Mathematical Models in Public Health: Forecasting Syphilis Deaths with Improved Testing Strategies

AUTHORS

Phong Ho - 1617975
R.J. Bilak - 1584507
Ruben Ross - 1131523
Santiago Arevalo - 1069167
Michael Zhang - 1616736

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Abstract

Background: Syphilis is a sexually transmitted infection (STI) caused by the Treponema pallidum bacteria (World Health Organization [WHO], 2024). It is primarily contracted via direct sexual contact; however, mother-to-child vertical transmission is also possible (WHO, 2024). Increasing rates of infectious syphilis in Canada are encouraging all provinces and territories to update their protocols to ensure more equitable access to testing, treatment, and follow-up medical visits. The objective of this study is to explore if an increase in syphilis testing and treatment reduces the health complication/health complication rate. Method: Data was obtained from the Government of Alberta's "Interactive Health Data Application" (2024) which assisted in providing a baseline for estimating parameter values for our model. We applied a mechanistic modeling approach to model how the number of health complications compared between individuals who received treatment and those who elected not to receive treatment.

Results: Although the reported number of health complications related to infectious syphilis are low, the model demonstrated that an increase in testing and treatment was associated with a decrease in the number of health complications when compared to those individuals who opted to not receive any form of treatment.

Conclusion: Current approaches to the treatment and management of infectious syphilis should be updated to encourage individuals to receive both appropriate testing and follow-up consultations. It is also recommended that such changes consider the unique needs and challenges faced by Canada's most disadvantaged and vulnerable populations who are often disproportionately affected by infectious syphilis.

Key words: infectious syphilis, testing, treatment, health concerns, health complication

Contents

1 Introduction

1.1 What is syphilis

The aim of this study is to establish a baseline understanding of syphilis—an infection caused by the bacterium Treponema pallidum—and to develop a model that projects the future trajectory of this disease under current conditions. Syphilis progresses through multiple stages: the primary stage typically involves a painless sore (chancre) appearing at the infection site; if left untreated, it advances to a secondary stage characterized by a more systemic rash and other flu-like symptoms. Subsequently, the disease can enter a latent period with no obvious symptoms, and, in some cases, progress to a tertiary stage, which can be associated with severe complications such as cardiovascular and neurological damage.

Fortunately, syphilis is highly treatable, especially when detected early. The preferred treatment involves antibiotic therapy, usually with penicillin, which can eradicate the bacterial infection and halt its progression. Early diagnosis and prompt treatment are crucial in preventing long-term complications. Regular testing, particularly for individuals who are sexually active with multiple partners or engage in unprotected sex, can help detect syphilis before it advances to later stages. Public health measures, awareness campaigns, and access to healthcare services also play vital roles in reducing the incidence and severity of syphilis worldwide.

The project begins by creating a baseline projection of syphilis cases in Alberta using historical data, assuming no extra interventions are in place. From this baseline, we will then adjust the model's parameters to estimate how increasing testing and ensuring treatment for all identified cases might reduce the overall number of infections. While we will not explore specific awareness strategies, we will discuss potential approaches for boosting testing rates in our concluding section.

1.2 Syphilis rates

From the 1930s to 1950s in Canada the rates of syphilis were quite high, peaking at 67 reported cases per 100,000. The 1960s saw a dramatic decrease in rates and fell steadily for the next three decades. In 1993, the reported cases were 3.22 per 100,000 people, approximately a 20-fold decrease in the reported cases from the peak in the 1940s. The cases remained quite low (under 10 cases per 100,000) until 2013. However, starting in 2013 cases began to rise, reaching a peak of 49 cases per 100,000 in 2022 (Government of Canada, 2024) which represented a 5-fold increase since 2013 (Canada wide). Figure 1 below shows the total number of cases in Canada since 1930.

Alberta has seen an even greater increase in syphilis rates than Canada, with a staggering 10,525% increase in reported cases from 2014 to 2023 (Government of Alberta[GOA], 2024), excluding infants. This alarming rise not only reflects a significant health concern but also highlights the potential consequences for both individual and community well-being. An area of particular concern is the 340 documented congenital cases identified between 2015 and 2024, resulting in 60 stillbirths. In 2014, there were zero documented congenital cases. The urgency of the situation demands heightened

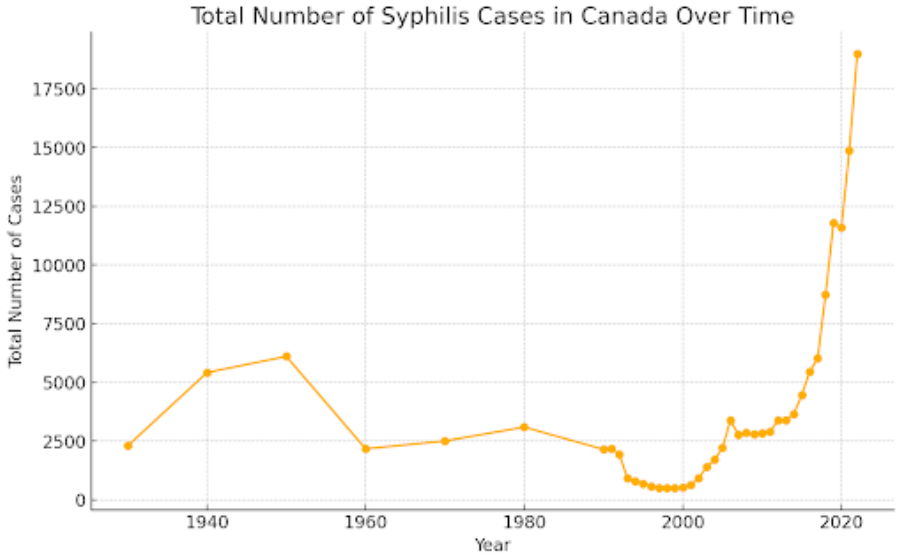


Figure 1: Total Syphilis Cases

awareness and proactive measures, especially among high-risk groups.

While effective treatment options exist, such as specific antibiotics, awareness, access to testing, and treatment are crucial for alleviating the ongoing outbreak. As seen from the historical data, the number of cases are likely to continue to rise unless some preventative actions are taken. The rise in cases is a significant concern and should be investigated. There could be several reasons for the rise in cases, including lack of awareness, increased use of online dating apps, or drug epidemics to name a few.

Advertising campaigns and encouraging expecting mothers to be tested for sexually transmitted infections (STI) are some of the measures that are currently used to identify infections. As of the latest data since 2022, rates of syphilis are continuing to increase (Public Health Agency of Canada, 2023), indicating the current measures are not enough to stop the increase of syphilis.

1.3 Project Objective

The intent of our model is to estimate the future trajectory of syphilis cases in Alberta and to determine whether increasing awareness and testing could identify and treat more cases, thereby reducing transmission and lowering overall case counts. We begin by establishing a baseline scenario in which no additional interventions are introduced. From this baseline, we then adjust model parameters to explore how enhanced testing and treatment might decrease the prevalence of the disease.

Since syphilis is treatable with antibiotics, we assume that all individuals who test positive will receive and complete their course of treatment. While the model's primary focus is on quantifying the impact of increased testing and treatment, rather than on the methods of raising awareness, we will

briefly discuss potential strategies for improving testing uptake in our concluding section.

