

# Analyzing the Control Measures for the Homeless Population and Quantifying their Impact on the COVID-19 Epidemic in New York City

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## Abstract

An outbreak of Coronavirus Disease 2019 (COVID-19) emerged in Wuhan, China in late December. Within four months, the World Health Organization (WHO) characterized the disease as a pandemic. As of July 10, 2020, the United States has reported more confirmed cases than any other country, and the count continues to increase. The state of New York has been the hardest hit with 404,000 confirmed cases, 223,000 of which are attributed to New York City (NYC) alone. Given that homelessness in NYC has reached its highest level since the Great Depression and many of the precautionary measures for preventing COVID-19 are not an option for the homeless, a major concern is the contribution of this group to the prolongation of the epidemic. In order to analyze the role of the homeless population in NYC's COVID-19 dynamics, we use a SEIR-like, two-group epidemic model, for the homeless and housed populations under preferential mixing. Parameter values and initial states, whenever possible, were defined using publicly available data about COVID-19 and homelessness in NYC. Our findings indicate that preferential mixing and compliance with precautionary measures have a significant influence on the final epidemic size for NYC. We use these results to discuss the importance of the homeless population when considering the wellness of the city.

**Keywords:** COVID-19; homelessness; reproduction number; control measures; New York City; epidemiology;

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# 1 Introduction

As of June 30, 2020, there were 10,185,374 globally confirmed cases of Coronavirus Disease 2019 (COVID-19) and among those 503,863 deaths. Currently the regions with the most reported cases include the North America, Europe, Eastern Mediterranean, and South-East Asia [8]. In the United States (U.S.) alone there were 2,537,636 confirmed cases with 126,203 deaths, the highest number recorded out of any country so far. Coronavirus is a single-stranded RNA belonging to the family coronaviridae. All human found variations of the virus have been shown to originate from animals. Similar to cases of Middle East Respiratory syndrome (MERS) and Severe Acute Respiratory Syndrome (SARS), it was discovered in Wuhan, China with patients showing symptoms of the virus ranging from mild flu like symptoms to severe symptoms such as pneumonia [3]. After 41 patients tested positive for this new form of the virus, it was named severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2).

Known to originate from bats, there is little known about the mediator responsible for transmitting the virus to humans. Although similar forms have been seen in the past, what makes this virus novel is that it originated in animals and has now been seen to be transmitted from human to human. Since our immune system is yet to develop immunity to this virus, we lack the necessary response system to fight it off [9]. This may be especially harmful for people with compromised immune systems, such as those taking medications or suffering from health conditions [9].

The virus is known to spread through droplets when an individual carrying the virus talks, sneezes, or coughs. It can also be transmitted through surfaces, this occurs when a non-carrier of the virus touches a surface and then touches an opening of their body such as their mouth, nose, or eyes [8]. Due to its novelty, there are currently no approved vaccinations available to the public. In the absence of vaccination, CDC suggests several control measures, few of which include social distancing, wearing cloth covering when in public, and hand washing.

A population that cannot consistently follow the recommended CDC guidelines is the homeless population. Furthermore, they are among those with underlying health conditions and most vulnerable for the disease. In 2018, 552,830 people in the U.S. were homeless on a given night with NYC accounting for a little over 14% of that population [1]. NYC alone accounts for 85% of the homeless population in New York State with the number of homeless people expected to grow [1]. Despite its high homeless population, it has the lowest unsheltered population with only 5% accounting for unsheltered homeless [1]. However, among those that are sheltered, one of the main concerns is the tight and closely confined spaces within shelters which make social distancing difficult. Adding to these conditions is the lack of sanitary bathrooms, dining areas, and bedrooms [6].

In 2010 homeless people with underlying health conditions accounted for 552,000 of emergency department visits in NYC. This is mainly due to the fact that homeless people are generally more vulnerable to disease. They suffer higher levels of physical illnesses such as cardiovascular disease, mental illness, hypertension and infectious diseases such as tuberculosis [3]. Since the need for clinical care is secondary to the need for shelter and food,

they often delay seeking medical attention until their conditions have become severe. Lack of access to appropriate medical care increase the frequency of medical care visits.

When considering frequent users of the healthcare system, reports have shown that the homeless population has a rate of hospitalization and use of emergency departments four and three times higher, respectively, in comparison to the U.S average [4]. Compared to visits by members of low income households, these visits incur an additional cost to the state of \$ 2,400 per hospital visit. In addition to hospital costs, the current spending on homeless shelters for the population is \$ 2.04 billion. This comes from a total of \$ 3.08 billion of the total projected spending on homelessness [4].

This study centers on the COVID-19 epidemic with a focus on the homeless population in NYC. The goal of this study is to quantify the homeless subpopulation’s ability to affect the dynamics of the virus. This particular group is of interest mainly due to the disproportionate impact of the epidemic on the homeless population. With 63,839 people sleeping in shelters every night, in May 2020, there were 926 confirmed cases of COVID-19 with 179 shelters affected [6]. The lack of sanitation equipment such as soap, hand sanitizer, and face masks made available to the population can intensify the vulnerability of the homeless population to the disease. Overall, because the homeless population does not have the means to follow the recommended precautionary measures for the epidemic, they are more likely to acquire and spread the virus.

## 2 Methods

### 2.1 Model Formulation

To better understand the dynamics between the homeless and housed population we use a two group Susceptible-Exposed-Asymptomatic-Infective-Removed (SEAIR) compartmental model. One population is the homeless population, while the other is composed of the housed population. Our Susceptible class (S) is comprised of individuals who have not yet been exposed to the disease. Exposure happens at rate  $\beta_i$ , and moves group  $i$ ’s individuals into the incubation stage after first encountering the virus. This represents their transition into the Exposed class (E), where they have the virus but are not yet infectious. They transition from this class with a rate,  $\phi$ . When exiting the exposed class, people become infectious. These people will either exhibit symptoms and enter the symptomatic Infected class (I), or they will not develop symptoms and instead enter the Asymptomatic infected class (A). We quantify this distinction by a term,  $\sigma$ , which is the proportion of individuals who do not develop symptoms.

Given the short time span in which COVID-19 spreads, taking only a few months to reach global pandemic levels, our model assumes a constant total population and labels the final class as Removed (R). All individuals who get exposed eventually reach the Removed class after having recovered or died of the disease. The recovery rate is  $\gamma$  and the disease-related mortality for a member of group  $i$  is  $\mu_i$ . Note that, due to the lack of symptom in the asymptomatic group, the disease does not typically affect asymptomatic individuals severely and so the rate of death for asymptomatic individuals is assumed to be negligible.

Common culture in NYC dictates the assumption that our groups exhibit preferential mixing; homeless people primarily interact within their own group, and housed individuals primarily mix among themselves. This is incorporated by including the  $p_{ij}$  terms, where  $i = 1, 2$  and  $j = 1, 2$ . Each one of these terms is a numerical account of the proportion of individuals from the  $i$  group which mixes with the  $j$  group. Note that the cross terms are marking the same event. That is,  $p_{12}$  denotes mixing of group 1 with group 2, and  $p_{21}$  denotes mixing of group 2 with group 1, which is the same event.

## 2.2 COVID-19 Epidemic Model

We begin by establishing that  $N_i$  is the total population for each group, where  $i = 1$  indicates the homeless group and  $i = 2$  refers to the housed group. The susceptible class is denoted by  $S_i$ , the exposed class by  $E_i$ , the symptomatic infected class by  $I_i$ , the asymptomatic infected by  $A_i$ , and recovered class by  $R_i$ .

The population that is infective will take the form  $\frac{I+A}{N}$ . That is, effective contact can occur with either an asymptomatic infective individual or with a symptomatic infective individual. One step further, we will find that in our model, these infectives can come from either the housed or homeless population, and in order to capture these dynamics we make use of preferential mixing terms which will be discussed in detail.

By considering the general impact the control measures such as mask-wearing, maintaining at least 6 feet of distance from others, and frequent thorough hand washing, might have, we include a term,  $(1 - \alpha_i)$ . The parameter  $\alpha_i$  measures the compliance of the control measures. We let  $0 \leq \alpha_i \leq 1$ , where  $\alpha_i = 0$  denotes no compliance and  $\alpha_i = 1$  denotes complete, perfect compliance with the control measures. We specify that  $\alpha_i$  values are group-specific because each population is a different practical ability to comply with suggested precautions. Notably, at  $\alpha_1 = \alpha_2 = 0$  we have no control measure being employed and get the control-free version of our model. On the other hand if  $\alpha_1 = \alpha_2 = 1$ , there is no possibility of new infections and the effective rate of exposure drops to 0.

A fuller description of  $\beta_i$  is given by investigating the standard incidence model of transmission. In this model,  $\beta_i = pc_i$ , where  $p$  is the probability of transmission given an effective contact and  $c_i$  is the rate of effective contacts for an individual in group  $i$ . We consider that the rate of effective contact is the only difference between contact with an asymptomatic person and contact with a symptomatic person[7]. To account for this, we establish the parameter  $\delta \geq 0$  which will scale our singular  $\beta_i$  value to the appropriate size for transmission from an asymptomatic person belonging to group  $i$ .

The mathematical expressions capturing the previously described dynamics are the following

$$\begin{aligned}
\dot{S}_1 &= -S_1\beta_1(1 - \alpha_1) \left( p_{11} \frac{\delta A_1 + I_1}{N_1} + p_{12} \frac{\delta A_2 + I_2}{N_2} \right), \\
\dot{E}_1 &= S_1\beta_1(1 - \alpha_1) \left( p_{11} \frac{\delta A_1 + I_1}{N_1} + p_{12} \frac{\delta A_2 + I_2}{N_2} \right) - \phi E_1, \\
\dot{A}_1 &= \sigma \phi E_1 - \gamma A_1, \\
\dot{I}_1 &= (1 - \sigma) \phi E_1 - (\mu_1 + \gamma) I_1, \\
\dot{R}_1 &= \gamma A_1 + (\mu_1 + \gamma) I_1, \\
\dot{S}_2 &= -S_2\beta_2(1 - \alpha_2) \left( p_{21} \frac{\delta A_1 + I_1}{N_1} + p_{22} \frac{\delta A_2 + I_2}{N_2} \right), \\
\dot{E}_2 &= S_2\beta_2(1 - \alpha_2) \left( p_{21} \frac{\delta A_1 + I_1}{N_1} + p_{22} \frac{\delta A_2 + I_2}{N_2} \right) - \phi E_2, \\
\dot{A}_2 &= \sigma \phi E_2 - \gamma A_2, \\
\dot{I}_2 &= (1 - \sigma) \phi E_2 - (\mu_2 + \gamma) I_2, \\
\dot{R}_2 &= \gamma A_2 + (\mu_2 + \gamma) I_2,
\end{aligned} \tag{1}$$

where  $N_i = S_i + E_i + I_i + A_i + R_i$  and  $i = \{1, 2\}$ .

