Abstract. The COVID-19 pandemic has posed a policy making crisis where efforts to slow down or end the pandemic conflict with economic priorities. This paper provides mathematical analysis of optimal disease control policies with idealized compartmental models for disease propagation and simplistic models of social and economic costs. The optimal control strategies are categorized as 'suppression' and 'mitigation' strategies and are analyzed in both deterministic and stochastic models. In the stochastic model, vaccination at an uncertain time is taken into account.

Key words. Optimal control, SIR model, COVID-19

AMS subject classifications. 92D30; 93C95, 90C90

1. Introduction. The spread of infectious diseases in a population can be modeled in a variety of ways. Here we focus on the optimization and control of stochastic and deterministic ‘compartmental’ models, where the population is grouped into a handful of states that represent the progression of the disease. These models have the benefit of having relatively few variables and parameters, and can be comprehended intuitively. Our analysis focuses on qualitative aspects of the optimal control policies with an aim to better understand the effective strategies for combating COVID-19. We find optimal control policies that fall into one of two categories that are widely used to describe real world responses: ‘suppression’ strategies that aim to eliminate the disease in a population, and ‘mitigation’ strategies that aim to reduce the negative impact of the disease.

1.1. Literature Review. There is a long history of ‘compartmental’ models including previous research into optimization and control and applications to the COVID-19 pandemic. We provide a non-exhaustive summary of relevant works. The optimal control of deterministic and stochastic Susceptible-Infectious-Recovered (SIR) compartmental models is analyzed in [17]. The control variable adjusts the rate at which to remove individuals from the infected population by isolation. The solutions that are found either expend all or none of the resources. Deterministic SIR and Susceptible-Exposed-Infectious-Recovered (SEIR) models are considered in [1] with control by vaccination, quarantine, screening, or health campaigns. It is found that in all cases the optimal policy is maximum effort on an initial time interval. A similar finding was made for an SEIR type model applied to COVID-19 in [12] with control on the transition rate. An SEIR model with logistic population growth is considered in [16], where the control found by numerical optimization appears to approach a state where the disease is endemic in the population. An SEIR type model has been applied to COVID-19 in [5] with the infection rate controlled up until a vaccine is developed. The optimal control found suppresses the disease until near the development of the vaccine when the control is relaxed. A variation is considered [8] where instead of...
vaccination it is assumed that control reverts back to the norm, and the result is a mitigation strategy that brings the susceptible population to herd immunity. A deterministic SIR model with control by vaccination and isolation is considered in [7]. In contrast with other results, cases are found when the optimal policy is not unique.

A discounted deterministic Susceptible-Infectious-Susceptible (SIS) model with two regions appears in [14], where the allocation of medical resources between the two resources is optimized. The main finding is that it is better to treat the region with less disease first. A review of epidemic models and control was undertaken in [11] with an emphasis of effects from the network of individuals (not seen in compartmental models). The report [6] provides an analysis of ‘suppression’ and ‘mitigation’ strategies for the COVID-19 pandemic, although optimality is not addressed. A spatial SIR model is considered in [9] with control of spatial dynamics.

The distinction between open loop and feedback control for a stochastic epidemic simulation model is studied in [2], where the feedback control is found to perform better because the approximate model loses accuracy across the entire time domain.

1.2. Model. Our main contribution is to study the optimal control of models pertaining to COVID-19 with control implemented through influencing the infection rate, e.g., by social distance policies. We consider a model that has six states: susceptible, exposed, infected, hospitalized, recovered, and dead. We optimize a cost comprised of the cost of hospitalizations, the cost of deaths, and the social/economic costs of reducing the infection rate by social distancing policies. The model terminates when the disease has become extinct in the population. We also account for future development of vaccines, which removes individuals from the susceptible population at a fixed rate.

A common theme of epidemic models is that the long time behavior is determined by a single ‘reproduction number,’ which when greater than 1 corresponds to exponential growth and when less than 1 corresponds to exponential decline of the disease in the population. This number will be frequently referenced in our analysis and can be computed easily from the model parameters and the control variable.

1.3. Results. Our first result is to find two locally optimal strategies in the deterministic model that we characterize as ‘suppression’ and ‘mitigation’ strategies. The ‘suppression’ strategy reduces the reproduction number well below 1 and it remains below 1 until the disease goes extinct. This strategy is qualitatively similar to the strategies in the literature that apply maximum control at the beginning and also resembles the strategy up to vaccination time found in [5]. The ‘mitigation’ strategy instead applies control around the peak of the epidemic rather than at the initial time. The reproduction number is then brought below 1 by a combination of control and the development of herd immunity. By the time the disease goes extinct, the majority of the population will have gotten infected when following the ‘mitigation’ strategy. This strategy is similar to what is found in [8]. We find both of these solutions exist as local optima with reasonable parameters for COVID-19, while the suppression strategy is the global minimum.

The stochastic model naturally provides a globally optimal feedback control, which yields threshold values of infected populations for which to apply different policies. This model captures the fluctuations of the extinction time, which leads to high variability of the cost for the ‘suppression’ strategy. The development of a vaccine allows the optimal feedback control to lessen in many cases, however, when the number of cases is still small, the optimal feedback control strengthens to reduce the infection rate upon development of the vaccine.
The code (written in Python with NumPy and Matplotlib for figures) and numerically generated data (JSON format) that we used is available at https://github.com/AaronZPalmer/SEIHRD.git.