2 Background

Rates of infectious and congenital syphilis have been on the rise in Canada since the early 2000s; however, a substantial increase in cases since 2017 coupled with outbreaks across Canada prompted the Public Health Agency of Canada (PHAC) to establish a Syphilis Outbreak Investigation Coordinating Committee (SOICC) with the aim of improving data collection, and enhancing communication among municipal, provincial and territorial agencies (Aho et al., 2022, p.53). Information collected through SOICC is then compiled with existing data from the Canadian Notifiable Disease Surveillance System (CNDSS) to create an enhanced profile of syphilis trends across key strata including age, sexual orientation, and pregnancy status (Aho et al., 2022, p.52).

Males continue to experience higher rates of infectious syphilis, however notable increases have been noted in recent years among females (Aho et al., 2022, p.55). Of particular concern is the magnitude of the increases with males experiencing a 73% increase and females experiencing a 773% increase between 2016 to 2020 (Aho et al., 2022, p.53). Although this trend among Canadian females is concerning, the spread of infectious syphilis was first detected among the gay, bisexual, and other men who have sex with men (gbMSM) populations (Shaw, Plourde, Klassen & Stein, 2022, p.95). These observations are significant for two reasons. First, they demonstrate that syphilis is a health issue that affects female and male populations regardless of sexual orientation. Second, Canadian women may be disproportionately affected underscoring the need for greater services and programs that consider unique factors associated with women's health. For example, diagnosis can be delayed or overlooked in women since anatomical differences can make it difficult to correctly identify specific areas requiring further medical testing and treatment (PHAC, as cited in Aho et al., 2022, p.56). Providing female-specific interventions early on may also play a role in reducing congenital syphilis which has also been steadily increasing in Alberta, Saskatchewan, and Manitoba (Aho et al., 2022, p.55).

An understanding of why infectious syphilis rates continue to rise among the gbMSM and heterosexual populations is still unclear (Aho et al., 2022, p.56). Some research suggests that structural issues pertaining to socioeconomic status and substance abuse may be contributing factors for the spread of syphilis (Shaw, Plourde, Klassen & Stein, 2022, p.95). For example, domestic violence, stigmatization, and discrimination may make it difficult for vulnerable populations to access the appropriate treatments and services from health practitioners (PHAC, as cited in Aho et al., 2022, p.56). Mathematical modeling confirms that for individuals in these circumstances the incidence rate of syphilis decreases when comprehensive and proactive screening measures are in place (Tuite et al., as cited in Shaw, Plourde, Klassen & Stein, 2022, p.96). Although many improvements can certainly be made to address some of these issues, region-specific challenges remain which could further complicate efforts to reduce infectious syphilis.

In Nunavut, the expansive territory, limited infrastructure, and shortage of qualified staff make it

difficult for people in remote communities to access testing and treatment services (Singh et al., 2022, p.103). In some instances, pregnant women are transferred out of the territory to receive additional care in neighbouring provinces (Singh et al., 2022, p.102). If a proportion of these pregnant women are from remote communities where neither English nor French are spoken, it may be difficult for them to properly express their health concerns to the treating physicians. Even in the presence of an interpreter, stigmatization or fear of violence, discrimination, and racism may also prevent some women from opening up about their sexual experiences. Consequently, some pregnant women with syphilis may return to their communities without ever having received additional testing and treatment. In an effort to address these issues, several modifications were made within the public health sector in Nunavut which included additional prevention and training activities, and a greater emphasis on managing syphilis cases via more in-person follow-ups with treating health practitioners (Singh et al., 2022, p.102).

3 Data Review

We were able to obtained important observations on the topic through data review. As it is essential for understanding and mitigating the impacts of syphilis, particularly in preventing complications and health complication from untreated infections. In our model, individuals are categorized into four groups: Susceptible (S), Infected (I), Tested (T), and health complication or Health Complication (D). As a starting point, data for the Edmonton Metropolitan Region was obtained from the Government of Alberta's "Interactive Health Data Application" (2024) which contained information on the Tested and health complication categories. An estimate of the Susceptible group was obtained by using a 0.541 multiplier based on Michelle Rotermann's (2020) findings on the percentage of sexually active individuals in the population. However, obtaining accurate data for the Infected group proved to be more challenging. Publicly available data on the total number of individuals infected with syphilis is often incomplete or under reported due to factors such as asymptomatic cases or unidentified cases. Moreover, data on testing or positivity rates could not be confirmed which hindered our ability to establish accurate case counts for the required categories. Although several estimates for the Infected category were considered, including those referenced from the Centers for Disease Control and Prevention, it was determined that a more flexible approach was required. Nevertheless, the overview of the data provided us with a glimpse of how reported cases of syphilis had changed over time across Canada.

To bypass the data-driven practices often found in statistical methodologies we turned to a mechanistic modeling approach which provided us with robust mathematical theories and tools to set up assumptions, pose a relevant question, and perform detailed analyses. The advantage of this method was twofold. First, it permitted us to consider extreme cases (i.e. an outbreak) where a larger portion of the population may be infected. Second, it allowed for comparison of different hypothetical scenarios with respect to treatment and health complication. These outcomes could be valuable to policymakers and health authorities who may be dealing with an outbreak of infectious syphilis or where

evidence-based mathematical modeling would be beneficial in providing deeper insights pertaining to public awareness, testing and treatment, and/or upgrading existing programs.

4 Model Formulations and Modeling Questions

4.1 Research question

'*Does an increase in testing and subsequent treatment for syphilis have a measurable impact on the transmission dynamics and the curve representing the number of individuals experiencing negative health effects or health complication?*'

4.2 Modeling Question

In traditional disease modeling frameworks, health complications are often represented as flows exiting the system, with little emphasis on the detailed information these events might provide. While this approach simplifies the modeling process, it may overlook valuable insights derived from analyzing aggregated data on health complications. By incorporating an explicit compartment for health complications, this method aims to investigate whether this representation can offer a more nuanced understanding of disease outcomes. Specifically, we seek to determine if measurable impacts can be observed within this compartment, providing additional context to inform public health interventions. Could this approach yield insights that are otherwise missed in traditional models? Or How does varying the testing rate γ affect the equilibrium points of the system, particularly the steady-state values of the infected population (I) and health complications (D)?

4.3 Practical Questions

Some practical questions that one may consider are:

- What level of testing γ is realistically achievable in a real-world scenario, and how does this compare to the model's predictions for reducing health complications or deaths?
- How can this model inform public health policies regarding resource allocation for testing and treatment programs?
- If certain subpopulations (e.g., those with limited healthcare access) have lower treatment efficacy ψ, how might the model adjust to reflect the disparity in outcomes?

4.4 Equations & Diagram

The transfer diagram can see in **Figure 2** with the following equations being

$$
\begin{aligned}
S' &= b + \psi T - \beta IS - d_3 S \\
I' &= \beta IS - \gamma I - d_1 I \\
T' &= \gamma I - \psi T - d_2 T \\
D' &= d_3 S + d_1 I + d_2 T - b
\end{aligned}
$$

4.5 Assumptions

The assumptions to make this model suitable are:

- All individuals in the treatment compartment have entered via testing at the specified testing rate.
- The rate of entry into the system equals the rate of exit from the system
- All treatment is assumed to be completed in its entirety
- S is comprised as a subset of the total population that is considered sexually active; thus, only this subset of the population is modeled.
- If an individual develops health complications, they remain in the D compartment until death.
- There is no immunity to syphilis.
- Those who have health complications are contained and cannot affect others.

