CX 4230 Mini Project 1

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Introduction:

This document is our write up for Mini Project 1: Contagion. In our report, we will discuss our answers for the problems along with our approach to these problems. We are asked to model an epidemic in which we can divide people into three different groups: susceptible, infected, and recovered. The first group consists of the people who can get the disease but haven't had it yet. The second group consists of people with the disease whereas the third group consists of people who no longer have the disease. For our model, we assume that the disease can only spread from infected people to susceptible people. We also assume that after some time, the infected people recover and develop immunity to the disease. Lastly, we assume that no one can be born or die in our population. In other words, $S(t) + I(t) + R(t) = 1$.

Question 1.1: Derivation

For 1.1, we are asked to use variable substitution along with the conservation law to derive equation 4 from equation 3. The model in equation 3 is a system of ordinary differential equations that uses τ and κ as parameters to measure the rate of spread and the rate of recovery in people.

$$\vec{f}(\vec{x}) = \begin{bmatrix} -\tau S(t) I(t) \\ \tau S(t) I(t) - \dfrac{I(t)}{\kappa} \\ \dfrac{I(t)}{\kappa} \end{bmatrix}.$$

$$\hat{D}\begin{bmatrix}\hat{S}\\ \hat{I}\end{bmatrix} = \begin{bmatrix}-\hat{S}\hat{I}\\ (\hat{S}-1)\hat{I}\end{bmatrix},$$

To derive equation 4, , we will proceed with using the conservation law and variable substitution to eliminate the parameters and simplify the equations. Using the conservation law, we can assume the total population doesn't change. We can then use algebraic manipulation to derive that $R(t) = 1 - S(t) - I(t)$. This allows us to simplify the system by reducing the number of variables. R can be determined from S and I, making it redundant in directly solving the system's dynamics.

Next, using variable substitution, we have S^ = S/K and I^ = I/K, then:

$$fx = \begin{Bmatrix} kS \\ kI \end{Bmatrix} = \begin{Bmatrix} -\tau(kS)(kI) \\ \tau(kS)(kI) - I \end{Bmatrix} = \begin{Bmatrix} -\tau(k^2)SI \\ \tau(k^2)SI - I \end{Bmatrix}$$

The above results in equation 4 when tau and k^2 are factored out of the matrix. These can be factored out since we are trying to find the result in terms of S^ and I^. In the above solution, the S and I are supposed to be hatted.

Question 1.2: Finding Fixed Points

When, finding the fixed points of the system

$dS/dt = 0\ implies\ -SI = 0$

$dI/dt = 0\ implies\ (S-1)I = 0$

From these equations, we can deduce the conditions for fixed points:

- Based on the first equation, for dS/dt to be zero, either $S = 0$ or $I = 0$
- Based on the second equation, for dI/dt to be zero, either $I = 0$ or $S = 1$

Hence, we have the fixed points as:

- $S = 1, I = 0$: This represents a disease-free state where all individuals are susceptible, and there are no infected individuals
- $S = 0, I = 0$: This point is not biologically relevant as it would imply no individuals in either category, which contradicts the model's premise

Classifying Stability

To classify the stability of these fixed points, we need to analyze the Jacobian matrix of the system at these points. The Jacobian matrix J for the system is:

$$J = \begin{bmatrix} \frac{\partial(-\hat{S}\hat{I})}{\partial\hat{S}} & \frac{\partial(-\hat{S}\hat{I})}{\partial\hat{I}} \\ \frac{\partial((\hat{S}-1)\hat{I})}{\partial\hat{S}} & \frac{\partial((\hat{S}-1)\hat{I})}{\partial\hat{I}} \end{bmatrix} = \begin{bmatrix} -\hat{I} & -\hat{S} \\ \hat{I} & \hat{S}-1 \end{bmatrix}$$

For each fixed point, we'll substitute the values into the Jacobian and analyze the eigenvalues to determine stability.

