CX 4230 - Computer Simulation - Project 1

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1 Question 1.1

First, using the conversation law, we can express $R(t) = 1 - S(t) - I(t)$. Equation 3 then becomes:

$$\frac{dS}{dt} = -\tau S(t)I(t)$$
$$\frac{dI}{dt} = \tau S(t)I(t) - \frac{I(t)}{\kappa}$$

We can drop $\frac{dR}{dT}$ since $R(t)$ is determined by $S(t)$ and $I(t)$.

From here, we'll define $\hat{S} = \tau S(t)$ and $\hat{I} = \frac{I(t)}{\kappa}$. We can now substitute these new variables into the equation to get:

$$\frac{d\hat{S}}{dt} = -\kappa\hat{S}\hat{I}$$
$$\frac{d\hat{I}}{dt} = \kappa\hat{S}\hat{I} - \hat{I}$$

Next, we need to rescale the time derivative: to eliminate κ from the derivative of $\hat{I}$, we can rescale time by defining $d\hat{t} = \kappa dt$. Thus, $\frac{d}{dt} = \frac{1}{\kappa}\frac{d}{d\hat{t}}$. Now, the system with the new variables becomes:

$$\frac{d\hat{S}}{d\hat{t}} = -\hat{S}\hat{I}$$
$$\frac{d\hat{I}}{d\hat{t}} = \hat{S}\hat{I} - \hat{I}$$

The above system is equivalent to Equation 4, which was derived from the original model. We now have a simplified system without τ or κ in the equations explicitly, which have been absorbed. The definitions for the appropriate hat-variables and the operator are as follows:

$$\hat{D} = \frac{1}{\kappa}\frac{d}{d\hat{t}}$$
$$\hat{S} = \tau S(t)$$
$$\hat{I} = \frac{I(t)}{\kappa}$$

The domain for the new variables, $\hat{S}$ and $\hat{I}$ is as follows:

$$\hat{S} = [0, \tau\kappa]$$
$$\hat{I} = [0, \tau\kappa]$$

2 Question 1.2

To find the fixed points of the system, we have to set the derivatives equal to 0 and solve for $\hat{S}$ and $\hat{I}$. The fixed points occur where the rate of change of both of these is equal to 0. From the following equations:

$$\frac{d\hat{S}}{d\hat{t}} = -\hat{S}\hat{I}$$
$$\frac{d\hat{I}}{d\hat{t}} = (\hat{S}-1)\hat{I}$$

Setting the RHS equal to 0 gives us the following cases:

1. $-\hat{S}\hat{I} = 0$ implies that either $\hat{S} = 0$ or $\hat{I} = 0$.
2. $(\hat{S}-1)\hat{I} = 0$ implies that either $\hat{S} = 1$ or $\hat{I} = 0$.

This gives us two fixed points for the system: $(\hat{S}, \hat{I}) = (1, 0)$ and $(\hat{S}, \hat{I}) = (0, 0)$. Let's now classify their stability. To address the stability of the two fixed points, we need to calculate the eigenvalues of the Jacobian matrix at each fixed point of the system. We'll reference the cases provided by the professor in this post help classify the stability of the fixed points. For the system:

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

The Jacobian Matrix J at any point $\left(\hat{S}, \hat{I}\right)$ is:

$$J = \begin{bmatrix}-\hat{I} & -\hat{S}\\ \hat{I} & \hat{S}-1\end{bmatrix}$$

At the fixed point $(1, 0)$, the Jacobian Matrix simplifies to:

$$J_{(1,0)} = \begin{bmatrix}0 & -1\\ 0 & 0\end{bmatrix}$$

The eigenvalues of this matrix are $\lambda_1, \lambda_2 = 0$. This aligns with the second case provided in the clarifications post by the professor, wherein both eigenvalues are 0. This is a degenerate case for the fixed point $(1, 0)$. Let's move on to the second fixed point, $(0, 0)$. For this fixed point, the Jacobian matrix is:

$$J_{(0,0)} = \begin{bmatrix}0 & 0\\ 0 & -1\end{bmatrix}$$

Here, the eigenvalues are $\lambda_1 = 0, \lambda_2 = -1$. This corresponds with the second case, degenerate case. However, $\lambda_2 < 0$, which means that this fixed point exhibits 'stable-adjacent' behavior. For the fixed point $(0, 0)$, with eigenvalues $\lambda_1 = 0$ and $\lambda_2 = -1$, and the corresponding eigenvectors $[1, 0]$ and $[0, 1]$ (respectively), we can classify its stability as follows:

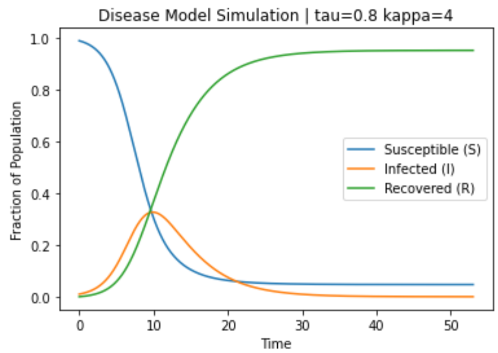
- The eigenvalue $\lambda_1 = 0$ with eigenvector $[1, 0]$ suggests that in the direction along the $\hat{S}$-axis, the system's behavior is indeterminate from the linear analysis. The system may either remain constant or change, but further analysis would be required to ascertain this.
- The eigenvalue $\lambda_2 = -1$ with eigenvector $[0, 1]$ indicates that in the direction along the $\hat{I}$-axis, any small perturbations will decay back towards the fixed point, implying stability in this direction.

Given this analysis, the fixed point $(0, 0)$ exhibits a type of "saddle" stability, where it's stable in one direction (along the $\hat{I}$-axis due to the negative eigenvalue) and neutral or indeterminate in the other direction (along the $\hat{S}$-axis due to the zero eigenvalue). This means that while the system will resist changes in the infected population, it's indifferent to changes in the susceptible population along the $\hat{S}$-axis. This behavior is characteristic of saddle points in two-dimensional systems, where one direction is stable and the other is not. The presence of a zero eigenvalue with a corresponding eigenvector that does not indicate movement (since it's $[1, 0]$, implying no change in $\hat{I}$) could also mean that there might be a whole line of fixed points along the $\hat{S}$-axis, or that the system could be influenced by higher-order terms not captured by the linear approximation at this fixed point.

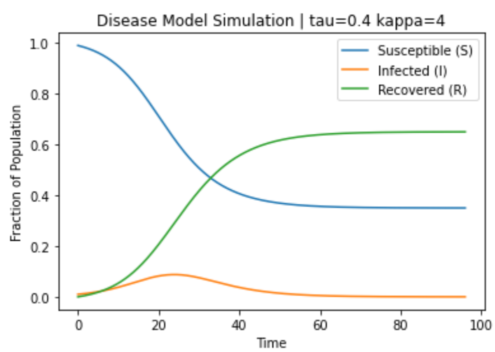
3 Question 1.3

3.1 Simulation Figures

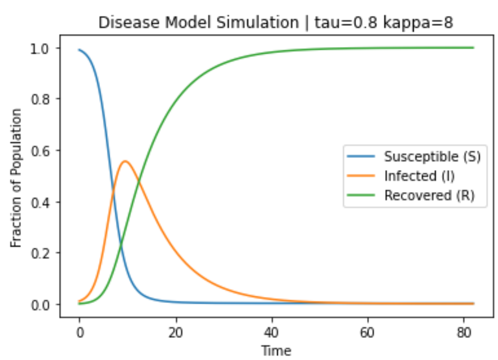
With $\tau = 0.8$ and $\kappa = 4$, the simulation was modeled with the following graph. In this simulation, the stopping point where $I(t) < 10e^{-4}$ was $t = 51.48$.



With $\tau = 0.4$ and $\kappa = 4$, the simulation was modeled with the following graph. In this simulation, the stopping point where $I(t) < 10e^{-4}$ was $t = 95.69$.