4.6 Derivation

The derivation of this model was guided by the objective of preserving the meaningful results and simplicity of the classical SIR framework while enhancing its ability to capture key dynamics of syphilis transmission, treatment, and health complication. Similar to the SIR model, our approach assumes that the incidence rate is proportional to transmission dynamics governed by the law of mass action, and that all other transitions are proportional to the size of the compartment from which they originate. However, unlike the SIR model, infected individuals only transition into the treatment compartment through testing and identification. Recovery in our framework is realized through transitions between compartments rather than through a dedicated recovery compartment, reflecting a structure more akin to that of an SIS model.

This model also introduces a treatment compartment, which is only accessible to individuals who have undergone proper testing and been referred to appropriate care, underscoring the critical role of testing as a gateway to effective treatment. By modeling treatment in this way, we aim to explore the effects of significantly increased testing rates, hypothesizing that such an intervention

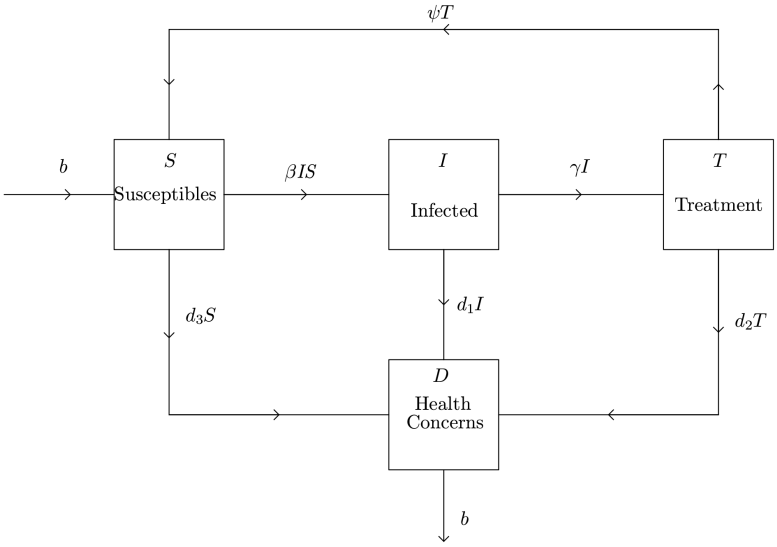


Figure 2: Syphilis Transfer Diagram

could lead to higher treatment uptake, a measurable reduction in infections at a given time, t, and a slower rate of increase in health complications over time. Additionally, to capture broader trends in health complication, the model includes a dedicated health complication compartment, providing a more comprehensive view of disease outcomes than models where health complications are simply treated as exits from the system. This formulation seeks to balance the simplicity of the SIR model with a richer understanding of testing, treatment, transmission, and health complication dynamics.

4.7 Variables & Parameter

Variables:

- $S = S(t) =$ Total number of susceptible individuals at time t
- $I = I(t) =$ Total number of infected individuals at time t
- $T = T(t) =$ Total number of individuals being treated at time t
- $D = D(t) =$ Total health complications at time t

Parameters:

- $\beta =$ Proportionality constant of the incidence rate governed by law of mass action

- $\gamma =$ Proportion of infected individuals who recieve treatment
- $\psi =$ Proportion of individuals who are treated and recover during each time step.
- $d_1 =$ Proportion of individuals who experience health concerns as a "hidden case"
- $d_2 =$ Proportion of individuals who experience health concerns during treatment
- $d_3 =$ Proportion of individuals who experience other health concerns as a susceptible
- $b =$ Birthrate and combined health complication rate

5 Mathematical Analysis

The analytical analysis includes finding the stability of the steady-state equilibrium points. Initially at time $t = 0 : S(0), I(0) > 0$ and $T(0) = D(0) = 0$. The parameters $b, \psi, \beta, \gamma, d_1, d_2, d_3$ are all positive. Recall the system of ODEs derived earlier:

- $S' = b + \psi T - \beta IS - d_1 S$
- $I' = \beta IS - \gamma I - d_1 I$
- $T' = \gamma I - \psi T - d_2 T$
- $D' = d_1 I + d_2 T + d_3 S - b$

Nota bene,

$$N(t) = S(t) + I(t) + T(t) + D(t) \forall t \geq 0 \Rightarrow S' + I' + T' + D' = (S + I + T + D)' = N' = 0.$$

The derivative of the total population N w.r.t. time is 0 , so the total population N remains constant, call it N_0. To find the steady-state equilibrium points, the four ODEs are set to 0 . Refer to the Appendix for thorough computations.

$$(S, I, T, D)$$
$$= \left(\frac{d_1 + \gamma}{\beta}, \frac{(d_3 S^* - b)(d_2 + \psi)}{(-d_1 d_2 - d_1 \psi - d_2 \gamma)}, \frac{(d_3 S^* - b)\gamma}{(-d_1 d_2 - d_1 \psi - d_2 \gamma)}, N_0 - \frac{d_1 + \gamma}{\beta} - \frac{(d_3 S^* - b)(d_2 + \psi + \gamma)}{(-d_1 d_2 - d_1 \psi - d_2 \gamma)} \right)$$

is the endemic steady-state equilibrium point and $(S, I, T, D) = \left(\frac{b}{d_3}, 0, 0, N_0 - \frac{b}{d_3} \right)$ is the disease free steady-state equilibrium point. To check for the stability of the steady-state equilibrium points, the Jacobian matrix taken w.r.t. S', I', T' gives

$$J(S,I,T)=\begin{bmatrix} \frac{\partial S'}{\partial S} & \frac{\partial S'}{\partial I} & \frac{\partial S'}{\partial T} \\ \frac{\partial I'}{\partial S} & \frac{\partial I'}{\partial I} & \frac{\partial I'}{\partial T} \\ \frac{\partial T'}{\partial S} & \frac{\partial T'}{\partial I} & \frac{\partial T'}{\partial T} \end{bmatrix}=\begin{bmatrix} -\beta I-d_3 & -\beta S & \psi \\ \beta I & \beta S-d_1-\gamma & 0 \\ 0 & \gamma & -(d_2+\psi) \end{bmatrix}$$

For the endemic steady-state equilibrium point, $I^*=\frac{(d_3S^*-b)(d_2+\psi)}{(-d_1d_2-d_1\psi-d_2\gamma)}$ and $T^*=\frac{(d_3S^*-b)\gamma}{(-d_1d_2-d_1\psi-d_2\gamma)}$ should both be positive inorder for an endemic to occur, so denominator $-d_1d_2-d_1\psi-d_2<0$

$$\begin{aligned} &\Leftrightarrow d_3S^*-b<0 \\ &\Leftrightarrow S^*=\frac{d_1+\gamma}{\beta}<\frac{b}{d_3} \\ &\Leftrightarrow 1<\frac{b\beta}{d_3\left(d_1+\gamma\right)}=\Re_0. \end{aligned}$$

$$J\left(S^*,I^*,T^*\right)=\begin{bmatrix} -\beta I^*-d_3 & -\beta S^* & \psi \\ \beta I^* & \beta S^*-d_1-\gamma & 0 \\ 0 & \gamma & -(d_2+\psi) \end{bmatrix}=\begin{bmatrix} -\beta I^*-d_3 & -\beta S^* & \psi \\ \beta I^* & 0 & 0 \\ 0 & \gamma & -(d_2+\psi) \end{bmatrix}$$