We assumed preferential mixing among the populations and used the following formulas to implement this:

$$\begin{aligned}
p_{11} &= \pi_1 + (1 - \pi_1)p_1 \\
p_{12} &= (1 - \pi_1)p_2 \\
p_{21} &= (1 - \pi_2)p_1 \\
p_{22} &= \pi_2 + (1 - \pi_2)p_2
\end{aligned}$$

$$p_i = \frac{(1 - \pi_i)\beta_i N_i}{(1 - \pi_1)\beta_1 N_1 + (1 - \pi_2)\beta_2 N_2}$$

where  $p_{ij}$ , as seen in the table below, indicates the proportion of contacts that an individual in group  $i$  has with an individual in group  $j$ . Additionally,  $\pi_i$  is the proportion of each group which has an own-group mixing preference and will thus only mix randomly among their own group. The portion of each group that does not have a preference is assumed to mix proportionally among either group. The expression for  $p_i$  gives this proportion in terms of effective contacts. That is,  $p_i$  is the proportion of effective contacts that is attributable to contact with an infective member of group  $i$ .

Parameters	Descriptions	Units	Values
$\beta_1$	Rate of transmission for homeless	days <sup>-1</sup>	0.25
$\beta_2$	Rate of transmission for housed	days <sup>-1</sup>	0.19
$p_{ij}$	Proportion of individuals in group $i$ who contact an individual in group $j$	N/A	[0,1]
$\phi$	Rate of entering infectious class	days <sup>-1</sup>	0.1923
$\mu_1$	Rate of COVID-19 related death homeless	days <sup>-1</sup>	0.00321
$\mu_2$	Rate of COVID-19 related death for housed	days <sup>-1</sup>	0.0027
$\gamma$	Rate of recovery	days <sup>-1</sup>	0.0556
$\sigma$	Proportion of infections that are asymptomatic	N/A	0.325
$\delta$	Asymptomatic factor, accounts for relative infectivity of asymptomatic individuals	N/A	[0.1, 10]
$\alpha_i$	Compliance factor, proportion of individuals in group $i$ taking recommended precautions	N/A	[0,1]

Table 1: Parameter Definitions and Units for ODEs

### 3 Mathematical Analysis

#### 3.1 Derivation of $\mathcal{R}_c$ and $\mathcal{R}_0$

The control reproductive number,  $\mathcal{R}_c$ , is the average number of secondary infections caused by a single infective individual over the course of the infected period, in a susceptible population where disease control measures are being implemented.

To begin the calculation of  $\mathcal{R}_c$  we write the next generation matrix (NGM)[5]. The  $\mathcal{R}_c$  value for the system is the dominant eigenvalue of this matrix. We obtain a 6x6 matrix however, we can reduce this matrix. We notice that first and fourth rows of this matrix provide us some information. Furthermore, the only relevant columns are, correspondingly, the first and the fourth columns. This gives us our reduced matrix with the corresponding values of  $p_{12}$  and  $p_{21}$ .

$$FV_{reduced}^{-1} = \begin{bmatrix} \frac{S_1\beta_1\delta p_{11}\sigma(1-\alpha_1)}{N_1\gamma} - \frac{S_1\beta_1 p_{11}(1-\alpha_1)(\sigma-1)}{N_1(\gamma+\mu_1)} & \frac{S_1\beta_1\delta p_{21}\sigma(1-\alpha_1)(1-\pi_1)}{N_2\gamma} - \frac{S_1\beta_1 p_{21}(1-\alpha_1)(1-\pi_1)(\sigma-1)}{N_2(\gamma+\mu_2)} \\ \frac{S_2\beta_2\delta p_{11}\sigma(1-\alpha_2)(1-\pi_2)}{N_1\gamma} - \frac{S_2\beta_2 p_{11}(1-\alpha_2)(1-\pi_2)(\sigma-1)}{N_1(\gamma+\mu_1)} & \frac{S_2\beta_2\delta p_{22}\sigma(1-\alpha_2)}{N_2\gamma} - \frac{S_2\beta_2 p_{22}(1-\alpha_2)(\sigma-1)}{N_2(\gamma+\mu_2)} \end{bmatrix}$$

It is now possible to define new variables which simplify our matrix even further. In order to do this, we let

$$\begin{aligned}\Gamma_1 &= \frac{\delta\sigma}{\gamma} + \frac{1-\sigma}{\gamma+\mu_1} \\ \Gamma_2 &= \frac{\delta\sigma}{\gamma} + \frac{1-\sigma}{\gamma+\mu_2}\end{aligned}$$

then the matrix becomes,

$$FV_{reduced}^{-1} = \begin{bmatrix} \Gamma_1\beta_1p_{11}(1-\alpha_1) & \Gamma_2\beta_2p_1(1-\alpha_1)(1-\pi_1) \\ \Gamma_1\beta_1p_2(1-\alpha_2)(1-\pi_2) & \Gamma_2\beta_2p_{22}(1-\alpha_2) \end{bmatrix}$$

Thus,

$$R_c = \frac{\Gamma_1\beta_1p_{11}(1-\alpha_1) + \Gamma_2\beta_2p_{22}(1-\alpha_2) + \sqrt{(\Gamma_1\beta_1p_{11}(1-\alpha_1) - \Gamma_2\beta_2p_{22}(1-\alpha_2))^2 - 4(\Gamma_1\Gamma_2\beta_1\beta_2p_1p_2(1-\alpha_1)(1-\alpha_2)(1-\pi_1)(1-\pi_2))}}{2}$$

$R_0$  is given when  $\alpha_1 = \alpha_2 = 0$ , where the system contains no control measures. Hence,

$$R_0 = \frac{\beta_1\Gamma_1p_{11} + \beta_2\Gamma_2p_{22} + \sqrt{(\beta_1\Gamma_1p_{11} - \beta_2\Gamma_2p_{22})^2 + 4\beta_1\beta_2\Gamma_1\Gamma_2p_{12}p_{21}}}{2} \quad (2)$$

These expressions correspond to the average number of secondary infections an infected person could cause in an otherwise entirely susceptible population.  $R_c$  relates to the case that the population is employing control measures, and  $R_0$  corresponds to the instance in which there are no control measures.

### 3.2 Equilibrium Points

All parameters and state variables of the model are non-negative and  $\alpha_i \in [0, 1]$  for  $i = 1, 2$  in which case

$$-S_i\beta_i(1-\alpha_i) \left( p_{ii}\frac{\delta A_i + I_i}{N_i} + p_{ij}\frac{\delta A_j + I_j}{N_j} \right) \leq 0,$$

where  $j = 1, 2$  and  $i \neq j$ . In this case both susceptible classes are continuously decreasing functions of time. This observation leads to the expectation no endemic equilibrium will be observed. That is, with the assumption of no new inflows of susceptibles, the disease must die out. To see this point demonstrated in greater detail, refer to Sections A and D in the Appendix.

Given that  $\alpha_i$  is the compliance rate with precautionary measures for group  $i$ , it is reasonable to assume  $\alpha_i \neq 1$ . With that assumption, there are only two solutions to the equation

$$-S_i\beta_i(1-\alpha_i) \left( p_{ii}\frac{\delta A_i + I_i}{N_i} + p_{ij}\frac{\delta A_j + I_j}{N_j} \right) = 0. \quad (3)$$



Let  $Q_k^*$  denote the  $k$ -th equilibrium point, then  $Q_k^*$  is found by choosing one of the solutions of (3) and substituting into the rest of the system. Assuming  $S_1^* = 0$  and  $S_2^* = 0$  the equilibrium point is  $Q_1^* = (0, 0, 0, 0, 0, 0, 0, 0)$ . Furthermore, if  $S_1^* = 0$  and

$$p_{21} \frac{\delta A_1^* + I_1^*}{N_1} + p_{22} \frac{\delta A_2^* + I_2^*}{N_2} = 0,$$

the equilibrium point is  $Q_3^* = (0, 0, 0, 0, f_2 N_2, 0, 0, 0)$ , where  $f_i$  is a proportion ( $0 < f_i \leq 1$ ) of the  $i$ -th group. Supposing

$$p_{11} \frac{\delta A_1^* + I_1^*}{N_1} + p_{12} \frac{\delta A_2^* + I_2^*}{N_2} = 0$$

and  $S_2^* = 0$  yields the equilibrium point  $Q_2^* = (f_1 N_1, 0, 0, 0, 0, 0, 0, 0)$ . Lastly, making the assumptions

$$p_{21} \frac{\delta A_1^* + I_1^*}{N_1} + p_{22} \frac{\delta A_2^* + I_2^*}{N_2} = 0,$$

and

$$p_{11} \frac{\delta A_1^* + I_1^*}{N_1} + p_{12} \frac{\delta A_2^* + I_2^*}{N_2} = 0$$

gives the disease-free equilibrium  $Q_4^* = (f_1 N_1, 0, 0, 0, f_2 N_2, 0, 0, 0)$ . In summary, the four equilibria of the system are

$$\begin{aligned} Q_1^* &= (0, 0, 0, 0, 0, 0, 0, 0), \\ Q_2^* &= (f_1 N_1, 0, 0, 0, 0, 0, 0, 0), \\ Q_3^* &= (0, 0, 0, 0, f_2 N_2, 0, 0, 0), \\ Q_4^* &= (f_1 N_1, 0, 0, 0, f_2 N_2, 0, 0, 0). \end{aligned}$$

Notably, the infectious compartments are empty at all equilibrium points, as hypothesized.

The Jacobian that is given in Section E of the Appendix has zero as an eigenvalue with multiplicity two when evaluated at the equilibrium points  $Q_1^*$ ,  $Q_2^*$ , and  $Q_3^*$ . As for what this finding adds to understanding the model, it is first important to draw a distinction between a hyperbolic equilibrium and a non-hyperbolic equilibrium. If all eigenvalues of the Jacobian have a non-zero real part, then small perturbations do not change the qualitative behaviour of the system near the equilibria and it is said to be hyperbolic [10]. On the other

hand, when the Jacobian has a zero eigenvalue the system is not structurally stable at that point and the equilibria is called non-hyperbolic. Given a small perturbation, the equilibria could disappear, change stability, or split into several equilibria. Unlike the previous three, the equilibrium point  $Q_4^*$  is hyperbolic; therefore, small perturbations will not qualitatively change the phase plane at this point.

## 4 Results

### 4.1 Numerical Evaluation of $\mathcal{R}_0$

A closed-form expression (2) for the basic reproductive number of (1) was obtained in Section 3.1. With the basic reproductive number expressed in terms of the parameters of the model, it is now possible to observe the behavior of  $\mathcal{R}_0$  as parameter values change. In particular, the purpose of this study is to examine the impact of preferential mixing between the homeless and housed populations of New York City. Two assumptions being made are that the homeless population is a high-risk group and face difficulties when trying to comply with the recommended precautions (e.g. social distancing). Bearing that in mind, it is natural to question what happens to the disease dynamics when the two groups come into contact more frequently or if the homeless population complies with precautionary measures at a higher rate. Mathematically, these questions are addressed by changing the values of the parameters for preferential mixing ( $\pi_i$ ) and for compliance with the recommended precautionary measures ( $\alpha_i$ ). Although there are many different outcomes that could change and are worth taking note of, the most fundamental aspect of any epidemic model is the basic reproductive number. Therefore, the first simulation undertaken focuses on how the basic reproductive number changes as the preference for mixing within each group changes. Table 2 presents values for the basic reproductive number when 80 percent of the homeless group prefers to mix among themselves, and the preference for mixing within the housed group is 0, 50, and 99 percent. Similarly, Table 3 lists values of the basic reproductive number when 80 percent of the housed group prefers to mix among themselves, and the preference for mixing within the homeless group is 0, 50, and 99 percent.

$\pi_1$	$\pi_2$	$\mathcal{R}_0$
0.8	0	2.4528
0.8	0.5	2.4538
0.8	0.99	2.5444

Table 2: Values of  $\mathcal{R}_0$  when  $\pi_1$  is fixed.