2. Epidemic Model.

2.1. Susceptible-Exposed-Infectious-Hospitalized-Recovered-Dead (SEIHRD) Model. We assume that individuals are indistinguishable and reside in one of six states: susceptible, exposed, infectious, hospitalized, recovered, and dead.

The state variables, which represent number of individuals in each state at a given time, are:
- \( S_t \), the number of ‘susceptible’ individuals at time \( t \);
- \( E_t \), the number of ‘exposed’ individuals at time \( t \);
- \( I_t \), the number of ‘infectious’ individuals at time \( t \);
- \( H_t \), the number of ‘hospitalized’ individuals at time \( t \);
- \( R_t \), the number of ‘recovered’ individuals at time \( t \);
- \( D_t \), the number of ‘dead’ individuals at time \( t \).

The total population size \( N \) accounts for all individuals: \( N = S_t + E_t + I_t + H_t + R_t + D_t \).

We assume that the following individual transitions occur at exponentially distributed times:
- Susceptible to exposed at rate \( \beta \frac{I_t}{N} \);
- Exposed to infectious at rate \( \alpha \);
- Infectious to hospitalized at rate \( \lambda_0 \);
- Infectious to recovered at rate \( \gamma_0 \);
- Infectious to dead at rate \( \delta_0 \);
- Hospitalized to recovered at rate \( \gamma_1 \);
- Hospitalized to dead at rate \( \delta_1 \).

The transition of any susceptible person to become infected, in other words \( S_t \) becomes \( S_t - 1 \) occurs as the first of \( S_t \) independent random times with exponential distributions of rate \( \beta \frac{I_t}{N} \). The first of these times that marks the transition from \( S_t \) to \( S_t - 1 \) is exponentially distributed with rate \( \beta S_t \frac{I_t}{N} \). The same holds for the other transitions. An illustration of the SEIHRD rate transition diagram is in Figure 1. Figure 1 also illustrates the simplified \( SIR \) model that we used in our stochastic analysis.

When \( N \) is large and the macroscopic state variables are of order \( N \), we can approximate the stochastic model by the system of differential equations

\[
\frac{dS_t}{dt} = -\beta S_t \frac{I_t}{N} \\
\frac{dE_t}{dt} = \beta S_t \frac{I_t}{N} - \alpha E_t \\
\frac{dI_t}{dt} = \alpha E_t - \gamma_0 I_t - \lambda_0 I_t - \delta_0 I_t \\
\frac{dH_t}{dt} = \lambda_0 I_t - \gamma_1 H_t - \delta_1 H_t \\
\frac{dR_t}{dt} = \gamma_0 I_t + \gamma_1 H_t \\
\frac{dD_t}{dt} = \delta_0 I_t + \delta_1 H_t.
\]
2.2. Equilibria. After a sufficiently long time the solutions of (2.1) will approach equilibrium points where the right hand sides of the differential equations are zero. An equilibrium requires that $E = I = H = 0$, since $\frac{dH}{dt} = 0$ and $\frac{dR}{dt} = 0$ imply $I = H = 0$, and then $\frac{dE}{dt} = 0$ implies $E = 0$. It is then clear that $S$, $R$, and $D$ can take any values in equilibrium and this allows us to determine all of the equilibrium points. To illustrate the SEIHHRD model reaching equilibria, Figure 2 graphs a particular solution.

We investigate the Jacobian matrix and its eigenvalues to parametrically characterize the system stability near equilibrium. The Jacobian matrix is

$$
\begin{pmatrix}
-\frac{\beta I}{N} & 0 & -\frac{\beta S}{N} & 0 & 0 & 0 \\
-\alpha & \frac{\beta I}{N} & 0 & 0 & 0 & 0 \\
0 & \alpha & -\left(\lambda_0 + \gamma_0 + \delta_0\right) & 0 & 0 & 0 \\
0 & 0 & \lambda_0 & -\gamma_1 - \delta_1 & 0 & 0 \\
0 & 0 & \gamma_0 & \gamma_1 & 0 & 0 \\
0 & 0 & \delta_0 & \delta_1 & 0 & 0
\end{pmatrix}.
$$

At an equilibrium point, the first column becomes zero because $I = 0$, and we can compute the eigenvalues $\epsilon$ by finding the roots of the characteristic polynomial, solving

$$
0 = det \begin{pmatrix}
-\epsilon & 0 & -\frac{\beta S}{N} & 0 & 0 & 0 \\
0 & -\alpha - \epsilon & \frac{\beta S}{N} & 0 & 0 & 0 \\
0 & \alpha & -\left(\lambda_0 + \gamma_0 + \delta_0\right) - \epsilon & 0 & 0 & 0 \\
0 & 0 & \lambda_0 & -\gamma_1 - \delta_1 - \epsilon & 0 & 0 \\
0 & 0 & \gamma_0 & \gamma_1 & -\epsilon & 0 \\
0 & 0 & \delta_0 & \delta_1 & 0 & -\epsilon
\end{pmatrix},
$$

which yields

$$
0 = \epsilon^3(\gamma_1 + \delta_1 + \epsilon)\left((\alpha + \epsilon)(\lambda_0 + \gamma_0 + \delta_0 + \epsilon) - \alpha \beta \frac{S}{N}\right).
$$
Fig. 2. Solution to SEIHRD model with parameter settings as in Table 1, using $\beta = 0.87$ so that $R_0 = 4$. The initial values are given in (2.4).

