Control Of Epidemic Spreads Via Testing And Lock-Down

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Abstract

Testing and lock-down are interventions that can combat the spread of an infectious disease. Testing is a targeted instrument that permits the isolation of infectious individuals. Lock-down, on the other hand, is blunt and restricts the mobility of all people. In this paper, we present a compartmental epidemic model that accounts for the impact of lock-down and different kinds of testing, motivated by the nature of the ongoing COVID-19 outbreak. We consider the testing of symptomatic, contact traced, and randomly chosen asymptomatic populations. Using the model, we first characterize static mobility levels and testing requirements that can dampen the spread asymptotically. We then characterize a threshold-type optimal lock-down policy that minimizes the social impact of an epidemic, modeled via a sum of infection and lock-down costs. Our results are contextualized with realistic parameter values for COVID-19.

1 Introduction

The COVID-19 pandemic has given rise to a global public health crisis. Significant mortality rates worldwide have fueled the overwhelming public health response to this crisis, which among other means, includes social distancing, restrictions on mobility, and various forms of testing and quarantining policies. This paper aims to derive a mathematical model for the epidemic dynamics with interventions (testing and lock-down) and optimize over the extent of these interventions in various settings to minimize the infection spread and the economic burden of an epidemic.

Motivated by the nature of COVID-19, we develop a compartmental model of an epidemic in Section 2 that accounts for asymptomatic infected individuals \cite{1, 2, 3}, testing using multiple channels \cite{4, 5}, and globally implemented lock-downs \cite{6, 7}. The latter two are especially relevant as they shape the disease dynamics. Specifically, testing allows isolation of infectious carriers, barring them from adding to the spread. On the other hand, lock-downs reduce the frequency of social interactions, including those responsible for infection transmission. Among testing strategies, we consider symptomatic, contact tracing-based, and random asymptomatic testing. Our paper’s first contribution stems from the development of a parsimonious dynamic epidemic model that allows inference of structural results while maintaining the essential features of COVID-19. This

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development complements the rich literature on compartmental models (see [8, 9] for surveys) that date back to Kermack and McKendrick’s work in [10].

In Section 3, we obtain sharp thresholds for constant lock-down levels and testing strategies necessary for asymptotic decay of the infection load—the second contribution of this paper. Our results reveal the complementary roles of lock-down and the different types of testing in containing the disease. We discuss our bounds with parameter estimates for COVID-19 in the United States from existing literature (US). We believe these estimates will provide valuable insights to policy-makers.

In practice, a mere asymptomatic decay of the disease may not be sufficient. Policy-makers must adopt interventions that carefully weigh both the costs associated with the infection spread (e.g., hospitalization costs, deaths, long-term health implications, investments in testing and vaccination infrastructure, etc.) and economic losses (e.g., impact on global GDP, business distress, supply chain disruption, rise in unemployment, etc.). See [11] for a review of the socio-economic impacts of COVID-19. To this end, with symptomatic and random asymptomatic testing in Section 4.1, we characterize an optimal threshold lock-down policy that minimizes such a cost over a finite horizon. This optimal policy defines the third and the central contribution of this work. We numerically explore the properties of said threshold and further demonstrate the impact of delay in implementing this optimal control policy on the overall cost. Then, upon augmenting testing strategies with contact tracing, in Section 4.2, we demonstrate that optimal lock-down measures are extremal. Leveraging this property, we design a possibly sub-optimal lock-down policy that closely mirrors the optimal strategy without contact tracing. We then numerically compare the advantage of lock-down with contact-tracing to the scenario without it. Section 5 concludes the paper.

2 The epidemic model

We build on the widely popular compartmental epidemiological SIR (susceptible–infectious–removed) model for the spread of an epidemic process with significant asymptomatic carriers such as COVID-19 [12]. Our model generalizes the conventional SIR model in that we account for the role of testing and lock-downs. Specifically, consider a population of \(N\) individuals, among which \(s^t\), \(i^t\) and \(r^t\) denote the number of susceptible, infected, and removed individuals, respectively, at time \(t\). We model the disease propagation in discrete time, where \(t\) stands for time, measured in days. The population segments satisfy \(s^t + i^t + r^t = N\). By removed, we represent the individuals who either recover from the infection or ultimately succumb to it. Let \(\Delta\) denote the deceased fraction of the removed population. We do not model repeat infections, i.e., a patient who recovers can never contract the virus again.

To account for testing, first segment the infected population at time \(t\) into two groups—confirmed positive \(p^t\) and untested yet infected \(u^t\). Thus, \(i^t = p^t + u^t\). Individuals who test positive (in \(p^t\)) are isolated, meaning that they do not contribute towards disease propagation. Therefore, testing directly impacts the dynamics of the disease itself. In what follows, we describe the dynamics of \((s^t, u^t, p^t, r^t)\) as visualized in Figure 1. For convenience, define \(\xi := 1 - \xi\) for any constant \(\xi\).

