COVID-19 Infection Dynamics

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Abstract

The coronavirus emerged in late 2019 and rapidly reached global pandemic status. Modelling a respiratory pathogen such as COVID-19 is essential in informing public policy decisions to curb infection spread, and it forms the motivation in this project. We create a SEIRS ODE system for COVID-19 infection dynamics with two modifications: 1) numerical methods for dynamical system time evolution and 2) population substructure to constrain infection behavior. Populations are split into categories of susceptible, exposed, infected, and recovered, with additional terms to incorporate behavioral strategies such as vaccination and quarantining. We seek to optimize these behavioral parameters under constraints to minimize either total infection and maximal infection at a given point to "flatten the curve". We further explore an agent-based hierarchical population substructure for our model to demonstrate network interactions among households, neighborhoods and districts, to account for variable population densities. We compare our model to allow for spatial dynamics and identify the conditions under which COVID-19 is most infectious in a more accurate model.

1 Introduction

The coronavirus disease (COVID-19) emerged from Wuhan in the Hubei province of China in late 2019, and quickly spread globally. By February of 2020, the World Health Organization (WHO) declared COVID-19 a global pandemic [1]. The response to the global pandemic was unprecedented, as countries imposed strict lockdowns while the research for a vaccination was greatly expedited.

COVID-19 is characterized by an array of symptoms, ranging from mild flu-like symptoms to severe respiratory system damage [2]. While the mortality rate of the disease is estimated to be 1.1% in the U.S., its ease of spreading as an airborne pathogen has enabled the disease to have immense costs on human life [3]. Hundreds of thousands of people have died from COVID-19 in the United States and millions have passed globally in less than three years.

Modeling and predicting the epidemiology of the COVID-19 disease spread is essential for determining appropriate responses to the pandemic. As countries have pursued varying policies and restrictions with varying degrees of success, it is important to understand the infection dynamics to determine the most effective strategies for combating such a disease. China, for instance, imposed strict lockdowns with obligatory quarantining, leading to immediate reductions in infection spread, but faced numerous resurgent waves of the

disease. Sweden, on the contrary, has taken far less intrusive steps in terms of restricting human movement and public gatherings without facing substantial higher incidence of disease [8]. Developing a more accurate model for the spread of such an infectious virus can enable a better understanding of the effects of policies such as quarantining, restricted social interactions, and vaccine distribution.

As a respiratory pathogen, the spread of COVID-19 is heavily dependent on population clustering and human movements. High population densities can catalyze the transmission of such respiratory pathogens as individuals come into closer proximity. Not only is transmission within households exceedingly prevalent, but interactions across neighborhoods and urban areas greatly contributed to the spread of the disease. Additionally, more urbanized areas faced greater and more immediate burden from the pandemic, as 90 percent of reported cases originated in such cities [4].

Hence, existing models which fail to account for population dynamics in the form of clustering result in predictions that do not closely reflect the true transmission dynamics of COVID-19. Our motivation in this paper is to construct an ODE model for the spread of COVID-19 while accounting for varying degrees of clustering and interaction across households, neighborhoods, and larger urban areas. We further seek to impose quarantining and vaccine distribution in such a model to determine how such policies can be utilized to curb infection spread and mortality rates for COVID-19. In particular, we seek to determine how such a model accounting for intervention policies can be leveraged to both minimize the total spread of disease and minimize the maximum infections at any given point to flatten the curve of infections and reduce burdens on healthcare facilities.

2 Methods

2.1 Data Analysis

To gain a better understanding in trends of COVID-19 propagation through time, we accessed the Massachusetts Bureau of Infectious Disease and Laboratory Sciences record of COVID cases in the state.

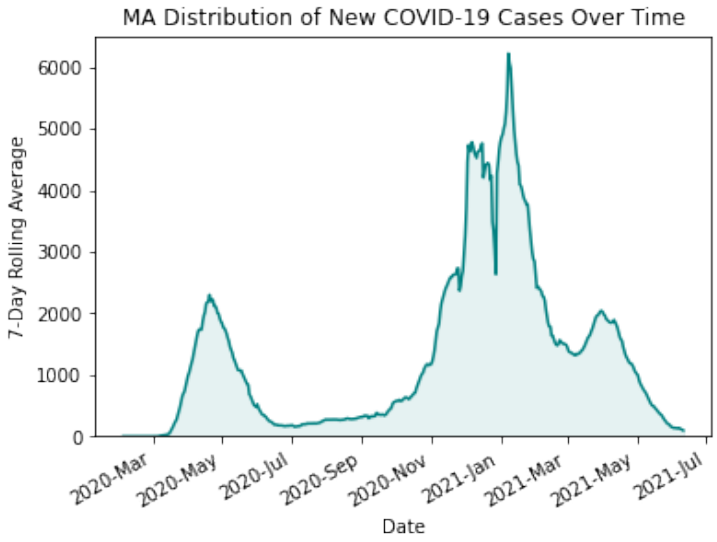

Figure 1: Massachusetts COVID-19 Cases From Jan 2020 to Jul 2021

The data are quite irregular, with complex influences of both environmental conditions and human behavioral decisions making a drastic impact on trends. In capturing general dynamics, however, we aimed to get a sense of the complexity of fit necessary to describe some of the important factors in the spread of the virus. Using the Least Squares method of minimizing residuals, we fit a set of varying degree polynomials and present a degree-12 model for expressivity.

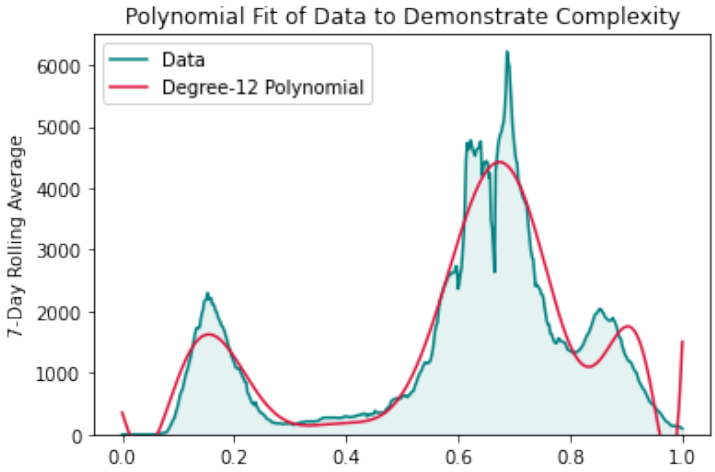

Figure 2: Degree-12 Polynomial Fit on MA COVID-19 Data

While this model does not describe the data well, it presents an initial insight into the complexity of the data we analyze in this study. This prompted our group to prioritize model expressivity while approaching the problem.