There is a zero eigenvalue with multiplicity three, $-\gamma_1 - \delta_1$ is an eigenvalue, and the other two eigenvalues solve

$$0 = \epsilon^2 + (\alpha + [\lambda_0 + \gamma_0 + \delta_0])\epsilon + \alpha [\lambda_0 + \gamma_0 + \delta_0] - \alpha \beta \frac{S}{N}$$

so

$$\epsilon = -\frac{(\alpha + [\lambda_0 + \delta_0 + \gamma_0])}{2} \pm \sqrt{\left(\alpha + [\lambda_0 + \delta_0 + \gamma_0]\right)^2 - 4 \alpha ([\lambda_0 + \delta_0 + \gamma_0] - \beta \frac{S}{N})}.$$ 

Both of these eigenvalues are negative if and only if

$$\lambda_0 + \delta_0 + \gamma_0 > \beta \frac{S}{N}.$$ 

As common in analysis of epidemic models, we define the reproduction number

$$(2.2) \quad R_0 = \frac{\beta}{\lambda_0 + \delta_0 + \gamma_0}$$

and the effective reproduction number

$$(2.3) \quad R_e = R_0 \frac{S}{N} = \frac{\beta \frac{S}{N}}{\lambda_0 + \delta_0 + \gamma_0}.$$ 

When $R_e < 1$, the disease will cease to propagate. When this is reached due to a decrease of the susceptible population, it is said that herd immunity has developed.

The following theorem classifies the long time behavior for nonnegative solutions to (2.1). We sketch an informal argument.
Fig. 3. Solution to SEIHRD model with parameter settings as in Table 1, using $\beta = 0.11$ so that $R_0 = 0.5$. The initial values are given in (2.4). Note that the susceptible population $S$ appears in a separate graph due to the difference in magnitude.

**Theorem 2.1.** Any equilibrium with $R_e > 1$ is unstable. If the initial condition has $E_0 + I_0 > 0$, then the solution will tend to an equilibrium with $S \leq N / R_0$.

If $R_0 < 1$ (and $S \leq N$) then every equilibrium is stable (but not asymptotically stable).

**Proof.** The calculation above shows that the Jacobian matrix at an equilibrium point has a positive eigenvalue if $R_e > 1$ making the equilibrium unstable. Whenever $R_e > 1$ and $I > 0$, the infected population $I$ will eventually increase because of the positive eigenvalue. The equation will eventually reach an equilibrium and it must have $R_e \leq 1$ so $S \leq N / R_0$.

When $R_e < 1$ the equilibrium is stable because $I, H, E$ will all tend to zero asymptotically, which causes the other variables to remain near their initial values. When $R_0 < 1$ and $S \leq N$, $R_e < 1$ so all such equilibrium points are stable.

An illustration of a solution with $R_0 = 1/2 < 1$ is given in Figure 3. Note that, in Figure 3, the equilibrium is reached with a susceptible population close to the initial population, consistent with $R_0$ being well below one, whereas, in Figure 2, nearly the entire population becomes infected, with the susceptible population near zero, consistent with $R_0 = 4$.

We plot the equilibrium value of $S$ at a large time $T$ for a range of $R_0$ values in Figure 4 as a result of simulation and the theoretical bound of Theorem 2.1. The $R_0$ value of COVID-19 without interventions has been estimated to be greater than 2, which would result in the majority of the population becoming infected. A clear qualitative feature shown in Figure 4 is that $S_T$ is near $N$ when $R_0 \leq 1$ and the final state of $S_T$ always lies below the bound of $N / R_0$. We find in Section 3.3 that the optimal ‘mitigation’ strategy achieves the end result of $S_T \approx N / R_0$. 
Fig. 4. The final state $S_T$ as function of $R_0$, the theoretical bound in red and simulations in yellow. Parameters are as in Table 1 with $\beta$ ranging as a function of $R_0$, $\beta = (\lambda_0 + \gamma_0 + \delta_0) R_0$. The end time for the simulations is taken to be $T = 10,000$ days.

2.3. Parameter settings for the SEIHRD model of COVID-19. We use the parameters from a prior study (see appendix in [10]). Each parameter of Table 1 is a rate per day per person except for $N$.

