

Postlockdown Dynamics of COVID-19 in New York, Florida, Arizona, and Wisconsin

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Abstract

The COVID-19 pandemic is widely studied as it continues to threaten many populations of people especially in the USA, the leading country in terms of both deaths and cases. More and more reports show that the spread of COVID-19 involves infected individuals first passing through a pre-symptomatic infectious stage in addition to the incubation period and that many of the infectious individuals are asymptomatic. In this study, we design and use a mathematical model to primarily address the question of who are the main drivers of COVID-19 - the symptomatic infectious or the pre-symptomatic and asymptomatic infectious in the states of Florida, Arizona, New York, Wisconsin and the entire United States. We emphasize the benefit of lockdown by showing that for all four states, earlier and later lockdown dates decrease the number of cumulative deaths. This benefit of lockdown is also evidenced by the decrease in the infectious cases for Arizona and the entire US when lockdown is implemented earlier. When comparing the influence of the symptomatic infectious versus the presymptomatic/asymptomatic infectious, it is shown that, in general, the larger contribution comes from the latter group. This is seen from several perspectives (1) in terms of daily cases, (2) in terms of daily cases when the influence of one group is targeted over the other by setting the effective contact rate(s) for the non-targeted group to zero, and (3) in terms of cumulative cases and deaths for the US and Arizona when the influence of one group is targeted over the other by setting the effective contact rate(s) for the non-targeted group to zero. The consequences of the difference in the contributions of the two infectious groups is simulated in terms of testing and these simulations show that an increase in testing and isolating for the pre-symptomatic and asymptomatic infectious group has more impact than an increase in testing for the symptomatic infectious. For example, for the entire US, a 50% increase in testing for the pre-symptomatic and asymptomatic infectious group results in a 25% decrease in deaths as opposed to a lower 6% decrease in deaths when a 50% increase in testing rate for the symptomatic infectious is implemented. We also see that if the testing for infectious symptomatic is kept at the baseline value and the testing for the pre-symptomatic and asymptomatic is increased from 0.2 to 0.25, then the control reproduction number falls below 1. On the other hand, to get even close to such a result when keeping the pre-symptomatic and asymptomatic at baseline fitted values, the symptomatic infectious testing rate must be increased considerably more - from 0.4 to 1.7. Lastly, we use our model to simulate an implementation of a natural herd immunity strategy for the entire U.S. and for the state of Wisconsin (the most recent epicenter) and we find that such a strategy requires a significant number of deaths and as such is questionable in terms of success. We conclude with a brief summary of our results and some implications regarding COVID-19 control and mitigation strategies.

NOTE: This preprint reports new research that has not been certified by peer review and should not be used to guide clinical practice.

Keywords: COVID-19, symptomatic infectious, asymptomatic infectious, pre-symptomatic infectious, natural herd immunity

1. Introduction

The coronavirus, known as COVID-19, emerged in Wuhan, China in December 2019, and has since then spread to all countries on earth. As of 12/10/2020, the United States has reported 15,535,565 cases and 291,403 deaths and although the leading country in both deaths and cases, the U.S. did not implement a national pandemic control initiative. Instead, many states and cities implemented their own initiatives at various times, and the transmission of COVID-19 has varied from state to state. States also implemented

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45 their own testing initiatives. Around 12-10-2020, the reported testing rates are as follows: Arizona testing at
46 a rate of 347.7 tests per 100,000 people, Florida testing at a rate of 497.2 tests per 100,000 people, New York
47 testing at a rate of 985.7 tests per 100,000 people, and Wisconsin testing at a rate of 562.4 tests per 100,000
48 people [2]. With regard to mask usage, the observed mask usage percentage in the US has increased since
49 the beginning of the pandemic. From March 1, 2020 to May 1, 2020, the mask use percentage increased from
50 5% to 40%. From May 1, 2020 to November 22, 2020, the mask use percentage increased from 71%. Social
51 distancing has also played a role in the transmission of COVID-19. From March 8, 2020 to April 4, 2020, the
52 US experienced a 53% decrease in mobility. From April 4, 2020 to June 24, 2020, the decrease in mobility
53 changed to only -22%. From June 24, 2020 to November 22, 2020, the mobility oscillated between -18% and
54 -25% [3]. The social distancing statistics are due to the fact that different states implemented stay-at-home
55 periods for various intervals of time. All of these factors play a part in the transmission of COVID-19 in the
56 US as a whole, and effectively in each state. Although we do not consider all of these measures directly, we
57 do discuss the effect that lockdown has on the number of cases and deaths. We do include mask usage, and
58 we use a different mask compliance value for each period per state [14]. To study social distancing, we fit
59 the values for the effective contact rate for each time period per state and per time period.

60 Mathematical modelling can help us better understand how COVID-19 cases and mortality is affected
61 based on when and how long lockdown periods are enforced. Modelling can also help us identify the main
62 drivers of the disease and give insight on how best to use the standard control and mitigation strategies. We
63 expand on the standard SIR model, which uses a system of ordinary differential equations to model disease
64 spread through multiple compartments over time. In this study, we do not consider a time-varying system.

65 Prior studies consider susceptible individuals transitioning to exposed and then to infectious. We include
66 a pre-symptomatic compartment for individuals who are infectious before the onset of symptoms. We also
67 consider individuals who were tested, resulting in an asymptomatic or symptomatic classification, as well
68 as the population of people who may have self-isolated after being exposed by an infected individual. It is
69 known that over the course of the pandemic, hospitals have been highly stressed and over-crowded due to the
70 influx of COVID-19 patients. In order to study mortality due to the pandemic, we also include compartments
71 that keep track of the number of patients admitted to the hospital and the ICU.

72 We explore scenarios where lockdown is started earlier or extended past the reported date. We show
73 that both scenarios decrease the number of deaths. We can use these type of simulations to predict future
74 mortality populations and use the information to help with determining when lockdown periods should start
75 and how long the lockdown period should be enforced.

76 Finally, in response to the increase in public discussions of a natural herd immunity approach, we use
77 our model to run some basic scenarios for implementing such a strategy in the state of Wisconsin and the
78 US as a whole. We simulate the number of deaths that would occur before herd immunity is achieved.

79 2. Main Questions

80 The overall purpose of this study is to explore the postlockdown dynamics of COVID-19 and in the
81 process, explore the notion that the main drivers of COVID19 are the asymptomatic and pre-symptomatic
82 infectious individuals. Because both the asymptomatic and pre-symptomatic infectious spread COVID-19
83 without showing symptoms we group these two compartments together and compare that group to the
84 symptomatic infectious. More specifically, we use a mathematical model to address the following questions
85 for New York, Arizona, Florida, Wisconsin, and in some cases, the entire US. The section of the paper in
86 which each question is addressed is given after the question.

- 87 • How do changes in lockdown dates (i.e., earlier or later lockdown dates) affect the number of deaths
88 and cases? (Section 4.3.1)
- 89 • How does the contribution of the pre-symptomatic and asymptomatic infectious individuals compare
90 to that of the symptomatic infectious in terms of cases and deaths? (Section 4.3.2)

- 91 • If we isolate all symptomatic infectious individuals or all asymptomatic and pre-symptomatic infectious
92 individuals, how will this impact deaths and cases? (Section 4.3.3)
- 93 • How might a minimum testing rate for pre-symptomatic and asymptomatic compare to that for the
94 symptomatic infectious individuals in order to obtain a reproduction number below 1? (section 4.3.4)
- 95 • How many deaths will occur in a natural herd immunity simulation for Wisconsin and the entire US?
96 (Section 4.4)

97 *2.1. COVID-19*

98 In order to assess and deliver guidelines for individuals to avoid coronavirus infection, the CDC has
99 implemented models equipped with the many stages of the virus [1]. A person who has been infected is
100 considered pre-symptomatic (an individual who is infectious but did not show symptoms at the time of
101 testing) or asymptomatic (a person who is infectious and does not show symptoms throughout the course
102 of the infection). The CDC built pandemic planning scenarios varying the infectiousness of asymptomatic
103 individuals compared to symptomatic, varying the percent of asymptomatic individuals, and varying the
104 percentage of transmission of pre-symptomatic individuals. In this paper, we use similar methods to build
105 our model and determine parameter values for the locations and time periods of interest. Figure 1 shows
106 the stages of COVID-19, especially the incubation period, infectious period, pre-symptomatic period, and
107 symptomatic period. It should be noted that a pre-symptomatic infectious individual can become either
108 asymptomatic or symptomatic infectious.