We consider testing the population in three ways: test \(x^t\) symptomatic individuals, \(y^t\) individuals from the pool of contacts of individuals who test positive at time \(t - 1\), and \(z^t\) randomly chosen asymptomatic individuals. At the start of day \(t\), the tests are conducted, but the test results are not revealed immediately. Then, the mobile part of the population mingles. The mobile
population comprises susceptible, untested but infected, and recovered individuals. Assume that each untested infected person meets \( f^t \) people among the mobile population each day, among which \( s^t/(s^t + u^t + r^t\Delta) \) defines the fraction of the susceptible population. In \( \beta \) fraction of these meetings between an infected individual and a susceptible individual, the latter becomes infected, resulting in the following change in \( s^t \).

\[
s^{t+1} = s^t - \beta f^t u^t \frac{s^t}{s^t + u^t + r^t\Delta}.
\]  

(1)

These new infections contribute to the increase in the untested yet positive segment. This segment, however, reduces when test results arrive. Let \( \phi^t \) define the number of individuals who test positive from the \( x^t + y^t + z^t \) tests. Those who test positive, add to \( p^t \) and are isolated. Accounting for the removal of a constant \( \gamma \) fraction of infected individuals at time \( t \) through recovery or death, we obtain

\[
u^{t+1} = \gamma \left( u^t + \beta f^t u^t \frac{s^t}{s^t + u^t + r^t\Delta} - \phi^t \right).
\]  

(2)

As a result, the dynamics of \( p^t \) becomes

\[
p^{t+1} = \gamma \left( p^t + \phi^t \right).
\]  

(3)

The removed part of the population (due to recovery or death) evolves as

\[
r^{t+1} = r^t + \gamma \left( u^t + \beta f^t u^t \frac{s^t}{s^t + u^t + r^t\Delta} + p^t \right).
\]  

(4)

Collectively, (1)–(4) capture the dynamics of the disease, given test results \( \phi^t \).

2.1 Computing the newly positive infections \( \phi^t \).

The total number of individuals who test positive is given by \( \phi^t = \phi^t_x + \phi^t_y + \phi^t_z \), where \( \phi^t_x, \phi^t_y, \) and \( \phi^t_z \) are positive cases discovered through symptomatic, contact tracing, and random asymptomatic testing, respectively. We now count \( \phi^t_x, \phi^t_y, \) and \( \phi^t_z \).

Segment the population to account for the prevalence or absence of symptoms. Symptoms such as respiratory distress and fever among the susceptible and recovered populations might
appear due to flu, common cold, etc. Suppose that \( \alpha_S \) fraction of the uninfected mobile population (of size \( s^t + \Delta r^t \)) exhibits COVID-like symptoms. Among the untested infected population, let the same fraction be \( \alpha_I \). Then, the symptomatic mobile population at time \( t \) is given by

\[
\psi^t := \alpha_S (s^t + \Delta r^t) + \alpha_I u^t. 
\tag{5}
\]

Testing \( x^t \) symptomatic individuals will confirm the infections of individuals in the same ratio as in the mobile population, provided these tests are administered randomly, and hence, we have

\[
\phi_x^t = \min \left\{ \frac{x^t}{\psi^t}, 1 \right\} \alpha_I u^t, 
\tag{6}
\]

Recall that \( \phi^{t-1} \) people test positive on day \( t - 1 \), who met with \( f^{t-1} \phi^{t-1} \) people at time \( t - 1 \). Among them, the number of susceptible, untested infected and recovered individuals were in proportions \( s^{t-1} \), \( u^{t-1} \), and \( \Delta r^{t-1} \), respectively. Among the meetings with susceptible people, \( \bar{\beta} \) fraction remained susceptible and the rest contracted the infection at time \( t - 1 \). Among these susceptible contacts, only \( \bar{\alpha}_S \) are asymptomatic, making the count of asymptomatic susceptible and possibly tested at time \( t \) as

\[
s_c^t := \bar{\alpha}_S f^{t-1} \phi^{t-1} \frac{s^{t-1}}{s^{t-1} + u^{t-1} + \Delta r^{t-1}}, 
\tag{7}
\]

The same among the recovered contacts becomes

\[
r_c^t := \bar{\alpha}_S f^{t-1} \phi^{t-1} \frac{\Delta r^{t-1}}{s^{t-1} + u^{t-1} + \Delta r^{t-1}}. 
\tag{8}
\]

Among the infected contacts (both already infected at time \( t - 1 \) and those who got the infection at time \( t - 1 \)), \( \gamma \) fraction is removed. This interaction yields the following population of asymptomatic infected contacts traced and possibly tested at time \( t \).

\[
u_c^t := \gamma \bar{\alpha}_I f^{t-1} \phi^{t-1} \frac{\beta s^{t-1} + u^{t-1}}{s^{t-1} + u^{t-1} + \Delta r^{t-1}}. 
\tag{9}
\]

Testing \( y^t \) contact traced asymptomatic individuals gives

\[
\phi_y^t = \min \left\{ \frac{y^t}{s_c^t + r_c^t + u_c^t / \gamma}, \frac{u_c^t}{s_c^t} \right\} 
\tag{10}
\]

new positive results.