$\pi_1$	$\pi_2$	$\mathcal{R}_0$
0	0.8	2.3012
0.5	0.8	2.3048
0.99	0.8	3.0126

Table 3: Values of  $\mathcal{R}_0$  when  $\pi_2$  is fixed.

When the proportion of the housed group with a preference for mixing among themselves is fixed at 0.8, Table 3 indicates the value of  $\mathcal{R}_0$  remains relatively consistent for small values of  $\pi_1$  but increases significantly when  $\pi_1$  becomes large. In other words, as the preference among the homeless group for contact within their group becomes large, the expected number of secondary cases produced by a single typical infection in a completely susceptible population increases. Figure 1 plots the basic reproductive number for all combinations of

preferences and shows that  $\mathcal{R}_0$  does not increase linearly with  $\pi_1$ . Rather, a threshold occurs at approximately  $\pi_1 = 0.8$ . That is, it is seen when the fraction of the homeless group that has a preference for mixing among themselves is less than 0.8,  $\mathcal{R}_0$  varies little as  $\pi_1$  changes. On the other hand, once this threshold is reached, increases in  $\mathcal{R}_0$  occur on a noticeable scale as  $\pi_1$  increases.

Unlike the preference parameter for the homeless, the results given in Table 2 and Figure 1 do not indicate that large fractions of the housed group having a preference for mixing among themselves has a negative affect on  $\mathcal{R}_0$ . As the value of  $\pi_2$  approaches the point at which the entire housed group has a preference for contact among themselves,  $\mathcal{R}_0$  increases slightly from 2.45 to 2.54. Figure 1 extends the results in Table 2 by showing that regardless of what fraction of the housed group prefers mixing with their own group, changes in  $\mathcal{R}_0$  do not occur on the same scale as when the preference parameter for the homeless changes. From an epidemiological perspective, this finding suggests the parameter  $\pi_2$  is not a substantial factor for preventing the spread of COVID-19 in NYC.

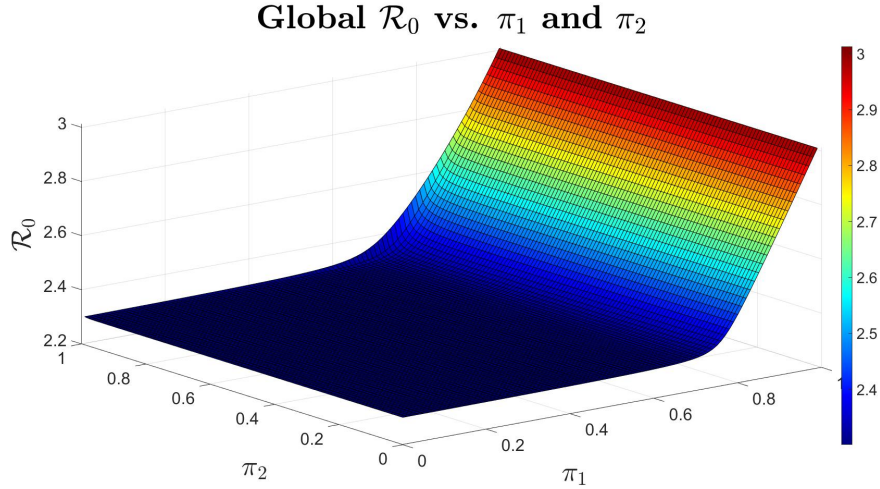


Figure 1: Changes in the reproductive number for different preferences.

Observing how the value of  $\mathcal{R}_0$  changes as the level of interaction between the homeless and housed group varies was the focal point of the simulation presented in Figure 1; yet, the issue of different rates of compliance with precautionary measures has been overlooked. Although the assumably different compliance rates presents a concern for ending the epidemic,  $\mathcal{R}_0$  is not the appropriate metric to use for exploring this idea. If the goal of the analysis is to determine how different compliance rates impact the epidemic, then changes in the control reproductive number ( $\mathcal{R}_c$ ) must be the focus of the analysis. More specifically, the analysis must determine for what values of the parameters, if any, that the control reproductive number is less than one. In this case the disease-free equilibrium is asymptotically stable. Rather than attempting to answer this question here, an alternative approach to exploring the potential consequences of low rates of compliance among the homeless and significantly higher compliance rates among the housed group is taken in later sections (4.3).

## 4.2 Peak Time

In epidemiology, "peak time" is the point at which the maximum number of infectious individuals is reached. For the system (1), there are two groups and time is being measured in days, so peak time refers to the day at which the maximum number of individuals in the homeless and housed groups are infectious. Much of the media attention surrounding the COVID-19 epidemic has emphasized "flattening the curve", so a potential concern if the homeless population is a high-risk group is that mixing could cause the curve to spike rather than flatten. The preciseness of this predicament is explored numerically by observing changes in the peak time and proportion of each group that is infectious as parameter values change. The first simulation attempts to quantify the effect of mixing between the homeless and housed groups by increasing the preference parameters simultaneously (i.e.  $\pi_1 = \pi_2$ ). When  $\pi_i$  ( $i = 1, 2$ ) takes on small values, the simulations indicate the dynamics when there is a substantial amount of mixing between the groups. For example,  $\pi_1 = \pi_2 = 0$  is the case of proportional mixing. At the other extreme, large values of  $\pi_1$  and  $\pi_2$  approach like-with-like mixing where the homeless only mix with others in the homeless group and vice versa. Results are displayed in Table 4 and Figure 2.

$\pi_1 = \pi_2$	Peak Time for $I_1$	Peak Time for $I_2$	$I_1$	$I_2$
0	11	107	0.00846	0.0156
0.1	13	106	0.0890	0.0157
0.2	15	106	0.0952	0.0159
0.3	22	105	0.1053	0.0160
0.4	32	104	0.1231	0.0161
0.5	35	103	0.1455	0.0160
0.6	36	103	0.1687	0.0157
0.7	36	103	0.1909	0.0154
0.8	35	104	0.2116	0.0150
0.9	34	106	0.2304	0.0145

Table 4: Peak times and proportion of each group that is infectious.

The results listed in Table 4 imply that increased levels of mixing within the groups is not ideal for the homeless. Namely, as the assumed values of the preference parameters tend towards both groups preferring to mix within their own group, the proportion of the homeless group that is infectious at the peak time increases monotonically. While the variation is not substantial, there is an initial increase in the fraction of infectious individuals in the housed group up until a peak is reached at  $\pi_1 = \pi_2 = 0.4$ . As for how to interpret this observation, first note it should be expected that large amounts of mixing with members of the same group is good for the housed group. An explanation for why the peak occurs at  $\pi_1 = \pi_2 = 0.4$  is not so clear-cut, but one possible explanation is there is just the right amount of mixing between groups for the housed to get the disease from the homeless and then infect others in their own group. For  $\pi_i > 0.4$  ( $i = 1, 2$ ), the proportion of the housed group that is infectious at the peak time is continuously decreasing. Although the numeric values are useful, the dynamics are more easily understood from Figure 2. For example, the case of proportional

mixing (i.e.  $\pi_1 = \pi_2 = 0$ ) results in a much flatter curve for the homeless group than when  $\pi_1 = \pi_2 = 0.9$ . Contrarily, the peak is minimized for the housed group when  $\pi_1 = \pi_2 = 0.9$ , or when a large fraction of each group has a preference for mixing with members in the same group.

It is clear from Figure 2 that mixing has more of an impact on the dynamics of COVID-19 for the homeless group, however, this does not necessarily mean proportional mixing is optimal for controlling the epidemic. When one takes into account the relative sizes of the homeless and housed groups in New York City, determining the ideal amount of mixing becomes more difficult. On the other hand, the ideal level of mixing for each group individually is straightforward. For the homeless, proportional mixing is optimal while like-with-like mixing is ideal for the housed group. That is, the homeless population of New York City is a high-risk group for COVID-19 and their interactions with the housed population can impact the overall dynamics of the disease within the city. The extent of this impact will be discussed further in Section 5.

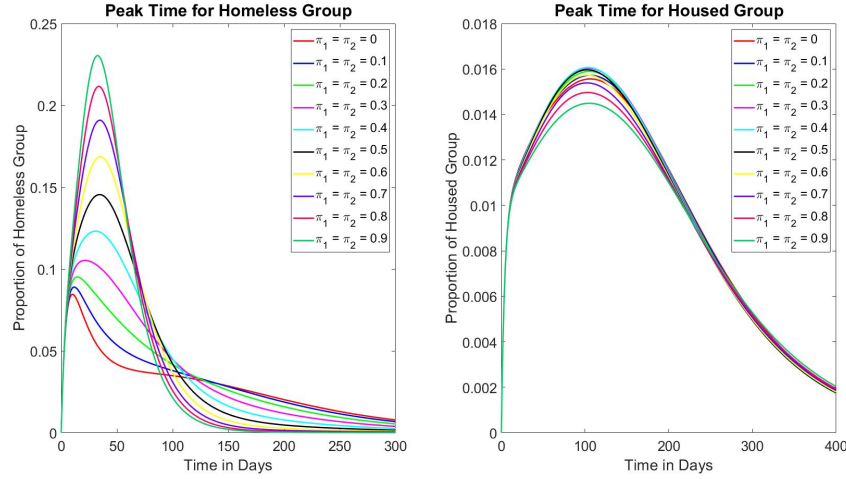


Figure 2: Peak time for different values of  $\pi_1 = \pi_2$ .

Regardless of what the model suggests is a desirable level of mixing, it is inevitable that the homeless and housed groups will come into contact with one another. Hence, an issue just as pressing as the impact of mixing is the affect of compliance with precautionary measures among the two groups on peak time and levels of infection. There is the potential that high rates of compliance among the housed group that would ordinarily flatten the curve are mitigated by low rates of compliance among the homeless group to the extent that a surge in the number of cases continues. To investigate this point mathematically, the ratio  $\frac{\alpha_2}{\alpha_1}$  is varied from 0.5 to 50 as peak time and the proportion of each group that is infectious is observed. Results are given in Table 5 and Figure 3.

$\frac{\alpha_2}{\alpha_1}$	Peak Time for $I_1$	Peak Time for $I_2$	$I_1$	$I_2$
0.50	10	105	0.0801	0.0141
0.56	11	105	0.0828	0.0142
0.63	12	105	0.0860	0.0143
0.71	13	106	0.0901	0.0145
0.83	15	106	0.0955	0.0146
1.00	21	106	0.1036	0.0148
1.25	30	106	0.1174	0.0150
1.67	34	106	0.1356	0.0151
2.50	36	105	0.1548	0.0152
5.00	36	104	0.1736	0.0153
50.00	36	103	0.1896	0.0154

Table 5: Peak time and the proportion of each group that is infectious for different values of  $\frac{\alpha_2}{\alpha_1}$ .

Note that for simulation results given in Table 5, the proportion of the housed group that is complying with precautionary measures is being held constant at  $\alpha_2 = 0.5$ . In other words, as the ratio  $\frac{\alpha_2}{\alpha_1}$  increases, the proportion of the homeless who are complying with precautionary measures is decreasing monotonically from 1 to 0.01. The constant rate of compliance being used is not intended to precisely reflect the compliance rate of the housed population in New York City; instead, the purpose of this simulation is to identify the impact on the disease dynamics when compliance among the homeless and housed groups take on significantly different values. That in mind, the value being assumed for  $\alpha_2$  is not of upmost importance.