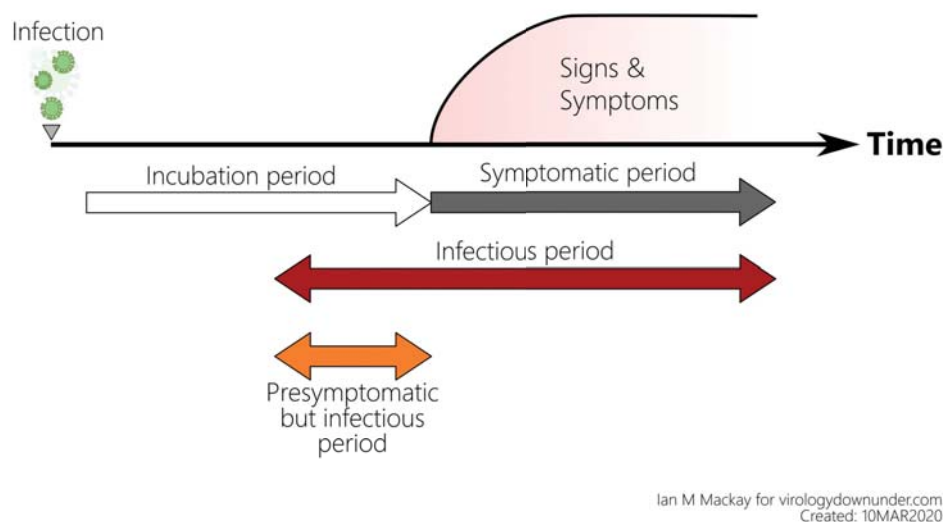


Figure 1: The Stages of COVID-19

109 We are interested in modeling COVID-19 in the United States as a whole, as well as focusing on four
110 states. We consider New York (Northeast), which started as an epicenter in the US. We consider Florida
111 (Southeast) and Arizona (Southwest) due to the fact that they became hot spots with significant numbers of
112 daily cases and overwhelming numbers in their healthcare facilities. Lastly, we consider Wisconsin (Midwest),
113 which became a hot spot later in the year. Table 1 shows the differences between the states with regards to
114 the number of cumulative deaths.

	Cumulative Deaths			
	Beginning of lockdown	End of lockdown	on August 11, 2020	on November 23, 2020
United States	16767	104803	164519	257779
Arizona	25	651	4199	6464
Florida	170	1399	8553	18085
New York	249	30482	32770	34339
Wisconsin	10	594	1006	3158

Table 1: This table gives the cumulative deaths for each state and the United States. The lockdown dates depend on the state.

115 3. Methods

116 3.1. Model with pre-symptomatic compartment and face masks

117 We use a modified Kermack-McKendrik-type epidemic (no human demography) model to better under-
 118 stand the dynamics of COVID-19. Since a main objective of this study is to analyze the role of infectious in-
 119 dividuals who show no symptoms, the model (2) includes a pre-symptomatic compartment as the foundation
 120 for parameter estimation. We use a deterministic susceptible, exposed, pre-symptomatic, symptomatically-
 121 infectious, asymptotically-infectious, self-isolated, hospitalized, recovered, and ICU patients modeling
 122 framework, with the classes denoted as $S(t)$, $E(t)$, $E_P(t)$, $I(t)$, $A(t)$, $J(t)$, $H(t)$, $R(t)$, $C(t)$ respectively;
 123 we also include $D(t)$ to track deaths. The model also includes the mitigation/control interventions of face
 124 mask usage (with a compliance parameter as well as a face mask efficacy parameter) and testing/detection
 125 implementation for each of the infectious classes.

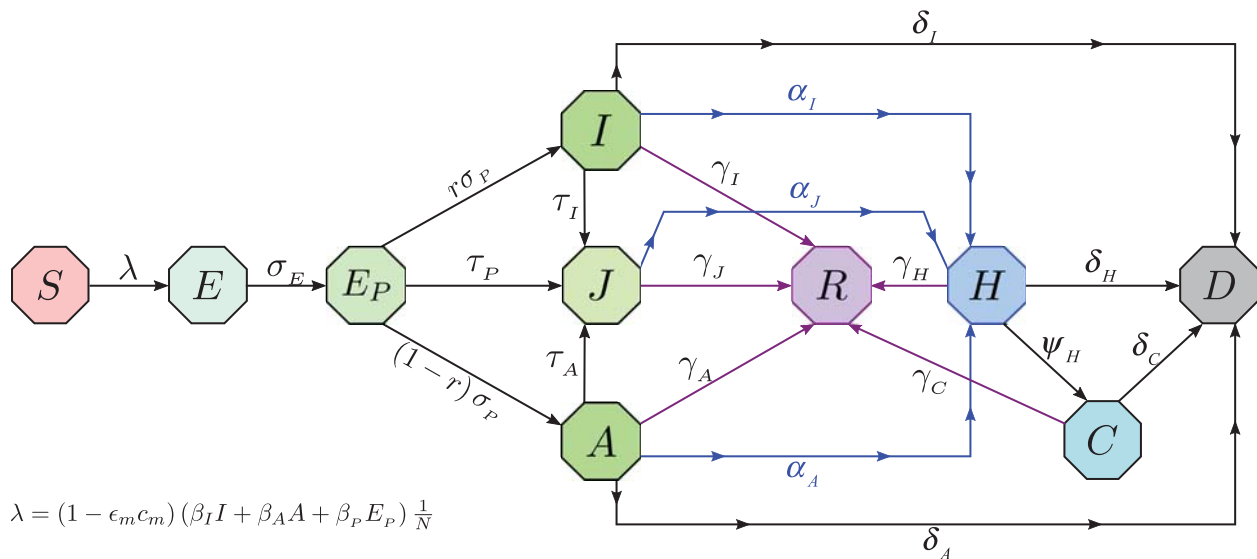


Figure 2: Schematic flow diagram of the model

126 3.2. Data Collection

127 We obtain the observed cumulative deaths and cases data for the states of Arizona, Florida, New York
 128 and Wisconsin from the COVID-19 Data Repository by the Center for Systems Science and Engineering
 129 (CSSE) at Johns Hopkins University (2020). The repository contains data beginning at January 22, 2020
 130 (the marked beginning of the pandemic in the US), however, we focus primarily on the data from March 1,
 131 2020 through October 13, 2020. Using these data sets we determine the initial conditions for the number of
 132 cases and deaths for a given time period.

133 The model has 24 parameters and we use values from the literature for 18 of these and estimate the
 134 remaining 6 by fitting the model to the observed cumulative mortality data for each state. The parameters
 135 that are estimated are as follows: the effective contact rate for the symptomatically-infectious individuals
 136 β_I , the effective contact rate for the asymptotically-infectious individuals β_A , the effective contact rate
 137 for pre-symptomatically-infectious individuals β_P , the rate at which presymptomatically-infectious indi-
 138 viduals self-isolate τ_P , the rate at which asymptotically-infectious individuals self-isolate τ_A , and the
 139 hospitalization rate for self-isolating individuals α_J . Parameter fitting was performed using a non-linear
 140 sum-of-squares estimate - i.e. determining the best parameters set that minimizes the sum of the square of
 141 the difference between the model outputs for death and the observed values for deaths.

142
 143 Our model for the transmission dynamics of COVID-19 is given by the following deterministic system of
 144 non-linear differential equations.

$$\begin{aligned}
 \frac{dS}{dt} &= -\beta_I(1 - \epsilon_m c_m) \frac{SI}{N} - \beta_A(1 - \epsilon_m c_m) \frac{SA}{N} - \beta_P(1 - \epsilon_m c_m) \frac{SE_P}{N} = -\lambda S \\
 \frac{dE}{dt} &= \beta_I(1 - \epsilon_m c_m) \frac{SI}{N} + \beta_A(1 - \epsilon_m c_m) \frac{SA}{N} + \beta_P(1 - \epsilon_m c_m) \frac{SE_P}{N} - \sigma_e E = \lambda S - \sigma_e E \\
 \frac{dE_P}{dt} &= \sigma_e E - \sigma_p E_P - \tau_p E_P \\
 \frac{dI}{dt} &= r \sigma_p E_P - \alpha_I I - \tau_I I - \gamma_I I - \delta_I I \\
 \frac{dA}{dt} &= (1 - r) \sigma_p E_P - \alpha_A A - \tau_A A - \gamma_A A - \delta_A A \\
 \frac{dH}{dt} &= \alpha_I I + \alpha_A A + \alpha_J J - \psi_H H - \gamma_H H - \delta_H H \\
 \frac{dC}{dt} &= \psi_H H - \gamma_C C - \delta_C C \\
 \frac{dJ}{dt} &= \tau_p E_P + \tau_A A + \tau_I I - \alpha_J J - \gamma_J J \\
 \frac{dR}{dt} &= \gamma_I I + \gamma_A A + \gamma_C C + \gamma_J J + \gamma_H H \\
 \frac{dD}{dt} &= \delta_A A + \delta_I I + \delta_H H + \delta_C C
 \end{aligned} \tag{1}$$

145 where $N(t) = S + E + EP + I + A + H + J + C + R$ is the total population at time t . Let ϵ_m be the efficacy
 146 of face masks to prevent transmission and acquisition of infection, c_m the compliance in face mask usage in
 147 the community ($0 < c_m \leq 1$), σ the progression rate from exposed (E) to infectious classes (I or A), r the
 148 fraction of exposed individuals who show clinical symptoms at the end of the incubation period, τ_I the rate
 149 self-quarantined symptomatically-infectious humans self-isolate, $\gamma_I, \gamma_A, \gamma_J, \gamma_H, \gamma_C$ the recovery rates for the
 150 subscribed population, ψ_H the rate of ICU admission for hospitalized individuals, α_I the hospitalization
 151 rate for symptomatically-infectious individuals, α_A the hospitalization rate for asymptotically-infectious
 152 individuals, δ_I the disease-induced mortality rate for symptomatically-infectious individuals, δ_A the disease-
 153 induced mortality rate for asymptotically-infectious individuals, δ_H the disease-induced mortality rate
 154 for hospitalized individuals, δ_C the disease-induced mortality rate individuals in ICU. We assume that
 155 hospitalized individuals do not come in contact with the general population.

