \frac{1}{6}, .9, .1, .173)$ and $N = 10000$ with $S(0) = 99999, I(0) = 0$, and $A(0) = 1$.

fewer individuals they can infect, and thus treatment regimes that would return an individual to susceptible faster would clearly benefit the population. The effects of using a condom more often, λ_2, getting tested more often, p, and using a condom more effectively, ε, are all inelastic. In other words massive changes would have to be made to these parameters to see any real difference in the susceptible population. This supports the result of Hethcote [4] that contact tracing of highly sexually active groups is effective in controlling an STD. Furthermore, the model exhibits a great deal of sensitivity to β and only a small amount to λ_1. A negative change in either of these parameters causes an increase in the susceptible population. Thus a very effective control on the number of infections would be to limit the number of risky sexual contacts one has in a year.

To measure the effect that total size has on the population we constructed the same sensitivity curves over a series of different total populations. In Figure 2 the long term sensitivity of each parameter does not seem to be affected by the population size but the maximum effect each parameter has on the susceptible population slowly increases as N does.

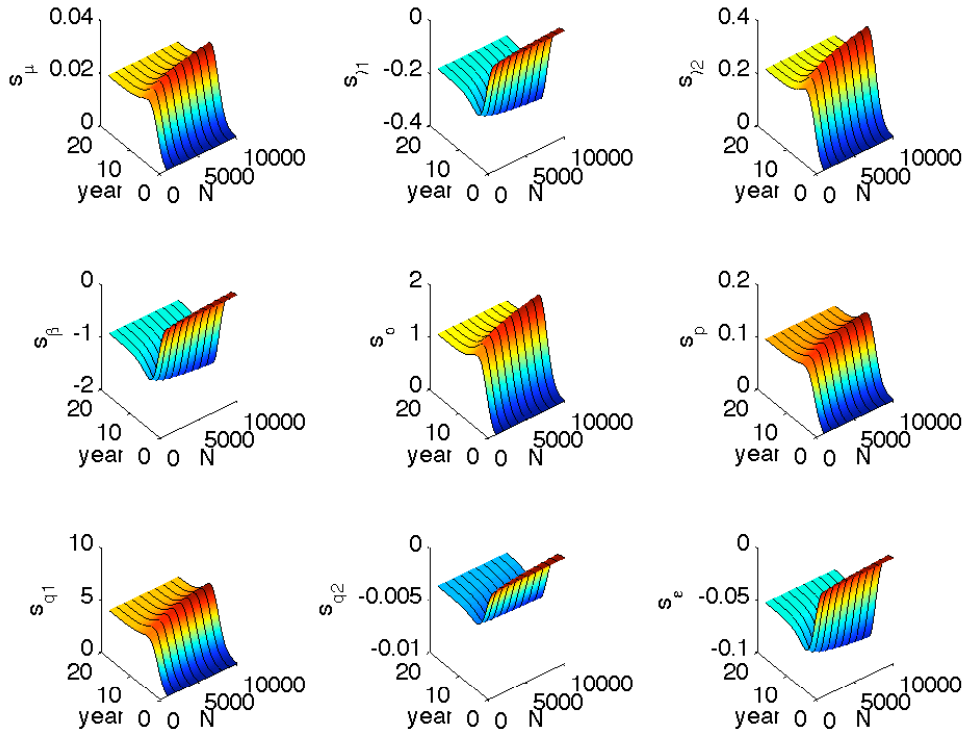

Figure 2: Sensitivity of the susceptible population with respect to each model parameter using the nominal values $(\mu, \lambda_1, \lambda_2, \beta, \alpha, p, q_1, q_2, \varepsilon) = (1/4, .744, .256, 25, 365/18, 1/6, .9, .1, .173)$ and $A(0) = 1$. N was varied from 1000 to 10000 incremented by 1000 each step.

The p value of $\frac{1}{6}$ that we've selected leads to the assertion that a very sexually active individual would get tested once every 3 months for STDs. Fixing the other parameters, with $N = 10000$, we varied p from 0 to 1. Figure 3 shows the results of varying p. Of note, when $p = 0$ then asymptomatic individuals remain in the system on average for four years. As a result there is a very large peak in the sensitivity for q_1. Intuitively if there is such a large infectious period for a particular infectious class then it would be desirable to avoid this infectious class. There is also a sustained dependance on μ that does not carry over for other values of p. Overall as p increases to 0.3 the peak sensitivity for each parameter decreases in magnitude and shifts to occur later in the time series. Also it appears that for $p \geq 0.4$ there is no change in sensitivity with respect to time for any of the parameters. However, there is a very tiny peak, on the order of 10^{-4} for each variable very early on in the epidemic. Thus it may be conjectured that for low p values the system is much more sensitive to the system parameters than otherwise.