As the ratio of compliance among the housed relative to the homeless group increases, the peak time increases monotonically for the homeless group and remains relatively the same for the housed group. Also, as this increase in the ratio of the compliance factors occurs, the proportion of both groups that are infectious at the peak point increases as well. Thus, unlike the prior simulation, a consensus can be reached on the optimal value of the ratio of compliance rates. If one of the goals for combating the COVID-19 epidemic is flattening the curve, Figure 3 suggests the housed group complying at half of the rate that the homeless are complying is the most ideal value that was tried in the simulation. Clearly, however, this is the result of perfect compliance among the homeless group and not an accurate indication of the optimal value for the ratio of compliance rates.

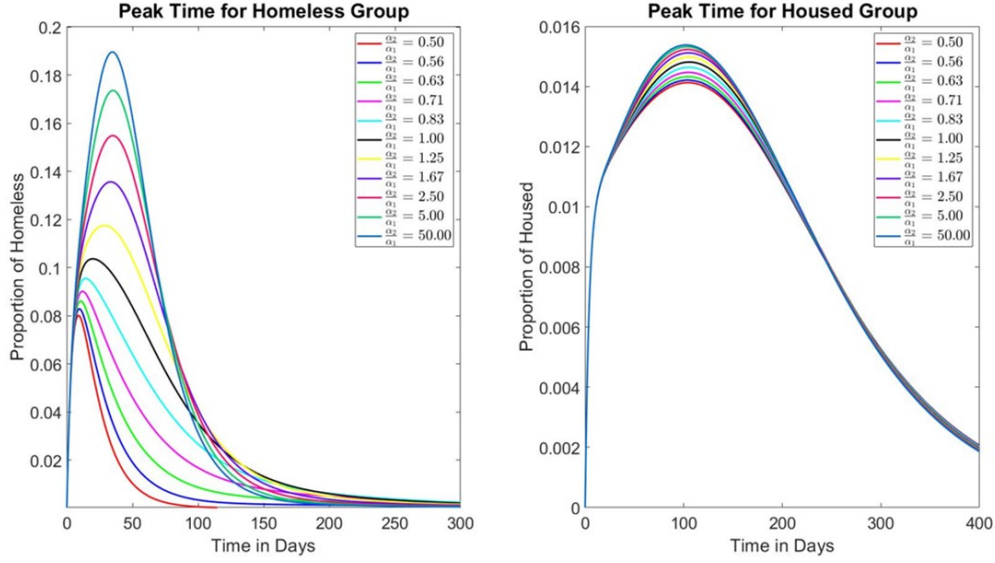


Figure 3: Peak time for different values of  $\frac{\alpha_2}{\alpha_1}$ .

### 4.3 FES

Another important aspect of any epidemic is the Final Epidemic Size (FES). Once the disease has died out, the FES is defined as the number of individuals in the population who are not susceptible. As with the previous metrics used for gauging the impact of an epidemic, mixing between the homeless and housed groups could cause the FES to be larger than it would be otherwise. Unlike the previous analyses, however, time is held constant in FES simulations. Specifically, a time value must be chosen that is large enough to ensure none of the effects of transient dynamics of the system are captured in the simulation.

Since the purpose of this study is to quantify the impact of control measures for the homeless group on COVID-19 dynamics in New York City, one matter of interest is whether changing control measures among the homeless can impact the FES for the city. It is assumed that the fraction of each group with a preference for mixing with others in their group along with differences in the compliance rates will affect the FES. Nonetheless, due to the relatively small size of the homeless group, it is not clear to what extent this assumption is true. Numerical simulations are one approach to quantifying the magnitude of the impact that different levels of mixing and compliance have on FES, but simulations do not illustrate what will happen under all possible circumstances. Compliance among the housed group could take on many values ( $\alpha_2 \in [0, 1]$ ), but only low ( $\alpha_2 = 0.5$ ), moderate ( $\alpha_2 = 0.8$ ), and high ( $\alpha_2 = 0.9$ ) compliance rates will be studied. Note that the definitions being used for low, moderate, and high compliance are not taken from the literature but are specific to this study. For every level of compliance among the housed group, three values for the compliance factor among the homeless are used such that the ratio of the two compliance rates are  $\frac{\alpha_2}{\alpha_1} = 4$ ,  $\frac{\alpha_2}{\alpha_1} = 2$ , and  $\frac{\alpha_2}{\alpha_1} = 1$ . In other words, the housed group complies at a rate 4 times greater, 2 times greater, and at the same rate as the homeless group. The preference each group has for mixing with others in the same group is assigned values by taking 50

equally spaced grid points in a unit square. Taking this approach indicates what happens to the FES for any amount of mixing.

## 4.4 Cases of Compliance

### 4.4.1 Case 1 ( $\alpha_2 = 0.5$ )

The first case is when there is a low rate of compliance among the housed group which is defined here to be when half of the group is complying with precautionary measures. Figures 4, 5, and 6 illustrate the case where the compliance rate among the homeless is 0.125, 0.25, and 0.5, respectively. These figures show that the FES for the homeless gets larger as the fraction of the homeless with a preference for mixing within their group increases. This result is expected since a higher rate of transmission is being assumed for the homeless group and they are complying with precautionary measures at a lower rate than the housed group. The housed group, on the other hand, benefits from a larger fraction of the homeless having a preference for mixing within their own group. That is, the FES for the housed group decreases as more of the homeless prefer to mix with others who are homeless.

If the ideal outcome is lowering the FES for the total population of New York City, there are two ways in which this is accomplished. Firstly, the most significant decrease in the total FES occurs when the fraction of the homeless with a preference for mixing with other homeless increases. To understand this result, it is important to recognize the relative sizes of the groups being considered. Namely, the homeless group makes up less than one percent of the total population of New York City. Thus, increased mixing among the homeless has a negative impact on the homeless FES but this is outweighed by the decrease in FES experienced by the housed group. The second way in which a lower total FES occurs is when the fraction of the housed group with a preference for mixing with others in their group gets large. This effect is most noticeable when very few of the homeless and almost all of the housed group have a preference for mixing within their group. Overall, the total FES is the lowest when contact between the homeless and housed groups is minimal.