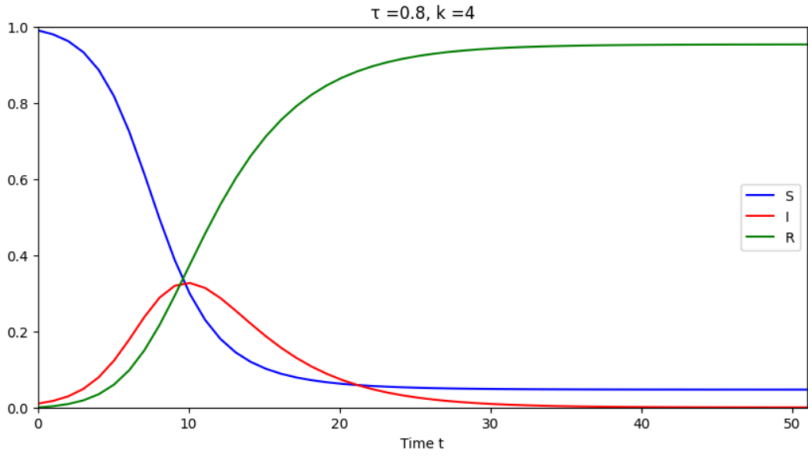
- For $S = 1, I = 0$: The jacobian simplifies to a matrix that will help us understand the nature of this equilibrium point.

det(Jλ) = (-Iλ)(Sλ-λ)-(-SI)
= -SIλ^2 + Iλ^2 + SI = 0
(-SI + I)λ^2 = -SI
λ^2 = (-SI)/(-SI + I)
Λ = sqrt((-SI)/(-SI + I)) = sqrt(-S/(-S + 1))

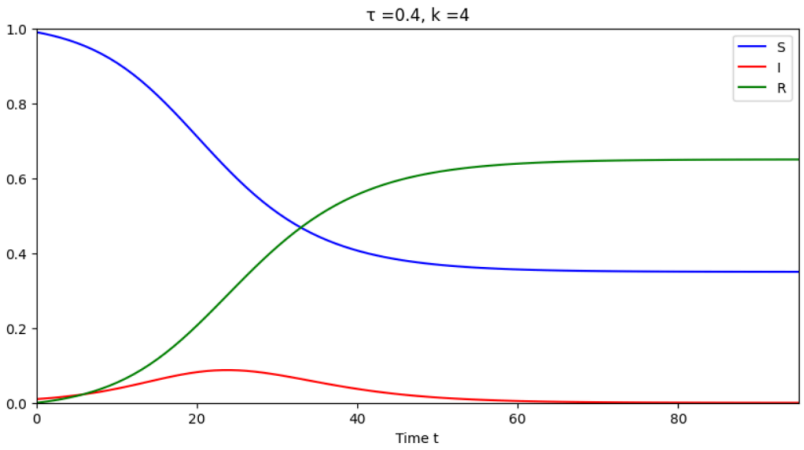
Hence, λ = sqrt(0/1) or λ = sqrt(-1/0)
This is a degenerate case where the imaginary case should not be considered. So, fixed points are semi-stable.

Question 1.3: The Three Simulations

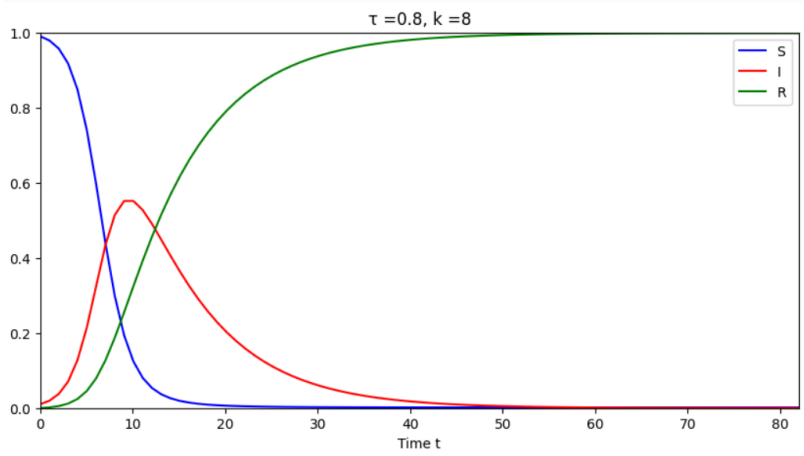
Simulation 1:



Simulation 2:



Simulation 3:



Objective:
To run simulations with varying parameters (τ, k) to observe their effects on the duration of an epidemic by solving a differential equation using Python (numpy and scipy).