| $\alpha$ | 0.192 | The exposed to infected rate is the reciprocal of the mean incubation period 5.2 days. [10] |
| $\beta$ | 0.11 $\sim$ 0.87 | A range of infection rates, corresponding to values of $R_0$ in the range 0.5 $\sim$ 4. [3, 15] |
| $\lambda_0 + \gamma_0 + \delta_0$ | 0.217 | The recovery is the reciprocal of the mean infectious period, 4.6 days. [10] |
| $\lambda_0$ | 0.0264 | The hospitalization rate is inferred from 12% hospitalized among confirmed cases. [4] |
| $\gamma_0$ | 0.189 | The recovery rate is inferred from 87% recovered on their own. |
| $\delta_0$ | 0.002 | The death rate is inferred from 1% die on their own. |
| $\gamma_1$ | 0.1 | The recovery in hospital rate is the reciprocal of the mean length of hospital stay 10 days. [10] |
| $\delta_1$ | 0.031 | The hospital death rate is inferred from 23.5% of those hospitalized die, so that $0.235 = \frac{\delta_1}{\gamma_1 + \delta_1}$. [10] |
| $N$ | 7,600,000 | Population of Washington State. |
We choose the initial values as reasonable values
\[
\begin{pmatrix}
S_0 \\
E_0 \\
I_0 \\
H_0 \\
R_0 \\
D_0
\end{pmatrix} =
\begin{pmatrix}
N - 5000 \\
2000 \\
3000 \\
0 \\
0 \\
0
\end{pmatrix}.
\]
This is used in the examples unless otherwise stated.

3. Optimal Control. We now pose an optimal control problem supposing that $\beta$ can be controlled over time through social distancing and other non-pharmaceutical interventions.

3.1. Deterministic dynamics. We assume the dynamics of (2.1), and we optimize over the end time $T$ and control policy $(\beta_t)_{t \in [0,T]}$. The cost consists of a running control cost $L$, a running hospitalization cost $F$, and a terminal death cost $G$, which has the form
\[
J[(\beta_t)_{t \in [0,T]}, T] = \int_0^T \left[ L(\beta_t) + F(H_t) \right] dt + G(D_T).
\]
We constrain the final state by
\[
E_T + I_T + H_T \leq e^{-1}
\]
to represent that the disease has gone extinct. The end time $T$ is the first time for which this constraint is satisfied, called the extinction time. Thus the optimal control problem has the following elements:
- Decision variables $T$ and $(\beta_t)_{t \in [0,T]}$;
- Dynamics determined by (2.1);
- Objective function (3.1);
- Terminal constraint (3.2).

Note that while the solution to the SEIHRD model scales linearly with the population size $N$, the extinction time is nonlinear in $N$. This is important in interpreting the optimal control policy, and its dependence on population size and extinction time.

We recall the dynamics (2.1) and define $(\Sigma_t)_{t \in [0,T]} = (S_t, E_t, I_t, H_t, R_t, D_t)$ and let $f(\Sigma, \beta) \in \mathbb{R}^d$ denote the right-hand side, i.e.,
\[
\frac{d\Sigma_t}{dt} = f(\Sigma_t, \beta_t).
\]
We let $(P_t)_{t \in [0,T]} = (P^S_t, P^E_t, P^I_t, P^H_t, P^R_t, P^D_t)_{t \in [0,T]}$ be the costate and define the Hamiltonian
\[
\mathcal{H}(\Sigma, P, \beta) = f(\Sigma, \beta) \cdot P - L(\beta) - F(H).
\]
Then we let $(P_t)_{t \in [0,T]}$ solve the costate equation, applying a differential operator to the Hamiltonian,
\[
\frac{dP_t}{dt} = -D_{\Sigma} \mathcal{H}(\Sigma_t, P_t, \beta_t),
\]
with
\[
\begin{pmatrix}
P^S_T \\
P^E_T + \sigma \\
P^L_T + \sigma \\
P^H_T + \sigma \\
P^R_T \\
P^D_T
\end{pmatrix}
= -D_2 G(\Sigma_T).
\]

Here \(\sigma \geq 0\) is a Lagrange multiplier for the target constraint. The free end time yields the additional transversality condition
\[
(3.5) \quad \sup_\beta \mathcal{H}(\Sigma_T, P_T, \beta) = 0.
\]

The Pontryagin maximum principle states:

**Theorem 3.1.** If \(T\) and \((\beta_t)_{t \in [0,T]}\) are optimal and \((\Sigma_t)_{t \in [0,T]}\) solves (2.1) with (3.2) satisfied, then there is \(\sigma \geq 0\) and \((P_t)_{t \in [0,T]}\) that solves (3.4) with terminal conditions, such that \(\sigma (I_T + E_T + H_T - e^{-1}) = 0\), the transversality condition (3.5) is satisfied, and for almost every \(t \in [0,T]\),
\[
\beta_t \in \text{argmax}\{\mathcal{H}(\Sigma_t, P_t, \cdot)\}.
\]

We let \(J_\sigma\) denote the augmented cost
\[
J_\sigma[(\beta_s)_{s \in [0,T]}, T] = J[(\beta_s)_{s \in [0,T]}, T] + \sigma (I_T + E_T + H_T - e^{-1}),
\]
and note that for \(\sigma\) of Theorem 3.1, \(T\) and \((\beta_t)_{t \in [0,T]}\) minimize \(J_\sigma\) over policies unconstrained by the extinction threshold (3.2). For any smooth \((\beta_t)_{t \in [0,T]}\), and solutions \((\Sigma_t)_{t \in [0,T]}\) and \((P_t)_{t \in [0,T]}\), we can calculate the functional derivative
\[
(3.6) \quad D_{(\beta_s)_{s \in [0,T]}\}J_\sigma[(\beta_s)_{s \in [0,T]}, T]}(t) = -D_\beta \mathcal{H}(\Sigma_t, P_t, \beta_t),
\]
and
\[
(3.7) \quad D_T J_\sigma[(\beta_s)_{s \in [0,T]}, T] = -\mathcal{H}(\Sigma_T, P_T, \beta_T).
\]

Note that even if \(L\), \(F\), and \(G\) are convex the problem may not be convex due to the nonlinear dynamics. However, we use a discretized version of the functional gradients to search for local optima.