The determinant is

$$\begin{aligned} &det\left(J(S^*,I^*,T^*)\right)=-\beta I^*\left[\beta S^*\left(d_2+\psi\right)-\gamma\psi\right] \\ &=-\beta I^*\left[\left(d_1+\gamma\right)\left(d_2+\psi\right)-\gamma\psi\right]<0. \end{aligned}$$

The trace is $\operatorname{tr}\left[J\left(S^*,I^*,T^*\right)\right]=-\beta I^*-d_3-\left(d_2+\psi\right)<0$. The sum of all 2×2 principal minors of $J\left(S^*,I^*,T^*\right)$ is

$$a_2=\beta I^*\beta S^*+\left(\beta I^*+d_3\right)\left(d_2+\psi\right)=\beta I^*\left(d_1+\gamma\right)+\left(\beta I^*+d_3\right)\left(d_2+\psi\right)$$

So,

$$\begin{aligned} &\operatorname{tr}[J(S,I,T)]a_2 \\ &=-\beta I^*\left(d_1+\gamma\right)\left(d_2+\psi\right)-\left(\beta I^*+d_3\right)\beta I^*\left(d_1+\gamma\right)-\left(\beta I^*+d_3\right)^2\left(d_2+\psi\right)-\left(\beta I^*+d_3\right)\left(d_2+\psi\right)^2 \\ &<-\beta I^*\left(d_1+\gamma\right)\left(d_2+\psi\right)+\beta I^*\gamma\psi=\det\left[J\left(S^*,I^*,T^*\right)\right] \end{aligned}$$

All the Routh-Hurwitz conditions for 3×3 matrices are satisfied, therefore the endemic steady-state equilibrium point is stable. Recall $D^*=N_0-\frac{d_1+\gamma}{\beta}-\frac{(d_3S^*-b)(d_2+\psi+\gamma)}{(-d_1d_2-d_1\psi-d_2\gamma)}=N_0-\frac{d_1+\gamma}{\beta}-\frac{[d_3(d_1+\gamma)-b\beta](d_2+\psi+\gamma)}{(-\beta d_1d_2-\beta d_1\psi-\beta d_2\gamma)}$. Increasing the γ testing parameter will increase $\frac{d_1+\gamma}{\beta}$. It will also increase $\left[d_3\left(d_1+\gamma\right)-b\beta\right]\left(d_2+\psi+\gamma\right)$ and decrease $-\beta d_1d_2-\beta d_1\psi-\beta d_2\gamma$, so $\frac{[d_3(d_1+\gamma)-b\beta](d_2+\psi+\gamma)}{(-\beta d_1d_2-\beta d_1\psi-\beta d_2\gamma)}$ will increase. So more is being subtracted from N_0, the value of D^* will decrease. Thus $D(t)$ will converge to a smaller limit D^* when the γ testing parameter is increased, which is what we want to

achieve. Nota bene, endemic occurs $\Leftrightarrow 1 < \Re_0 = \frac{b\beta}{d_3(d_1+\gamma)} \Leftrightarrow \gamma < \frac{b\beta}{d_3} - d_1$. So increasing γ will decrease D^* so long as it stays below the $\frac{b\beta}{d_3} - d_1$ threshold.

For the disease free steady-state equilibrium point,

$$J(S^*, 0, 0) = \begin{bmatrix} -d_3 & -\beta S^* & \psi \\ 0 & \beta S^* - d_1 - \gamma & 0 \\ 0 & \gamma & -(d_2 + \psi) \end{bmatrix}$$

Cofactor expansion along the first column gives

$$\det[J(S^*, 0, 0)] = d_3(\beta S^* - d_1 - \gamma)(d_2 + \psi) < 0$$
$$\Leftrightarrow \quad \beta S^* - d_1 - \gamma < 0 \Leftrightarrow S^* = \frac{b}{d_3} < \frac{d_1 + \gamma}{\beta} \Leftrightarrow \frac{b\beta}{d_3(d_1 + \gamma)} = \Re_0 < 1.$$

The trace is $\operatorname{tr}[J(S^*, 0, 0)] = -d_3 + \beta S^* - d_1 - \gamma - (d_2 + \psi) < 0$ because $\beta S^* - d_1 - \gamma < 0$ from above. The sum of all 2×2 principal minors of $J(S^*, 0, 0)$ gives

$$a_2 = -d_3(\beta S^* - d_1 - \gamma) - (d_2 + \psi)(\beta S^* - d_1 - \gamma) + d_3(d_2 + \psi).$$

Let $x = \beta S^* - d_1 - \gamma < 0$. Then $\operatorname{tr}[J(S^*, 0, 0)] a_2$

$$= d_3(d_2 + \psi)x + (-d_3 - d_2 - \psi)^2 x + (-d_3 - d_2 - \psi)\left[x^2 + d_3(d_2 + \psi)\right] < d_3(d_2 + \psi)x = \det[J(S^*, 0, 0)].$$

All the Routh-Hurwitz conditions for 3×3 matrices are satisfied, therefore the disease free steady-state equilibrium point is stable. This happens when $\Re_0 = \frac{b\beta}{d_3(d_1+\gamma)} < 1 \Leftrightarrow \frac{b\beta}{d_3} - d_1 < \gamma$. Lo, once γ surpasses $\frac{b\beta}{d_3} - d_1$, increasing it anymore would not result in decreasing $D^* = N_0 - \frac{b}{d_3}$ as $I^* = T^* = 0$ will remain 0.

6 Model Simulation

We opted for a model simulation rather than parameter estimation due to the complexity of the data. Simulation not only allows us to create an idealized scenario for our model, but also helps us clearly identify whether any errors arise from the system itself rather than from the data. To fit the model, we used the Matlab code provided in Appendix C. Simulating the model requires us to first define the parameters and initial conditions. Initially, we chose a population size $N_0 = 9.1$, where 99% (9) of the population is susceptible, and the remaining $1\%(0.1)$ is initially infected, with no individuals in the other compartments. We aim to simulate both equilibrium states to compare whether the trends align or differ drastically.

For our syphilis model, we begin by examining the disease-free equilibrium. The chosen parameters are $\psi = 0.3, \gamma = 0.5, \beta = 0.8, d_1 = 0.4, d_2 = 0.2, d_3 = 0.7$, and $b = 0.5$, where β represents the transmission rate between susceptible and infected individuals. The other parameters correspond

to the rates at which individuals transition between compartments, and b refers to the birth and health complication rates. Due to the structure of the model, it's important to note that the reciprocal of each rate corresponds to the time it takes to move between compartments. These parameters were specifically selected to align with the mathematical analysis, which defines the disease-free equilibrium as stable when the condition $\frac{b\beta}{d_3(d_1+\gamma)} < 1$ holds true. Simulating the model with these parameters and initial conditions yields **Figure 3**.