Scenarios:

- (τ=0.8, k=4): Fast spread and moderate recovery rate.
- (τ=0.4, k=4): Slower spread and moderate recovery rate.
- (τ=0.8, k=8): Fast spread and slow recovery rate.

Stopping Condition: Simulation runs until $I(t)$ < 10^-4, indicating the epidemic is nearly over.

Results:

- For (τ=0.8, k=4): Epidemic ends quickest with t = 51.
- For (τ=0.4, k=4): Epidemic lasts longest with t = 95.
- For (τ=0.8, k=8): Intermediate duration with t = 82.

Observations:

- Scenario 1 (τ=0.8, k=4): Represents a balanced approach with a quick spread but also a quick recovery, leading to the shortest epidemic duration.
- Scenario 2 (τ=0.4, k=4): Exhibits a slower virus spread and a quicker recovery, resulting in the longest epidemic due to fewer infections and a slower reach to the population.
- Scenario 3 (τ=0.8, k=8): Characterized by a quick spread and slower recovery, had an intermediate duration. It suggests that a faster exposure to the virus, despite slower recovery, can contribute to a quicker end to the epidemic due to widespread immunity.

Graphical Analysis:

- Image 1: Presentes scenario 1’s moderate infection peak and how it balances susceptible and recovery curves.
- Image 2: Highlights scenario 2's lower infection peak and longer duration to peak.
- Image 3: Shows scenario 3's higher infection peak and quicker descent in the number of susceptible individuals.

Initial Intuition vs. Outcome:

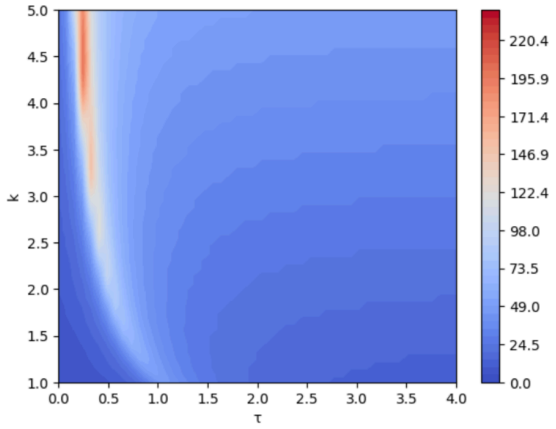
Initially, it was assumed maximizing τ and minimizing k would be ideal for ending the epidemic quickly. This hypothesis was partially correct but nuanced, as further discussed in section 1.4, indicating that the interplay between the rate of spread and recovery significantly influences epidemic duration.

Question 1.4: 2D Contour Plot

Initial Strategy and Reevaluation:

Initially, we thought that minimizing the recovery rate parameter (k) and maximizing the infection rate parameter (τ) would minimize the time to meet the epidemic's stopping condition. However, upon analyzing 2-D contour plots for ranges $\tau \in (0, 4]$ and $\kappa \in [1, 5]$, this strategy proved to be overly simplistic and not universally valid.

2D Contour Plot:



Key Observations from 2-D Contour Plots:

- The color gradient (red to blue) indicates time duration to meet the stopping condition, with blue representing shorter times.
- Lower k values and higher τ values generally correlate with shorter durations, but exceptions exist based on the specific τ range.
- The critical observation involves the stability condition given by $1/\tau k S_0$, separating stable and unstable fixed points in the system, impacting outbreak occurrence and duration.