2.2 Basic SEIRS Model

The SEIRS model is a staple of classical epidemiology, as it divides the population into subsectors and models the transmission of disease across these groups [1]. It extends a more basic SIR model to account for the incubation time of a disease, as well as the possibility to lose immunity after a period of time recovering from the infection. The model is defined as a dynamical system of ODEs below, where S corresponds to the population of susceptible individuals, E corresponds to exposed, I corresponds to infected, and R corresponds to recovered. We note that the total population is defined N, where under the below assumptions $N = S+E+I+R$, meaning the total population remains constant throughout the evolution of the system. Note that we will revisit this assumption as we modify the below model.

$$\begin{cases} \dot{S} = -\frac{\beta SI}{N} + \xi R \\ \dot{E} = \frac{\beta SI}{N} - \sigma E \\ \dot{I} = \sigma E - \gamma I \\ \dot{R} = \gamma I - \xi R \end{cases} \tag{1}$$

where we have defined the parameters as follows:

Parameter Description	Symbol	Default Value
Rate of transmission from I to S	β	0.24
Rate of Incubation	σ	0.25
Rate of Recovery	γ	0.1
Rate of Immunity	ξ	0.01

Table 1: SEIRS parameters

The infection parameter β is drawn from literature and experimentation to satisfy the condition derived for $\mathcal{R}_0 = \frac{\beta}{\gamma}$ in equation (9). The rate of incubation σ is defined as the inverse of incubation time, which is roughly 4 days for COVID-19. The infectious period is 10 days, leading to a rate of recovery $\gamma = 0.1$ [10]. Finally, the parameter ξ reflects the expected immunity period of about 100 days.

The Jacobian for the above system can be defined as follows, and is useful for determining the equilibria and their respective stabilities:

$$J(S,E,I,R) = \begin{pmatrix} -\frac{\beta I}{N} & 0 & -\frac{\beta S}{N} & \xi \\ \frac{\beta I}{N} & -\sigma - \lambda & \frac{\beta S}{N} & 0 \\ 0 & \sigma & -\gamma - \lambda & 0 \\ 0 & 0 & \gamma & -\xi - \lambda \end{pmatrix} \tag{2}$$

An equilibrium of this system is defined as a point at which the respective subgroups are unchanging in size, meaning $\dot{S} = \dot{E} = \dot{I} = \dot{R} = 0$. This provides valuable information for determining conditions under which the infection spread can be halted and prevented from progressing to worse levels in terms of total infections. We note that an equilibrium point of this system occurs where $\gamma I = \xi R = \sigma E = \frac{\beta SI}{N}$. This represents a maintenance of the infection spread, without complete eradication, making it an interesting result for the goals of flattening the curve, or minimizing a spike in infections.

Alternatively, there is also a equilibrium point at which the population is entirely susceptible, representing a complete eradication of the infection. Thus, we observe our system will stabilize to either an eradication of infection, or a constant number of infected individuals, motivating our introduction of behavioral parameters discussed below (Section 2.3).

The above defined system can be further expanded to include births and natural cause deaths, represented by parameters λ and δ below. Further, we include deaths caused by infection, represented by μ. We can capture this modified system with the below figure, noting that the total population is no longer constant as in the assumptions of standard SEIR models.

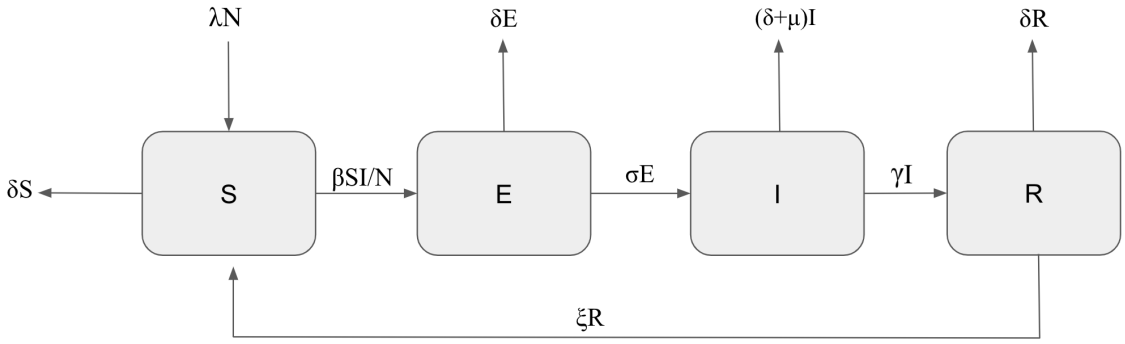

Figure 3: Modified SEIRS model with Variable Total Population

Our set of ODEs becomes the following, with the addition of the birth and death terms. See Table 2 for a complete summary of the parameters.

$$\begin{cases} \dot{S} = \lambda N - \frac{\beta SI}{N} + \xi R - \delta S \\ \dot{E} = \frac{\beta SI}{N} - \sigma E - \delta E \\ \dot{I} = \sigma E - \gamma I - (\delta + \mu) I \\ \dot{R} = \gamma I - \xi R - \delta R \end{cases} \tag{3}$$

From this modified infection dynamics system, we can derive the $\mathcal{R}_0$ term, also called the basic reproductive number. If this value is greater than 1, the virus's inherent reproductive rate will allow it to increase in population size over time. For classical SIR dynamics, we have

$$\mathcal{R}_0 = \frac{\beta}{\gamma}, \tag{4}$$

where β is the rate of infection and γ is the rate of recovery. This definition can be drawn from the construction of the next-generation matrix $\mathbf{G}$ [5]. To determine the state of the system at the next time step, we need transition from non-infected to infected F as well as the transitions between states V. This gives $\mathbf{G} = FV^{-1}$, where $\mathcal{R}_0$ is the leading eigenvalue of $\mathbf{G}$. For the SEIRS model, we need only consider the transition of exposed and infected populations:

$$F = \begin{bmatrix} 0 & \frac{\beta\lambda}{\delta} \\ 0 & 0 \end{bmatrix} \tag{5}$$

$$V = \begin{bmatrix} \delta + \sigma & 0 \\ -\sigma & \gamma + \delta + \mu \end{bmatrix} \tag{6}$$

$$FV^{-1} = \begin{bmatrix} \frac{\beta\lambda\sigma}{\delta(\delta+\sigma)(\gamma+\delta+\mu)} & 0 \\ 0 & \frac{\beta\lambda}{\delta(\gamma+\delta+\mu)} \end{bmatrix} \tag{7}$$

$$\mathcal{R}_0 = \frac{\beta\lambda\sigma}{\delta(\delta + \sigma)(\gamma + \delta + \mu)} \tag{8}$$

In the case that we lack vital dynamics, we have the following simplification for $\mathcal{R}_0$:

$$\mathcal{R}_0 = \frac{\beta}{\gamma} \tag{9}$$

This form is equivalent to SIR dynamics. We can later use this result to constrain our optimization of the system on realistic values of $\mathcal{R}_0$, which is between 2.2 and 4 for COVID-19 [9].