With $\tau = 0.8$ and $\kappa = 8$, the simulation was modeled with the following graph. In this simulation, the stopping point where $I(t) < 10e^{-4}$ was $t = 81.95$.



3.2 Analysis of Figures

Analyzing the simulation figures provided, we observe the dynamics of an SIR model under different conditions of the rate of infection (τ) and rate of recovery (κ):

1. $\tau = 0.8, \kappa = 4$: The peak of the infected curve ($I(t)$) is relatively low and occurs early in the simulation. The disease spreads moderately but is quickly brought under control due to a moderate recovery rate.

2. $\tau = 0.4, \kappa = 4$: The peak is much flatter, suggesting a slower spread of infection and a more extended period before the disease begins to decline, likely due to the lower infection rate.

3. $\tau = 0.8, \kappa = 8$: The peak of $I(t)$ is higher and occurs later than in the first case. This indicates a faster spread and a longer-lasting outbreak due to the slower recovery rate (longer infectious period).

To summarize the analysis of the figures for a public policy expert, or someone who does not have a computing background:

- Infection Rate Impact: The figures show that a higher infection rate leads to a steeper rise in cases, indicating a more aggressive outbreak. For policy, this suggests the need for rapid and robust intervention measures such as social distancing, mask mandates, and possibly lockdowns to flatten the curve and prevent healthcare system overload.

- Recovery Rate Impact: A lower recovery rate results in a prolonged outbreak with a higher peak, necessitating sustained healthcare resources. Policies might include increasing hospital capacity, ensuring adequate supplies for long-term patient care, and vaccination strategies to prevent healthcare system saturation.

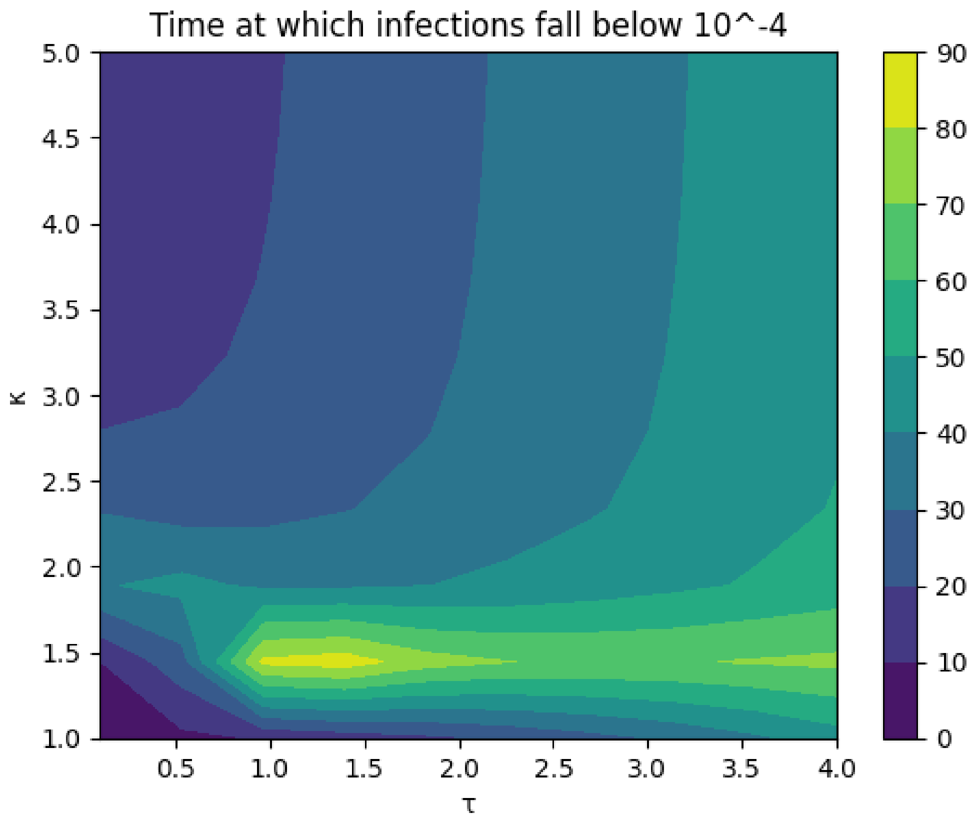
- Stopping Time Differences: The time it takes for the infection to reduce to negligible levels varies significantly with these parameters (infection and recovery rates). This affects how long interventions should be kept in place. Short-term measures might suffice for high recovery rates, while long-term strategies are crucial for slower recoveries.