The total number of asymptomatic people that have not made it to the pool of contacts is given by \( \bar{\alpha}_S (s^t + \Delta r^t) - s_c^t - r_c^t \). The same number among the infected and undetected population is \( \bar{\alpha}_I u^t - u_c^t \). Thus, positives from testing \( z^t \) people among this population number

\[
\phi_z^t = \min \left\{ \frac{z^t}{\bar{\alpha}_S (s^t + \Delta r^t) - s_c^t - r_c^t + \bar{\alpha}_I u^t - u_c^t}, 1 \right\} (\bar{\alpha}_I u^t - u_c^t). 
\tag{11}
\]
2.2 Simplified dynamics in the large population limit

The dynamical system described in (1)–(4) simplifies considerably under the assumption that $s^t \approx N$, i.e., most of the population remains susceptible, implying $u^t \ll N$, $r^t \ll N$. This assumption is valid at the beginning of an epidemic. For COVID-19, the total number of confirmed cases in the US crossed 6.7M ($\approx 2\%$ of the US population) in mid-September 2020. Even accounting for asymptomatic carriers, the assumption appears justified over March-September 2020, our timeline of interest for controlling the epidemic in Section 4. Using this assumption, we infer $s^t / (s^t + u^t + \Delta r^t) \approx 1$ and $\psi^t \approx a_s N$ that yields

$$\phi_x^t \approx \min \left\{ \frac{x^t}{a_s N}, 1 \right\} \alpha_I u^t, \quad (12)$$

Under the additional assumption $\beta \gg u^t / N$, we obtain

$$s_c^t \approx \beta a_s f^{t-1} \phi^{t-1},$$
$$r_c^t \approx \alpha_s f^{t-1} \phi^{t-1} \Delta r^{t-1} / N \approx 0,$$
$$u_c^t \approx \gamma \alpha_I f^{t-1} \phi^{t-1} \left( \beta + \frac{u^{t-1}}{N} \right) \approx \beta \gamma \alpha_I f^{t-1} \phi^{t-1},$$

using which we derive

$$\phi_y^t \approx \min \left\{ y^t \frac{\beta \gamma \alpha_I}{\beta \alpha_I + \beta a_s}, \beta \gamma \alpha_I f^{t-1} \phi^{t-1} \right\}, \quad (13)$$

$$\phi_z^t \approx \alpha_I \left( u^t - \beta \gamma f^{t-1} \phi^{t-1} \right) \cdot \min \left\{ z^t / a_s N, 1 \right\}$$
$$= \frac{1}{N} \frac{\alpha_I}{a_s} z^t \left( u^t - \beta \gamma f^{t-1} \phi^{t-1} \right). \quad (14)$$

The last line is derived with $z^t \ll N$. This assumption is certainly justified, given that less than 2M COVID-19 tests (including all testing modes) are being currently administered daily in the US, which is less than 0.5% of the US population. To support our modeling choice of $\beta \gg u^t / N$, notice that 30M cases have been confirmed over a year in the US. Even accounting for asymptomatic infections, the average daily infection load remains well below 0.1%. The probability of COVID-19 transmission $\beta$, however, is 16-25% with mask use and 50% without them (see [13, 14]) that is much higher than $u^t / N$.

Classical Kermak-McKendrick-style compartmental models are derived for the dynamics of populations’ fractions (see [10]). For each group, denote the fraction of the population in that group by the corresponding capital letter, e.g., $U^t := u^t / N$. Then, testing strategies dictate the choice of $(X^t, Y^t, Z^t)$. One can also choose to partially lock-down the population that amounts to choosing a mobility ratio of $F^t \in [0, 1]$ and setting $f^t = f_0 F^t$. Introducing the notation $\beta_0 := \beta f_0$,
the dynamics of $U, P, R$ becomes
\begin{align*}
U^{t+1} &= \gamma (U^t + \beta_0 F^t U^t - \Phi^t), \\
P^{t+1} &= \gamma (P^t + \Phi^t), \\
R^{t+1} &= R^t + \gamma (U^t + \beta_0 F^t U^t + P^t),
\end{align*}
(15)
where
\begin{align*}
\Phi^t &= \Phi^t_x + \Phi^t_y + \Phi^t_z \\
&= \min \left\{ \frac{\alpha_I}{\alpha_S} X^t U^t, \alpha_I U^t \right\} \\
&\quad + \min \left\{ \zeta Y^t, \beta_0 F^{-1} \frac{\alpha_I}{\alpha_S} \Phi^t \right\} \\
&\quad + \frac{\alpha_I}{\alpha_S} Z^t (U^t - \beta_0 F^{-1} \gamma \Phi^t). 
\end{align*}
(16)
Without contact tracing, the above relation simplifies to
\begin{align*}
\Phi^t &= \min \left\{ \frac{\alpha_I}{\alpha_S} X^t U^t, \alpha_I U^t \right\} + \frac{\alpha_I}{\alpha_S} Z^t U^t, 
\end{align*}
(17)
by setting $y^t = s^t_c = u^t_c = r^t_c = 0$ in (10)–(11).

Our compartmental model, by its very nature, is a mean-field approximation to a stochastic infection dynamics. Such a model is valid only for large populations. Stochastic infection dynamics in small populations, as those within a university or a small town, can exhibit large excursions from the mean. We remark that generalizations of our model to consider multiple areas connected via a network with heterogeneous mobilities within and outside of the areas, and with exogenous shocks, offer no conceptual barriers.