To evolve this dynamical system over time, we compared a variety of integrator methods against the SciPy implementation of explicit 5th-order Runge-Kutta.

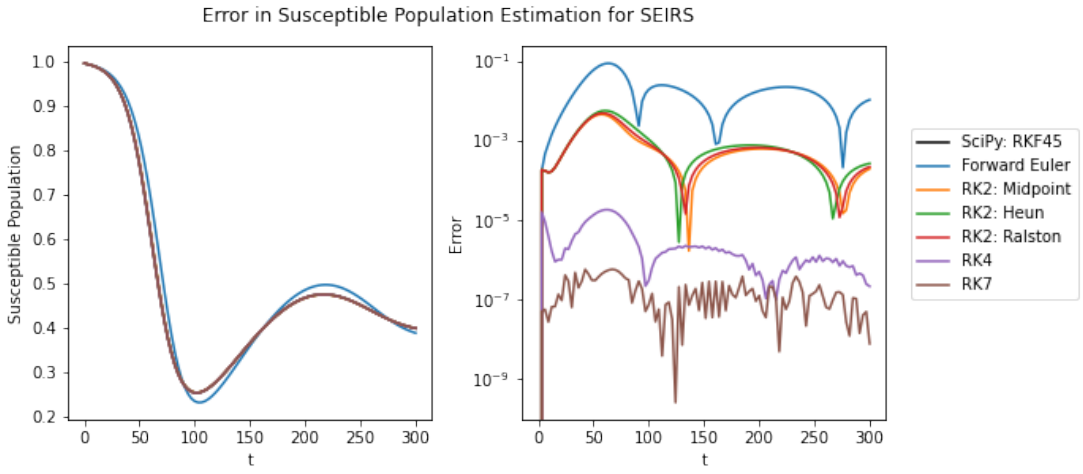

Figure 4: Comparison of Integrator Effectiveness on SEIRS

The dynamical system is not computationally intensive, so lower stability methods suffice for this process. For the SEIRS model, we use the SciPy integrator implementation `solve_ivp()` (RKF45). For stochastic evaluations, we use the 4th-order Runge-Kutta method, a balance of accuracy and computational intensity. Note that error is absolute, so the 7th-order Runge-Kutta may be closer to the true solution than the SciPy implementation.

2.3 Quarantine & Social Distancing

To model the effects of quarantining, social distancing, and other policies directed at reducing spread of infection of COVID-19, we incorporated a variable interaction rate between individuals. By modifying parameter β_0 to be time-evolved $\beta(t)$, our model can reflect policies that are time-dependent. We sought to determine the varying efficacy of strict initial quarantining, compared to prolonged, less stringent social distancing, with a benchmark of no intervention. Hence, in order to determine whether a strict quarantine at the start of outbreak lowers overall infection compared to a more moderate, longer-duration social distancing policy, we determined the following infection parameters.

$$\beta_q(t) = \begin{cases} 0.30\beta_0, & \text{if } 0 < t < 50 \\ \beta_0, & \text{if } t > 50 \end{cases}$$

$$\beta_s(t) = 0.6\beta_0$$

where β_q represents the strict initial quarantine policy resulting in limited interactions by 70 percent that return to normal after a period of 50 days. Likewise, β_s represents the social distancing policy which features a consistent 40 percent reduction of interactions, held for the duration of the simulation.

The resulting system with time-dependent infection parameter $\beta(t)$ becomes:

$$\begin{cases} \dot{S} = \lambda N - \frac{\beta(t)SI}{N} + \xi R - \delta S \\ \dot{E} = \frac{\beta(t)SI}{N} - \sigma E - \delta E \\ \dot{I} = \sigma E - \gamma I - (\delta + \mu) I \\ \dot{R} = \gamma I - \xi R - \delta R \end{cases} \tag{10}$$

2.4 Vaccination

We further included the rollout of a vaccination in our model to determine how various characteristics of a vaccination are significant in curbing infection from COVID-19. A vaccine introduces immunity similar to how individuals who have recovered from the disease itself. In modelling the rollout and efficacy of the COVID-19 vaccines, first distributed in December of 2020, we incorporated a delayed rollout and limited efficacy. As a vaccine was not available from the start of infection outbreak, modelling a delayed vaccine availability was important for the accuracy of our system, meaning vaccine administrations were time dependent. Thus, we introduce term $v(t)$ to represent the vaccination rate, which will be nonzero only after the time period required for the development of such a vaccine. Further, we assume limited efficacy of a vaccine. As current research estimates the efficacy of Moderna and Pfizer vaccines at 90 percent, with reduced efficacy over time [6], we introduce term ρ to represent vaccine failure, or the rate at which vaccinated individuals become susceptible for infection.

The resulting population subsectors along with their transitions is surmised in Figure 5.