156 4. Results

157 4.1. Computation of Reproduction Numbers

The basic reproduction number \mathcal{R}_0 is the average number of secondary infections produced when one
 infected individual is introduced into a host population of susceptible individuals. For our model's \mathcal{R}_0 , we
 have $\mathcal{R}_0 = \mathcal{R}_{0A} + \mathcal{R}_{0I} + \mathcal{R}_{0P}$ where

$$\mathcal{R}_{0A} = \frac{\beta_A(1 - r)}{\alpha_A + \gamma_A + \delta_A}$$

$$\mathcal{R}_{0I} = \frac{\beta_I r}{\alpha_I + \gamma_I + \delta_I}$$

and

$$\mathcal{R}_{0P} = \frac{\beta_P}{\sigma_P}.$$

158 The control reproduction number \mathcal{R}_C is the average number of new cases generated by a typical in-
 159 fectious individual introduced into a host population of susceptible individuals with some control mea-
 160 sures/interventions in place. Using the next generation operator method and notation found in [17] we
 161 have the following computation for the control reproduction number \mathcal{R}_C . If we take the column vector
 162 $(E \ E_P \ I \ A)$ representing the compartments of infected, we have the associated next generation matri-
 163 ces, F and V , for the new infection terms and the transition terms are given respectively as

$$\mathcal{F} = \begin{bmatrix} 0 & (1 - \epsilon_m c_m) \beta_P & (1 - \epsilon_m c_m) \beta_I & (1 - \epsilon_m c_m) \beta_A \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad \mathcal{V} = \begin{bmatrix} \sigma_e & 0 & 0 & 0 \\ -\sigma_e & G_1 & 0 & 0 \\ 0 & -r\sigma_P & G_2 & 0 \\ 0 & -(1-r)\sigma_P & 0 & G_3 \end{bmatrix}$$

where, $G_1 = \sigma_P + \tau_P$, $G_2 = \alpha_I + \gamma_I + \delta_I + \tau_I$, $G_3 = \alpha_A + \gamma_A + \delta_A + \tau_A$. \mathcal{R}_C is the spectral radius of the next generation matrix given by,

$$\mathcal{R}_C = \rho(FV^{-1}) = \mathcal{R}_I + \mathcal{R}_A + \mathcal{R}_P$$

164 where $\mathcal{R}_I = \frac{(1-\epsilon_m c_m)\beta_I r \sigma_P}{G_1 G_2}$, $\mathcal{R}_A = \frac{(1-\epsilon_m c_m)(1-r)\beta_A \sigma_P}{G_1 G_3}$, and $\mathcal{R}_P = \frac{(1-\epsilon_m c_m)\beta_P}{G_1}$. In absence of interventions,
 165 i.e. with $\tau_I = \tau_A = \tau_P = \tau_A = \epsilon_m = c_m = 0$, we get the basic reproduction number for model (1), \mathcal{R}_0 , as
 166 given earlier.

167 **Theorem 1.** *The disease-free equilibrium (DFE) of the model 1 is locally-asymptotically stable if $\mathcal{R}_C < 1$,
 168 and unstable if $\mathcal{R}_C > 1$.*

169 A basic implication of Theorem 1 is that in order to control the COVID-19 outbreak (to not generate
 170 more), it suffices to keep $\mathcal{R}_C < 1$.

171 4.2. Goodness of Fit

We analyze how well our model fits the data from John Hopkins using the T-Test in *R*. The T-Test measures the difference between two sets of continuous data. We want to compare two sample means to determine if there is a statistically significant difference between the model and the data. We use a paired T-test where each vector has the same amount of entries and the values are taken from the same independent variable. The T-Test assumes that the data in each vector is normally distributed and that they have approximately equal variances. To test for normality, we use the Shapiro-Wilk Test in *R*. The null hypothesis for this test is that the values in the vector are normally distributed. If the p-value is greater than 0.05, then the distribution of the values in the vector are not significantly different from the normal distribution. I.e. we fail to reject the null hypothesis. We can visualize this using a Q-Q plot. We assume normality if all the points fall along the reference line. We then check the variances of the values in the two vectors using the standard variance equation from probability. Let the null hypothesis for the T-Test be that the difference between the two groups is 0. The T-statistic is defined as

$$T = \frac{\bar{x}_1 - \bar{x}_2}{\sqrt{(s_1^2/n_1) + (s_2^2/n_2)}}$$

172 where, \bar{x}_1 is the mean of the data, \bar{x}_s is the mean of the model values, s_1^2 is the variance of the data,
 173 s_2^2 is the variance of the model values, n_1 is the sample size of the data, and n_2 is the sample size of the
 174 model values. We conclude that the model for each state, cumulative death population, during the lockdown
 175 period does indeed fit the data. Table 2 shows the resulting p-values of the T-Test for each state and the
 176 United States as a whole during the lockdown period. The goodness of fit plots can be found in the appendix
 177 (section 6.3) in Figures 17, 18, 19, 20, and 21.

	Daily p-value	Daily T-statistic	Cumulative p-value	Cumulative T-statistic
United States	0.9722	0.035037	0.7839	0.27581
Florida	0.9083	0.11621	0.4947	0.69165
Arizona	0.6002	-0.52796	0.0004622	3.7845
New York	0.8781	-0.15396	0.007702	2.749
Wisconsin	0.02353	-2.323	2.2e-16	-17.953

Table 2: Goodness of Fit for the T-Test. This table gives the p-values and T-statistic for each test for each state and the United States during the lockdown period. The daily column denotes the model vs the data of the daily deaths. The cumulative column denotes the model vs the data of the cumulative deaths.

178 *4.3. Simulated Epidemics*

179 We fit our model using the cumulative and daily mortality data for Florida, Arizona, Wisconsin, and New
 180 York over the pre-lockdown, lockdown, and post-lockdown periods based on the state’s respective decisions.
 181 Florida, Arizona, New York, and Wisconsin pre-lockdown period is defined from March 10, 2020 to April 3,
 182 2020, March 21, 2020 to March 31, 2020, March 2, 2020 to March 22, 2020, and March 19, 2020 to March
 183 25, 2020 respectively. The lockdown period is defined from April 3, 2020 to May 4, 2020, March 31, 2020
 184 to May 15, 2020, March 22, 2020 to May 28, 2020, March 25, 2020 to May 26, 2020 respectively. For the
 185 entire US the pre-lockdown and postlockdown dates are January 22, 2020 to April 7, 2020 and April 7, 2020
 186 to May 28, 2020, as given in [14]. The post-lockdown periods begin at the end of the prescribed lockdown
 187 period for each state and for most of our results ends on August 11, 2020. Figure 3 shows the fit for our
 188 model with the cumulative deaths data during the lockdown period.

189

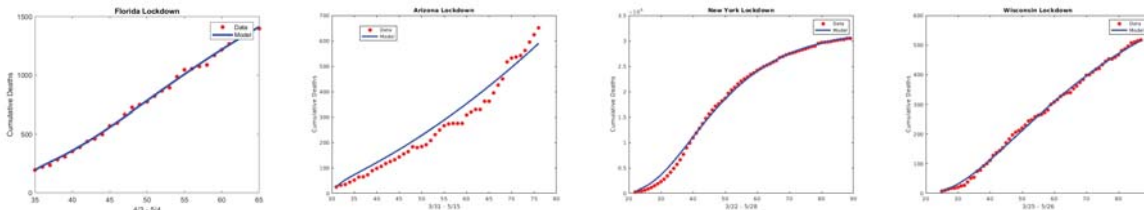


Figure 3: Data fitting of the model using COVID-19 mortality data for the states of Florida, Arizona, New York and Wisconsin during their respective lockdown periods. The plots show the model (blue) against the data (red).

190 The state variables and the parameters for our model are given in the Appendix in Tables 3 and 4
 191 respectively.

192 *4.3.1. Lockdown starting earlier or later*

193 For all of the states, the first lockdown (and for most states, the only lockdown so far) was implemented in
 194 the month of March. Since an earlier lockdown would have most likely prevented the disease from gaining a
 195 foothold in these areas, when we simulate an earlier lockdown, we find that the number of cumulative deaths
 196 decreases significantly for each state. This decrease occurs each time we add one week to the beginning of
 197 the lockdown period - see Figure 4.