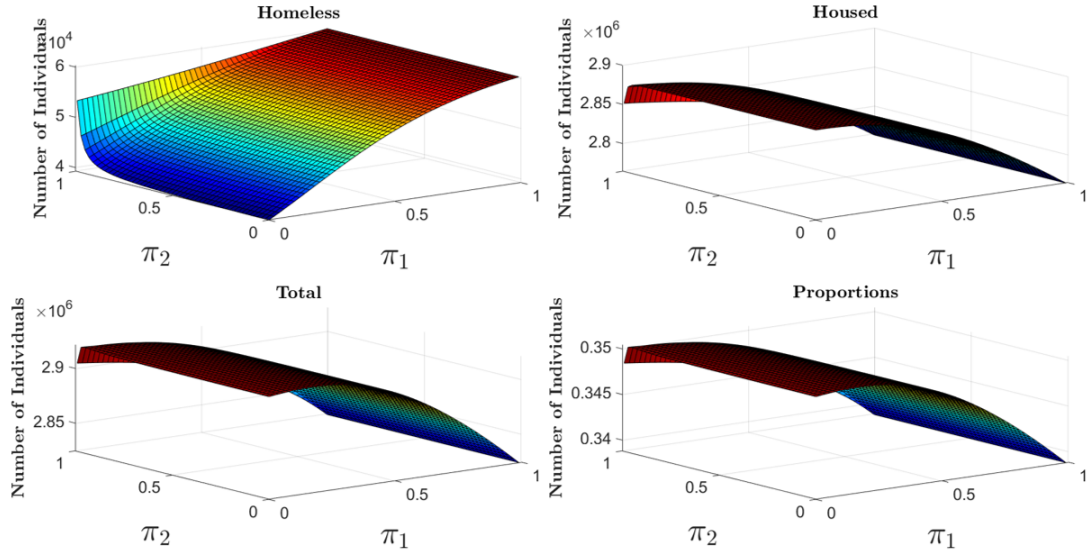


Figure 4:  $\frac{\alpha_2}{\alpha_1} = 4$

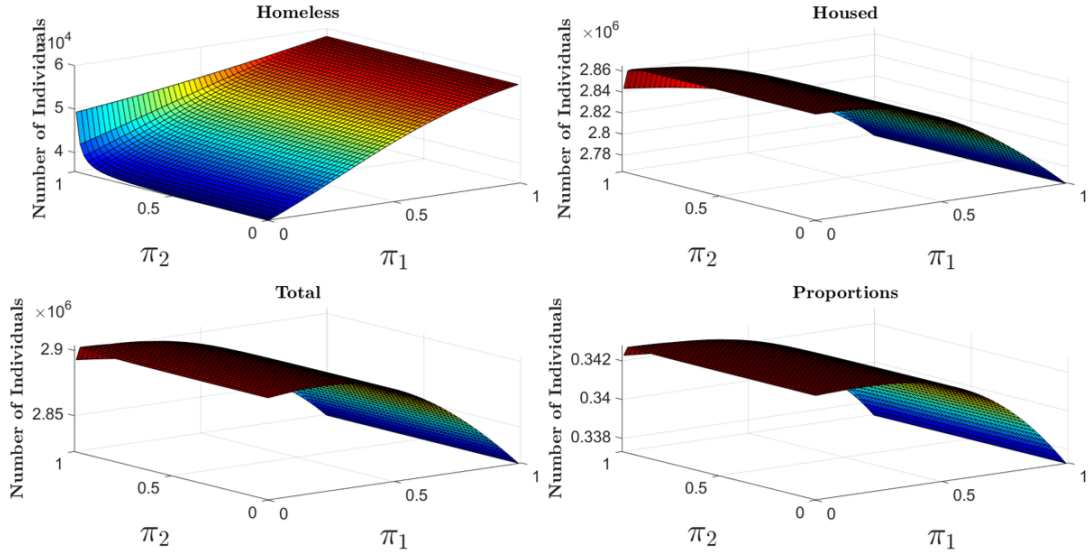


Figure 5:  $\frac{\alpha_2}{\alpha_1} = 2$

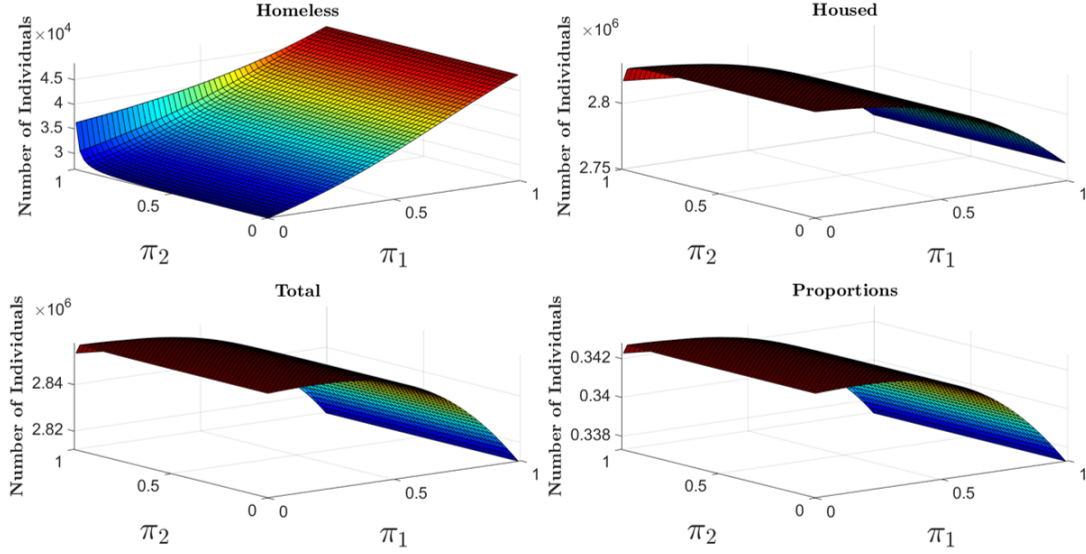


Figure 6:  $\frac{\alpha_2}{\alpha_1} = 1$

#### 4.4.2 Case 2 ( $\alpha_2 = 0.8$ )

Case 2 studies changes in the FES as a greater fraction of the housed group comply with the recommended precautionary measures. Specifically, an 80% compliance rate among the housed group is referred to as moderate in this study. Figures 7, 8, and 9 show how the FES changes for different values of the mixing parameters and when compliance among the homeless group is 0.2, 0.4, and 0.8, respectively. As was the case for low levels of compliance, the figures indicate negative consequences for the homeless group's FES as the fraction of the homeless that prefer mixing with other homeless increases. With lower rates of compliance and a higher rate of transmission being assumed for the homeless group, the result is again anticipated. Interestingly, there is a different relationship between the housed group and the optimal amount of mixing than was the case for low compliance. When there was low compliance among the housed group, the FES for the group decreased monotonically as more of the homeless group gained a preference for mixing with other homeless. For the case of moderate compliance among the housed group, both Figures 7 and 8 indicate a peak occurs for the housed group's FES. Initially, the FES increases as more of the homeless prefer mixing among themselves but then declines continuously after reaching the peak point at  $\pi_1 \approx 0.4$  for Figure 7 and  $\pi_1 \approx 0.5$  for Figure 8. When the compliance rate for the two groups are the same, the FES for the housed group again declines monotonically as more of the homeless prefer mixing among themselves.

A noticeable difference between this simulation and the previous is the magnitude of the FES. For example, a low value of the FES for the housed group is roughly 2,850,000 individuals in Figure 4 but drops to 320,000 for the case of moderate compliance displayed in Figure 7. Similarly, a low value of the FES for the homeless group is about 40,000 in Figure 4 but declines to 20,000 with moderate compliance in Figure 7. Clearly, this result is good for reducing the total FES for New York City, however, there is another implication of increased compliance. For the case of low compliance, the FES for the total population

strongly reflects the FES for the housed group. When the compliance rate of the housed group is increased, the total FES no longer decreases monotonically as more of the homeless prefer mixing within their own group. In Figures 7 and 8 both the housed and total FES initially increase, peak, and then decline as more of the homeless prefer to mix with other homeless. Since changes in the FES for the homeless is now comparable in scale to changes in the FES for the housed group, the homeless have a noticeable impact on total FES but this is moderated by the housed group. In other words, the total FES does not increase monotonically as more of the homeless prefer mixing among themselves but instead peaks and then declines due to the housed group.

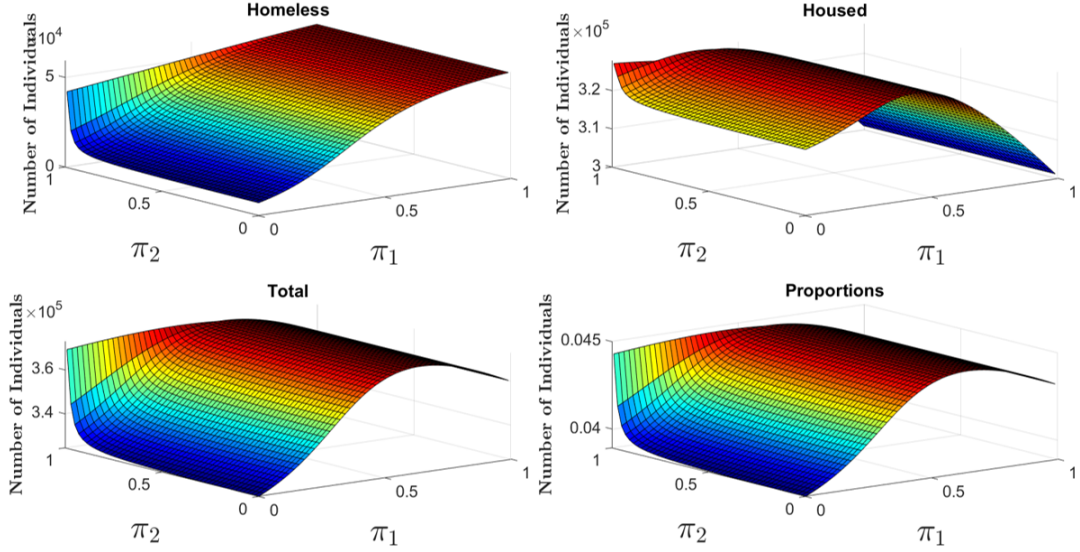


Figure 7:  $\frac{\alpha_2}{\alpha_1} = 4$

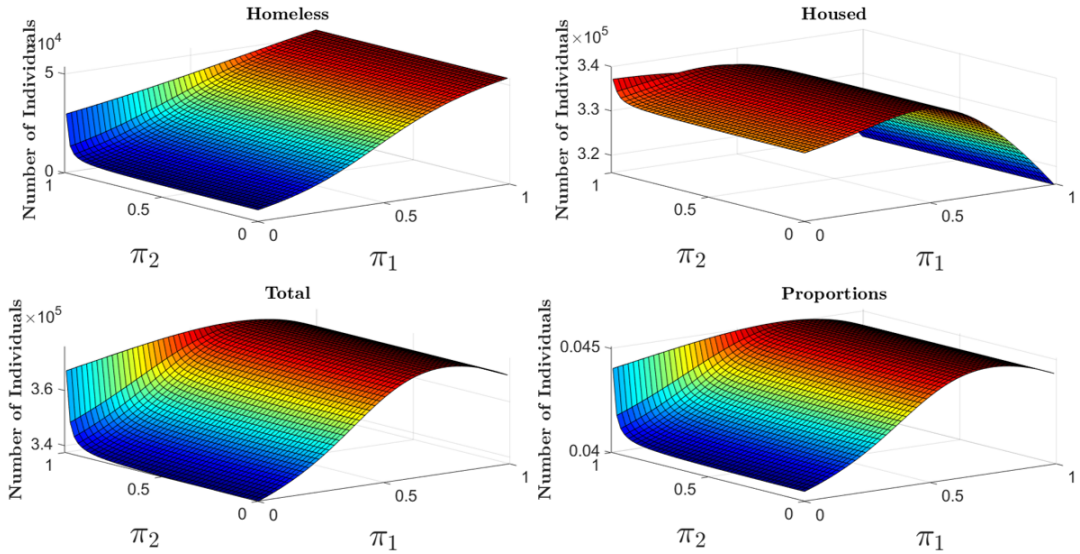


Figure 8:  $\frac{\alpha_2}{\alpha_1} = 2$

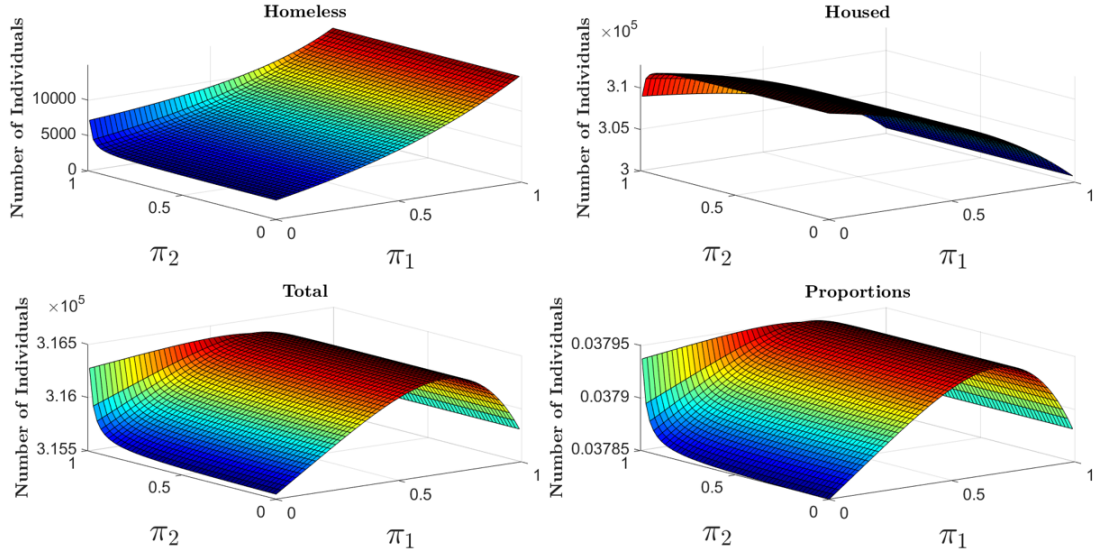


Figure 9:  $\frac{\alpha_2}{\alpha_1} = 1$

#### 4.4.3 Case 3 ( $\alpha_2 = 0.9$ )

The final case that is studied assumes that there is a 90% rate of compliance with precautionary measures among the housed group. To make the ratio of the compliance factors the desired values, compliance rates of 0.225, 0.45, and 0.9 are used for the homeless group. Like in previous cases, Figures 10, 11, and 12 show that increases in the fraction of the homeless group with a preference for mixing within their group has a negative consequence for the FES of the homeless group. Thus, all three simulations indicate large amounts of mixing within the homeless group is not ideal for controlling the COVID-19 epidemic among the homeless. As for members of the housed group, the figures suggest the FES is lowest when the majority of the homeless prefer mixing with themselves. While the ideal strategy for lessening the impact on each group individually is contradictory, it is noticeable that the FES for the housed group peaks when approximately half of the homeless group has a preference for mixing with other homeless. In other words, the FES for the housed group does not decrease monotonically with the preference parameter for the homeless.

If the population of New York City is considered as a whole, Figures 10, 11, and 12 suggest dynamics that are similar to the case of moderate compliance. Although the housed group makes up the majority of the population in New York City, only a minor proportion of this group becomes infectious when there is moderate or high compliance. Hence, unlike the case of low compliance, the FES for the total population of New York City does not primarily reflect only the FES for the housed group. Due to the FES for the homeless and housed groups now being similar in scale, total FES does not decrease as more of the homeless have a preference for mixing with other homeless. In fact, total FES now increases under those circumstances.