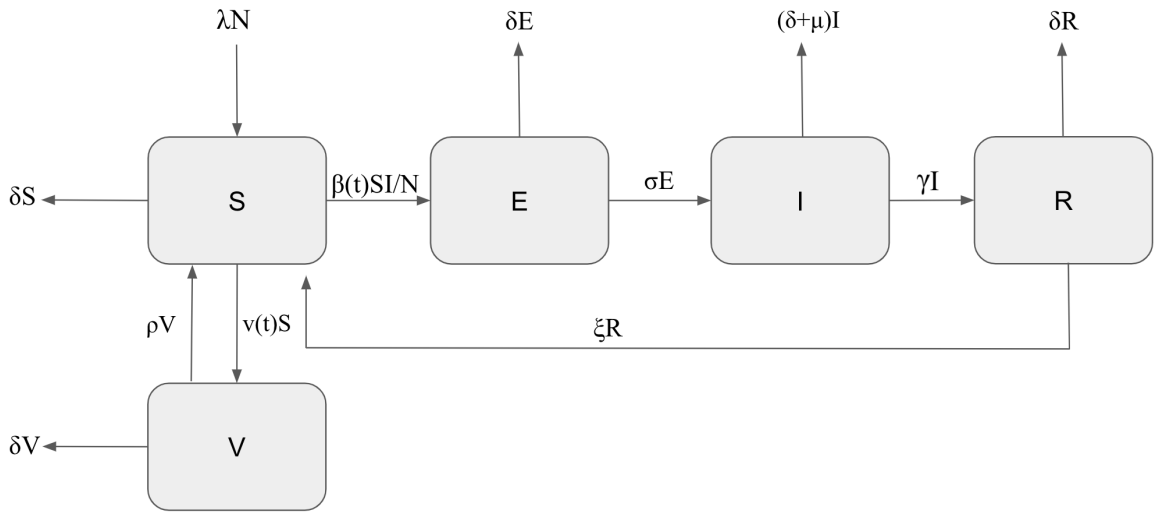

Figure 5: Modified SEIRS model w/Variable Total Population

$$\begin{cases} \dot{S} = \lambda N - \frac{\beta SI}{N} + \xi R - \delta S - v(t)S \\ \dot{E} = \frac{\beta SI}{N} - \sigma E - \delta E \\ \dot{I} = \sigma E - \gamma I - (\delta + \mu)I \\ \dot{R} = \gamma I - \xi R - \delta R \\ \dot{V} = v(t)S - \rho V I - \delta V \end{cases} \tag{11}$$

The parameters for the above system are defined below:

Parameter Description	**Symbol**	**Default Value**
Rate of transmission from I to S	$\beta(t)$	0.24
Incubation time	σ	0.25
Rate of recovery	γ	0.1
Rate of Immunity	ξ	0.01
Natural Cause Birth Rate	λ	0.001
Natural Cause Death Rate	δ	0.001
Coronavirus Related Death Rate	μ	0.005
Vaccine Administration Rate	$v(t)$	0.04 if $t > 100$
Vaccine Failure Rate	ρ	0.02

Table 2: Modified SEIRVS parameters

With the introduction of policies for vaccine administration and quarantining or social distancing, we are able to test the hypothetical effects of a combination of these policy implementations on the rate of infection. In Figure 6 below, we compare varying policies for reduced interactions alongside the time period of vaccine rollout.

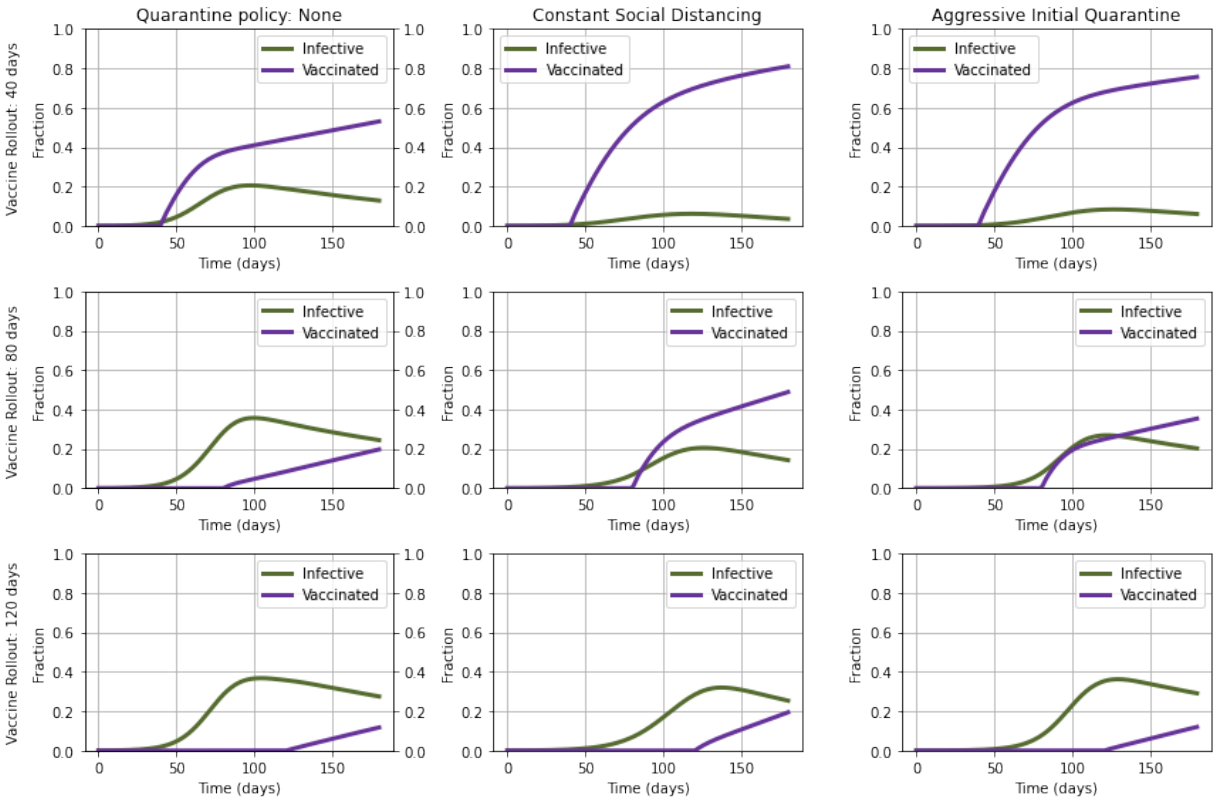

Figure 6: Comparison of Distancing Interventions & Vaccine Rollout

Interestingly, we observe that restricted social interactions in the form of social distancing or quarantine can actually increase vaccination rates. This is a result of such distancing efforts reducing the infective population and thus maintaining a high susceptible population. As only non-infected individuals may become vaccinated, by reducing infection rate, vaccinations can be further streamlined through restrictions on social interactions. Moreover, we observe lower on average and maximal rates of infection with the implementation of a social distancing policy as compared to an aggressive initial quarantine. This result informs us that prolonged, less intrusive intervention is more effective in minimizing the spread of infection as compared to strict policies at the start of an outbreak which are later loosened.

We can similarly compare the speed of vaccine rollout, and observe that faster vaccine administration results in considerably reduced rates of infection. This is an expected result, as the other characteristics of the vaccine were held constant in the above simulations, and thus earlier vaccination would begin to combat infection more rapidly.