198

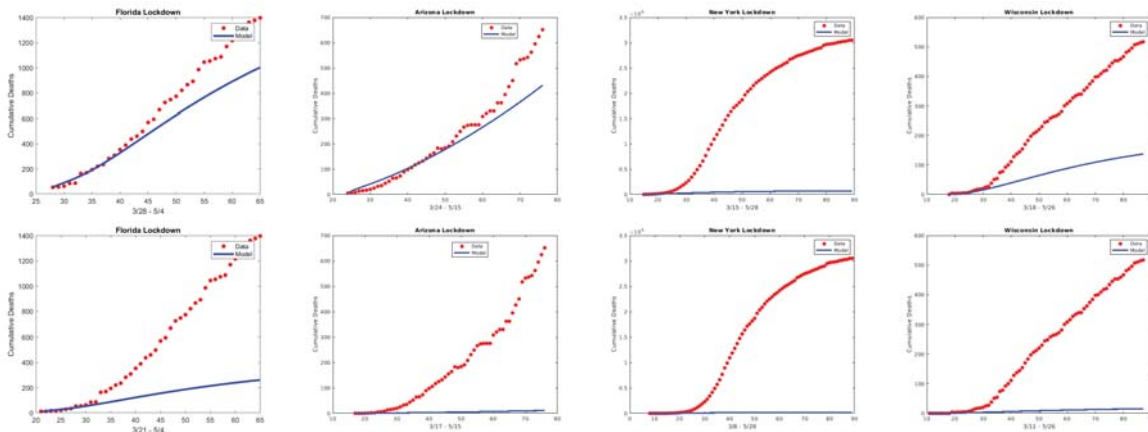


Figure 4: **Row 1:** The cumulative deaths for each state if lockdown began 1 week earlier. **Row 2:** The cumulative deaths for each state if lockdown began 2 weeks earlier.

199 In general, an earlier lockdown also results in a decrease in daily cases for both the symptomatic and
 200 asymptomatic/pre-symptomatic compartments, as shown in the daily cases plots for Arizona and the entire
 201 US in Figure 5.

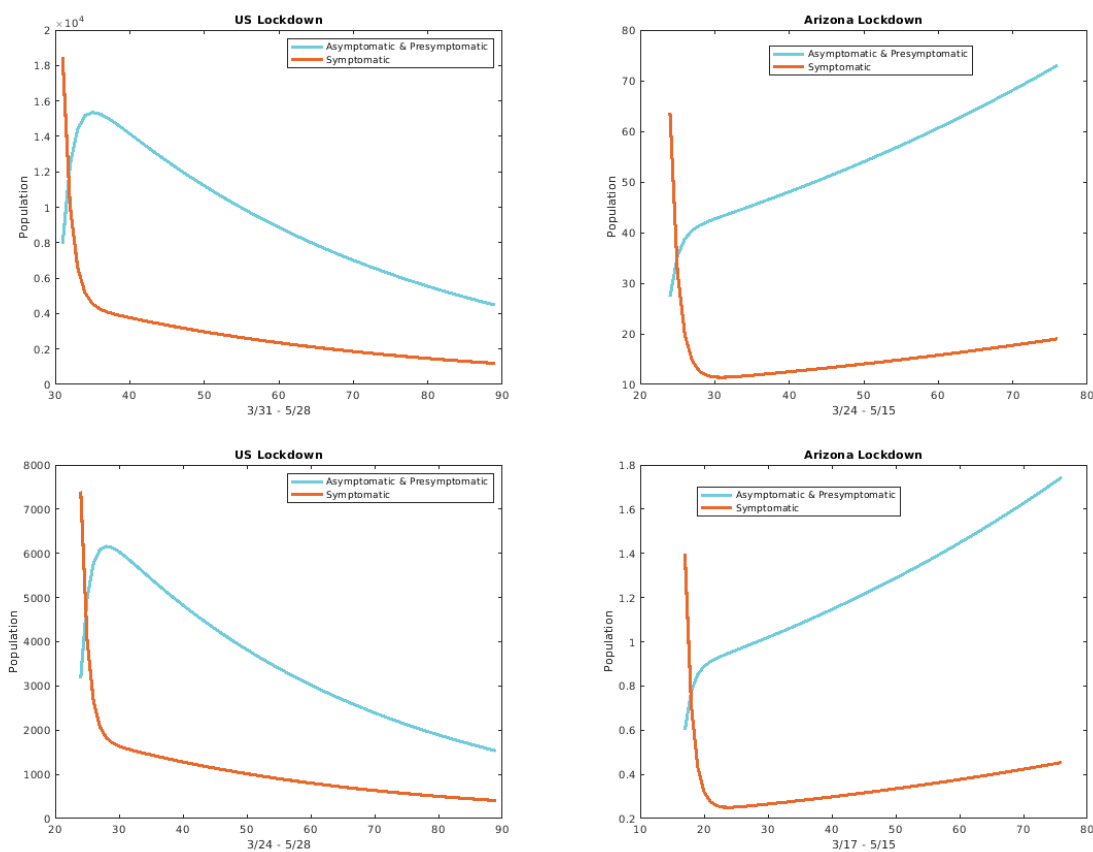


Figure 5: Daily cases for the asymptomatic/pre-symptomatic individuals (cyan curve) versus the symptomatic individuals (red curve) for the entire US and Arizona. **Row 1:** If lockdown began 1 week earlier. **Row 2:** If lockdown began 2 weeks earlier.

202 On the other hand, if we use the original lockdown start dates but extend the lockdown periods by 2 and
 203 4 weeks for each state, we again see a decrease in cumulative deaths as shown in Figure 6.

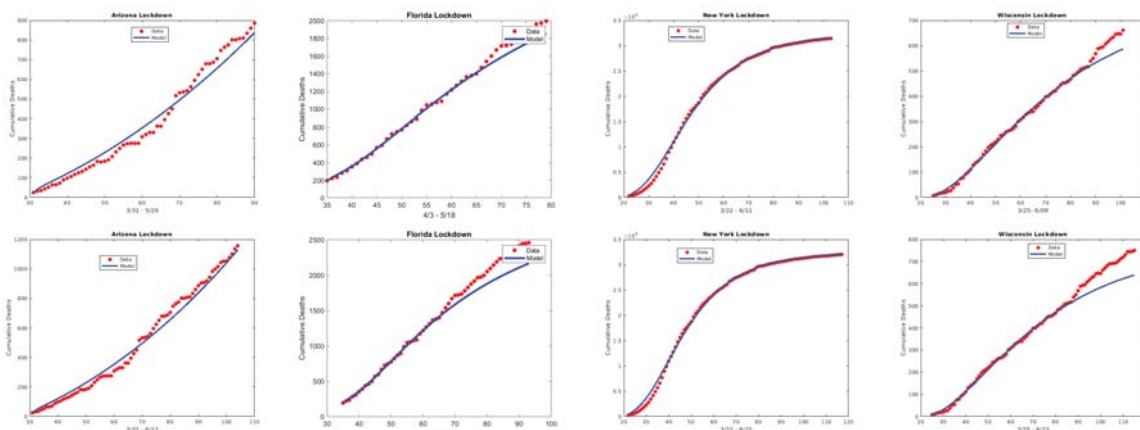


Figure 6: **Row 1:** The cumulative deaths for each state if lockdown was lifted 2 weeks later. **Row 2:** The cumulative deaths for each state if lockdown was lifted 4 weeks later.

204 These varying lockdown dates scenarios can be used to assess the efficacy of lockdown periods and to
 205 guide the manner in which the lockdown periods are implemented. For example, one interesting lockdown
 206 scenario is to consider what would happen if the entire U.S. implemented a lockdown during the month of
 207 December 2020 for two weeks. In order to simulate such a strategy we reduce the contact rate parameters
 208 - i.e., the betas - by 40% [11] and run the simulation forward. As shown in Figure 7 below, the simulations
 209 give that such a lockdown would result in a marked decrease in the number of deaths.

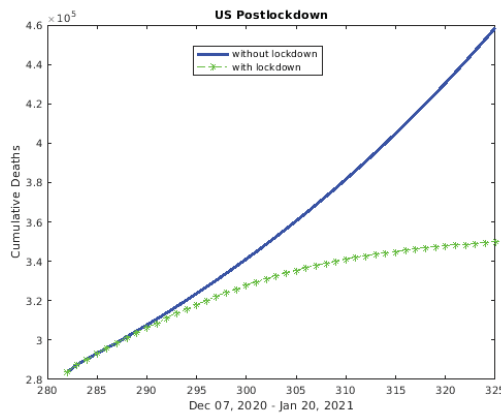


Figure 7: Cumulative deaths in US with lockdown in December (green) versus without lockdown (blue)

210 4.3.2. Simulations for Asymptomatic, pre-symptomatic, and Symptomatic

211 In this section, we focus more on comparing the contributions of the symptomatic infectious group versus
 212 the asymptomatic and pre-symptomatic infectious group. To this end, we first consider plotting the daily
 213 cases for the two groups against each other. In all states and time periods, we find that the number of daily
 214 cases for the asymptomatic and pre-symptomatic is more than the daily cases for the symptomatic as shown
 215 in the following figure.