3 Testing and lock-down strategies for asymptomatic decay of epidemic spread

Using the epidemic dynamics from Section 2, we now study conditions for testing and (partial) lock-down that make the disease decay in the long run—a question that has long been studied for established epidemic models, e.g., in [8]. Here, we only consider static lock-down measures that hold mobility ratio $F^t$ constant at $F \in [0, 1]$.

3.1 Can the epidemic disappear without testing?

Without any testing, the dynamics simplifies to
\begin{align*}
U^{t+1} &= \gamma (1 + \beta_0 F) U^t.
\end{align*}
(18)
The disease will dissipate without testing if
\begin{align*}
1 + \beta_0 F < \gamma^{-1}.
\end{align*}
(19)
Violating the above condition, either testing or (partial) lock-down or both is essential to mitigate the disease spread. The above condition is reminiscent of the well-known result for uncontrolled SIR dynamics, which requires the “reproduction number” of the disease to be smaller than unity. According to [15], $\beta_0 \in [0.75, 1.25]$ across US counties for COVID-19. With partial lock-down measures, $F \in [0.5, 1.0]$. Given the estimated recovery rate of $\frac{1}{17}$ to $\frac{1}{120}$ days$^{-1}$ (as in [16]), we get $\gamma(1 + \beta_0 F) \in [1.27, 2.14]$ without any intervention, suggesting the need for interventions to dampen the spread. In the absence of testing, one roughly requires a stringent mobility ratio of $F \approx 0.11$ for the same.

3.2 When will testing fail to contain the disease?

Testing cannot discover more infections than are present at the time the test is conducted at the start of day $t$, i.e., $\Phi^t \leq U^t$, implying that

$$U^{t+1} = \overline{\gamma} (U^t + \beta_0 F U^t - \Phi^t) \geq \overline{\gamma} \beta_0 F U^t. \quad (20)$$

If mobility is high enough to make $\overline{\gamma} \beta_0 F > 1$, then the delay between testing at the beginning of day $t$ and isolating positively tested patients at the end of day $t$ may fail to contain the disease, owing to the emergence of new infections at time $t$. This simple analysis highlights the role of delay in revealing test results on the dynamics of the disease. In the sequel, assume that $\overline{\gamma} \beta_0 F < 1$.

3.3 With symptomatic-only testing policy

Several states in the US have adopted a symptom-based testing strategy. Can such a strategy mitigate the disease? With perfect symptomatic-only testing ($X^t = \alpha_I U^t, Y^t = Z^t = 0$), the epidemic dynamics becomes

$$U^{t+1} = \gamma (1 + \beta_0 F - \alpha_I) U^t. \quad (21)$$

Thus, the disease disappears asymptotically if

$$1 + \beta_0 F < \gamma^{-1} + \alpha_I. \quad (22)$$

Symptomatic-only testing helps dissipate the disease if $\alpha_I$ is large enough, i.e., when more carriers of the disease are symptomatic. Diseases such as Ebola fit this description, and therefore, symptomatic-only testing had proven efficient to contain these epidemics. Containing COVID-19, however, has proven much more challenging. Following [17], $\alpha_I \in [0.55, 0.64]$. With testing all symptomatic patients, the allowable mobility ratio can be improved from 0.11 (without testing) to around 0.80 to contain the spread.

3.4 With symptomatic and random asymptomatic testing

Consider the addition of asymptomatic testing of a small fixed fraction $Z$ at all periods. The disease then disappears if

$$1 + \beta_0 F < \gamma^{-1} + \alpha_I + \frac{\alpha_I}{\alpha_S} Z, \quad (23)$$

Notice that $\beta$ and $\beta^0$ are different parameters. The former is the probability of infection transmission, while the latter includes mobility of the population.
i.e., random testing provides more flexibility in the required mobility ratio from the symptomatic-only testing paradigm in the form of \(\frac{\alpha I}{\alpha S}Z\) to bound \(1 + \beta_0 F\). The effectiveness of random testing is affected by \(\alpha_S\), the prevalence of similar symptoms as the disease among the uninfected population. Higher \(\alpha_S\) implies a smaller uninfected asymptomatic population, increasing the chances of random asymptomatic testing to discover the disease’s prevalence. With \(X^t + Y^t + Z^t \approx 0.5\%\) of the US population (with 1.5M daily tests), \(Z^t\) appears negligible.

### 3.5 Impact of contact tracing

Finally, we add perfect contact tracing to the mix, i.e., test all contacts of people who test positive on the previous day. With \(\Phi\) in (17), the disease dynamics simplifies to

\[
\begin{pmatrix}
U^{t+1} \\
\Phi^t
\end{pmatrix} = A(F, Z) \begin{pmatrix}
U^t \\
\Phi^{t-1}
\end{pmatrix},
\]

where

\[
A(F, Z) := \begin{pmatrix}
\gamma [\beta_0 F + \overline{\alpha}_I(Z)] & -\beta_0 F\overline{\alpha}^2 I(Z) \\
1 - \overline{\alpha}_I(Z) & \beta_0 F\overline{\gamma} I(Z)
\end{pmatrix},
\]

\[
\overline{\alpha}_I(Z) := \overline{\alpha}_I (1 - Z/\alpha_S).
\]