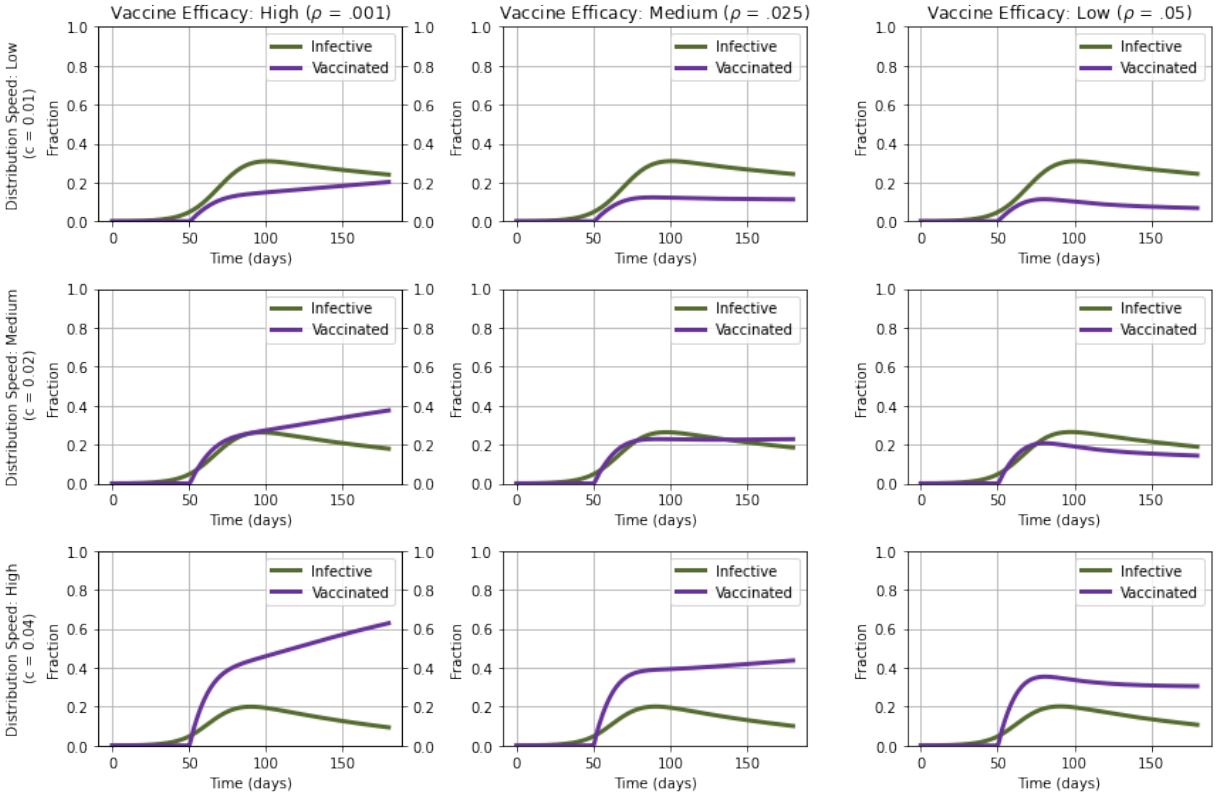

Figure 7: Comparison of Vaccine Speed & Efficacy

We further compared the effects of vaccine speed of distribution and vaccine efficacy, shown in Figure 7. The speed of distribution, also the rate at which vaccinations are administered upon rollout, appears to have a stronger effect on reducing overall infection rates and curbing the maximum proportion of infected individuals on a given date. As shown above, the vaccine efficacy has a more minimal effect in reducing infection rates, although does substantially improve rates of vaccination if more efficacious.

2.5 Constrained Optimization of Vaccine

To determine the most effective manner of vaccine development, we developed a constrained optimization problem. We considered three characteristics of a vaccine's development: ρ – the vaccine failure rate, d – the length of time required for development, and c – the speed of distribution upon development. We first noted our standard parameter values for the above characteristics, which were derived from a scaled version of the actual COVID-19 vaccine development (to a shorter timeline). By utilizing our default parameter values, we formulated the following constraint:

$$g(\rho, d, c) = \frac{(1-\rho)}{0.95} + \frac{d}{100} + 200c - 3 = 0 \tag{12}$$

where we make use of $(1-\rho)$ to correspond to the vaccine efficacy, being the complement of the vaccine failure rate. We formulated this constraint under assumptions regarding the tradeoff between the speed of development and efficacy of a COVID-19 vaccine [7].

We utilize a constrained optimization via `scipy.optimize.minimize` with the trust-constr method. This is a trust-region algorithm for constrained optimization that allows us to assert bounds on parameter values and ensure that the optimized vaccine characteristics are within realistic ranges. As an equality constrained optimization, this makes use of the Byrd-Omojokun Trust-Region SQP method.

We choose two optimizations schemes for the vaccination constraints defined. Firstly, we seek to minimize the total infection throughout the duration of the simulation. Secondly, we seek to minimize the date with the maximum number of infected individuals, a result that will allow us to "flatten the curve" and minimize burden on healthcare facilities.

The results of optimization for total number of infected individuals:

$$\rho = 0.0017$$

$$d = 101$$

$$c = 0.0047$$

Results of optimization for maximum infected individuals at any single time:

$$\rho = 0.042$$

$$d = 100$$

$$c = 0.0069$$

The above results indicate that a goal of minimizing the maximum number of infected individuals on a given day, akin to flattening the curve, results in a prioritization of a vaccine's speed of distribution over the vaccine efficacy. On the contrary, minimizing the total number of infections results in a solution that prioritizes a more efficacious vaccine, with a lower corresponding value of ρ.

2.6 Stochastic Model

To decrease the deterministic quality of the SEIRS model with and without modification, we applied a stochastic term to the system of ODEs based on the Wiener process. In general, this stochastic term has the following form in differential equations:

$$dX_t = f(X_t)X_t dt + \sigma X_t dW_t \tag{13}$$

White noise given by the Wiener process is scaled by the value of X as well as variation parameter σ. This noise term adjusts the original equation. The model still tends toward the deterministic stable state of the SEIRS model, but varies to more accurately reflect real-world data and for more useful comparison to alternative stochastic models.

2.7 Agent-Based Infection Dynamics on Graph

The classical SIR model is expressed through a system of differential equations that describe movement between distinct population groups: susceptible, infected, and recovered. Even in the more complex SEIRS model, this formulation provides three primary advantages. The model is easy to interpret, easy to modify, and relatively straightforward to evolve through time. However, the model assumes a well-mixed population and

nearly deterministic update, even with the addition of the Wiener process. Thus, we propose a stochastic agent-based infection dynamics simulation with population substructure through graph theory to address the limitations of a continuous dynamical system. This model discretizes the system of ODEs into a probabilistic setting, allowing for careful treatment at each time step.