We select cost functions for the optimal control criterion in (3.1) by a phenomenological approach:

\[
(3.8) \quad L(\beta) = N k \left( - \log \left( \frac{\beta}{b} \right) + \frac{\beta}{b} - 1 \right),
\]
\[
(3.9) \quad F(H) = c \left( H + \frac{1}{2N} H^2 \right),
\]
\[
(3.10) \quad G(D) = d D.
\]

We assume that \(L(\beta) = +\infty\) if \(\beta \leq 0\). We note that \(L\) is convex, \(L(\beta) \geq 0\) with \(L(b) = 0\). The values of \(L\) for \(\beta \geq b\) are not important as they are never optimal. The choice of a logarithmic term in \(L\) is consistent with the phenomenon that independent methods of intervention have additive costs and multiplicative reduction of \(\beta\). A quadratic term is included in \(F\) to reflect the cost of passing the hospital occupancy threshold. The linear death cost reflects each human life being of equal value.
3.2. Control parameter settings. The cost functions of (3.8), (3.9) and (3.10) have four parameters: the control cost coefficient \( k \), the baseline infection rate \( b \), the hospitalization cost rate \( c \) and the death cost coefficient \( d \). We attempt to choose reasonable values in USD, shown in Table 2, though further specification is ultimately subjective. It is hopeful that more efficient handling of the epidemic would significantly lower the control cost coefficient, \( k \).

| \( d \) | \$1,000,000 per person | US department of transportation values life at \$9,600,600; this accounts for approximately 10% shortened lifespan. |
| \( b \) | \( 0.87 \) per day per person | For \( R_0 = 4 \). |
| \( c \) | \$3,500 per day per person | Calculated for a total cost per patient of \$35,000 divided by an average stay of 10 days. [13] |
| \( k \) | \$100 per day per person | A subjective value chosen to be a round ballpark number; at \( R_0 = 1/2 \), the cost is \$120 a day per person; at \( R_0 = 1 \), the cost is \$64 a day per person. |

3.3. Deterministic dynamics results. Numerically, we find two locally optimal solutions, the ‘suppression’ strategy where \( \beta \) stays low enough so that \( R_0 < 1 \) and the ‘mitigation’ strategy where \( \beta \) is near \( b \) except at the peak of the epidemic, and the epidemic runs its course. This qualitative finding is not sensitive to the choices of parameters.

The two locally optimal solutions, ‘suppression’ strategy and ‘mitigation’ strategy, are plotted in Figure 5 and Figure 6, respectively. The globally optimal solution is the ‘suppression’ strategy, with a cost about a third of the cost of the ‘mitigation’ strategy. We find:

- The cost of ‘suppression’ strategy is \$12,535 per person, a total cost of nearly \$100 billion, and is mostly from the control cost and the cost of the ‘mitigation’ strategy is \$33,694 per person, a total cost of over \$250 billion, and mostly from the cost of deaths.
- In the ‘suppression’ strategy \( T = 74 \) and in the ‘mitigation’ strategy \( T = 5274 \) (only the first 365 days are shown in Figure 6). The long time to extinction in the ‘mitigation’ strategy is due to the end state being very near to \( S_T = \frac{N}{R_0} \), making \( R_e = 1 \) (recall this is the theoretical upper bound of Theorem 2.1 and Figure 4). If, for example, the death cost coefficient \( d \) was lower, the solution would reach an end state \( S_T < \frac{N}{R_0} \) in a much shorter period of time.
- The cost of the ‘suppression’ strategy is proportional to \( NT \), and \( T \) is determined by the extinction threshold. The control variable \( \beta \) is kept low so that \( I_t, E_t, \) and \( H_t \) decay exponentially, and extinction time is achieved relatively quickly. If the whole model is scaled by \( N \), and the extinction threshold remains constant, then the end time \( T \) is proportional to \( \log N \). The cost of the ‘mitigation’ strategy is proportional to \( N \) but less influenced by the extinction time, so the control variable \( \beta \) can reach the initial, uncontrolled, infection rate \( b \).
- The Lagrange multiplier \( \sigma \) is \$2,865,200,000 for the ‘suppression’ strategy, and \$4,256,000 for the ‘mitigation’ strategy. This reflects the marginal cost
of the terminal constraint, which is heuristically the savings for being allowed to stop while one person is still infected.

Recall we have computed the effective reproduction number $R_e = \frac{\beta}{\lambda_0 + \gamma_0 + \delta_0}$. This gives a useful perspective on the qualitative nature of the two solutions. Figure 7 compares the effective reproduction number of the two strategies over time.