The eigenvalues of \(A\) are nonnegative and are given by

\[
\lambda_1 = \overline{\gamma} \overline{\alpha}_I(Z)(1 + \beta_0 F), \quad \lambda_2 = \overline{\gamma} \beta_0 F.
\]

Assuming \(\overline{\gamma} \beta_0 F < 1\) (the condition for testing to be effective), we get \(\lambda_2 < 1\). Then, the disease asymptotically vanishes if

\[
1 + \beta_0 F < \overline{\gamma}^{-1} \overline{\alpha}_I^{-1} (1 - Z/\alpha_S)^{-1}.
\]

With \(Z = 0\), the right-hand-side (RHS) of the above relation becomes \(\overline{\gamma}^{-1} \overline{\alpha}_I^{-1}\). Notice that

\[
\overline{\gamma}^{-1} \overline{\alpha}_I^{-1} > \overline{\gamma}^{-1} + \alpha_I,
\]

that together with (22) imply that the addition of contact tracing allows a higher mobility ratio as compared to symptomatic-only testing. Indeed with perfect contact tracing (even with \(Z = 0\)), our estimates indicate that (27) will hold in the US without any lock-down (\(F = 1\)). However, contact tracing is seldom perfect; efficiencies above 70% are difficult, as per [18]. Even such efficiencies are challenging to achieve nationally. With 70% efficiency, one needs \(Z\) that equates to 10M-14M daily COVID-19 tests in the US per (26) to retain \(F = 1\). This level far exceeds the current daily testing levels of <2M. Current testing levels demand a mobility ratio of < 85% via partial lock-down for the same.

Random asymptomatic testing of \(Z\) fraction of the population buys a factor of \((1 - Z/\alpha_S)^{-1}\) in bounding \(1 + \beta_0 F\), further reducing the severity in lock-down requirements. This factor approaches \(\infty\) with \(Z \uparrow \alpha_S\). This is in sharp contrast to (23), where the RHS remains bounded above. Thus, equipped with contact tracing, ramping up asymptomatic random testing can obviate the need for lock-downs.
4 Minimizing the impact of an epidemic

A policymaker must balance between the economic consequences of lock-down and that of infection spread. In this section, we formulate the question of minimization of the impact of an epidemic over a finite horizon $T$, modeled as

$$J = gU^T + \sum_{t=0}^{T-1} \left[ (1 - F^t) + gU^t \right].$$

Here, we allow $F^t \in [F_{\text{min}}, 1]$, where $F_{\text{min}}$ denotes the minimum enforceable mobility ratio, while $F^t = 1$ corresponds to opening up the economy. The economic impact of lock-down at time $t$ given by $1 - F^t$ thus increases with the extent of lock-down. The term $gU^t$ increases with the untested infection load at each time $t$. This load drives the growth in hospitalizations among the $P^t$ positively tested patients and deaths among the $R^t$ removed populations. The constant $\rho > 0$ models the ratio of costs associated with the epidemic spread and economic losses due to lock-down. That is, a larger $\rho$ in minimizing $J$ will prioritize arresting the infection growth more as compared to lock-down measures. The results in this section are presented with random asymptomatic testing levels held constant at $Z^t = Z$, while all symptomatic patients are tested.\(^2\)

In (29), $T$ encodes the planning horizon for the policy-maker. In Section 4.1, we study the optimal lock-down policy without any testing via contact tracing. In Section 4.2, we study the case with contact tracing.

4.1 Optimal lock-down policy without contact tracing

The minimum-cost lock-down sequence solves

$$\begin{align*}
\text{minimize} & \quad J, \\
\text{subject to} & \quad U^{t+1} = \tau \left( 1 + \beta_0 F^t - \alpha I - \frac{\alpha I}{\alpha S} Z \right) U^t, \\
& \quad F_{\text{min}} \leq F^t \leq 1, \\
& \quad \text{for } t = 0, \ldots, T - 1,
\end{align*}$$

over $F^0, \ldots, F^{T-1}$ and $U^1, \ldots, U^T$, given $U^0$.

For this optimal control problem, we now characterize the optimal admissible lock-down policy sequence $F_0^*, \ldots, F_{T-1}^*$ that respectively map $U_0^*, \ldots, U_{T-1}^*$ to $[F_{\text{min}}, 1]$.

**Theorem 1.** An optimal lock-down policy for (30) is single threshold-type, given by

$$F_t^*(U_t^*) = \begin{cases} F_{\text{min}}, & \text{if } U_t^* \geq \nu_t, \\ 1, & \text{otherwise,} \end{cases}$$

where

$$\nu_t := \frac{F_{\text{min}}}{g (\eta_{\text{max}} - \eta_{\text{min}})} \left( \frac{\eta_{\text{max}} - 1}{\eta_{\text{max}} - 1} \right),$$

and $\eta_{\text{max}}, \eta_{\text{min}}$ are the maximum and minimum values of $\tau \left( 1 + \beta_0 F - \alpha I - \frac{\alpha I}{\alpha S} Z \right)$ over $F \in [F_{\text{min}}, 1]$.