We adapted a nested network Markov chain model from Rader et al. 2020 [9] to create a hierarchy of interaction strengths in the population. The model was originally designed for SIR dynamics by Dr. Alison Hill. An interaction term drives the transition from susceptible to infected; once an individual is infected, they are assigned a randomly sampled exponential waiting time dependent on the average infection duration (recovery parameter γ). Likelihood of infection depends on contact, whose probability is determined by a structured graph underlying the population. The total population is separated into a hierarchy of 3 interaction groups: households, neighborhoods, and districts. Interaction is highest in households, drops by two orders of magnitude in neighborhoods, and drops again by an order of magnitude in districts. Connections between districts are not uniform; only a small number of individuals have these connections, reflective of the power law-like trends seen in social networks.

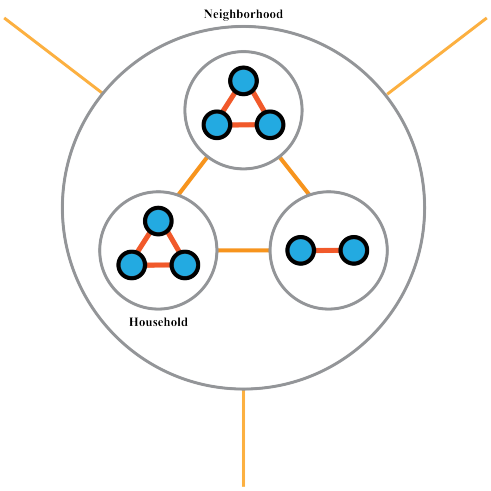

Figure 8: Graph Structure of Agent-Based Network Model

We constructed a SEIRS model from this groundwork by adding an exposed population compartment and allowing the immunity of recovered individuals to wear off over time, which is exactly analogous to the stochastic SEIRS ODE system. The parameterization of the models are equivalent. Deprecated functions and comments were updated for clarity. Networks were visualized using Fruchterman-Reingold force-directed algorithm to separate nodes based on closeness, which is built into the Python package `networkx`. We additionally applied a contact-reducing intervention (social distancing), equivalent to the constant reduction applied to the infection factor β in the SEIRS ODE system. Critically, this intervention only decreased the strength of neighborhood- and district-level interactions, not household interactions.

3 Results

3.1 SEIRS ODE System

From our optimization of vaccine development, we determined the optimal set of parameters to both minimize the total number of infections and to minimize the maximum number of infections on a given day. The resulting outcomes for the each of these respective interventions is shown in the below figure.

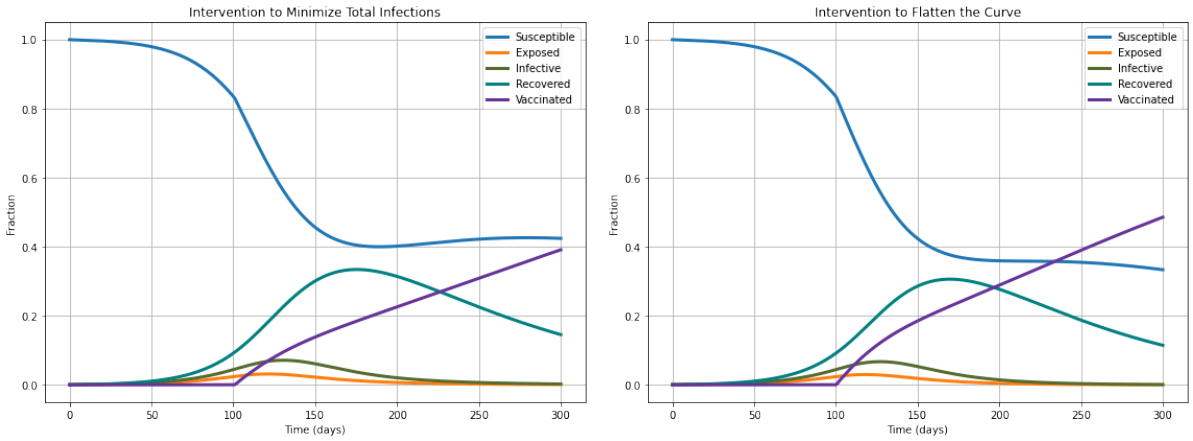

Figure 9: Comparison of Intervention Priorities

While the difference between the respective trajectories for either intervention appears minimal, optimizing for the minimization of total infections results in 12.6 percent fewer total infections as opposed to the alternative intervention. However, optimizing to flatten the curve, or minimize the maximum number of infected individuals on a given day, results in a maximum percentage of infected individuals that is 6.05 percent less than when minimizing for total infections.