- Public Health Policy: Vaccination campaigns should be prioritized to alter both infection and recovery rates positively. Reducing infection rates through vaccines would decrease transmission, and improving recovery rates by reducing the time individuals are infectious through effective treatment and care management can significantly alter the outbreak dynamics, as seen in the simulations.

As Software Engineers simulating health models, it's essential to communicate to public policy and health experts that these simulations show how changing the speed of disease spread and recovery can drastically change the outbreak's course. This highlights the importance of quick, decisive actions and the value of public health interventions in controlling an epidemic.

4 Question 1.4

4.1 Heatmap of Infections Based on τ, κ



4.2 Analysis

We can observe that when τ is between $[1.0, 1.5]$ and $\kappa = 1.5$, it takes the longest time for the infections to fall below 10^{-4}, or essentially for the infection to stop spreading. This is interesting because faster rates of spread (aka a greater τ) are not necessarily correlated to to a higher time for the disease to pass. Also, we found it interesting that having a super low rate of recovery ($\kappa \approx 1.0$) was not where the longest infection run time was.

5 Question 1.5

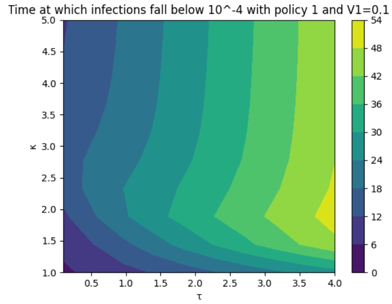
The two vaccination policies try to find different ways to allocated the limited vaccine resources in two different ways.

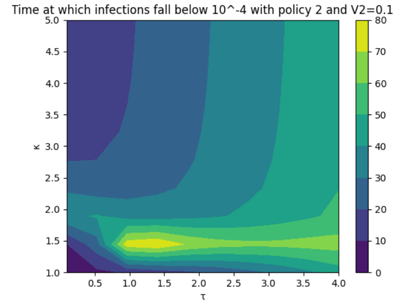
1. Policy 1 allocates vaccines proportional to the current number of susceptible individuals, S. It's a straightforward policy that aims to vaccinate as many susceptible individuals as possible based on the limited resources. This policy would directly reduce the susceptible portion of the population at a rate that is proportional to its size.

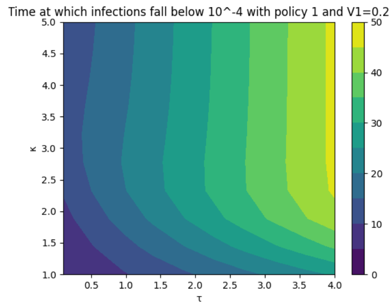
2. Policy 2 is more strategic and aims to vaccinate susceptible individuals based on the fraction of interactions between susceptible and infected individuals. The $\frac{SI}{S+I}$ term in this policy is based on the concept that the rate of contact between susceptible and infected individuals will be the driver for the spread of the disease itself. This expression doesn't give a probability but is proportional to the chance of contact between susceptible and infected individuals. The product of susceptible and infected individuals gives a raw measure of the potential interactions between these two population groups, and their sum provides a simple measure of the total number of individuals in the two groups that could potentially interact. Their ratio then normalizes the raw potential for interaction by the size of the population that could have such interactions. Thus, it assumes that the more infected individuals there are, and the more susceptible individuals there are, the higher chance that any one susceptible individual will come into contact with an infected individual. This ratio then helps scale the vaccination resources to focus on the part of the population where the interaction, and also the risk, is highest. This can be more efficient in preventing new infections from spreading, as it targets the vaccination effort at the interface between the susceptible and infected populations, possibly reducing the spread of the infection more effectively.