3.4. Stochastic considerations. The deterministic model is an approximation to a stochastic model, and the quality of the approximation is impacted by population size $N$. The error of the deterministic approximation to the stochastic model at a given time is proportional to the standard deviation of the macroscopic state variables, which is of order $\sqrt{N}$ so that the relative error, of order $\frac{1}{\sqrt{N}}$, becomes small when $N$ is large. However, a feature that is not captured in the deterministic model is that the time to reach equilibrium is finite in the stochastic model and infinite in the deterministic model. We have accounted for this by introducing a threshold to mark extinction of the disease. If $R_0 < 1$, the time to extinction is of order $\log(N)$, and the error of the deterministic approximation is again proportional to the standard deviation, which is of order $\sqrt{\log(N)}$. At least for the ‘suppression’ type strategy, the cost is of order $NT \sim N \log(N)$ and the relative error grows if we scale by $N$.

A second interesting note is that when approaching this problem from a dynamic programming perspective, the discretization of the deterministic model naturally leads to a stochastic interpretation by addition of numerical viscosity. We proceed to take
this dynamic programming perspective and properly account for the fluctuations of the stochastic SEIRHD model.

An alternative approach to account for the stochastic fluctuations, which we do not consider here, is to approximate the problem near the deterministic solution as a linear quadratic Gaussian (LQG) stochastic optimal control problem. The LQG problem can then be solved as a system of Ricatti differential equations. It is not clear if the LQG approach can account for the fluctuations of the extinction time.

**Fig. 6.** The ‘mitigation’ strategy solution. Parameters and approximation are the same as Figure 5.

**Fig. 7.** The effective reproduction number in each strategy. In the mitigation strategy, $R_e$ approaches 1 asymptotically with these parameters.
since it is usually done with a fixed end time, but it has proven very effective in many applications and can also account for noisy and incomplete observations of the state variables.

3.5. **Stochastic simplified problem.** We now use the dynamic programming approach to better understand the stochastic nature of the epidemic model. This approach also has the feature that it finds the globally optimal strategy, where as the approach of Section 3.1 centered around finding local optima.

The dynamic programming approach is not practical to solve with six state variables, as it would require a computational complexity on the order of $N^6$. Instead we reduce to a simplified three state model $\tilde{S}, \tilde{I}, \tilde{R}$, as depicted in Figure 1, which can then be solved by reducing the dimension to two, by setting $\tilde{R} = N - \tilde{S} - \tilde{I}$ and finally applying a courser discretization to the remaining state variables. We approximate the parameters for the simplified model so that the solution can be fed back to the full stochastic SEIHRD model.

For the simplified model there are two states $\tilde{S}$ and $\tilde{I}$. A transition occurs from $(\tilde{S}, \tilde{I})$ to $(\tilde{S}-1, \tilde{I}+1)$ at rate $\beta \tilde{S} \frac{\tilde{I}}{N}$ (neglecting the effect of the incubation period), and a transition occurs from $(\tilde{S}, \tilde{I})$ to $(\tilde{S}, \tilde{I}-1)$ at rate $\gamma \tilde{I}$, where $\gamma = \lambda_0 + \gamma_0 + \delta_0 \approx 0.217$. We can then approximate $\tilde{H} = \lambda_0 \tilde{I}$ with $\lambda_0 = \frac{\lambda}{\gamma_0 + \delta_0} \approx 0.202$ by considering the quasi-equilibrium when $I$ is near constant, and similarly $\tilde{D} = \delta \tilde{R}$ with $\delta = \frac{\delta_0 + \gamma_0 + \lambda_0}{N} \approx 0.038$.

We run the model until the infection dies out, i.e., when $\tilde{I} = 0$ and $T = \inf\{t; \ I_t = 0\}$. We now assume $\beta$ has a feedback form, $(\beta) = (\beta(\tilde{S}, \tilde{I}))_{(\tilde{S}, \tilde{I}) \in \{0, \ldots, N\}^2}$. The cost is

$$J[(\beta)] = \mathbb{E}\left[ \int_0^T \left( (\beta(\tilde{S}_t, \tilde{I}_t)) + F(\tilde{H}_t) \right) dt + G(\tilde{D}_T) \right],$$

where $\tilde{H}$ and $\tilde{D}$ are approximated as above, and $L, F$, and $G$ are the same as (3.8), (9), and (3.10).

We solve for the value function

$$V(\tilde{S}, \tilde{I}) = \sup_{(\beta)} \left\{ - \mathbb{E}\left[ \int_0^T \left( (\beta(\tilde{S}_t, \tilde{I}_t)) + F(\tilde{H}_t) \right) dt + G(\tilde{D}_T) \right] ; \ (\tilde{S}_0, \tilde{I}_0) = (\tilde{S}, \tilde{I}) \right\},$$

which satisfies $V(\tilde{S}, 0) = -G(\tilde{D})$ and solves the Bellman equation

$$\max_{\beta} \left\{ \beta \tilde{S} \frac{\tilde{I}}{N} (V(\tilde{S}+1, \tilde{I}-1) - V(\tilde{S}, \tilde{I})) + \gamma \tilde{I} \left( V(\tilde{S}, \tilde{I}-1) - V(\tilde{S}, \tilde{I}) \right) - L(\beta) - F(\tilde{H}) \right\} = 0.$$

It is an interesting challenge how to approximate the solution at a coarser discretization of the population variables in order to handle large $N$. Special care must be taken near $\tilde{I} = 0$ to ‘renormalize’ the coefficients and accurately take into account the logarithmic behavior of the extinction time. More details are given in Appendix B.