\(^2\)The results can be generalized to the case with a fixed sequence of random asymptomatic testing $Z_0, \ldots, Z^{T-1}$. 9
Proof. We break the proof into three steps. First, we show that \( F_t \) is extremal for all \( t \), and hence, it suffices to search over such extremal policies. Second, we present the dynamic programming framework for (30) and characterize the optimal value functions in terms of the minimum over a particular discrete set. Third, we deduce the optimal policy using the nature of that discrete optimization problem.

- **Step 1. Showing that** \( F_t(U^t) \in \{F_{\min}, 1\} \). The dynamics of \( U^t \) in (30) dictates that \( U^{t+1} \) depends linearly in \( F_t \). Also, \( U^{t+2}, \ldots, U^T \) vary linearly in \( U^{t+1} \), that in turn is linear in \( F_t \). The objective function \( J \) is jointly linear in \( F_t \) and in \( U^{t+1}, \ldots, U^T \), where the latter are linear in \( F_t \). From this linearity of the objective function in \( F_t \), we infer that an optimal choice of \( F_t \) is extremal, i.e, \( F_t(U^t) \in \{F_{\min}, 1\} \).

- **Step 2. Computing the optimal value functions.** Define \( J_t^*(U^t) \) as the optimal value function at day \( t \) that models the optimal cost from that day onward, starting with an untested population of size \( U^t \), for each \( t = 0, \ldots, T \). Using dynamic programming, \( J_t^* \) then satisfies

\[
J_t^*(U^T) = qU^T, \\
J_t^*(U^t) = qU^t + \min \left\{ T_{\min} + J_{t+1}^*(\eta_{\min}U^t), J_{t+1}^*(\eta_{\max}U^t) \right\}.
\]

The optimal value of (30) is \( J_0^*(U^0) \). We now show that

\[
J_t^*(U^t) = \min_{\tau \in \{0, \ldots, T-t\}} Q_t(U^t; \tau),
\]

where

\[
Q_t(U^t; \tau) := T_{\min} + qU^t \left[ \sum_{i=0}^{\tau-1} \eta_{\min}^i + \eta_{\min}^{\tau-t} \sum_{i=0}^{T-t-\tau} \eta_{\max}^i \right].
\]

We proceed via backward induction. In (35) with \( t = T \), we have \( \tau \in \{0\} \), at which

\[
Q^T(U^T, 0) = qU^T = J_T^*(U^T).
\]

The last equality verifies the base case. Assume that (34) holds for times \( t + 1, \ldots, T \). We now show that the same holds at time \( t \). By induction hypothesis, we have

\[
J_t^*(U^t) = qU^t + \min \left\{ T_{\min} + \min_{\tau \in \{0, \ldots, T-t-1\}} Q_{t+1}(\eta_{\min}U^t; \tau), \min_{\tau \in \{0, \ldots, T-t-1\}} Q_{t+1}(\eta_{\max}U^t; \tau) \right\}.
\]

By altering the order of the minimizations and using the definition of \( Q_t \) in (35), we get

\[
J_t^*(U^t) = \min \left\{ qU^t + Q_{t+1}(\eta_{\max}U^t; 0), qU^t + T_{\min} + Q_{t+1}(\eta_{\min}U^t; T-t-1), \right. \] 
\[ \left. W_t(0), \ldots, W_t(T-t-2) \right\}
\]

\[
= \min \{ Q_t(U^t; 0), Q_t(U^t; T-t), W_t(0), \ldots, W_t(T-t-2) \},
\]

where we define \( W_t \)'s as

\[
W_t(\tau) := \min \left\{ qU^t + T_{\min} + Q_{t+1}(\eta_{\min}U^t; \tau), U^t + Q_{t+1}(\eta_{\max}U^t; \tau + 1) \right\}
\]

for \( \tau = 0, \ldots, T-t-2 \). In what follows, we prove that \( W_t(\tau) = Q_t(U^t; \tau + 1) \), leveraging which in (36), gives the desired relation in (34) for time \( t \), completing the backward induction argument.
To simplify $\mathcal{W}_t$, notice that the difference of the two terms in (37) after elementary algebra yields

$$[g U^t + Q^{t+1}(\eta_{\max} U^t; \tau + 1)] - [g U^t + F_{\min} + Q^{t+1}(\eta_{\min} U^t; \tau)] = g U^t(\eta_{\max} - \eta_{\min}) \sum_{i=0}^{\tau} \eta^i_{\min} \geq 0.$$  

(38)

Hence, one term dominates the other, implying

$$\mathcal{W}_t(\tau) = g U^t + F_{\min} + Q^{t+1}(\eta_{\min} U^t; \tau) = Q^{t}(U^t; \tau + 1).$$  

(39)

The last line follows from the definition of $Q^{t}$ in (35).