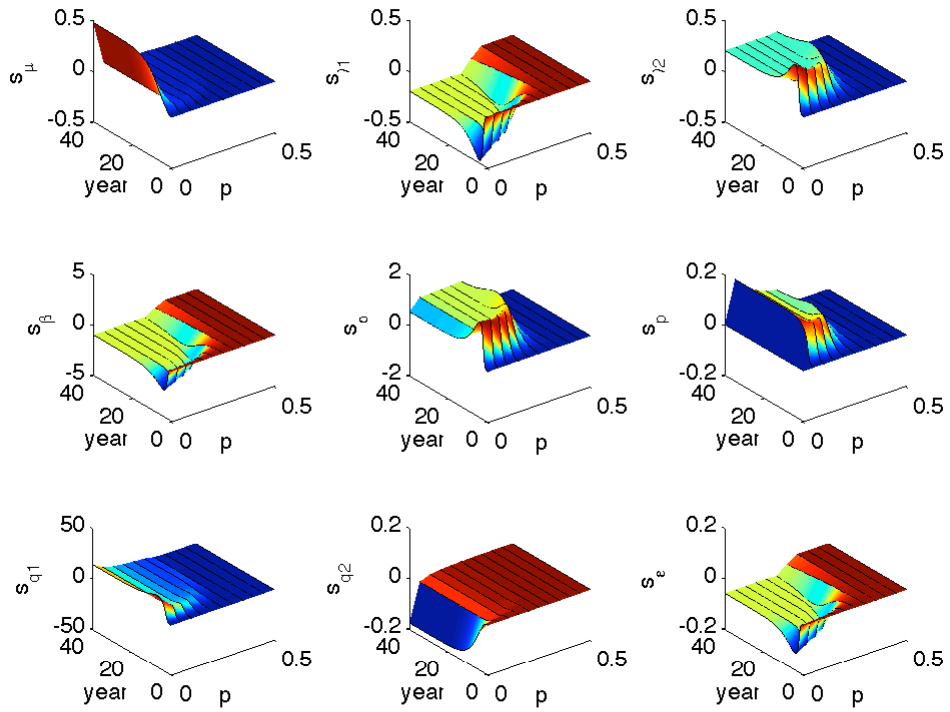

Figure 3: Sensitivity of the susceptible population with respect to each model parameter using the nominal values $(\mu, \lambda_1, \lambda_2, \beta, \alpha, p, q_1, q_2, \varepsilon) = (1/4, .744, .256, 25, 365/18, 1/6, .9, .1, .173)$, $N = 10000$, and $A(0) = 1$. P was varied from 0 to 1 incremented by .05 each step.

3.3 Reducing $\mathfrak{R}_E$

Holding all but one parameter constant we varied the free parameter until $\mathfrak{R}_E < 1$. With an $\varepsilon = 0.03$, implying that a condom is used properly 100% of the time, $\mathfrak{R}_E \approx .9949$. If $p = .32$, roughly meaning getting tested for STD's every 57 days, or once every other month, then $\mathfrak{R}_E \approx .9986$. While unrealistic to expect from a population at large, this would be a simple measure to suggest to a highly sexually active population. If regular condom use, despite its relatively high failure rate, increased to approximately 31%, a change to $\lambda_2 \approx .31$, then $\mathfrak{R}_E \approx .9972$. Finally, the nominal β value assumes 50 risky contacts per year, if we reduce that to 47.6 then $\mathfrak{R}_E \approx .9854$. If each control parameter is varied a very little we find that $\lambda_2 = .271, \beta = 24.78, p = .192$ and $\varepsilon = .148$ then $\mathfrak{R}_E < 1$. What these parameters imply is that if one reduces their annual risky contacts from 50 to 49.56, the percentage of time one uses a condom from 25.6% to 27.1%, getting tested every 93.75 days down from 108 days and using a condom so that the failure changes from 17.3% of the time to 14.8%. This multifaceted approach includes many changes, but each of which are relatively minor.

4 Conclusion and Discussion

In this paper, a simple model of homosexual gonorrhea transmission is analyzed and the parameter sensitivities are determined. In section 3.2, it is shown that disease education is useful in disease control, however education has to be multifaceted and include reductions in the average number of contacts and awareness that leads to testing. In detail, the system is most sensitive to q_1 (the portion of contacts with symptomatic infected individuals that lead to symptomatic infection), however this parameter is hard to be impacted via public policy then it is not the most useful parameter to focus on. As shown in Section 3.2, in magnitude, p (increased duration of asymptomatic infection) and λ_2 (the portion of time the population spends in good behavior) are the next most sensitive education related parameters. The analysis suggests that public health efforts should be concentrated on encouraging people to practice safe sex more often and to be tested more frequently. These initiatives would be better aimed at a reducing β, thus impacting the highly active core subpopulation, a result coinciding with Hethcote & Yorke's.

We have shown that some single methods of education, within realistic bounds, are not completely effective at reducing disease prevalence. Since people generally pay for their STD testing, while from a public health perspective frequent testing is best, on an individual level, testing is too costly and inconvenient. Along this line, work has been done from a game-theoretic perspective by Reluga [10].

In this paper we have considered the average, safe and risky behavior of the population. This has given insights into how education affect disease transmission. However, the population is not homogenous and in fact there are subpopulations each that interact differently and have different behaviors. Future work should be done to consider the different subpopulations and not aggregate the populations. Moreover, the question of how education affects disease transmission can be approached from an agent-based perspective.

5 Acknowledgements

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