3.6. **Stochastic simplified problem results.** We plot five simulations of the SEIHRD model under ‘optimal control’ from the simplified $\tilde{SIR}$ model (the correspondence between the simulations and the value function is only approximate), see Figure 8. As expected there is variability in the cost due to the fluctuations of the end time. For this approach we find only the globally optimal, ‘suppression’, strategy. To illustrate the ‘mitigation’ strategy we plot the same simulation but with $d = \$100,000$.
Fig. 8. Five simulated optimal solutions, with the same parameters as in Figure 5, except initial condition $E_0 = 20,000$ and $I_0 = 30,000$. Note the fluctuations in the end time, while the other fluctuations are not noticeable. Individual simulations can be identified by matching the time of extinction with the point the cost flattens.

(maybe unreasonably low) in Figure 9. The low death coefficient cost significantly reduces the cost of the ‘mitigation’ strategy but has little effect on the cost of ‘suppression’ strategy. While there is still variability in the end time with the ‘mitigation’ solution, it no longer has much effect on the cost.

The main qualitative difference in the solutions from the finite time horizon, deterministic dynamics problem is that there is now a finite time where the infection dies out and the control returns to $\beta = b$. The feedback control $\beta$ is plotted as a function of the infected population in Figure 10, where ‘suppression’ and ‘mitigation’ regimes can be identified.

We also plot the optimal $\beta$ for differing values of $\tilde{S}$ and $\tilde{I}$ in Figure 10.

3.7. Switching times for discrete $\beta$. Instead of allowing continuous values for $\beta$ we now consider when $\beta$ is restricted to only four values calculated from $R_0 = 0.5, 1, 2,$ and $4$. The motivation for discretizing $\beta$ is to reflect four policies that impact $R_0$ through non-pharmaceutical interventions. The “switching time” between policies is shown in terms of infected and susceptible population size, providing feedback information on when to change policies. Figure 10 shows how this affects the feedback control policy. Indeed, the optimal policy is very close to the policy with continuous $\beta$ rounded to the nearest admissible value. There is little qualitative difference in the
Fig. 9. Simulated optimal solutions, with a much smaller death cost coefficient, \( d = 100,000 \). The other parameters are as in Figure 8.

Fig. 10. The optimal \( \beta \) values for a range of infected population sizes with the susceptible population fixed on the left, and a range of susceptible population values with the infected population size fixed on the right. Same parameters as Figure 8.

Simulated solutions from Figures 8 and Figure 9 so we do not plot them. In particular, the suppression strategy maintains \( \beta \) at the lowest possible value until the disease has gone extinct, whereas the mitigation strategy with reduced death cost coefficient \( d = 100,000 \) only reduces \( R_0 \) to 2 at the peak of the epidemic.

4. Vaccinations. We can include vaccinations by adding an additional discrete variable \( u \) with binary values, \( \{0, 1\} \). Here, \( u = 0 \) represents that the vaccine is under development and not available, while \( u = 1 \) represents that the vaccine is being dispensed to the susceptible population at rate \( o_1 N \). We add this to the \( S \) dynamics so that when \( u = 1 \), \((S, I, 1)\) also transitions to \((S - 1, I, 1)\) at rate \( o_1 N \). We assume that \((S, I, 0)\) transitions to \((S, I, 1)\) at rate \( w_0 \). (Treating the time till vaccinations become available as an exponential random variable seems to be reasonable since there
is so much uncertainty). The Bellman equation becomes

\begin{equation}
0 = \sup_\beta \left\{ \beta \tilde{S} \tilde{I} N (V(\tilde{S} + 1, \tilde{I} - 1, u) - V(\tilde{S}, \tilde{I}, u)) \\
+ \tilde{\gamma} \tilde{I} (V(\tilde{S}, \tilde{I} - 1, u) - V(\tilde{S}, \tilde{I}, u)) \\
+ w_0 (V(\tilde{S}, \tilde{I}, 1) - V(\tilde{S}, \tilde{I}, u)) \\
+ u o_1 N (V(\tilde{S} - 1, \tilde{I}, u) - V(\tilde{S}, \tilde{I}, u)) - L(\beta) - F(\tilde{H}) \right\}.
\end{equation}

4.1. Vaccination Results. The vaccination coefficient \( o_1 = 1/100 \) represents the case where the entire susceptible population is vaccinated in roughly 100 days. As shown in the top graph in Figure 11, the susceptible population \( S \) drops off linearly to near zero, as the vaccination takes effect. Comparing Figure 8 with Figure 11, it is clear that in the simulation the control relaxes when the vaccine is developed early, due to the decrease in the susceptible population. The cost in Figure 11 is less than the cost in Figure 8, primarily due to a shorter extinction time.