3.2 Stochastic Graph Model

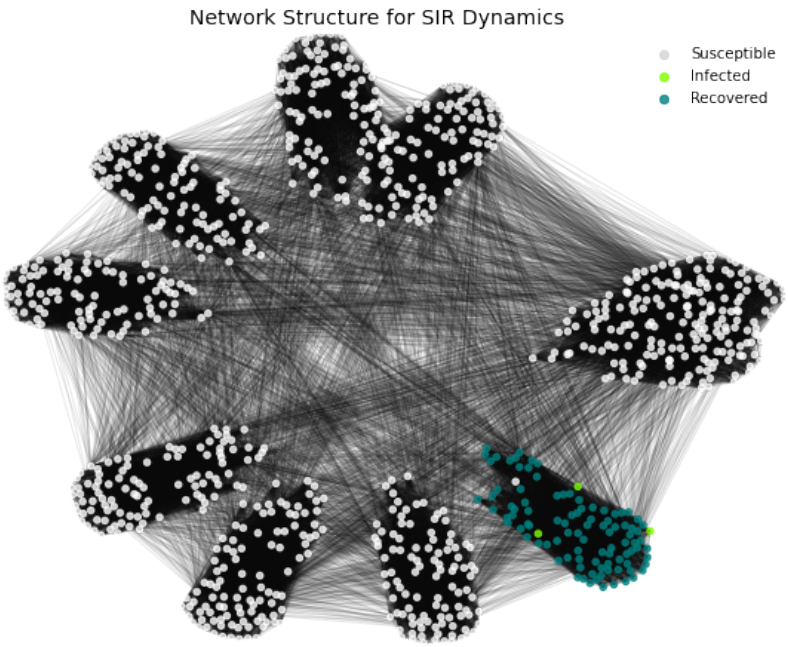

Figure 10: Population State After 50 Days with SIR Dynamics

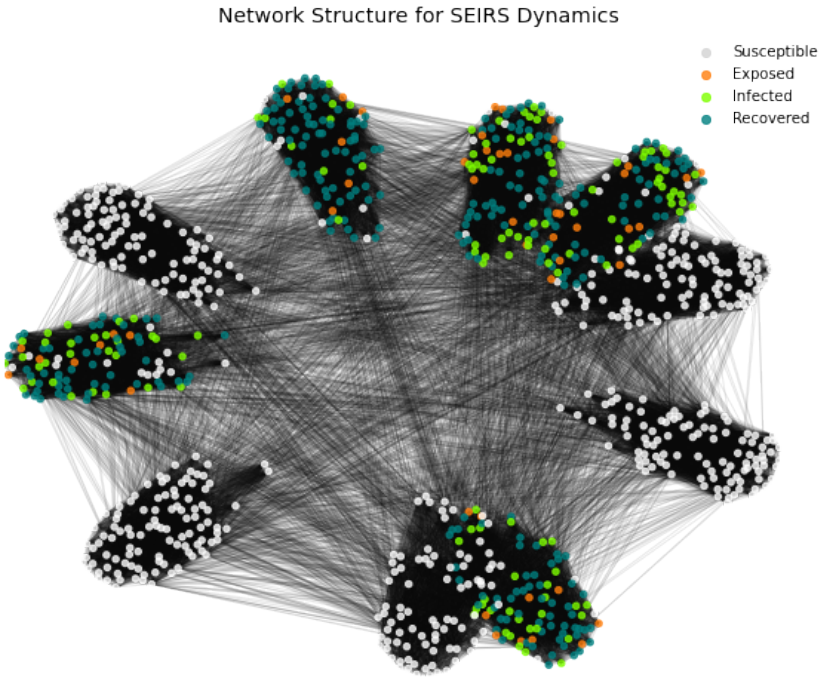

Figure 11: Population State After 50 Days with SEIRS Dynamics

Figure 9 exhibits clustering of infected and recovered individuals in a single neighborhood. Figure 10 demonstrates a broader spread of infectious disease, although the network was initialized with equivalent parameters.

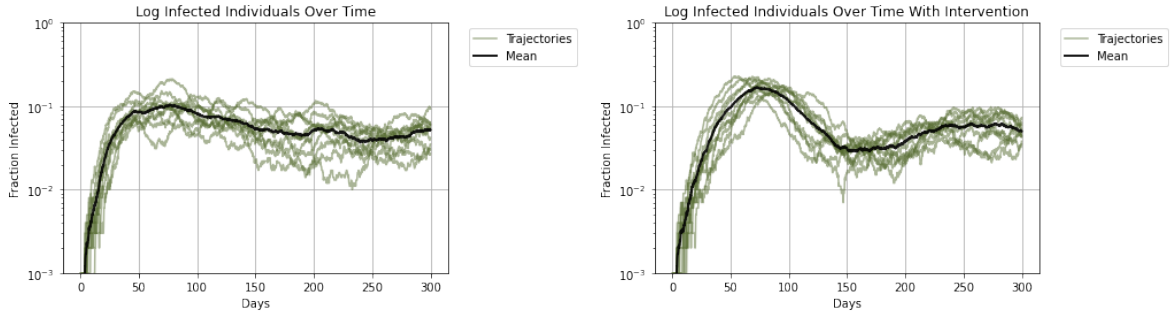

Figure 12: Infection Trajectories for SEIRS Models With and Without Intervention

In Figure 12, we note the complex influence the infection rate parameter β has on the infection dynamics. The intervention dynamics face a 40% reduction in contact rate, but still exhibit a large number of cases.

3.3 Comparative Analysis

Below, we present two figures which compare the two models used in this study: an ODE system and a modified Markov chain with population substructure. Figure 13 is simulated with the standard infection rate of $\beta = .24$, while Figure 14 is simulated with the reduced infection rate of $\beta = .144$.

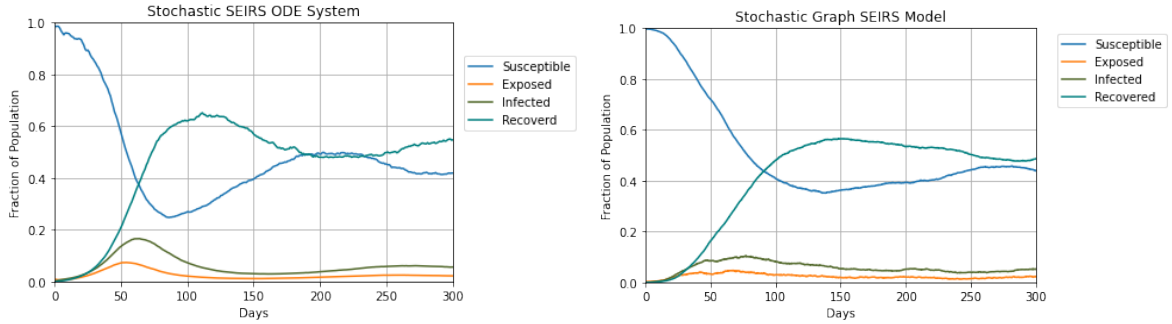

Figure 13: Comparison of Stochastic ODE and Stochastic Graph Models with Normal Contact

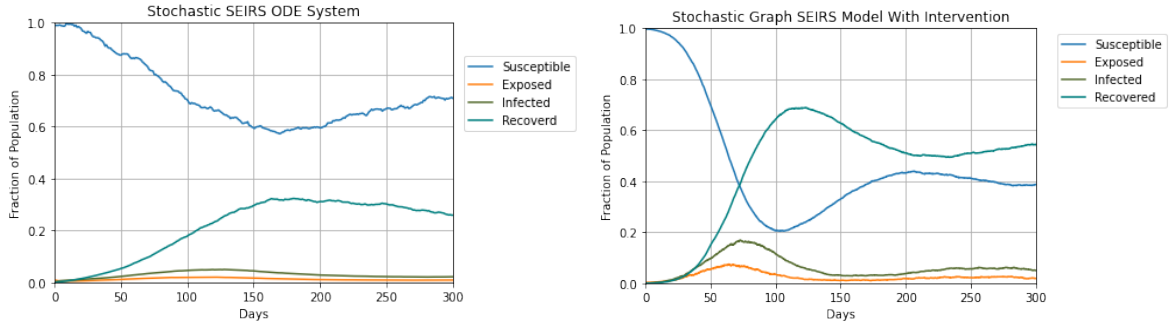

Figure 14: Comparison of Stochastic ODE and Stochastic Graph Models with Reduced Contact

4 Discussion

4.1 Results Interpretation

4.1.1 Optimization of Parameters

Our investigation into the optimization of parameters to curb infection spread seeks to provide information regarding optimal policy interventions. In particular, we focused on both reducing the total infections as well as reducing the maximum infection count on a given day. This allows us to seek policies that both reduce COVID-19 spread and associated deaths as well as flatten the curve to alleviate resource burden on healthcare facilities.