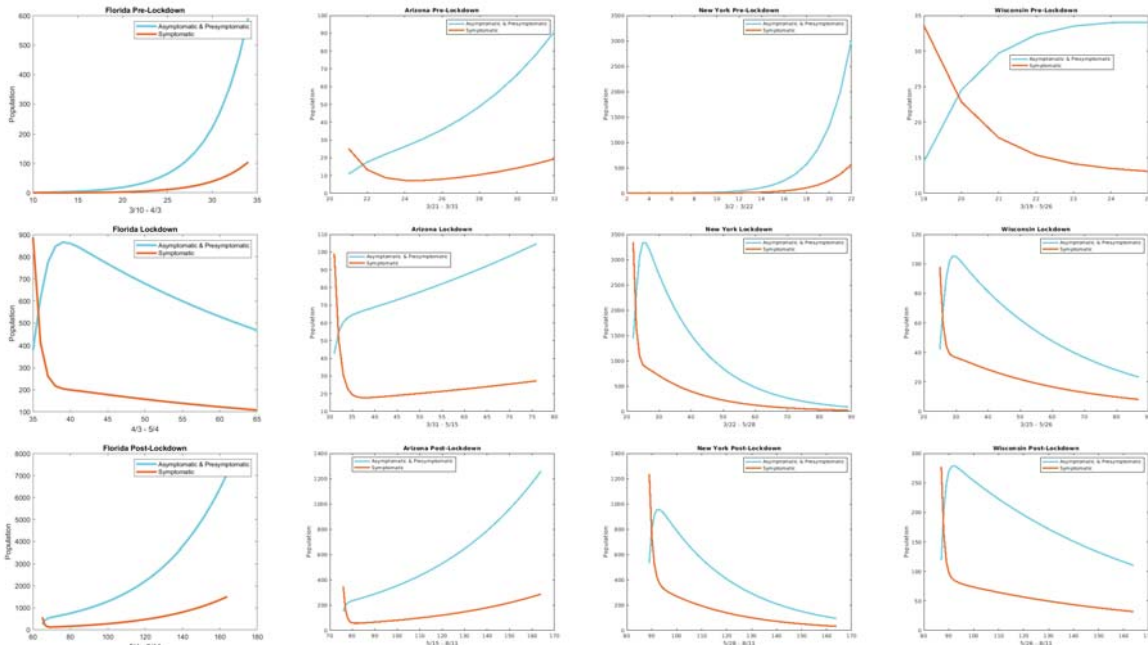


Figure 8: The plots show the pre-symptomatic and asymptomatic versus symptomatic individuals for each state during their respective time period. **Row 1:** Pre-lockdown period. **Row 2:** Lockdown period. **Row 3:** Post-lockdown period.

216 The parameter β_I represents the contacts by the symptomatically infectious individuals that could
 217 transmit the disease. By setting $\beta_I = 0$, we eliminate that transmission and focus on the effect of the
 218 pre-symptomatic and asymptomatic individuals. Similarly, fixing $\beta_A = \beta_P = 0$, targets the effect of the
 219 symptomatically infectious. In order to further discern the role of the asymptomatic and pre-symptomatic
 220 infectious versus that of the symptomatic infectious, we consider these zero contact rate scenarios in terms
 221 of the number of daily cases for all of the states and the entire US. In Figure 9, we show the results for the
 222 pre-lockdown and post-lockdown periods and for those periods, we again get that the larger contribution
 223 comes from the asymptomatic and pre-symptomatic individuals. For the lockdown period we obtain mixed
 224 results and these are considered in the appendix (Figure 16).

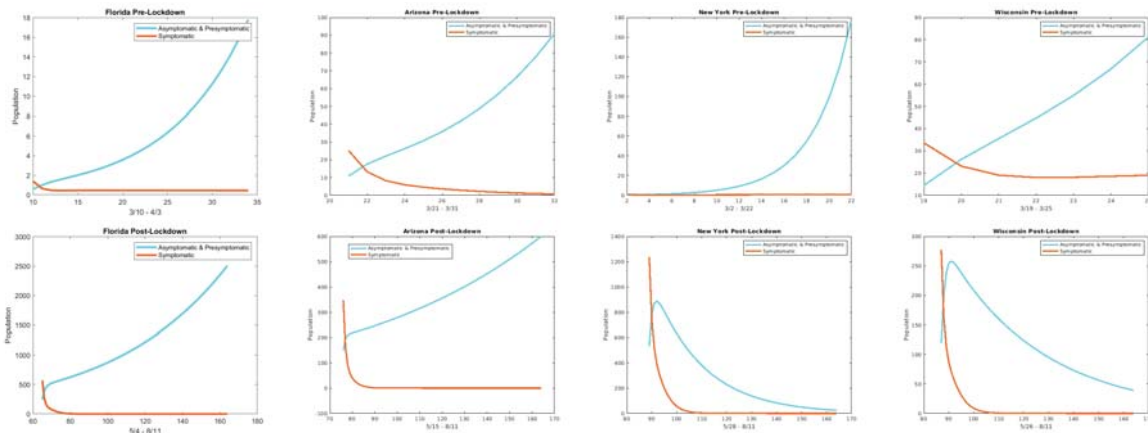


Figure 9: The plots show the daily cases for each state with $\beta_I = 0$ (cyan curve) and with $\beta_A = \beta_P = 0$ (red curve) during their respective **Row 1**: Pre-lockdown period and **Row 2**: Post-lockdown period.

225 In Figure 10, we again take the $\beta_I = 0$ (green curve) versus the $\beta_A = \beta_P = 0$ (magenta curve) scenario,
 226 but here we consider the effect in terms of cumulative cases and cumulative deaths for the entire US and
 227 the state of Arizona. Both types of plots indicate that the main drivers are the asymptomatic and pre-
 228 symptomatic individuals.

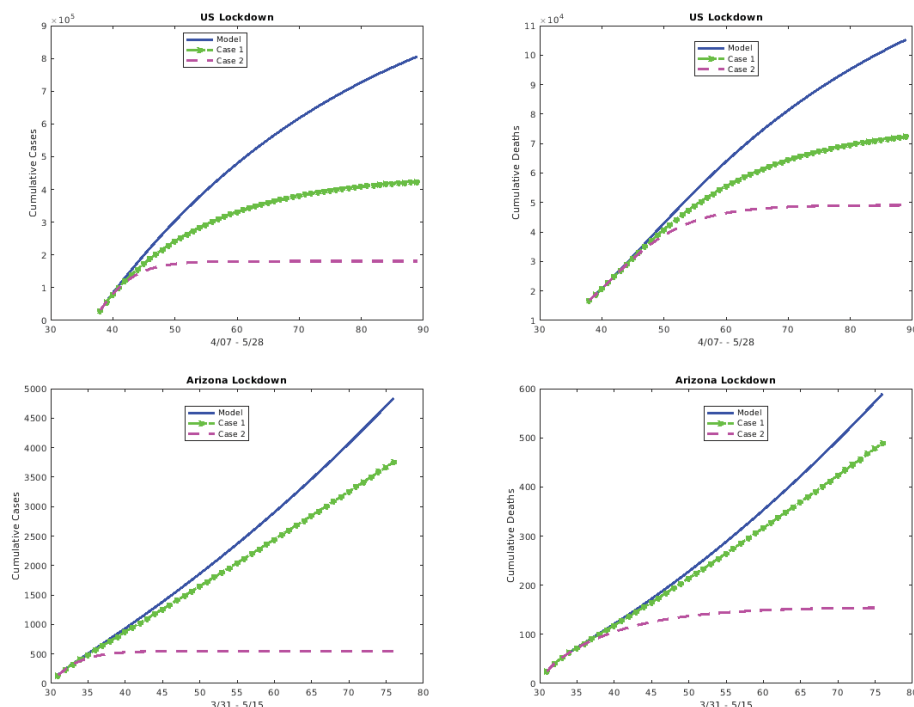


Figure 10: The cumulative cases and deaths for the US and Arizona over the respective lockdown period where **Case 1** corresponds to $\beta_I = 0$ (green curve; so focuses on the impact of asymptomatic/pre-symptomatic individuals) and **Case 2** corresponds to $\beta_A = \beta_P = 0$ (magenta curve; so focuses on the impact of symptomatic individuals)

229 4.3.3. Isolating Symptomatic and Asymptomatic/Pre-symptomatic

230 Recall that the τ values give the rate for testing/detection of infected individuals and subsequent self-
 231 isolation. In this section we compare and contrast the effect of isolating the symptomatic against the effect
 232 of isolating the asymptomatic and pre-symptomatic by changing the appropriate τ values. For example, to
 233 address the effect of isolating symptomatic infectious individuals, we can increase the value of τ_I (the rate
 234 that symptomatically infectious individuals self-isolate) while keeping all other rates the same. We consider
 235 this scenario for the entire US and find that when τ_I increases (so that the number of symptomatically
 236 infectious individuals that are self-isolated and taken out of the general population increases), the number of
 237 cumulative deaths decreases by as much as 12.5%. Alternatively, if the rate of self-isolation for the asymp-
 238 tomatic and pre-symptomatic infectious individuals - τ_A and τ_P respectively - are increased and the τ_I is
 239 kept at the baseline value for the US, the number of cumulative deaths decreases by about 35% at the end
 240 of the period - see Figure 11. Thus, the decrease is more pronounced when the τ_A and τ_P are increased than
 241 when the τ_I is increased which indicates that the asymptomatic and pre-symptomatic have more impact.
 242 Figure 12 focuses on the difference in impact with 50% increase in testing and shows that the asymptomatic

243 and pre-symptomatic testing gives up to about four times as much percent decrease as the symptomatic.