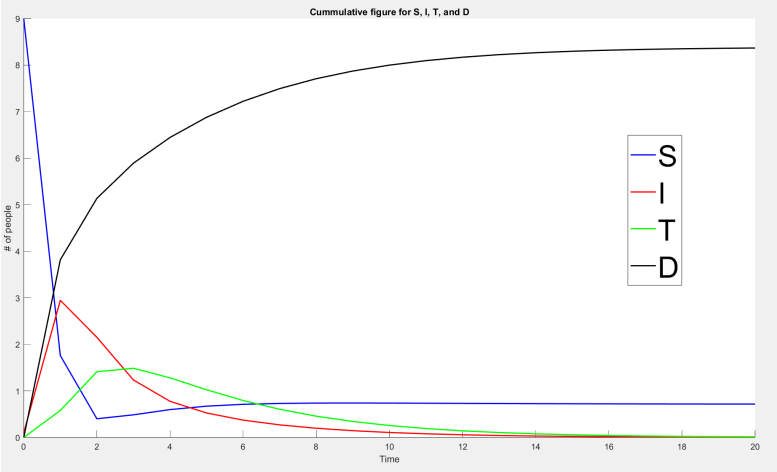


Figure 3: Cumulative Curve for Disease Free Equilibrium

Figure 3 shows that the susceptible curve starts high at the initial condition and decreases as the number of infected individuals increases. This decline is a result of disease transmission between those who have syphilis and those who are susceptible. During this period, we also observe an increase in health complications, which continues to rise but eventually plateaus, similar to the decline in the susceptible curve. In contrast to the health complications compartment, the infected compartment reaches a peak before gradually decreasing toward zero. This decline is likely influenced by testing rates, as the peak of the infected curve is shifted compared to that of health complications. This suggests that as the number of infected individuals rises, more people are likely to be tested. However, as the number of infected individuals decreases, testing becomes less prominent, leading to a corresponding decline in the number of individuals tested.

The health complications, influenced by varying testing rates gives **Figure 4**.

Similar to the cumulative plot in **Figure 3**, we observe that for all values of γ, the rate of health complications initially increases drastically but then reaches a peak before quickly decreasing and tending toward zero. This suggests that, eventually, no more individuals are experiencing negative health effects. However, when varying the value of γ or the testing rate, the peak of the curve shifts, although the overall shape remains consistent. A lower γ value corresponds to a longer time for an individual to get tested after being infected, while a higher γ value results in a shorter time to testing. Therefore, as γ increases, the trend of health complications remains the same, but the curve shifts downward, indicating a faster response to testing and, consequently, a quicker resolution of health

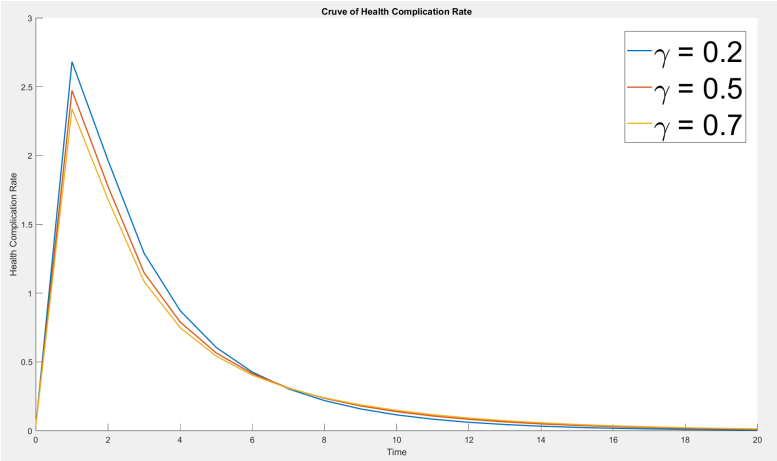


Figure 4: Heath Complication Rates for Disease Free Equilbrium

issues. Next, we look at the endemic equilibrium, with the parameters $\psi = 0.1, \gamma = 0.5, \beta = 0.7, d_1 = 0.3, d_2 = 0.2, d_3 = 0.4$, and $b = 0.8$, where the condition $\frac{b\beta}{d_3(d_1+\gamma)} > 1$ is satisfied. This yields the following **Figure 5**.

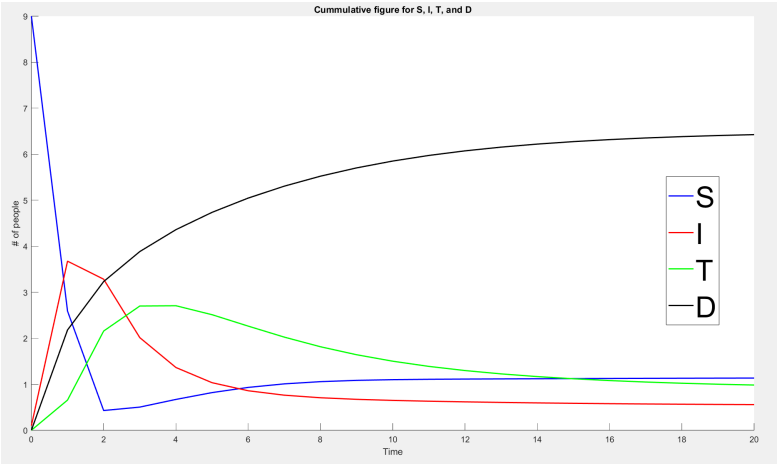


Figure 5: Cumulative Curve for Endemic Equilibrium

Similar to the disease-free scenario, the trends for all compartments are the same. However, instead of the infected and testing curves tending toward zero, they tend toward the endemic equilibrium described in the mathematical analysis. In this case, the disease does not completely die out and remains present in the population, which is a more realistic scenario for syphilis. When we examine the health complications rate for this model, we obtain the following **Figure 6**.

Varying the testing rate in the same way as for the disease-free scenario, we observe that the general shape of the curves remains the same across different values of γ, but the peak of each curve

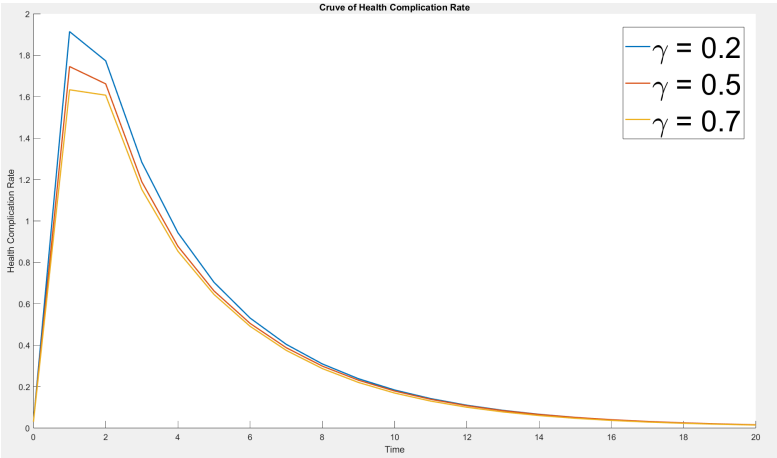


Figure 6: Health Complication Rates for Endemic Equilibrium

varies. For larger values of γ, the peak is lower, and for smaller values of γ, the peak is higher. Thus, between the two equilibrium states, we find that the rate at which health complications occur is lower as the testing rate γ increases.

7 Discussion

7.1 Summary

In this study, we developed a modified SIR model to explore the impact of increased testing and treatment on the dynamics of syphilis infection. Our results suggest that increase testing decreases the peak of the health complications rate that is associated with the disease. This highlights the importance of early detection and timely intervention in controlling syphilis outbreaks.