With the same parameters we view how the vaccination affects the \( \beta \) thresholds in Figure 12. The overall solution remains close to the solution without vaccinations of Figure 8. Figure 12 plots the effect on the threshold of \( \beta \) values, before vaccination is available, as in Figure 10, and in the vaccination model. The triggers for policy changes are very similar, even though the susceptible population decreases significantly once a vaccination is available. The most notable feature is that, when the vaccine is developed, the control variable \( \beta \) decreases, so more strict interventions are optimum, when the susceptible population is large, and when infected population is small (below \( \sim 250,000 \)).

5. Conclusion. We have analyzed the optimal control of an idealized epidemic model of COVID-19. We found two locally optimal strategies, ‘suppression’ and ‘mitigation’, which correspond to qualitatively distinct approaches to combat the epidemic. By considering a stochastic model and variations with discrete control thresholds as well as vaccinations, we find the solutions to be fairly robust.

There are many additional features that we have not attempted to model. One feature is that the control cost will likely depend on the number of infected individuals. In particular, targeted contact tracing and quarantine may serve to reduce the infection when the number of infections is small with less cost than overarching social distancing policies. Similarly, targeted vaccination can also effectively reduce the infection rate as well as remove individuals from the susceptible population.

Another feature is the network dependence of epidemic spread, either through social networks or geographic distance. This is an active area of research with many different existing approaches.

A final feature to mention is the role of information. Our idealized model has assumed perfect information about the state of the disease, which is not realistic. Gaining accurate information about the parameters and progression the disease is also essential for optimal epidemic control.

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Appendix A. Deterministic Control Numerics.
We discretize time in fixed increments of \( \Delta t = 1 \). Dynamics are approximated by a first order Euler scheme. The target constraint is relaxed by adding the quadratic
Fig. 11. Simulations for the stochastic model with vaccinations. Parameters are \( w_0 = 1/100 \) and \( o_1 = 1/100 \) with the other parameters the same as Table 1 and Table 2. The time at which a vaccine is developed can be identified by the decrease in the susceptible population as well as a qualitative change in \( \beta \) and the control cost.

penalty function to the cost:

\[
\frac{N}{2\mu} \left( E_t + I_t + H_t - e^{1-1} \right)^2_+
\]

and the Lagrange multiplier is retrieved simply as \( \sigma = \frac{N}{\mu} \left( E_T + I_T + H_T - e^{1-1} \right)_+ \). We use \( \mu = 0.01 \).

We then calculate the gradient of the cost with respect the the control variable by back propagation (equivalent to (3.6) and a discretization of costate equations) and employ momentum gradient descent (momentum factor is 0.9). The gradient step is chosen to be between \( 10^{-5}/N \sim 10^{-7}/N \) for different parameter values. Larger steps lead to instabilities due to the long time horizon.

To find the optimal end time we implement the simple algorithm that if the Hamiltonian at the current end time \( T \) is positive, we increase the end time by one increment, and otherwise, if the Hamiltonian at \( T - \Delta t \) is negative we decrease the end time to \( T - \Delta t \).

Appendix B. Stochastic Control Numerics.

The Bellman equations (4.1) can be solved in a single sweep of value iterations making sure that we first increase \( \tilde{S} \) then increase \( \tilde{I} \), and with vaccinations we start with \( u = 1 \) and then do \( u = 0 \). We discretize \( \beta \) in increments of 0.001.
Fig. 12. Optimal $\beta$ thresholds before and after the vaccine with the susceptible population fixed in the first figure at $\tilde{S} = 6,800,000$ and with the infected population fixed at $\tilde{I} = 150,000$ in the second figure. Parameters for vaccination are $w_0 = 1/100$ and $o_1 = 1/100$ with the other parameters the same as Table 1 and Table 2.

When $N$ is much larger than 1000 we do not solve the equations directly as the computational cost is of order $N^2$. Instead we discretize the population variable in 1000 increments. We let $\Delta k = 0.001*N$ denote the discretization increment (which we assume is greater than 1). It is then possible to solve approximate Bellman equations, where we replace

$$V(\tilde{S}, \tilde{I} - 1, u) - V(\tilde{S}, \tilde{I}, u) \approx \frac{V(\tilde{S}, \tilde{I} - \Delta k, u) - V(\tilde{S}, \tilde{I}, u)}{\Delta k}.$$  

However, this leads to a bad approximation when $\tilde{I}$ is small. For example the expected time to transition from $\tilde{I} = \Delta k$ to $\tilde{I} = 0$ with $\tilde{S} = 0$ is approximated by $\frac{1}{\alpha}$, whereas the correct expected time can be computed as

$$\frac{1}{\alpha} \sum_{j=1}^{\Delta k} \frac{1}{j},$$

which is about 7.5 times larger when $\Delta k = 1000$. Since $\tilde{I} = \Delta k$ must be visited by any solution before extinction, this error would propagate through the whole problem. Because of this we renormalize the coefficient $\alpha$ at state $\tilde{I}$ using the formula

$$\frac{1}{\alpha} \rightarrow \frac{1}{\alpha} \left( \sum_{j=\tilde{I}-\Delta k+1}^{\tilde{I}} \frac{1}{j} \right).$$

The sum is approximated using the standard formula

$$\sum_{j=1}^{k} \frac{1}{j} \approx \log(k) + \gamma_e + \frac{1}{2k} - \frac{1}{12k^2}$$

where $\gamma_e \approx 0.577$ is the Euler-Mascheroni constant.

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