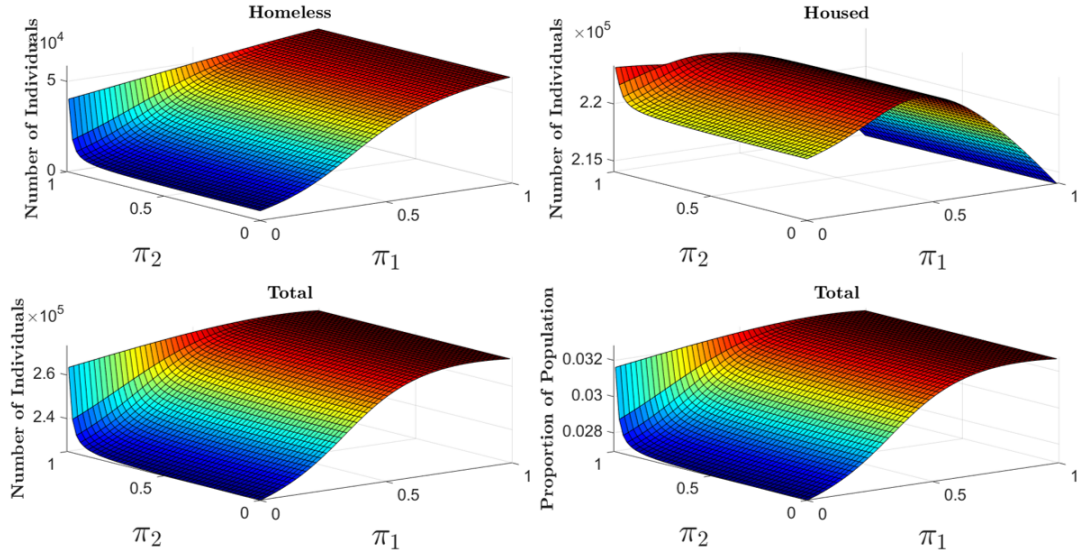


Figure 10:  $\frac{\alpha_2}{\alpha_1} = 4$

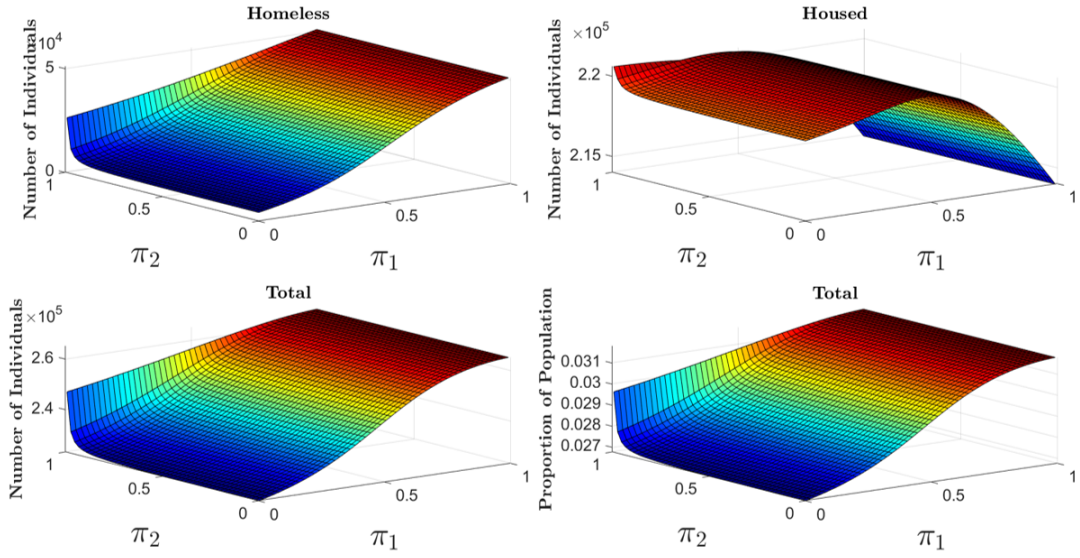


Figure 11:  $\frac{\alpha_2}{\alpha_1} = 2$



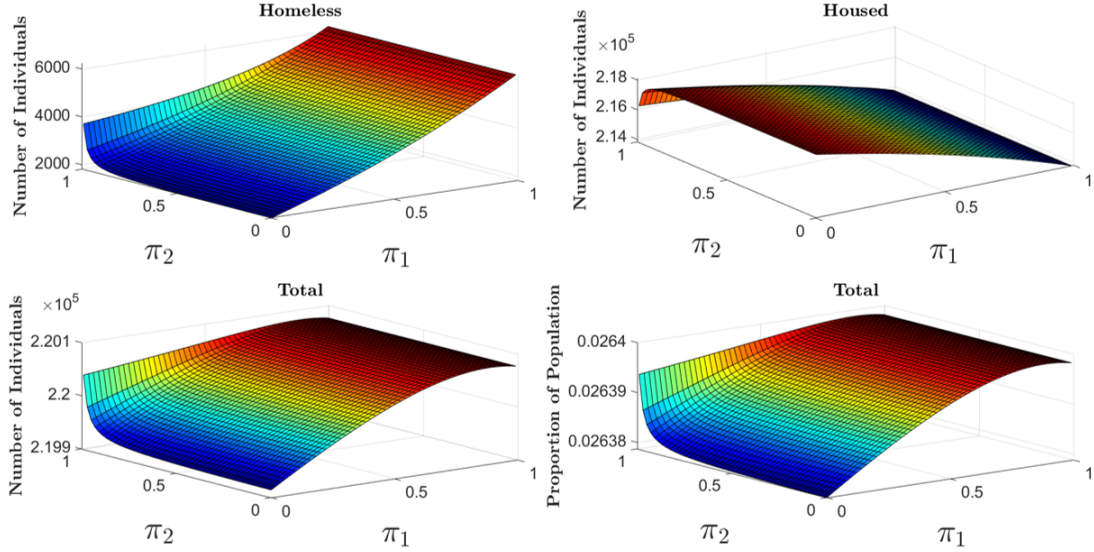


Figure 12:  $\frac{\alpha_2}{\alpha_1} = 1$

## 5 Conclusions and Discussion

This study sought to quantify the impact of preferential mixing between the homeless and housed groups of New York City on the dynamics of COVID-19. In Section (1), a ten-dimensional system of ODEs was derived based on the assumptions made for interactions between the homeless and housed groups. Specifically, the standard SEIR model was expanded to include an asymptomatic compartment intended to account for one of the unique aspects of COVID-19. A factor,  $\delta$ , was introduced to capture the assumed relative rate of infectivity of individuals in the asymptomatic compartment. Along with the assumption of preferential mixing and reduced infectivity of asymptomatic individuals, a distinguishing point of this model is the compliance factor ( $\alpha_i$ ). With  $(1 - \alpha_i)$  being the proportion of group  $i$  that is not complying with precautionary measures, this factor reduces the inferred rate of transmission,  $\beta_i$ .

To address our research question, a series of numerical simulations were undertaken. First, the behavior of the expected number of secondary cases produced by a typical single infection in a completely susceptible population for various mixing preferences was studied using the analytical expression for the global reproductive number obtained in Section 3.1. Second, simulations were run to evaluate the peak time and proportion of each group that was infectious at that point. In this second series of simulations, changes in the preference each group has for mixing among itself and the ratio of the compliance rates were considered. Third, three cases for compliance among the housed group were defined in order to study changes in the FES for varying degrees of compliance and mixing.

In the first set of simulations, we saw that when the number of homeless people with a preference for their own group increases (greater  $\pi_1$ ) there was an increase in the  $R_0$  value. On the other hand, large variations in the housed population's preference for mixing with their own group (greater  $\pi_2$ ) had a negligible effect on the global rate of infection, relative to

the homeless group. This is because the housed population follows the guidelines outlined by CDC for preventing the spread of COVID-19. So, even in the chance that they come into contact with each other, they have lowered the chances of passing the virus to other individuals. Therefore, as more non-infective people mix amongst each other this impacts the spread less. On the other hand, the homeless population continues to interact with each other while maintaining the conditions under which the virus could persist. If the number of homeless people with a preference for their own group increases, the virus spreads further.

Analysis of the peak time revealed that the greater value of the peak within the housed population depended on the compliance of the housed population. As a result, we see that when compliance is lower for the housed population, the peak time occurs at a later day with a higher proportion of the population, meaning the infection is spreading longer and to more individuals. Conversely, high compliance with control measures in the housed population results in a lower peak, meaning less spread of the infection.

In the case that there is more compliance with the housed population than the homeless population, we saw that increased mixing between the two groups would be beneficial for the homeless population. In the case when there is more compliance with the homeless than the housed population, we saw that mixing between groups would be beneficial for the housed population while being detrimental to the homeless population.

From this we see a relationship holding in the case of more compliance with the housed or homeless group. If one population group is more infective than the other due to a lack of control measures or otherwise, increased mixing between the groups would be beneficial for controlling the disease within the infective group. In the case of this study, because the size of the housed population is larger than the size of the homeless population, the homeless population is more likely to display impacts of the mixing. From this, we infer that the size of the population is also influential on the spread of COVID-19.

Since the housed population is larger than the homeless population we saw that large variations in the compliance of the housed population has a significant impact on the FES. Our model accounts for 3 million housed groups while the homeless group accounts for about 60,000 individuals. Overall, we determined that the FES is lowest when there is minimal mixing within their own group.

Furthermore, we assumed the housed population have greater compliance than the homeless group because they are equipped with the necessary tools to follow the guidelines to contain the epidemic. Therefore, the proportional FES of the housed population is lower than the proportional FES of the homeless population. FES of the homeless population would benefit from mixing with the housed population, while the housed population would be negatively impacted. If we consider the total population, increased mixing between the two groups is expected to lower the FES and would be the best choice if the general population is considered. However this is not the case for low compliance of the housed group, because FES would increase for the homeless group.

Although one theoretical solution is to increase mixing amongst both groups, this style of intervention is not likely to be implemented. Instead, one possible control measure could be providing housing and sanitary facilities to the homeless population, while maintaining

that the housed population continues to follow CDC guidelines for preventing the spread of COVID-19. If homes were provided to the homeless group, this would solve the issue of social distancing. Similar to the housed population, if the homeless were given the necessary equipment to take precautionary measures, they reduce the chances of getting infectious.

Two limitations of our study should be noted. Our first limitation is accurately determining the amount of mixing happening amongst both groups. Though we were not able to determine exact values associated with the mixing, we ran our simulation for various values of the mixing parameters to gain a sense of the effect that mixing has overall. The second limitation is in determining the level of compliance in both groups. Again, the ability to determine an exact parameter value associated with this aspect of the real world system alluded us, we were able to simulate the general effect that control measures would have on disease spread. Despite these limitations, we have successfully quantified the impact of the epidemic by using simulations and diagrams that would present results for the cases provided. We determined that although the housed population has a significant impact on the epidemic, the homeless group still has an influence on the course of the FES. To improve the public health of the general population, interventions which mitigate the epidemic in the homeless population should be implemented.