7.2 Future Direction

Future work could expand this model to varying rates of entry and exits, which would make it applicable to more dynamic and realistic scenarios such as urban communities or regions with significant migration. By incorporating population flux in births, health complications unrelated to the disease, and migration, the model could simulate a wider range of contexts. Additionally, integrating behavioral factors like healthcare access, stigma, and cultural influences, as well as network-based approaches to account for diverse contact patterns could provide deeper insights into disease dynamics. These enhancements would enable the model to evaluate large-scale public health strategies such as mass testing campaigns or targeted interventions, and explore their cost-effectiveness. Overall, refining this model for open systems would broaden its utility, allowing for a more comprehensive understanding of syphilis transmission and control in varied real-world settings.

7.3 Limitations

Despite its strengths, our model has limitations. For instance, it assumes uniform testing rates across a whole population, and does not account for varying levels of access to healthcare across different populations. Additionally, behavioral factors influencing the likelihood of individuals seeking testing and treatment were not included, which could impact the accuracy of the predictions.

Another limitation of this study is that insufficient data was available for comparison with the model outcomes. For example, data on individuals' pre-existing health conditions, lifestyle, and mental health may have provided greater insight as to what populations are being most affected by syphilis, and where interventions are most needed. Moreover, the lack of appropriate data prevented the researchers from performing statistical testing to determine whether the reduction in health complication rates as espoused by the model were significant. Thus, while the results of the model are promising, more research and data collection is required to determine what, if any, effect testing and treatment have in the long-run.

7.4 Applications

Our modified "SIT" model is particularly well-suited to scenarios that approximate a system, where the dynamics of disease transmission and intervention strategies can be observed with mini-

mal external interference. One such context is a retirement home or similar residential care facility, which offers unique characteristics that align with the assumptions of our model: Constant population dynamics, inflated STI rates, or a population of vulnerable individuals, where treatment/testing can be realistically implemented. While retirement homes represent an possible context for applying this model, it could also be adapted to other settings with similar characteristics, such as military barracks, college dormitories, and prisons. Each of these scenarios presents opportunities to tailor the model's parameters, such as testing frequency or contact rates to reflect the specific population dynamics.

As a result, the model does not fully reflect the true prevalence of syphilis. We relied upon whatever reported data was available and epidemiological estimates, but further research would be needed to obtain a more accurate representation of the infected population, and the efficacy of testing and treatment outcomes.

8 Conclusion

While there were challenges in identifying and fitting the actual number of syphilis cases, our model has demonstrated that increasing testing rates reduces syphilis rates and associated negative outcomes. By increasing testing, public health agencies can identify and treat more cases of syphilis, leading to improved health outcomes.

Modeling is an important first step, but developing strategies to translate the model's conclusions into actionable solutions is critical for addressing real-world problems. Based on the results of our analysis, increasing testing is the next logical step to decrease syphilis rates.

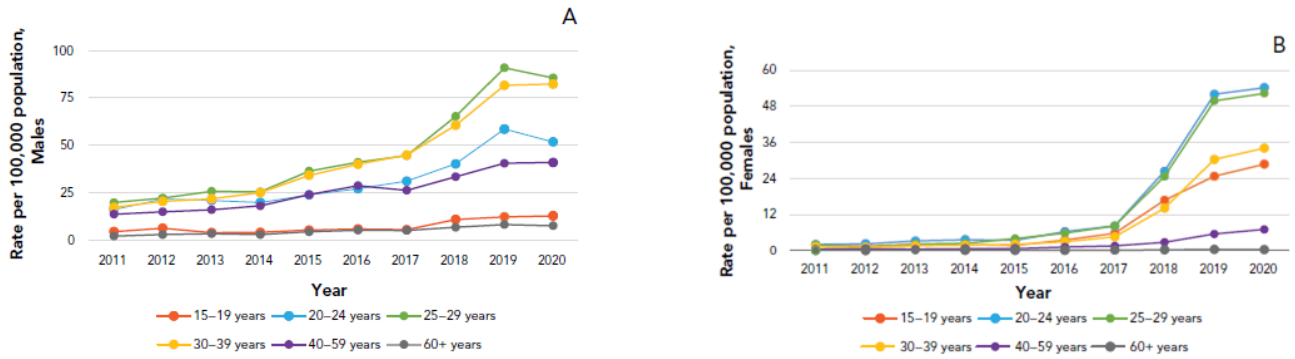
8.1 Importance of testing and Targeted Interventions

Increasing testing, and thereby identifying more cases of syphilis, has two major benefits. First, it enables timely treatment for individuals suffering from syphilis. Second, it enhances understanding of the disease's prevalence across different populations. As shown in the graphs depicting syphilis rates across various age groups and by sex, the disease is not equally distributed; certain age groups and males are more likely to be affected. The relevant charts are displayed below:

While the charts above focus on age and sex demographics, other demographic factors likely influence syphilis rates. For example, geographic location—specific neighborhoods or cities—may show higher prevalence, and certain socioeconomic brackets may be at greater risk.

Increased testing will provide a better understanding of these factors, enabling more targeted public health campaigns. This targeted approach would enhance awareness and improve the likelihood of reaching individuals at higher risk through focused marketing efforts.

If we consider an extreme scenario, such as testing an entire city's population, this could be effective in identifying cases but would also be prohibitively expensive and likely to face significant societal resistance. Therefore, more targeted approaches would be both cost-effective and less politically controversial.



(a) Males (b) Females

Figure 7: Rate of infectious syphilis in Canada, 2011-2020

Potential strategies to increase awareness and testing include identifying high-risk demographics and utilizing targeted advertising through platforms such as Facebook and other social media apps. Another possibility is to collaborate with dating apps to include badges on profiles of users who have recently been tested for STIs, encouraging conversations about sexual health and testing.

8.2 Moving Forward with Action and Research

While there were challenges in finding and fitting the actual number of syphilis cases, our model shows the clear benefits of increasing testing to lower syphilis rates and reduce negative outcomes. By identifying more cases and gaining a better understanding of demographic and geographic risk factors, public health agencies can allocate resources more effectively and develop targeted interventions.

These findings have wider implications for public health, demonstrating the importance of combining data-driven modeling with practical implementation. Increasing testing for syphilis is not just about controlling one disease; it can serve as a framework for tackling other public health issues.

Going forward, collaboration among public health agencies, policymakers, and community organizations will be essential to increasing testing, running targeted awareness campaigns, and finding creative ways to reduce stigma around STI testing. Further research will also be important to refine our understanding of risk factors, develop cost-effective strategies, and track the long-term impact of these efforts.

The findings from this report highlight the importance of taking a proactive and focused approach to reducing syphilis rates. Acting on these insights can lead to meaningful progress in reducing the disease's prevalence and improving health outcomes for communities.