244

245 We can also compare the effect of these type of testing scenarios for different states. For example,
 246 although Wisconsin and Florida had similar testing rates around 12/10/2020, we find that a 50% increase
 247 in τ_A and τ_P , results in roughly a 20% decrease in cumulative deaths for Wisconsin compared to a 50%
 248 decrease in cumulative deaths for Florida - Figure 13. Note that the results for both states show the greater
 249 influence of the asymptomatic and pre-symptomatic infectious.

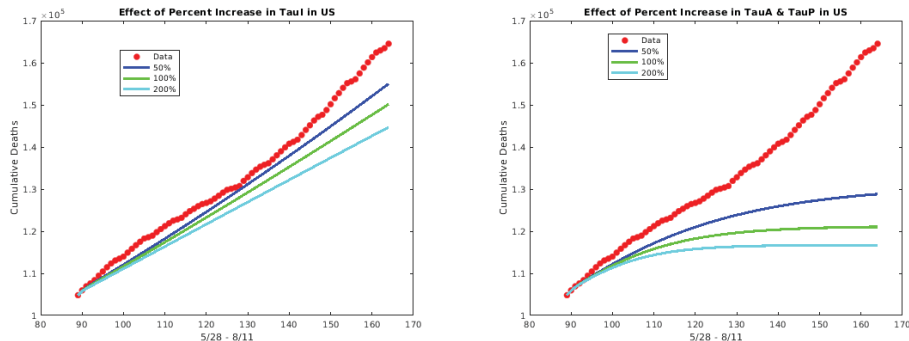


Figure 11: The effect of an increase in testing of symptomatic individuals versus asymptomatic and pre-symptomatic individuals on the cumulative deaths in the US during post-lockdown period 5/15 to 8/11.

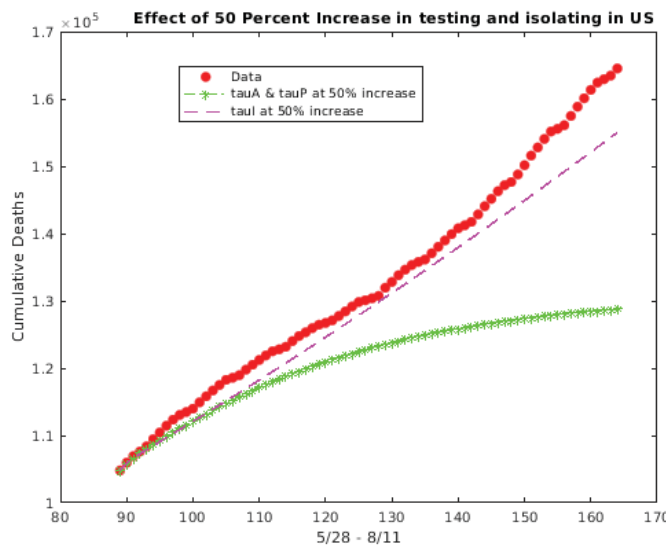


Figure 12: The effect of a 50% increase in testing of asymptomatic and pre-symptomatic versus symptomatic on the cumulative deaths in the US during post-lockdown period 5/28 to 8/11.

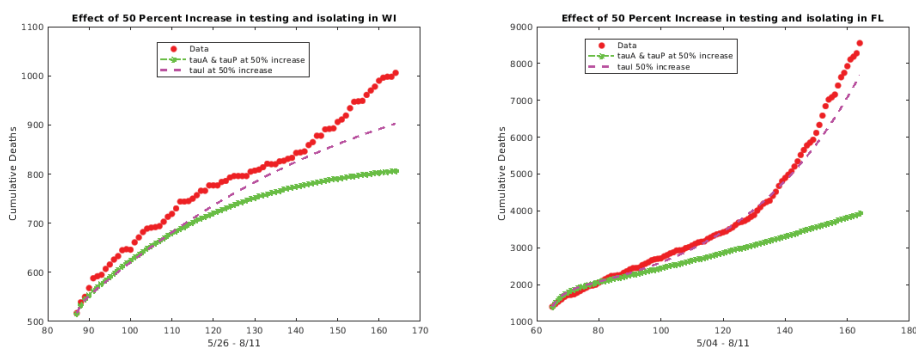


Figure 13: The effect of a 50 percent increase in testing of asymptomatic and pre-symptomatic (green curve) versus symptomatic (magenta curve) individuals on the cumulative deaths for Wisconsin and Florida.

250 4.3.4. Minimum testing needed

251 In this section we consider the question concerning the minimum rate of testing/detection required to
 252 lower the control reproduction number, \mathcal{R}_C below one. This question is more difficult to answer directly,
 253 but we can begin to get at an answer by again considering the effect of increasing testing. For example, for
 254 the entire US from May 28 to Aug 11 (after lifting of lockdown) if the testing rate for asymptomatic and

255 pre-symptomatic is 0.2 - that is, if τ_A and τ_P are both set to 0.2, while $\tau_I = 0.4$, then the reproduction
 256 control number \mathcal{R}_C is 1.0346 (so just above 1). If τ_A and τ_P are bumped above 0.2, for example to 0.25,
 257 while τ_I remains at 0.4, then \mathcal{R}_C falls below 1 - to 0.9628 - and the resulting decrease in cumulative deaths
 258 and daily cases is shown in Figure 14. To get a better appreciation of the impact of testing for asymptomatic
 259 and pre-symptomatic cases, note that if τ_A and τ_P are kept at their baseline fitted values, then τ_I must be
 260 increased to 1.7 to get $\mathcal{R}_C = 0.9945$. Of course, the challenge with all of these testing scenarios is that the
 261 testing of asymptomatic and pre-symptomatic individuals is more difficult since those individuals do not have
 262 symptoms, so in order to achieve these testing rates, widespread and frequent testing should be implemented.
 263

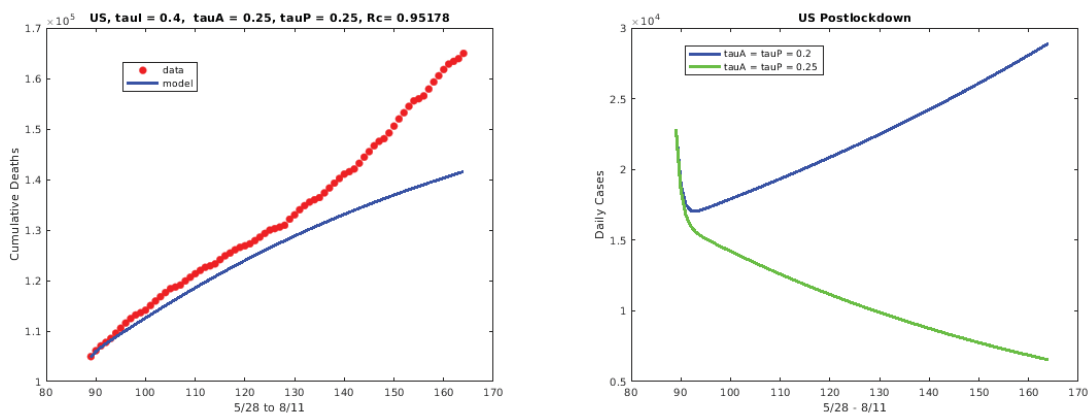


Figure 14: The effect of an increase in τ_A and τ_P

264 4.4. Forward Simulation of the Pandemic

265 Natural herd immunity refers to the process by which enough of the population achieves immunity
 266 via natural recovery from the disease, so that the remaining population that is not immune also receives
 267 protection against the acquisition of the disease. The process involves a natural herd immunity threshold,
 268 that is, the minimum number of people required to achieve disease-acquired immunity. For a disease with
 269 a basic reproduction number of \mathcal{R}_0 , the necessary minimum fraction of people who must achieve immunity
 270 can be given as $1 - 1/\mathcal{R}_0$. We simulate for both Wisconsin (a state which was considered an epicenter
 271 later in 2020) and the entire US, a scenario in which COVID-19 is allowed to run its natural course (with
 272 no implementation of any new control or mitigation strategies) until natural herd immunity is achieved.
 273 For both Wisconsin and the entire US, the number of deaths that occur before reaching the herd immunity
 274 threshold, is considerably high; thus, making the success of a natural herd immunity strategy for COVID-19
 275 questionable (at the very least). For the state of Wisconsin, the number of deaths that occurs is approximately
 276 290,000 and for the entire U.S., the number of deaths is more than 5 million - see Figure 15.