- **Step 3. Deducing the optimal policy.** From (34), it follows that $F^*_i(U^t) = 1$, if $\tau = 0$ is a minimizer of $Q^{t}(U^t; \tau)$. Next, we show that the sequence $Q^{t}(U^t; \tau)$ is a discrete convex sequence, i.e., it satisfies

$$Q^{t}(U^t; \tau - 1) + Q^{t}(U^t; \tau + 1) \geq 2Q^{t}(U^t; \tau).$$  

(40)

Using (35), we get

$$Q^{t}(U^t; \tau - 1) + Q^{t}(U^t; \tau + 1) - 2Q^{t}(U^t; \tau)$$

$$= \eta^{\tau-1}_{\min}(\eta_{\max} - \eta_{\min}) \sum_{i=0}^{T-\tau} \eta^i_{\min} - \sum_{i=0}^{T-\tau} \eta^i_{\max} \sum_{i=0}^{T-\tau-1} \eta^i_{\max}$$

$$= \eta^{\tau-1}_{\min}(\eta_{\max} - \eta_{\min}) \left[ 1 + (\eta_{\max} - \eta_{\min}) \sum_{i=0}^{T-\tau-1} \eta^i_{\max} \right]$$

$$\geq 0.$$  

Owing to the discrete convex nature of the sequence, it suffices to check $Q^{t}(U^t; 0) \leq Q^{t}(U^t; 1)$ to certify optimality of $\tau = 0$ in (34). This inequality simplifies to $U^t \leq v^t$, where $v^t$ is given in (32). The algebra is omitted due to space constraints.

In the left panel of Figure 2, we plot the optimal threshold $v^t$ for two different values of $Z$. Theorem 1 advocates to lock-down when the infection level $U^t$ grows beyond $v^t$. As the random asymptomatic testing level $Z$ increases, the threshold increases as more testing allows the economy to remain open at higher infection levels. Thus, testing and lock-down assume complementary roles to combat the epidemic. Our parameter values, detailed in Figure 2, are consistent with existing literature (as presented in Section 3). Roughly, our plots correspond to March 21, 2020 as $t = 0$, when the US reported a total death toll of 600. Assuming a mortality rate of $\approx 1.8\%$, this indicates $v^0 \approx 33K$, that is 0.01% of the US population, equivalently $U^0 = 10^{-4}$. We consider a planning horizon of $T = 120$ days (4 months). In practice, a policy-maker will likely operate with a horizon in mind and revise those estimates with time.

We offer additional insights into the construction of the threshold policy of Theorem 1. Notice that we choose $F^*_i(U^t) = 1$, when $Q^t(U^t; \tau)$ in (35) is minimized at $\tau = 0$. One can identify $Q^t(U^t; \tau)$ as the cost associated with $\tau$ periods with maximal lock-down, followed by $T - t - \tau$ periods without lock-down, starting at time $t$ with $U^t$ fraction of untested positive infections. The proof builds on the premise that the least-cost path on day $t$ from $U^t$ comprises said front-loading.
of the closures. The increasing nature of $\nu^t$ with $t$ in Figure 2 conveys the same. Our optimal policy depends on the knowledge of $U^t$. Given asymptomatic carriers, $U^t$ is unobservable and can only be estimated from $Z$ in practice. The higher the $Z$, the lower will be the error in that estimate. A formal analysis of the optimal control policy with a stochastic testing process as considered in [9, Chapter 4] is left for future work.

![Figure 2: Numerical experiments with $T = 120$ days, $\alpha_I = 0.5, \alpha_S = 0.08, \beta_0 = 0.9, \gamma = 1/13, \varphi = 15, F_{\text{min}} = 0.6$ and $U^0 = 10^{-4}$. Plot on the left shows the optimal infection threshold $\nu^t$. Plot on the right depicts the costs $J$ obtained by implementing the optimal control policy with a delay.](image)

It is natural for policymakers to carefully weigh the economic consequences of a lock-down and, therefore, delay imposing travel restrictions. The optimal control policy, however, advocates earlier lock-downs. In the right half of Figure 2, we capture the effect of delay in implementing the optimal lock-down policy. That is, we plot $J$, when the optimal policy is implemented from time $T_{\text{delay}}$ onward, keeping the economy open before that. Formally, $F^t = 1$ for $t < T_{\text{delay}}$ and $F^t = F^t(U^t)$ for $t \geq T_{\text{delay}}$. Delay in implementation of necessary lock-downs can lead to sharp increases in overall costs, given the logarithmic scale of the plot. The rise is sharper and happens at smaller $T_{\text{delay}}$, when testing capability is limited (with $Z \approx 0$).

### 4.2 Lock-down policy with contact tracing

Recall that with contact tracing, the testing at day $t$ becomes a function of the number of positive tests on the day $t-1$. As a result, one cannot reduce the epidemic dynamics to that of $U^t$ alone; rather, it requires us to describe the dynamics of $U^t := (U^t, \Phi^t)^\top$. Here, we use the notation $H^\top$ to denote the transpose of any matrix/vector $H$. With a fixed level of asymptomatic testing $Z$, the optimal control problem to find the minimum cost lock-down policy is given by

\[
\begin{align*}
\text{minimize} & \quad J, \\
\text{subject to} & \quad U^{t+1} = \tilde{A}(F^{t-1}, F^t)U^t, \\
& \quad F_{\text{min}} \leq F^t \leq 1, \\
& \quad \text{for } t = 0, \ldots, T - 1,
\end{align*}
\]

starting from $U^0$, where

\[
\tilde{A}(F^{t-1}, F^t) := \begin{pmatrix} \gamma \left[ \beta_0 F^t + \alpha_I(Z) \right] & -\gamma^2 \alpha_I(Z) \beta_0 F^{t-1} \\ 1 - \alpha_I(Z) & \gamma \alpha_I(Z) \beta_0 F^{t-1} \end{pmatrix}.
\]
We have used the convention that $F^{-1} = 1$ and the notation $\bar{\pi}_I(Z) = \bar{\pi}_I(1 - Z/\bar{\alpha}_S)$. The following result characterizes an important property of the optimal lock-down sequence.