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Appendix A

To find the steady-state equilibrium points, the four ODEs are set to 0 .

$$I' = \beta IS - \gamma I - d_1 I = I(\beta S - \gamma - d_1) = 0 \Rightarrow I^* = 0, S^* = \frac{d_1+\gamma}{\beta}.$$

$$S^* = \frac{d_1+\gamma}{\beta} \Rightarrow S' = b + \psi T - (d_1+\gamma) I - d_3 \frac{d_1+\gamma}{\beta} = 0 \Rightarrow \psi T - (d_1+\gamma) I = d_3 \frac{d_1+\gamma}{\beta} - b.$$

$$T' = \gamma I - \psi T - d_2 T = 0 \Rightarrow I = \frac{(d_2+\psi)}{\gamma} T$$

$$\Rightarrow \psi T - \frac{(d_1+\gamma)(d_2+\psi)}{\gamma} T = \frac{\gamma\psi - (d_1+\gamma\gamma(d_2+\psi)}{\gamma} T = d_3 \frac{d_1+\gamma}{\beta} - b = \frac{d_3(d_1+\gamma) - b\beta}{\beta}$$

$$\Rightarrow T^* = \frac{[d_3(d_1+\gamma) - b\beta]\gamma}{\beta[\gamma\psi - (d_1+\gamma)(d_2+\psi)]} = \frac{(d_3 S^* - b)\gamma}{(-d_1 d_2 - d_1\psi - d_2\gamma)}$$

$$\Rightarrow I^* = \frac{(d_2+\psi)}{\gamma} \frac{(d_3 S^* - b)\gamma}{(-d_1 d_2 - d_1\psi - d_2\gamma)} = \frac{(d_3 S^* - b)(d_2+\psi)}{(-d_1 d_2 - d_1\psi - d_2\gamma)}$$

$$\Rightarrow D^* = N_0 - S^* - I^* - T^* = N_0 - \frac{d_1+\gamma}{\beta} - \frac{(d_3 S^* - b)(d_2+\psi)}{(-d_1 d_2 d_1\psi - d_2\gamma')} - \frac{(d_3 S^* - b)\gamma}{(-d_1 d_2 - d_1\psi - d_2\gamma)}$$

$$= N_0 - \frac{d_1+\gamma}{\beta} - \frac{(d_3 S^* - b)(d_2+\psi+\gamma)}{(-d_1 d_2 - d_1\psi - d_2\gamma)}$$

Thus $(S, I, T, D) = \left(\frac{d_1+\gamma}{\beta}, \frac{(d_3 S^* - b)(d_2+\psi)}{(-d_1 d_2 - d_1\psi - d_2\gamma)}, \frac{(d_3 S^* - b)\gamma}{(-d_1 d_2 - d_1\psi - d_2\gamma)}, N_0 - \frac{d_1+\gamma}{\beta} - \frac{(d_3 S^* - b)(d_2+\psi+\gamma)}{(-d_1 d_2 - d_1\psi - d_2\gamma)}\right)$ is the endemic steady-state equilibrium point.

$$I^* = 0 \Rightarrow T' = -\psi T - d_2 T = (-\psi - d_2) T = 0 \Rightarrow T^* = 0 \Rightarrow S' = b - d_3 S = 0$$

$\Rightarrow S^* = \frac{b}{d_3} \Rightarrow D^* = N_0 - \frac{b}{d_3}$. Thus $(S, I, T, D) = \left(\frac{b}{d_3}, 0, 0, N_0 - \frac{b}{d_3}\right)$ is the disease free steady-state equilibrium point.

To check for the stability of the endemic steady-state equilibrium point:

$$J(S^*, I^*, T^*) = \begin{bmatrix} -\beta I^* - d_3 & -\beta S^* & \psi \\ \beta I^* & \beta S^* - d_1 - \gamma & 0 \\ 0 & \gamma & -(d_2+\psi) \end{bmatrix} = \begin{bmatrix} -\beta I^* - d_3 & -\beta S^* & \psi \\ \beta I^* & 0 & 0 \\ 0 & \gamma & -(d_2+\psi) \end{bmatrix}$$

Cofactor expansion along the second row gives $\det[J(S^*, I^*, T^*)] = -\beta I^* [\beta S^* (d_2+\psi) - \gamma\psi]$

$$= -\beta I^* [(d_1+\gamma)(d_2+\psi) - \gamma\psi] = -\beta I^* (d_1 d_2 + d_1\psi + d_2\gamma) < 0$$

Its trace is $\mathrm{tr}\left[J\left(S^*, I^*, T^*\right)\right] = -\beta I^* - d_3 - \left(d_2 + \psi\right) = -\left(\beta I^* + d_3 + d_2 + \psi\right) < 0$. The sum of all 2×2 principal minors of $J\left(S^*, I^*, T^*\right)$ gives

$$a_2 = \beta I^* \beta S^* + \left(\beta I^* + d_3\right)\left(d_2 + \psi\right) = \beta I^*\left(d_1 + \gamma\right) + \left(\beta I^* + d_3\right)\left(d_2 + \psi\right)$$

So, $\mathrm{tr}\left[J\left(S^*, I^*, T^*\right)\right] a_2 = -\left[\left(\beta I^* + d_3\right) + \left(d_2 + \psi\right)\right]\left[\beta I^*\left(d_1 + \gamma\right) + \left(\beta I^* + d_3\right)\left(d_2 + \psi\right)\right]$

$$= -\beta I^*\left(d_1 + \gamma\right)\left(d_2 + \psi\right) - \left(\beta I^* + d_3\right)\beta I^*\left(d_1 + \gamma\right) - \left(\beta I^* + d_3\right)^2\left(d_2 + \psi\right) - \left(\beta I^* + d_3\right)\left(d_2 + \psi\right)^2$$

$$< -\beta I^*\left(d_1 + \gamma\right)\left(d_2 + \psi\right) + \beta I^* \gamma\psi = -\beta I^*\left[\left(d_1 + \gamma\right)\left(d_2 + \psi\right) - \gamma\psi\right] = \det\left[J\left(S^*, I^*, T^*\right)\right]$$

To check for the stability of the disease free steady-state equilibrium point:

$$J\left(S^*, 0, 0\right) = \begin{bmatrix} -d_3 & -\beta S^* & \psi \\ 0 & \beta S^* - d_1 - \gamma & 0 \\ 0 & \gamma & -\left(d_2 + \psi\right) \end{bmatrix}$$

Cofactor expansion along the first column gives $\det\left[J\left(S^*, 0, 0\right)\right] = d_3\left(\beta S^* - d_1 - \gamma\right)\left(d_2 + \psi\right) < 0$

$$\Leftrightarrow \beta S^* - d_1 - \gamma < 0 \Leftrightarrow S^* = \frac{b}{d_3} < \frac{d_1 + \gamma}{\beta} \Leftrightarrow \frac{b\beta}{d_3\left(d_1 + \gamma\right)} = \Re_0 < 1.$$

The trace is $\mathrm{tr}\left[J\left(S^*, 0, 0\right)\right] = -d_3 + \beta S^* - d_1 - \gamma - \left(d_2 + \psi\right) < 0$ because $\beta S^* - d_1 - \gamma < 0$ from above. The sum of all 2×2 principal minors of $J\left(S^*, 0, 0\right)$ gives

$a_2 = -d_3\left(\beta S^* - d_1 - \gamma\right) - \left(d_2 + \psi\right)\left(\beta S^* - d_1 - \gamma\right) + d_3\left(d_2 + \psi\right)$. Let $x = \beta S^* - d_1 - \gamma < 0$.