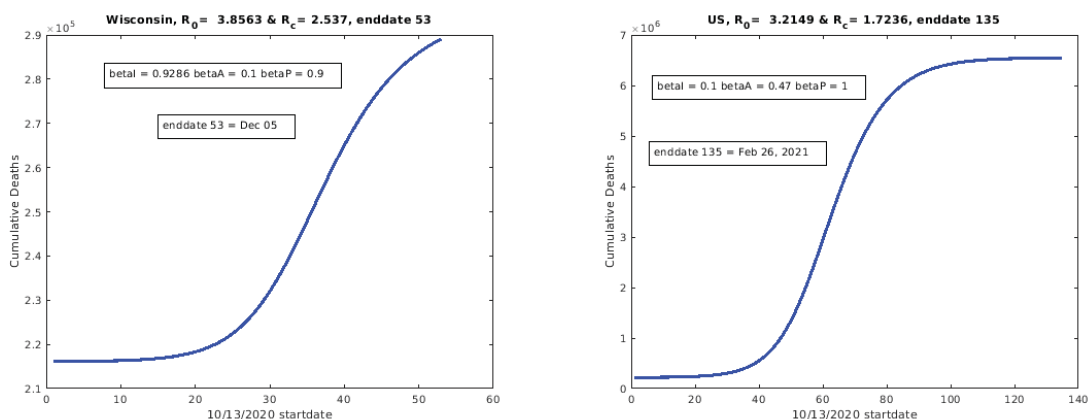


Figure 15: The number of deaths that will occur before natural herd immunity threshold is achieved for the state of Wisconsin and the entire US. The simulations are run starting on October 13.

277 5. Discussion

278 From various perspectives, our analysis indicates that overall, the drivers of COVID-19 are the asymp-
 279 tomatic and pre-symptomatic infectious individuals. We see that if testing is increased, these asymptomatic
 280 and pre-symptomatic infectious have a greater impact than the symptomatic infectious; hence, widespread
 281 and frequent testing is key to controlling the spread of the disease. This is especially true after holiday
 282 periods and large gathering events which are often notorious for reduced and lax implementation of control
 283 and mitigation strategies such as social distancing. Thus, in the schools and workplaces, testing should be
 284 intentionally and systematically increased after such occurrences in order to control the silent transmission
 285 by the newly infectious individuals who do not (yet) have symptoms. Our simulations also show that al-
 286 though lockdown might be difficult in terms of economic impact, the benefit of even a short lockdown (2
 287 weeks) in reducing deaths is considerable.

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 293 (DMS-1915954 and DMS-2016406), and National Security Agency (H98230-20-1-0015).

294 7. Appendix A

295 7.1. Tables of State Variables and Parameter Values

State Variable	Description
S	Population of susceptible individuals
E	Population of non-quarantined exposed individuals (infected but not showing symptoms and cannot transmit infection; newly-infected but not infectious)
E_p	Population of pre-symptomatic (infectious) individuals
I	Population of symptomatically-infectious individuals
A	Population of asymptotically-infectious individuals
J	Population of self-isolated individuals
H	Population of hospitalized individuals
R	Population of recovered individuals
C	Population of individuals in intensive care unit (ICU)
D	Population of COVID-19 deceased individuals

Table 3: State variables for the Model 1

Parameter	Description	Range	Baseline value	References
β_I	Effective contact rate for symptomatically-infectious individuals	Fitting	Fitting	Fitting
β_A	Effective contact rate for asymptotically-infectious individuals	Fitting	Fitting	Fitting
β_P	Effective contact rate for pre-symptomatically-infections individuals	Fitting	Fitting	Fitting
ϵ_m	Efficacy of face masks to prevent transmission and acquisition of infection ($0 < \epsilon_m \leq 1$)	0.4 – 0.6	0.5	[14]
c_m	Compliance in face mask usage in the community ($0 < c_m \leq 1$)	0.0190-0.1835	per state	[14]
σ_e	Progression rate from exposed (E) to pre-symptomatic infectious class (E_p) infectious class	$1/2.5 \text{ day}^{-1}$	$1/2.5 \text{ day}^{-1}$	[14]
σ_P	Progression rate from pre-symptomatic infectious class (E_p) to asymptotically infectious or symptomatically infectious	$1/2.5 \text{ day}^{-1}$	$1/2.5 \text{ day}^{-1}$	[14]
r	Fraction of exposed individuals who show clinical symptoms at the end of the incubation period	0.4 – 0.6	0.6	[13, 10, 6]
τ_P	Rate at which pre-symptomatic infectious individuals self-isolate	Fitting	Fitting	Fitting
τ_I	Rate self-quarantined symptomatically-infectious humans self-isolate	0.07 - 0.4681 day^{-1}	$1/2.5 \text{ day}^{-1}$	[18, 5, 15, 12, 16, 11]
τ_A	Rate at which asymptotically-infectious humans self-isolate	Fitting	Fitting	Fitting
γ_I	Recovery rate for individuals in the I class	$1/30 - 1/3 \text{ day}^{-1}$	$1/7 \text{ day}^{-1}$	[13, 10, 6]
γ_A	Recovery rate for individuals in the A class	$1/14 - 1/3 \text{ day}^{-1}$	$1/7 \text{ day}^{-1}$	[13, 10, 6]
γ_J	Recovery rate for self-isolated individuals	$0.0714 - 0.1667 \text{ day}^{-1}$	$1/8 \text{ day}^{-1}$	[13]
γ_H	Recovery rate for hospitalized patients	$1/30 - 1/3 \text{ day}^{-1}$	$1/14 \text{ day}^{-1}$	[13, 10, 6]
γ_C	Recovery rate for ICU patients	$0.018 - 0.14 \text{ day}^{-1}$	0.0225 day^{-1}	[13, 9]
ψ_H	Rate of ICU admission for hospitalized individuals	$0.02 - 0.1667 \text{ day}^{-1}$	0.083 day^{-1}	[13, 6, 11]
α_I	Hospitalization rate for symptomatically-infectious individuals	$0.1111 - 0.3333 \text{ day}^{-1}$	0.2199 day^{-1}	[6, 8, 10]
α_A	Hospitalization rate for asymptotically-infectious individuals	$0.1667 - 0.3333 \text{ day}^{-1}$	$1/4 \text{ day}^{-1}$	[4, 13?]
α_J	Hospitalization rate for self-isolated individuals	Fitting	Fitting	Fitting
δ_I	Disease-induced mortality rate for symptomatically-infectious individuals	$0.001 - 0.1 \text{ day}^{-1}$	0.0225 day^{-1}	[6, 11, 13, 7]
δ_A	Disease-induced mortality rate for asymptotically-infectious individuals	$0.001 - 0.1 \text{ day}^{-1}$	0.0075 day^{-1}	[6, 7]
δ_H	Disease-induced mortality rate for hospitalized individuals	$0.001 - 0.1 \text{ day}^{-1}$	0.015 day^{-1}	[10, 6, 11]
δ_C	Disease-induced mortality rate individuals in ICU	$0.001 - 0.1 \text{ day}^{-1}$	0.0225 day^{-1}	[10, 6, 7]

Table 4: **Parameter notation, description, values and sources for the Model 1.** Some values change depending on the state and time period and for simulations that are post post-lockdown or involve testing implications, the testing parameter values are chosen to moreso align with reported testing trends.

296 7.2. Lockdown period with separated symptomatic and asymptomatic individuals

297 In Figure 9, the results for the pre-lockdown and post-lockdown periods confirm the hypothesis that the
 298 asymptomatic and pre-symptomatic are the primary contributors to the spread of COVID-19. However,
 299 when we consider this scenario of targeting the asymptomatic and pre-symptomatic (by setting $\beta_I = 0$)
 300 versus the symptomatic (with $\beta_A = \beta_P = 0$) for lockdown, we get mixed results. In particular for New
 301 York and Wisconsin we get more symptomatic infectious cases than asymptomatic and pre-symptomatic -
 302 see Figure 16.

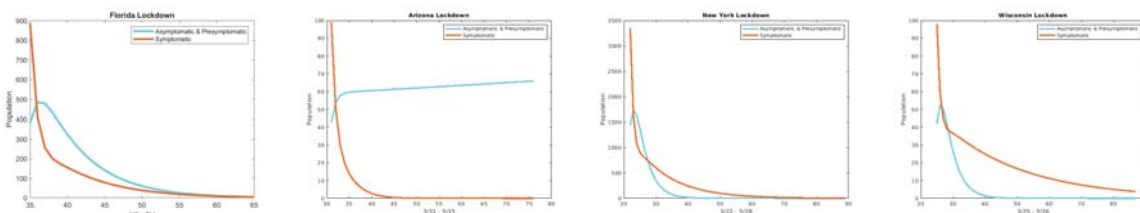


Figure 16: The plots show the daily cases for each state with $\beta_I = 0$ (cyan curve) and $\beta_A = \beta_P = 0$ (red curve) for the lockdown period.

303 7.3. Goodness of Fit

304 In this section, we show the plots for the goodness of fit of the lockdown period for each state and the
 305 US as a whole.