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## Appendix A Reducing the System

A ten-dimensional system of ODEs (1) was proposed for modeling the dynamics of COVID-19 among the homeless and housed populations of New York City. However, for the purpose of analysis, the system can be reduced to eight dimensions without any loss of information. To do this, we first notice  $R_1$  and  $R_2$  do not impact the dynamics of the system. Furthermore,

$$\dot{N}_1 = \dot{S}_1 + \dot{E}_1 + \dot{A}_1 + \dot{I}_1 + \dot{R}_1 = 0,$$

and

$$\dot{N}_2 = \dot{S}_2 + \dot{E}_2 + \dot{A}_2 + \dot{I}_2 + \dot{R}_2 = 0,$$

which shows  $N_1$ ,  $N_2$ , and  $N = N_1 + N_2$  are all constant with respect to time. Therefore, only the following eight ODEs must be considered in the analysis of system (1)

$$\begin{aligned} \dot{S}_1 &= -S_1\beta_1(1 - \alpha_1) \left( p_{11} \frac{\delta A_1 + I_1}{N_1} + p_{12} \frac{\delta A_2 + I_2}{N_2} \right), \\ \dot{E}_1 &= S_1\beta_1(1 - \alpha_1) \left( p_{11} \frac{\delta A_1 + I_1}{N_1} + p_{12} \frac{\delta A_2 + I_2}{N_2} \right) - \phi E_1, \\ \dot{A}_1 &= \sigma \phi E_1 - \gamma A_1, \\ \dot{I}_1 &= (1 - \sigma) \phi E_1 - (\mu_1 + \gamma) I_1, \\ \dot{S}_2 &= -S_2\beta_2(1 - \alpha_2) \left( p_{21} \frac{\delta A_1 + I_1}{N_1} + p_{22} \frac{\delta A_2 + I_2}{N_2} \right), \\ \dot{E}_2 &= S_2\beta_2(1 - \alpha_2) \left( p_{21} \frac{\delta A_1 + I_1}{N_1} + p_{22} \frac{\delta A_2 + I_2}{N_2} \right) - \phi E_2, \\ \dot{A}_2 &= \sigma \phi E_2 - \gamma A_2, \\ \dot{I}_2 &= (1 - \sigma) \phi E_2 - (\mu_2 + \gamma) I_2. \end{aligned} \tag{4}$$

## Appendix B NGM

$$FV^{-1} = \begin{bmatrix} \frac{S_1\beta_1\delta p_{11}\sigma(1-\alpha_1)}{N_1\gamma} - \frac{S_1\beta_1p_{11}(1-\alpha_1)(\sigma-1)}{N_1(\gamma+\mu_1)} & \frac{S_1\beta_1\delta p_{11}(1-\alpha_1)}{N_1\gamma} & \frac{S_1\beta_1p_{11}(1-\alpha_1)}{N_1(\gamma+\mu_1)} & \frac{S_1\beta_1p_{12}\sigma(1-\alpha_1)}{N_2\gamma} - \frac{S_1\beta_1p_{12}(1-\alpha_1)(\sigma-1)}{N_2(\gamma+\mu_2)} & \frac{S_1\beta_1\delta p_{12}(1-\alpha_1)}{N_2\gamma} & \frac{S_2\beta_2p_{12}(1-\alpha_1)}{N_2(\gamma+\mu_2)} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{S_2\beta_2\delta p_{21}(1-\alpha_2)}{N_1\gamma} - \frac{S_2\beta_2\delta p_{21}(1-\alpha_2)(\sigma-1)}{N_1(\gamma+\mu_1)} & \frac{S_2\beta_2\delta p_{21}(1-\alpha_2)}{N_1\gamma} & \frac{S_2\beta_2p_{21}(1-\alpha_2)}{N_1(\gamma+\mu_1)} & \frac{S_2\beta_2p_{22}\sigma(1-\alpha_2)}{N_2\gamma} - \frac{S_2\beta_2p_{22}(1-\alpha_2)(\sigma-1)}{N_2(\gamma+\mu_2)} & \frac{S_2\beta_2\delta p_{22}(1-\alpha_2)}{N_2\gamma} & \frac{S_2\beta_2p_{22}(1-\alpha_2)}{N_2(\gamma+\mu_2)} \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

## Appendix C NGM Reduction

Recalling that the cross-mixing must be reciprocal, that is, each contact between a member of group 1 with a member of group 2 is also an contact between a member of group 2 with a member of group 1, we can write mathematically that  $\frac{\beta_2 p_1}{N_1} = \frac{\beta_1 p_2}{N_2}$ . Thus we can also equate,  $\beta_1((1 - \pi_1)p_2)\frac{S_1}{N_2} = \beta_2((1 - \pi_1)p_1)\frac{S_1}{N_1}$ . Similarly, we also find it to be true that,  $\beta_2((1 - \pi_2)p_1)\frac{S_2}{N_1} = \beta_1((1 - \pi_2)p_2)\frac{S_2}{N_2}$ . And when these substitutions are made in our matrix, we get the following:

$$FV_{reduced}^{-1} = \begin{bmatrix} \frac{S_1 \beta_1 \delta p_{11} \sigma (1 - \alpha_1)}{N_1 \gamma} - \frac{S_1 \beta_1 p_{11} (1 - \alpha_1) (\sigma - 1)}{N_1 (\gamma + \mu_1)} & \frac{S_1 \beta_2 \delta p_1 \sigma (1 - \alpha_1) (1 - \pi_1)}{N_1 \gamma} - \frac{S_1 \beta_2 p_1 (1 - \alpha_1) (1 - \pi_1) (\sigma - 1)}{N_1 (\gamma + \mu_2)} \\ \frac{S_2 \beta_1 \delta p_2 \sigma (1 - \alpha_2) (1 - \pi_2)}{N_2 \gamma} - \frac{S_2 \beta_1 p_2 (1 - \alpha_2) (1 - \pi_2) (\sigma - 1)}{N_2 (\gamma + \mu_1)} & \frac{S_2 \beta_2 \delta p_{22} \sigma (1 - \alpha_2)}{N_2 \gamma} - \frac{S_2 \beta_2 p_{22} (1 - \alpha_2) (\sigma - 1)}{N_2 (\gamma + \mu_2)} \end{bmatrix}$$

Now, because we are interested in finding expressions for the  $R_c$  and  $R_0$  values of our system, we must approximate an entirely susceptible population. Hence we substitute in  $S_1 = N_1$  and  $S_2 = N_2$ ,

$$FV_{reduced}^{-1} = \begin{bmatrix} \frac{\beta_1 \delta p_{11} \sigma (1 - \alpha_1)}{\gamma} - \frac{\beta_1 p_{11} (1 - \alpha_1) (\sigma - 1)}{\gamma + \mu_1} & \frac{\beta_2 \delta p_1 \sigma (1 - \alpha_1) (1 - \pi_1)}{\gamma} - \frac{\beta_2 p_1 (1 - \alpha_1) (1 - \pi_1) (\sigma - 1)}{\gamma + \mu_2} \\ \frac{\beta_1 \delta p_2 \sigma (1 - \alpha_2) (1 - \pi_2)}{\gamma} - \frac{\beta_1 p_2 (1 - \alpha_2) (1 - \pi_2) (\sigma - 1)}{\gamma + \mu_1} & \frac{\beta_2 \delta p_{22} \sigma (1 - \alpha_2)}{\gamma} - \frac{\beta_2 p_{22} (1 - \alpha_2) (\sigma - 1)}{\gamma + \mu_2} \end{bmatrix}$$

It is now possible to define new variables which simplify our matrix even further. In order to do this, we let

$$\begin{aligned} \Gamma_1 &= \frac{\delta \sigma}{\gamma} + \frac{1 - \sigma}{\gamma + \mu_1} \\ \Gamma_2 &= \frac{\delta \sigma}{\gamma} + \frac{1 - \sigma}{\gamma + \mu_2} \end{aligned}$$

then the matrix becomes,

$$FV_{reduced}^{-1} = \begin{bmatrix} \Gamma_1 \beta_1 p_{11} (1 - \alpha_1) & \Gamma_2 \beta_2 p_1 (1 - \alpha_1) (1 - \pi_1) \\ \Gamma_1 \beta_1 p_2 (1 - \alpha_2) (1 - \pi_2) & \Gamma_2 \beta_2 p_{22} (1 - \alpha_2) \end{bmatrix}$$

Remembering that in order to find  $R_c$  we want to find the dominant eigenvalue of this matrix, we produce the following expression whose solutions correspond to the both the eigenvalues of this reduced matrix and the non-zero eigenvalues of our original next generation matrix.

$$\lambda_{\pm} = \frac{\Gamma_1 \beta_1 p_{11} (1 - \alpha_1) + \Gamma_2 \beta_2 p_{22} (1 - \alpha_2) \pm \sqrt{(\Gamma_1 \beta_1 p_{11} (1 - \alpha_1) + \Gamma_2 \beta_2 p_{22} (1 - \alpha_2))^2 - 4(Det)}}{2}$$

Where, for compactness, we use  $Det$  to mean the determinant of the reduced matrix. The expression for which is

$$Det = -\Gamma_1\Gamma_2\beta_1\beta_2p_1p_2(1-\alpha_1)(1-\alpha_2)(1-\pi_1)(1-\pi_2) + \Gamma_1\Gamma_2\beta_1\beta_2p_{11}p_{22}(1-\alpha_1)(1-\alpha_2)$$

If we expand out all terms under the radical and then recombine we can get a simpler expression,

$$\lambda_{\pm} = \frac{\Gamma_1\beta_1p_{11}(1-\alpha_1) + \Gamma_2\beta_2p_{22}(1-\alpha_2) \pm \sqrt{(\Gamma_1\beta_1p_{11}(1-\alpha_1) - \Gamma_2\beta_2p_{22}(1-\alpha_2))^2 - 4(\Gamma_1\Gamma_2\beta_1\beta_2p_1p_2(1-\alpha_1)(1-\alpha_2)(1-\pi_1)(1-\pi_2))}}{2}$$

## Appendix D Equilibria Computations

Equilibria are found by solving the system of equations

$$-S_1^*\beta_1(1-\alpha_1) \left( p_{11}\frac{\delta A_1^* + I_1^*}{N_1} + p_{12}\frac{\delta A_2^* + I_2^*}{N_2} \right) = 0, \quad (5)$$

$$S_1^*\beta_1(1-\alpha_1) \left( p_{11}\frac{\delta A_1^* + I_1^*}{N_1} + p_{12}\frac{\delta A_2^* + I_2^*}{N_2} \right) - \phi E_1^* = 0, \quad (6)$$

$$\sigma\phi E_1^* - \gamma A_1^* = 0, \quad (7)$$

$$(1-\sigma)\phi E_1^* - (\mu_1 + \gamma)I_1^* = 0, \quad (8)$$

$$-S_2^*\beta_2(1-\alpha_2) \left( p_{21}\frac{\delta A_1^* + I_1^*}{N_1} + p_{22}\frac{\delta A_2^* + I_2^*}{N_2} \right) = 0, \quad (9)$$

$$S_2^*\beta_2(1-\alpha_2) \left( p_{21}\frac{\delta A_1^* + I_1^*}{N_1} + p_{22}\frac{\delta A_2^* + I_2^*}{N_2} \right) - \phi E_2^* = 0, \quad (10)$$

$$\sigma\phi E_2^* - \gamma A_2^* = 0, \quad (11)$$

$$(1-\sigma)\phi E_2^* - (\mu_2 + \gamma)I_2^* = 0. \quad (12)$$

As a preliminary step, observe that the relations

$$A_1^* = \frac{\sigma\phi}{\gamma}E_1^*, \quad (13)$$

$$I_1^* = \frac{(1-\sigma)\phi}{\mu_1 + \gamma}E_1^*, \quad (14)$$

$$A_2^* = \frac{\sigma\phi}{\gamma}E_2^*, \quad (15)$$

$$I_2^* = \frac{(1-\sigma)\phi}{\mu_2 + \gamma}E_2^*, \quad (16)$$

can be obtained from equations (7), (8), (11), and (12). Given that  $\alpha_i$  ( $i = 1, 2$ ) is the rate of compliance with precautionary measures among the homeless and housed populations,

respectively, it is feasible to assume  $\alpha_i \neq 1$ . Thus, equation (5) implies

$$S_1^* = 0 \quad \text{or} \quad p_{11} \frac{\delta A_1^* + I_1^*}{N_1} + p_{12} \frac{\delta A_2^* + I_2^*}{N_2} = 0.$$