**Proposition 1.** An optimal lock-down sequence for (41) comprises extremal actions, i.e., $F^*_t(\mathcal{U}^t) \in \{F_{\min}, 1\}$.

**Proof.** Similar to the proof of part (a) of Theorem 1, we argue that $\mathcal{U}^{t+1}, \ldots, \mathcal{U}^T$ is affine in $F^t$ for each $t = 0, \ldots, T - 1$. Rewrite (42) as

$$\tilde{A}(F^{t-1}, F^t) = F^{t-1}Q_1 + F^tQ_2 + Q_3, \quad (43)$$

where

$$S(Q_1) = \begin{pmatrix} 0 & * \\ 0 & * \end{pmatrix}, \quad S(Q_2) = \begin{pmatrix} * & 0 \\ 0 & 0 \end{pmatrix}, \quad (44)$$

encode the sparsity patterns of $Q_1$ and $Q_2$, respectively. The relation in (43) implies that $\mathcal{U}^{t+1} = \tilde{A}(F^{t-1}, F^t)\mathcal{U}^t$ is affine in $F^t$. Then, we have

$$\mathcal{U}^{t+2} = \tilde{A}(F^t, F^{t+1})\tilde{A}(F^{t-1}, F^t)\mathcal{U}^t$$

$$= [F^tQ_1 + F^{t+1}Q_2 + Q_3] \cdot [F^{t-1}Q_1 + F^tQ_2 + Q_3] \cdot \mathcal{U}^t$$

for $t < T - 1$. The only term in $\mathcal{U}^{t+2}$, possibly nonlinear in $F^t$ in the above relation, is given by $(F^t)^2Q_1Q_2\mathcal{U}^t = 0$, owing to the sparsity patterns of $Q_1$ and $Q_2$ shown in (44). States $\mathcal{U}^{t+2}, \ldots, \mathcal{U}^T$ for $t < T - 2$ do not depend on $F^t$ directly, but are linear in $\mathcal{U}^{t+2}$, that is affine in $F^t$. As a result, the cost $J$ becomes affine in $F^t$. Its unconstrained optimization over a bounded interval yields extremal optimal solutions. ■

Proposition 1 does not identify an optimal policy, but characterizes a crucial property that narrows its search space. We surmise that a proof strategy similar to that for Theorem 1 can be used to identify an optimal policy with contact tracing. In what follows, we numerically explore a (possibly sub-optimal) lock-down policy inspired by the proof of Theorem 1. At time $t$, we compute $Q^*_c(\mathcal{U}; \tau)$ as the cost from time $t$ to $T$, obtained by maximally locking down for the first $\tau$ consecutive periods and opening the economy after that. Then, at time $t$, we follow the policy

$$\tilde{F}^t(\mathcal{U}^t) := \begin{cases} 1, & \text{if } 0 \in \arg\min_{\tau=0,\ldots,T-t} Q^*_c(\mathcal{U}; \tau), \\ F_{\min}, & \text{otherwise}. \end{cases} \quad (45)$$

We compare this lock-down policy in the presence of contact tracing with the optimal control policy without contact tracing for the same example studied in Section 4.1. Figure 3 reveals that perfect contact tracing essentially drowns the infection load; this is not surprising, given the discussion in Section 3.5. Table 1 confirms that contact tracing yields lesser costs than without it, keeping other testing levels the same across the two experiments. Lock-downs are also relaxed earlier with contact tracing (where $U^t$ changes sharply).
Figure 3: Plot portrays $U_t$ with and without contact tracing for two different values of $Z$. The parameter values for the experiments are those used for Figure 2 and $\Phi^0 = 0$.

<table>
<thead>
<tr>
<th>Testing strategy</th>
<th>$Z = 0$</th>
<th>$Z = 0.2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Without contact tracing</td>
<td>35.37</td>
<td>20.9</td>
</tr>
<tr>
<td>With contact tracing</td>
<td>17.61</td>
<td>9.61</td>
</tr>
</tbody>
</table>

Table 1: Comparison of costs under optimal lock-down policy without contact tracing and the possibly sub-optimal lock-down policy in (45) with contact tracing.

5 Conclusions

We derived a compartmental epidemic model in discrete time that captures the role of asymptomatic infection carriers and intervention strategies. Under mild assumptions, we established conditions for static lock-down and testing to asymptotically mitigate the epidemic. We then studied the optimal control problem to minimize the impact of the disease. The results are contextualized with parameters that describe COVID-19 spread. We are keen to compare our computed optimal intervention strategies with policies adopted in practice in future research. We also aim to pursue a generalization of our model with vaccines and analyze it.

References