Epidemic Dynamics Insights:

- A significant insight is the delineation between scenarios where an outbreak does or does not occur, based on the value of $\tau k S_0$ relative to 1. This ratio is a predictor of outbreak occurrence and system stability.
- Stable System: When $\tau k S_0 <= 1$, indicating no outbreak and stable fixed points, the epidemic ends quickly due to the slow spread.
- Unstable System: When $\tau k S_0 > 1$, indicating an outbreak, achieving a quick end to the epidemic requires the disease to spread and recover quickly, despite the system's instability making precise predictions challenging.

Unexpected Findings:

- The analysis revealed that certain combinations of τ and k values (e.g., (1.5, 1.5) vs. (1.5, 1)) resulted in counterintuitive outcomes, underscoring the system's instability and the difficulty in predicting the exact impact of varying these parameters.

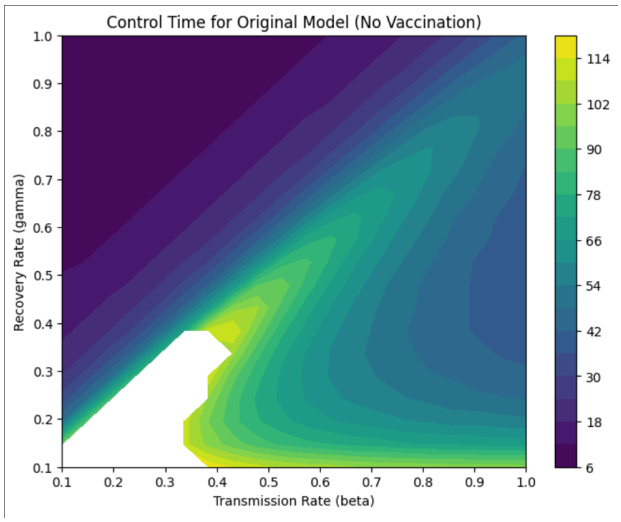
- It was observed that not all strategies that seem to minimize the time to reach the stopping condition are effective due to the complex interplay between the rate of spread, recovery rate, and system stability.

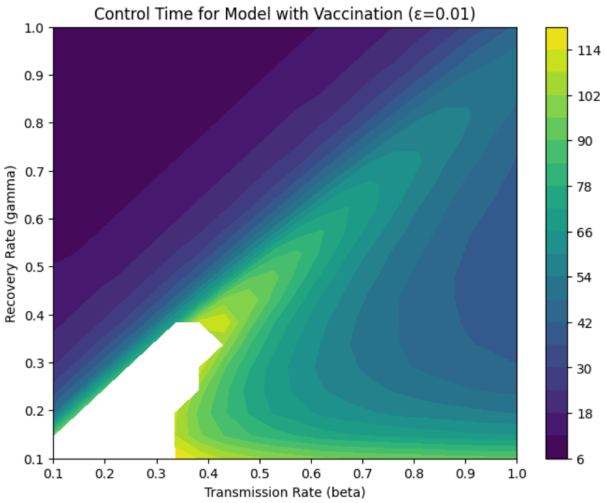
Conclusion:
While a general trend suggests that lower recovery rates and higher infection rates may shorten the epidemic under specific conditions, the system's inherent instability and the critical role of the $\tau k S_0$ ratio in determining outbreak occurrence and epidemic duration complicate universal application of this strategy.

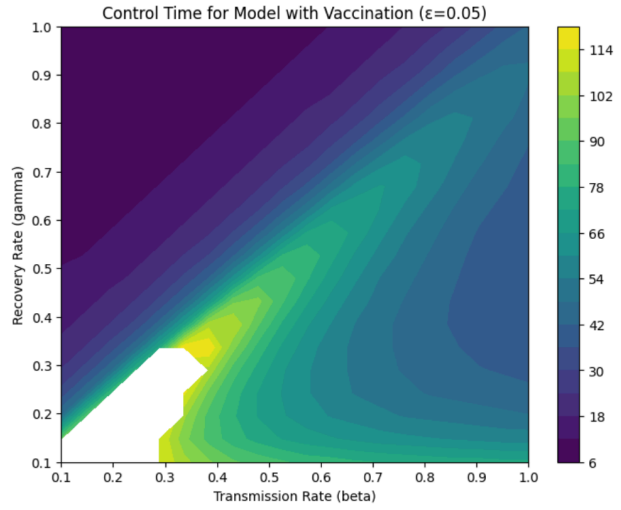
Question 1.5:

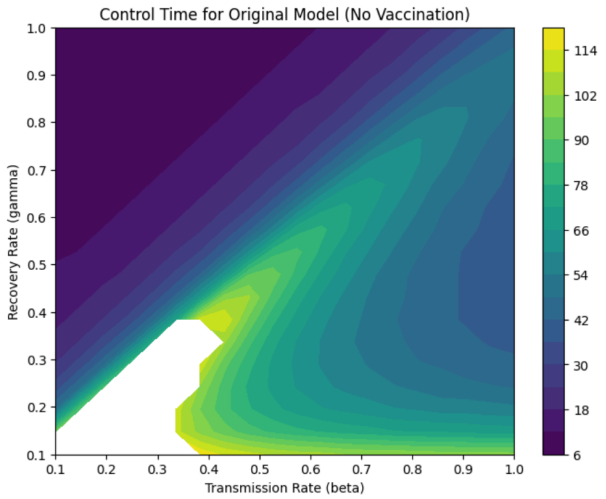
Policy 2 is defined by the formula $g(x) = g_2(x) = v_2 SI/S + I$. This formula suggests that the rate of vaccination is proportional to the product of the susceptible group and the infected populations divided by the sum of susceptible and infected groups. This approach reflects a target approach to vaccination in response to higher levels of infection in the population. In other words, it prioritizes the use of vaccinations when the threat of transmission is the greatest by analyzing the group of infected people and the susceptible group. When comparing this policy to Policy 1 which vaccinates at a rate proportional to the susceptible population, Policy 2 considers the current state of infections. Policy 2 adjusts the rate based on a ratio of susceptible to infected groups which reflects a more targeted approach responding to varying conditions during an outbreak. For instance, in a situation when the infection is already widespread, Policy 2 would prioritize vaccination even if the susceptible population isn't as large. In contrast, in a situation when the infection rate is low with a large susceptible group, Policy 2 may not prioritize vaccination as much as Policy 1.

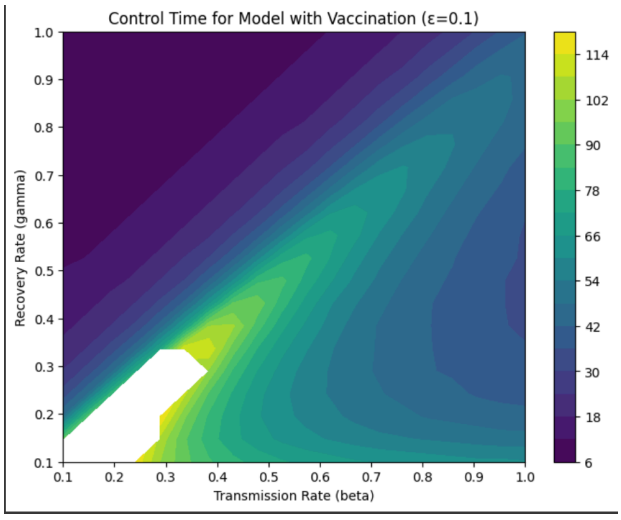
After running the simulation to visualize the differences between the two models, we generated these graphs for various parameters.











These contour plots help us visualize how vaccinations with varying rates affect the time required to control the epidemic. By comparing the two

models across different parameters, we can garner the impact of vaccinations. As seen in the graphs, vaccination makes it easier to control the disease in quicker time across all ranges of beta and gamma. This conclusion is supported from the shift in time toward the purple end of the spectrum whenever vaccination is used. However, the higher vaccination rates also imply higher costs for these efforts. We can see that even a small vaccination rate can greatly affect control times. Vaccination helps the population achieve a steady state sooner, especially when considering scenarios with high transmission rates and low recovery rates. In conclusion, vaccination can be a great solution for controlling epidemics; however, they come with higher costs. Hence, it is imperative to use a balanced strategy that addresses its benefits, its costs, and its logistical implications.