First, assume  $S_1^* = 0$ , then equation (6) gives

$$\begin{aligned} -\phi E_1^* &= 0, \\ \implies E_1^* &= 0, \\ \implies A_1^* &= I_1^* = E_1^* = 0, \end{aligned} \quad \left[ (13), (14) \right].$$

In other words, if  $S_1^* = 0$ , the equilibrium point has the form  $(0, 0, 0, 0, S_2^*, E_2^*, A_2^*, I_2^*)$  with the final four values being determined based on the solutions to the equations (9), (10), (11), and (12). Recalling the assumption  $\alpha_i \neq 1$  ( $i = 1, 2$ ), it follows from equation (9) that

$$S_2^* = 0 \quad \text{or} \quad p_{21} \frac{\delta A_1^* + I_1^*}{N_1} + p_{22} \frac{\delta A_2^* + I_2^*}{N_2} = 0.$$

If  $S_2^* = 0$ , then equation (10) implies

$$\begin{aligned} -\phi E_2^* &= 0, \\ \implies E_2^* &= 0, \\ \implies A_2^* &= I_2^* = E_2^* = 0, \end{aligned} \quad \left[ (15), (16) \right]$$

meaning that the trivial solution  $(0, 0, 0, 0, 0, 0, 0, 0)$  is the equilibrium point. On the other hand, if

$$p_{21} \frac{\delta A_1^* + I_1^*}{N_1} + p_{22} \frac{\delta A_2^* + I_2^*}{N_2} = 0,$$

it still follows from (10), (15), and (16) that  $A_2^* = I_2^* = E_2^* = 0$ ; yet, there is now no constraint on  $S_2^*$ . In this case, the equilibrium point is given by  $(0, 0, 0, 0, f_2 N_2, 0, 0, 0)$  where  $0 < f_2 \leq 1$ . This would imply that  $(1 - f_2)N_2$  is the removed population whom have either recovered or died of the disease.

The two equilibria that have been obtained up to this point account for all solutions of equations (5)-(12) when  $S_1^* = 0$ . When  $S_1^* \neq 0$ , then

$$p_{11} \frac{\delta A_1^* + I_1^*}{N_1} + p_{12} \frac{\delta A_2^* + I_2^*}{N_2} = 0,$$

which means that

$$\begin{aligned}
-\phi E_1^* &= 0, & \left[ (6) \right] \\
\implies E_1^* &= 0, \\
\implies A_1^* &= I_1^* = E_1^* = 0, & \left[ (13), (14) \right].
\end{aligned}$$

Although  $A_1^*$ ,  $I_1^*$ , and  $E_1^*$  must all equal zero, no constraint has been placed on  $S_1^*$ . Therefore, the equilibria have the form  $(S_1^*, 0, 0, 0, S_2^*, E_2^*, A_2^*, I_2^*)$  with the final four values again being determined based on how equations (9), (10), (11), and (12) are solved. Since all possible solutions of (9), (10), (11), and (12) are already known, it is clear that when

$$p_{11} \frac{\delta A_1^* + I_1^*}{N_1} + p_{12} \frac{\delta A_2^* + I_2^*}{N_2} = 0,$$

the equilibrium points are  $(f_1 N_1, 0, 0, 0, 0, 0, 0, 0)$  and  $(f_1 N_1, 0, 0, 0, f_2 N_2, 0, 0, 0)$  where  $0 < f_1 \leq 1$  and  $0 < f_2 \leq 1$ . Hence, the equilibria of this dynamical system are

$$\begin{aligned}
Q_1^* &= (0, 0, 0, 0, 0, 0, 0, 0), \\
Q_2^* &= (f_1 N_1, 0, 0, 0, 0, 0, 0, 0), \\
Q_3^* &= (0, 0, 0, 0, f_2 N_2, 0, 0, 0), \\
Q_4^* &= (f_1 N_1, 0, 0, 0, f_2 N_2, 0, 0, 0).
\end{aligned}$$

## Appendix E Jacobians

$$J = \begin{bmatrix}
-\beta_1(1-\alpha_1) \left( p_{11} \frac{\delta A_1 + I_1}{N_1} + p_{12} \frac{\delta A_2 + I_2}{N_2} \right) & 0 & -S_1 \beta_1 (1-\alpha_1) p_{11} \frac{\delta}{N_1} & -S_1 \beta_1 (1-\alpha_1) \frac{p_{11}}{N_1} & 0 & 0 & -S_1 \beta_1 (1-\alpha_1) p_{12} \frac{\delta}{N_2} & -S_1 \beta_1 (1-\alpha_1) \frac{p_{12}}{N_2} \\
\beta_1(1-\alpha_1) \left( p_{11} \frac{\delta A_1 + I_1}{N_1} + p_{12} \frac{\delta A_2 + I_2}{N_2} \right) & -\phi & S_1 \beta_1 (1-\alpha_1) p_{11} \frac{\delta}{N_1} & S_1 \beta_1 (1-\alpha_1) \frac{p_{11}}{N_1} & 0 & 0 & S_1 \beta_1 (1-\alpha_1) p_{12} \frac{\delta}{N_2} & S_1 \beta_1 (1-\alpha_1) \frac{p_{12}}{N_2} \\
0 & \sigma \phi & -\gamma & 0 & 0 & 0 & 0 & 0 \\
0 & (1-\sigma)\phi & 0 & -(\mu_1 + \gamma) & 0 & 0 & 0 & 0 \\
0 & 0 & -S_2 \beta_2 (1-\alpha_2) p_{21} \frac{\delta}{N_1} & -S_2 \beta_2 (1-\alpha_2) \frac{p_{21}}{N_1} & -\beta_2 (1-\alpha_2) \left( p_{21} \frac{\delta A_1 + I_1}{N_1} + p_{22} \frac{\delta A_2 + I_2}{N_2} \right) & 0 & -S_2 \beta_2 (1-\alpha_2) p_{22} \frac{\delta}{N_2} & -S_2 \beta_2 (1-\alpha_2) \frac{p_{22}}{N_2} \\
0 & 0 & S_2 \beta_2 (1-\alpha_2) p_{21} \frac{\delta}{N_1} & S_2 \beta_2 (1-\alpha_2) \frac{p_{21}}{N_1} & \beta_2 (1-\alpha_2) \left( p_{21} \frac{\delta A_1 + I_1}{N_1} + p_{22} \frac{\delta A_2 + I_2}{N_2} \right) & -\phi & S_2 \beta_2 (1-\alpha_2) p_{22} \frac{\delta}{N_2} & S_2 \beta_2 (1-\alpha_2) \frac{p_{22}}{N_2} \\
0 & 0 & 0 & 0 & 0 & \sigma \phi & -\gamma & 0 \\
0 & 0 & 0 & 0 & 0 & (1-\sigma)\phi & 0 & -(\mu_2 + \gamma)
\end{bmatrix}$$

$$J \Big|_{Q_1^*} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\phi & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi\sigma & -\gamma & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi(1-\sigma) & 0 & -(\gamma+\mu_1) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\phi & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \phi\sigma & -\gamma & 0 \\ 0 & 0 & 0 & 0 & 0 & \phi(1-\sigma) & 0 & -(\gamma+\mu_2) \end{bmatrix}$$

$$J \Big|_{Q_2^*} = \begin{bmatrix} 0 & 0 & -f_1\beta_1\delta p_{11}(1-\alpha_1) & -f_1\beta_1p_{11}(1-\alpha_1) & 0 & 0 & -\frac{f_1N_1\beta_1\delta p_{12}(1-\alpha_1)}{N_2} & -\frac{f_1N_1\beta_1p_{12}(1-\alpha_1)}{N_2} \\ 0 & -\phi & f_1\beta_1\delta p_{11}(1-\alpha_1) & f_1\beta_1p_{11}(1-\alpha_1) & 0 & 0 & \frac{f_1N_1\beta_1\delta p_{12}(1-\alpha_1)}{N_2} & \frac{f_1N_1\beta_1p_{12}(1-\alpha_1)}{N_2} \\ 0 & \phi\sigma & -\gamma & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi(1-\sigma) & 0 & -(\gamma+\mu_1) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\phi & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \phi\sigma & -\gamma & 0 \\ 0 & 0 & 0 & 0 & 0 & \phi(1-\sigma) & 0 & -(\gamma+\mu_2) \end{bmatrix}$$

$$J \Big|_{Q_3^*} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\phi & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi\sigma & -\gamma & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi(1-\sigma) & 0 & -(\gamma + \mu_1) & 0 & 0 & 0 & 0 \\ 0 & 0 & -\frac{f_2 N_2 \beta_2 \delta p_{21}(1-\alpha_2)}{N_1} & -\frac{f_2 N_2 \beta_2 p_{21}(1-\alpha_2)}{N_1} & 0 & 0 & -f_2 \beta_2 \delta p_{22}(1-\alpha_2) & -f_2 \beta_2 p_{22}(1-\alpha_2) \\ 0 & 0 & \frac{f_2 N_2 \beta_2 \delta p_{21}(1-\alpha_2)}{N_1} & \frac{f_2 N_2 \beta_2 p_{21}(1-\alpha_2)}{N_1} & 0 & -\phi & f_2 \beta_2 \delta p_{22}(1-\alpha_2) & f_2 \beta_2 p_{22}(1-\alpha_2) \\ 0 & 0 & 0 & 0 & 0 & \phi\sigma & -\gamma & 0 \\ 0 & 0 & 0 & 0 & 0 & \phi(1-\sigma) & 0 & -(\gamma + \mu_2) \end{bmatrix}$$

$$J \Big|_{Q_4^*} = \begin{bmatrix} 0 & 0 & -f_1\beta_1\delta p_{11}(1-\alpha_1) & -f_1\beta_1p_{11}(1-\alpha_1) & 0 & 0 & -\frac{f_1N_1\beta_1\delta p_{12}(1-\alpha_1)}{N_2} & -\frac{f_1N_1\beta_1p_{12}(1-\alpha_1)}{N_2} \\ 0 & -\phi & f_1\beta_1\delta p_{11}(1-\alpha_1) & f_1\beta_1p_{11}(1-\alpha_1) & 0 & 0 & \frac{f_1N_1\beta_1\delta p_{12}(1-\alpha_1)}{N_2} & \frac{f_1N_1\beta_1p_{12}(1-\alpha_1)}{N_2} \\ 0 & \phi\sigma & -\gamma & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi(1-\sigma) & 0 & -(\gamma+\mu_1) & 0 & 0 & 0 & 0 \\ 0 & 0 & -\frac{f_2N_2\beta_2\delta p_{21}(1-\alpha_2)}{N_1} & -\frac{f_2N_2\beta_2p_{21}(1-\alpha_2)}{N_1} & 0 & 0 & -f_2\beta_2\delta p_{22}(1-\alpha_2) & -f_2\beta_2p_{22}(1-\alpha_2) \\ 0 & 0 & \frac{f_2N_2\beta_2\delta p_{21}(1-\alpha_2)}{N_1} & \frac{f_2N_2\beta_2p_{21}(1-\alpha_2)}{N_1} & 0 & -\phi & f_2\beta_2\delta p_{22}(1-\alpha_2) & f_2\beta_2p_{22}(1-\alpha_2) \\ 0 & 0 & 0 & 0 & 0 & \phi\sigma & -\gamma & 0 \\ 0 & 0 & 0 & 0 & 0 & \phi(1-\sigma) & 0 & -(\gamma+\mu_2) \end{bmatrix}$$