Then $\mathrm{tr}\left[J\left(S^*, 0, 0\right)\right] a_2 = \left[x + \left(-d_3 - d_2 - \psi\right)\right]\left[\left(-d_3 - d_2 - \psi\right) x + d_3\left(d_2 + \psi\right)\right]$

$$= d_3\left(d_2 + \psi\right) x + \left(-d_3 - d_2 - \psi\right)^2 x + \left(-d_3 - d_2 - \psi\right) x^2 + \left(-d_3 - d_2 - \psi\right) d_3\left(d_2 + \psi\right)$$

$$= d_3\left(d_2 + \psi\right) x + \left(-d_3 - d_2 - \psi\right)^2 x + \left(-d_3 - d_2 - \psi\right)\left[x^2 + d_3\left(d_2 + \psi\right)\right]$$

$$< d_3\left(d_2 + \psi\right) x = d_3\left(d_2 + \psi\right)\left(\beta S^* - d_1 - \gamma\right) = \det\left[J\left(S^*, 0, 0\right)\right]$$

Appendix B

```
    % Plotting solutions of ODEs in time plots

clear all % Clear all variables
close all % Close all figures

% Define parameters
 % psi = 0.1;
 % beta = 0.7;
 % gamma = 0.5;
 % d1 = 0.3;
 % d2 = 0.2;
 % d3 = 0.4;
 % b = 0.8;

  psi = 0.3;  % disease free
 beta = 0.8;
 gamma = 0.5;
 d1 = 0.4;
 d2 = 0.2;
 d3 = 0.7;
 b = 0.5;


% Define the system of ODEs
% f = @(t,x) [psi * x(3) - beta * x(2) * x(1);
%             beta * x(2) * x(1) - gamma * x(2) - d1 * x(2);
%             gamma * x(2) - psi * x(3) - d2 * x(3);
%             d1 * x(2) + d2 * x(3)];

f = @(t,x) [b - d3*x(1)+ psi * x(3) - beta * x(2) * x(1);
            beta * x(2) * x(1) - gamma * x(2) - d1 * x(2);
            gamma * x(2) - psi * x(3) - d2 * x(3);
            d3*x(1) + d1 * x(2) + d2 * x(3)-b];

% g = @(t,x) [psi * x(3) - beta * x(2) * x(1);
%             beta * x(2) * x(1) - gamma2 * x(2) - d1 * x(2);
%             gamma2 * x(2) - psi * x(3) - d2 * x(3);
%             d1 * x(2) + d2 * x(3)];
```

```
g = @(t,x) [b - d3*x(1)+ psi * x(3) - beta * x(2) * x(1);
            beta * x(2) * x(1) - gamma * x(2) - d1 * x(2);
            gamma * x(2) - psi * x(3) - d2 * x(3);
            d3*x(1) + d1 * x(2) + d2 * x(3)-b];

% gamma_values = [0.2, 0.5, 0.7];  % Example gamma values
gamma_values = [0.2, 0.5, 0.7];  % disease free


% Solve the system using ode45
[t, xa] = ode45(f, [0:1:20], [9, 0.1, 0, 0]);

% Evaluate d1*x2 + d2*x3 for the solution xa
p = psi * xa(:,3) - beta * xa(:,2) .* xa(:,1); % Calculate the expression for each time s
i = beta * xa(:,2) .* xa(:,1) - gamma * xa(:,2) - d1 * xa(:,2);
T = gamma * xa(:,2) - psi * xa(:,2) - d2 * xa(:,3); % Fixed here
d = d1 * xa(:,2) + d2 * xa(:,3);

figure;
hold on

% Loop through the gamma values to solve and plot the expression for each
for i = 1:length(gamma_values)
    gamma2 = gamma_values(i);  % Update gamma2 for the current iteration
    g = @(t,x) [psi * x(3) - beta * x(2) * x(1);
                beta * x(2) * x(1) - gamma2 * x(2) - d1 * x(2);
                gamma2 * x(2) - psi * x(3) - d2 * x(3);
                d1 * x(2) + d2 * x(3)];

    % Solve the system for the current gamma2 value
    [t, xb] = ode45(g, [0:1:20], [9, 0.1, 0, 0]);

    % Compute d1 * x(2) + d2 * x(3)
    dd = d1 * xb(:,2) + d2 * xb(:,3);

    % Plot the result for this gamma
    plot(t, dd, 'LineWidth', 1.5);
end
```

```
hold off
xlabel('Time');
ylabel('Health Complication Rate');
title('Cruve of Health Complication Rate');
legend(arrayfun(@(gamma) ['\gamma = ' num2str(gamma)], gamma_values, 'UniformOutput', fals
legendFontSize = 35; % Set the desired font size
set(gcf, 'CurrentAxes', gca); % Get the current axes
legend('FontSize', legendFontSize); % Apply the font size to the legend

figure;

% First subplot for the ODE solutions (S, I, T, D)

hold on
plot(t, xa(:,1), 'b', 'LineWidth', 1.5) % Plot S
plot(t, xa(:,2), 'r', 'LineWidth', 1.5) % Plot I
plot(t, xa(:,3), 'g', 'LineWidth', 1.5) % Plot T
plot(t, xa(:,4), 'k', 'LineWidth', 1.5) % Plot D
hold off
xlabel('Time');
ylabel('# of people');
title('Cummulative figure for S, I, T, and D');
lgd = legend('S', 'I', 'T', 'D', 'Location', 'best');
set(lgd, 'FontSize', 35); % Set the font size for the legend


figure;

% First subplot for the ODE solutions (S, I, T, D)
subplot(2, 1, 1); % This will place the first plot in the upper half (2 rows, 1 column)
hold on
plot(t, xa(:,1), 'b', 'LineWidth', 1.5) % Plot S
plot(t, xa(:,2), 'r', 'LineWidth', 1.5) % Plot I
plot(t, xa(:,3), 'g', 'LineWidth', 1.5) % Plot T
plot(t, xa(:,4), 'k', 'LineWidth', 1.5) % Plot D
hold off
xlabel('Time');
ylabel('# of people');
title('Cummulative figure for S, I, T, and D');
legend('S', 'I', 'T', 'D', 'Location', 'best');
```

```
legendFontSize = 35; % Set the desired font size

% Second subplot for the expression d1*x2 + d2*x3 for different gammas
subplot(2, 1, 2); % This will place the second plot in the lower half (2 rows, 1 column)
hold on

% Loop through the gamma values to solve and plot the expression for each
for i = 1:length(gamma_values)
    gamma2 = gamma_values(i);  % Update gamma2 for the current iteration
    g = @(t,x) [psi * x(3) - beta * x(2) * x(1);
                beta * x(2) * x(1) - gamma2 * x(2) - d1 * x(2);
                gamma2 * x(2) - psi * x(3) - d2 * x(3);
                d1 * x(2) + d2 * x(3)];

    % Solve the system for the current gamma2 value
    [t, xb] = ode45(g, [0:1:20], [9, 0.1, 0, 0]);

    % Compute d1 * x(2) + d2 * x(3)
    dd = d1 * xb(:,2) + d2 * xb(:,3);

    % Plot the result for this gamma
    plot(t, dd, 'LineWidth', 1.5);
end

hold off
xlabel('Time');
ylabel('Health Complication Rate');
title('Cruve of Health Complication Rate');
legend(arrayfun(@(gamma) ['\gamma = ' num2str(gamma)], gamma_values, 'UniformOutput', fals
legendFontSize = 14; % Set the desired font size
set(gcf, 'CurrentAxes', gca); % Get the current axes
legend('FontSize', legendFontSize); % Apply the font size to the legend
```