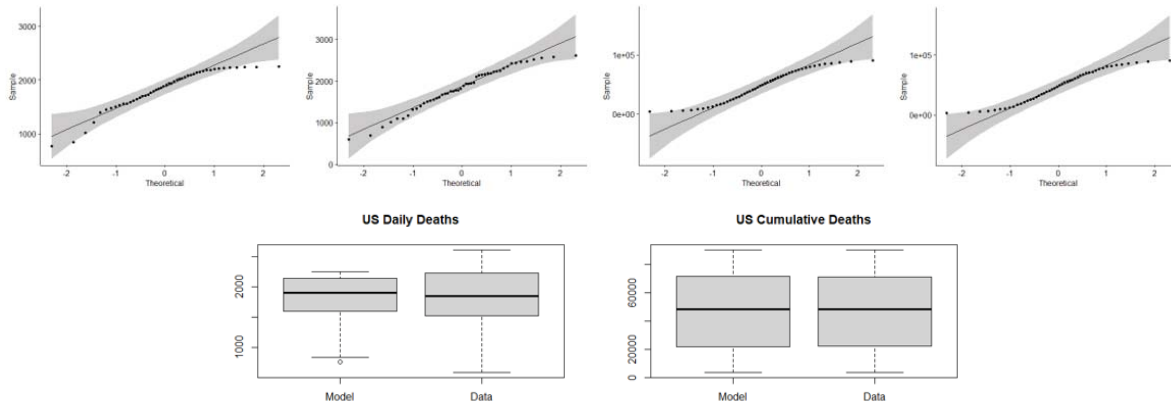


Figure 17: US **Row 1**: Q-Q plots for the (1) Daily death values from the model (2) Daily death values from the data (3) Cumulative death values from the model (4) Cumulative death values from the data. **Row 2**: Box plot of the (1) Daily death values from the model and data (2) Cumulative death values from the model and data.

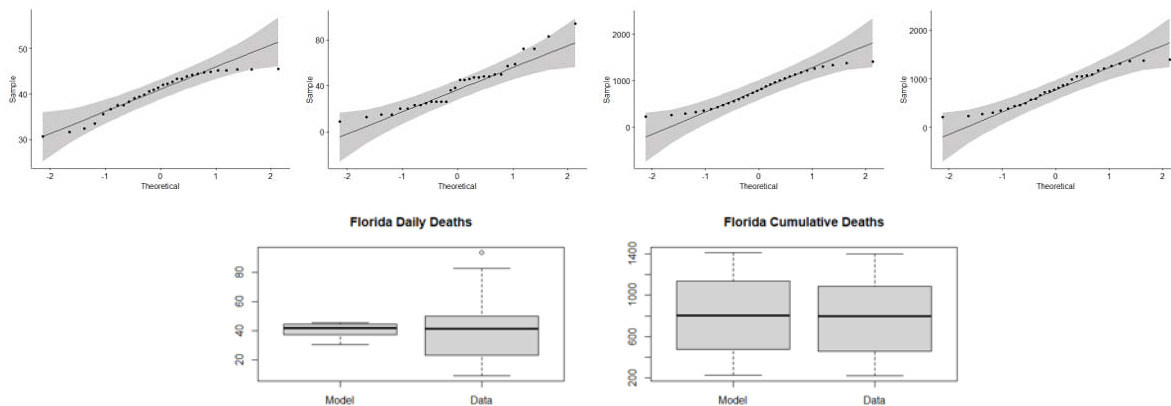


Figure 18: Florida **Row 1**: Q-Q plots for the (1) Daily death values from the model (2) Daily death values from the data (3) Cumulative death values from the model (4) Cumulative death values from the data. **Row 2**: Box plot of the (1) Daily death values from the model and data (2) Cumulative death values from the model and data.

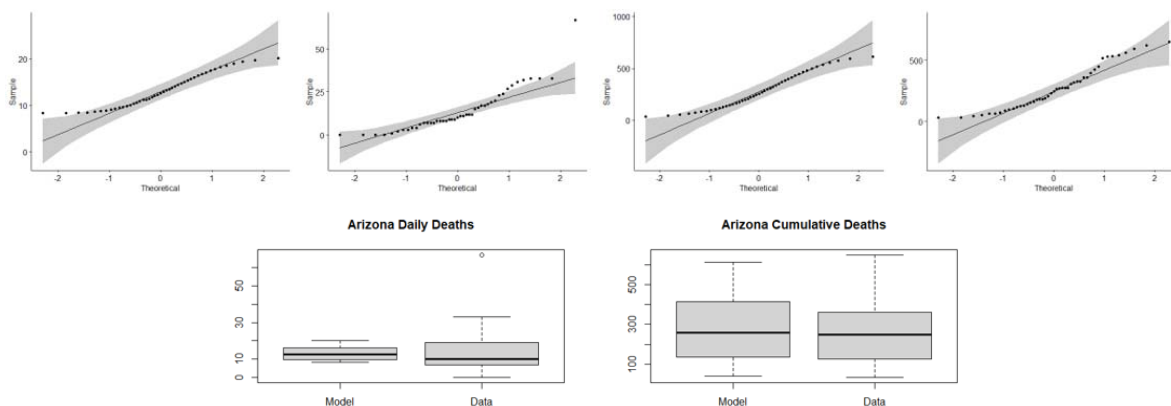


Figure 19: Arizona **Row 1**: Q-Q plots for the (1) Daily death values from the model (2) Daily death values from the data (3) Cumulative death values from the model (4) Cumulative death values from the data. **Row 2**: Box plot of the (1) Daily death values from the model and data (2) Cumulative death values from the model and data.

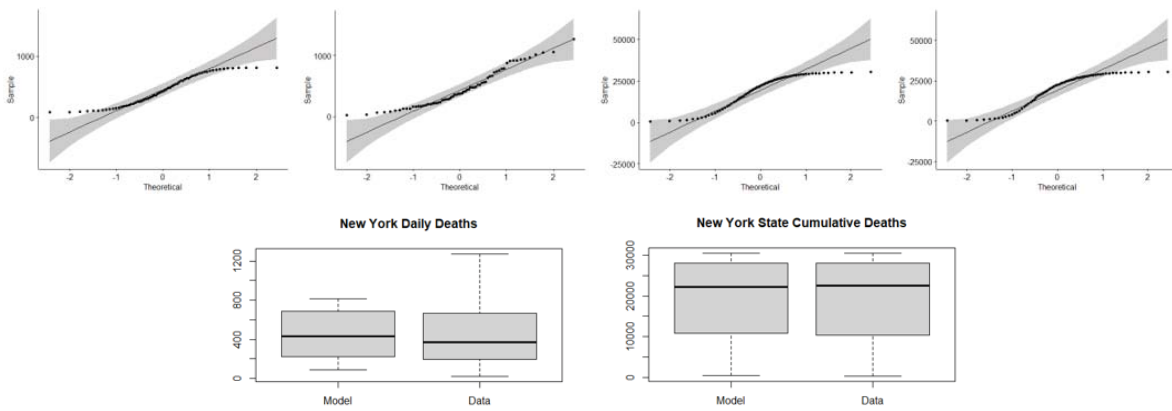


Figure 20: New York **Row 1**: Q-Q plots for the (1) Daily death values from the model (2) Daily death values from the data (3) Cumulative death values from the model (4) Cumulative death values from the data. **Row 2**: Box plot of the (1) Daily death values from the model and data (2) Cumulative death values from the model and data.

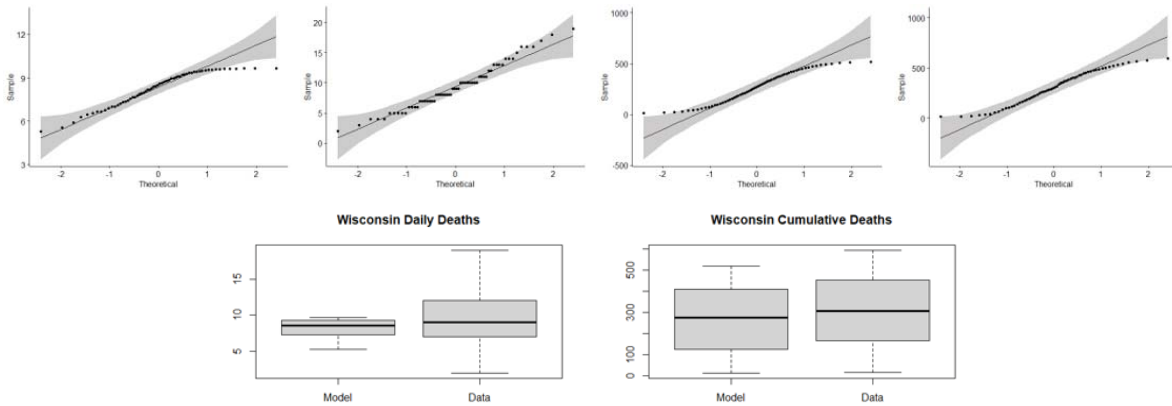


Figure 21: Wisconsin **Row 1**: Q-Q plots for the (1) Daily death values from the model (2) Daily death values from the data (3) Cumulative death values from the model (4) Cumulative death values from the data. **Row 2**: Box plot of the (1) Daily death values from the model and data (2) Cumulative death values from the model and data.

306 Acknowledgements

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