In seeking to implement an intervention to reduce interactions and curb the spread of infection, we observed that a sustained less strict policy is more effective than an immediate, short-duration, strict policy. Our results therefore indicate that the duration of interventions such as quarantining or social distancing is more significant, meaning a prolonged, sustainable social distancing policy should be prioritized over more intensive, shorter-duration quarantine mandates.

Further, as we optimized the development and administration of a vaccine under the constraints outlined in equation (12), we determined the appropriate prioritization of vaccine efficacy and speed of distribution. We observed that in prioritizing the reduction of total infection spread we place greater emphasis on the efficacy of the vaccine as compared to its speed of distribution. On the contrary, prioritizing to reduce the maximum number of infections on a given day results in developing a vaccine that is of comparatively lower efficacy but can be distributed more rapidly. In particular, by optimizing for total infection reduction, we determined a vaccine administration plan that reduces total infection count by 12.6 percent as compared to the alternative intervention. By optimizing to flatten the curve, we reduced the maximum number of infections on a given day by 6.05 percent as compared to the alternative intervention. Hence, our results indicate that determining the desired effect of policy intervention, whether it be reducing total infections or flattening the curve, must be properly determined as even slightly different prioritizations result in substantially different infection outcomes.

4.2 Specifics of SEIRS Model

By nature, a SEIRS model adds a built-in time delay to the infectious behavior. This is demonstrated most clearly in Figs. 10 & 11, where the virus is much more stable with the exposed compartment. While this interim incubation period prevents the virus from reproducing as rapidly, it more consistently is able to find a host and spread between neighborhoods, instead of just within a single one. This shift in time scale is also reflected by the greater number of infected individuals present in Figure 11.

4.2.1 Comparison of SEIRS ODE and Graph Model

The stochastic ODE system and graph model match surprisingly closely in Figure 13; the simulations are instantiated with equal parameters. We see slightly greater magnitude changes in the system of differential equations, but the trends in both are quite similar. This is generally expected, as the models are analogous. However, when introducing the constant reduction to interaction term β, we see drastically different results. In the ODE

system, infections over time are drastically reduced and the infection quickly disappears. In the graph model, we see a trend very similar to the previous infection rate, with a larger initial bump and a lower consistent value.

This result is critical at the level of policy and model interpretation. For the ODE system, decreasing contact (social distancing) seems to drastically reduce the number of infections on its own. However, incorporating population substructure, we see that the infection still spreads throughout households; with the additional incubation period and long infectious period, the simulated virus can escape these hotspots and spread throughout a community at large scale. We also see this trend in Figure 12, where a 40% decrease in contact rate β leads to unexpected changes in infection dynamics. Lower β leads to greater clustering, and a larger number of infected individuals at a single time. It also fails to significantly reduce the total number of cases.

4.3 Limitations

There are inherent limitations in the structure of our SEIRS model, even with the incorporation of additional parameters to account for time-evolved interactions and vaccination policies. In particular, the ODE implementation assumes homogeneity amongst these population subsectors, meaning all individuals within a category are treated the same. Our inclusion of an agent-based network model to demonstrate the differences in such an approach is an attempt to account for some of these assumptions. Furthermore, our model does not account for various different environmental factors, such as a seasonal surge of the COVID-19 virus, or different propensities for illness among individuals given pre-existing conditions.

4.4 Outlook & Future Work

Tuning constant parameters and creating more dynamic time-dependent parameters are likely the most effective directions this project could go. Most classical infection models are effective at capturing a single trend, but lack the expressivity to model a viral history as complex as COVID-19. The network model also suffers from this lack of complexity, although both showed strong results in their respective contexts.

Future models could combine the hierarchical nature of the network with a more direct spatial component, such as a modified reaction-diffusion equation with weights according to probability of interaction.

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References

[1] Mwalili, Samuel, et al. "SEIR model for COVID-19 dynamics incorporating the environment and social distancing." BMC Research Notes 13.1 (2020): 1-5.

[2] "Basics of COVID-19." *CDC* (Nov 2021). https://www.cdc.gov/coronavirus/2019-ncov/your-health/about-covid-19/basics-covid-19.html

[3] "Mortality Analysis." *Johns Hopkins Medicine*. https://coronavirus.jhu.edu/data/mortality

[4] Guterres, António. "COVID-19 in an Urban World." *United Nations*. https://www.un.org/en/coronavirus/covid-19-urban-world

[5] Heffernan, Jane M., Robert J. Smith, and Lindi M. Wahl. "Perspectives on the basic reproductive ratio." Journal of the Royal Society Interface 2.4 (2005): 281-293.

[6] Katella, Kathy. "Comparing the COVID-19 Vaccines: How Are They Different?" *Yale Medicine* (Dec 9, 2022). https://www.yalemedicine.org/news/covid-19-vaccine-comparison

[7] Paltiel, A. David, Amy Zheng, and Jason L. Schwartz. "Speed versus efficacy: quantifying potential tradeoffs in COVID-19 vaccine deployment." Annals of internal medicine 174.4 (2021): 568-570.

[8] Yan, B., Zhang, X., Wu, L., Zhu, H., & Chen, B. (2020). "Why Do Countries Respond Differently to COVID-19? A Comparative Study of Sweden, China, France, and Japan." *The American Review of Public Administration*, 50(6–7), 762–769. https://doi.org/10.1177/0275074020942445

[9] Rader, Ben et al. "Crowding and the Shape of COVID-19 Epidemics." Nature Medicine 26 (Oct 2020): 1829-34. https://doi.org/10.1038/s41591-020-1104-0

[10] "Ending Isolation and Precautions for People with Covid-19." Centers for Disease Control and Prevention, Centers for Disease Control and Prevention, https://www.cdc.gov/coronavirus/2019-ncov/hcp/duration-isolation.html.