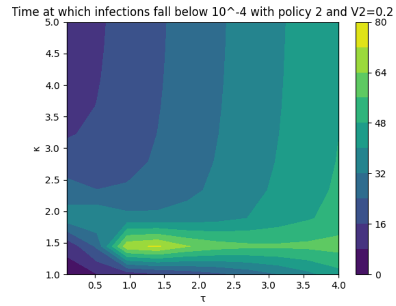
Policy 1 is far simpler and would be much easier to implement, while Policy 2 is more nuanced, and provides a method to interrupt transmission of the disease by focusing on those who are at the highest risk of contracting the disease due to contact with an infected individual.

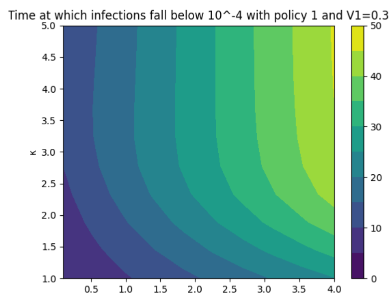
5.1 Policy 1 vs Policy 2 Heatmaps ($v_1, v_2 = 0.1$ to 0.5)

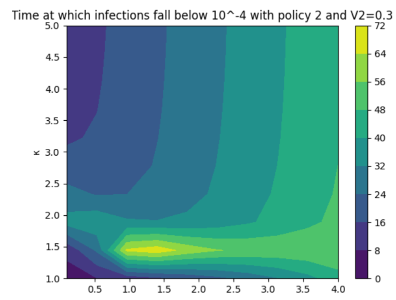


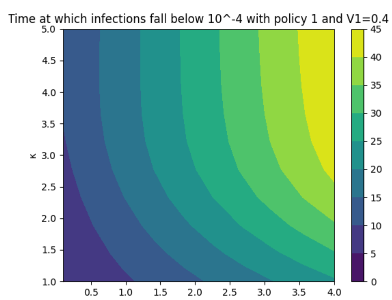


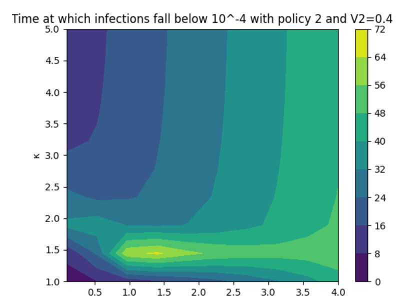


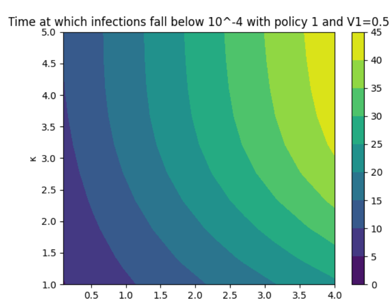


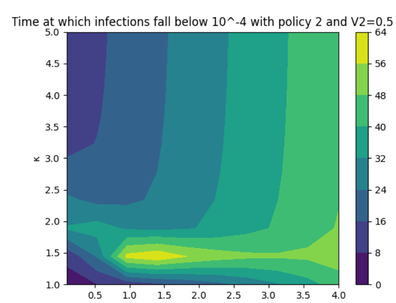




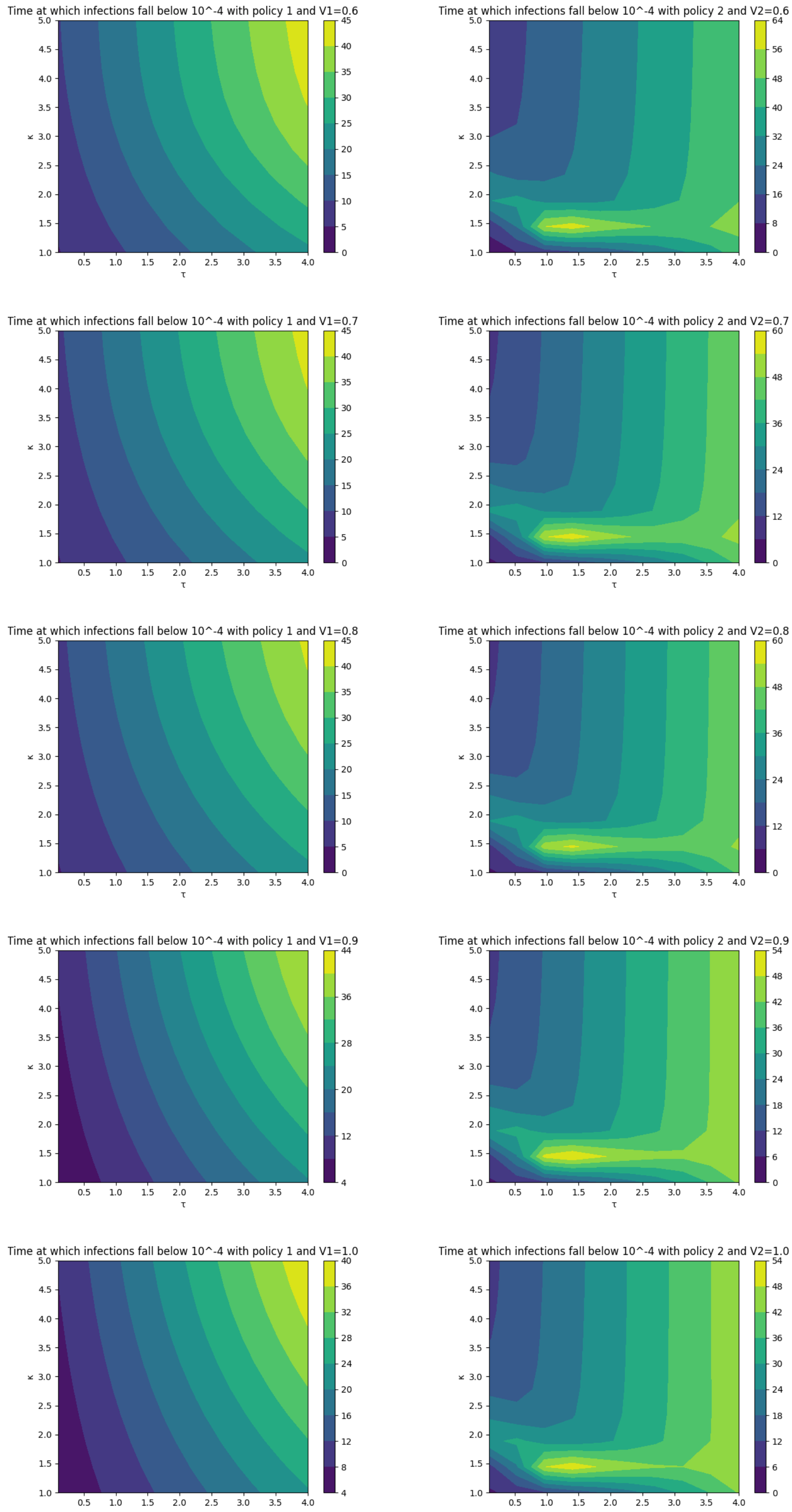








5.2 Policy 1 vs Policy 2 Heatmaps ($v_1, v_2 = 0.6$ to 1.0)



5.3 Analysis

Based on the comparisons between the graphs shown in 5.1 and 5.2, we can see that there are certain trade-offs that may cause one policy to be better than the other. One interesting thing we noticed is that policy 2 is able to better account for the rate of recovery (κ), not really increasing the time to reach steady state as the rate of recovery increases.

If we are considering total number of people vaccinated, then Policy 1 would be greater because it vaccinates a percentage of the susceptible population. On the contrary, Policy 2 targets individuals with a high likelihood of spreading the infection, so its focused efforts may still yield similar results in terms of the overall time to reach a steady state. If we are considering number of infected people, then Policy 2 is more effective because it targets both susceptible and infected people. Finally, if we are considering the time to reach steady state, then Policy 1 would lead to a faster reduction in the susceptible population if v_1 is high. However, this is not really realistic because vaccinations are expensive, so Policy 2 would be better overall.