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\section*{Abstract}

Nations are struggling to decide when to end COVID-19 inspired lockdowns, with sharply divergent views between those arguing for a resumption of economic activity and those arguing for continuing the lockdown. We examine this choice within a simple optimal dynamic control model that encompasses both health and economic outcomes, and pays particular attention to when need for care exceeds hospital capacity. The model shows that very different strategies can perform similarly well and even both be optimal for the same relative valuation on work and life because of the presence of a so-called Skiba threshold. Qualitatively the alternate strategies correspond to trying to essentially eradicate the virus or merely to flatten the curve so fewer people urgently need healthcare when hospitals are already filled to capacity.

\textbf{Keywords:} COVID-19, Lockdown, Skiba threshold, SIR model

\section*{1. Introduction}

The novel SARS-CoV-2 has literally swept around the globe. A prominent countermeasure has been to “lock down” non-essential parts of the economy to reduce contagious spread through social interaction. Lockdowns have succeeded in “flattening the curve” but at a high price; job losses in many places are the highest ever recorded. That raises the question of when such measures should be relaxed. Too soon and the epidemic will bounce back; too late and there is needless economic hardship.

This paper analyzes an optimal dynamic control model of that difficult balancing. It supplements a standard SIR epidemic model with controls that temporarily remove a proportion of the population from both social interaction and the workforce. The objective function encompasses both health and economic considerations. Specifically, it recognizes benefits from people being free to work and costs when infected people need hospitalization, with an extra penalty when the number needing hospitalization exceeds hospital capacity.

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The modeling of policy here is simple, involving just picking the beginning and ending times of the lockdown, denoted by $\tau_1$ and $\tau_2$, respectively. Most jurisdictions have already begun their lockdown, so we initially planned to optimize only over $\tau_2$, for varying values of $\tau_1$. The results were surprising.

As might be expected, the optimal duration of the lockdown depends sharply on when it began, but not always in the expected way. Whereas one might have thought that starting too late would require keeping the lockdown in place longer, we find that starting later can sometimes make it optimal to end the lockdown sooner.

To understand the origins of this behavior, we formulate the model more generally to optimize over both $\tau_1$ and $\tau_2$. Doing so reveals alternative optimal strategies that differ only modestly concerning timing of lockdown initiation, but which differ markedly in terms of health and economic outcomes. Later starts lead to health outcomes that are just enough worse to offset for the earlier re-opening. Remarkable is that both optimal solutions prevail under the same prioritization regarding health versus economic growth. Alternative optimal solution trajectories in dynamic models have variously been called Skiba, Sethi-Skiba, DNS, and DNSS points recognizing the contributions of various pioneers in the field (Grass et al., 2008). The one here appears to be of a new type. Rather than emerging from interactions between the state and control variables, here path dependence is generated by an interaction between timing and duration.

The literature on COVID-19 models is exploding almost as fast as the virus itself, and some papers do model the balancing of health and economic interests (see Layard et al., 2020, for a careful evaluation). Gonzalez-Eiras and Niepelt (2020) depart from the SIR dynamics and have economic activity as a control variable. Increasing this variable on the one hand raises output, but on the other hand it feeds the number of infections, raising the burden on the health care system. Calibrating their model based on the U.S. situation, they suggest a lockdown of 50 days in which economic activity is reduced by two-thirds.

Alvarez et al. (2020) also employ the SIR dynamics, but their control variable is the fraction of the population going into lockdown. They particularly include the effect of testing. Conditional on the possibility of testing and after parametrizing their model using data from the World Health Organization (WHO), they find that it is optimal to start a lockdown one week after the outbreak, and after one month it will be gradually withdrawn. Absence of testing increases the economic costs of the lockdown and shortens its optimal duration.

Acemoglu et al. (2020) investigate a heterogeneous SIR model distinguishing between the “young”, the “middle-aged” and the “old”. They find that it is especially beneficial to have stricter lockdown policies on the oldest group. Huberts and Thijssen (2020) consider a stochastic version of the SIR model. They use a continuous-time Markov chain model to study the value and optimal timing of two (sequential) options: the option to intervene and, after intervention has started, the option to end it.

We, like they, recognize that locking down the population in order to reduce transmission also reduces employment. Our primary additional innovation on the modeling side is distinguishing between the health consequences of infection when proper care is received from those when the healthcare system’s capacity to render appropriate care has been exceeded. The number of deaths is not simply proportional to the number of infections; there is an extra penalty for infections that happen when hospitals are overwhelmed. Our primary methodological contribution is exploration of threshold effects when the optimal solution “tips” from one strategy to another.

Piguillem and Shi (2020) extend the SIR framework to an SEIR framework, where E stands for individuals exposed to the virus but not symptomatic. Like us, they also consider limited capacity of the health system. Their main result is that testing generates considerable welfare gains.

Where the just mentioned papers, including ours, formulate planner problems, Eichenbaum et al. (2020) consider a competitive equilibrium in which a consumption tax is used to slowdown economic activity and epidemic diffusion. Krueger et al. (2020) extend Eichenbaum et al. (2020) by distinguishing goods by the degree to which they can be consumed at home rather than in a social (and thus possibly contagious) context.
The model analyzed here is quite simple. It does not, for example, distinguish between symptomatic and asymptomatic cases, exposed vs. infected individuals, or between young and old. So although we choose parameters that produce epidemic curves broadly consistent with those of other models, we stress the qualitative conclusions only.

The paper is organized as follows. Section 2 presents the model and elaborates the choice of the parameter values. The numerical results and their implications are discussed in Section 3. Section 4 concludes.

2. The Model

2.1. SIR model

The backbone of the model is a standard open-population SIR model (Kermack and McKendrick, 1927) with a birth rate \( \nu \) and extra mortality for individuals who are infected (\( \mu_I \)) above and beyond that for those who are susceptible or recovered (\( \mu \)).\(^1\) Given the state variables:

\[ S(t): \text{Number of susceptible individuals at time } t \]

\[ I(t): \text{Number of infected and infectious individuals at time } t \]

\[ R(t): \text{Number of recovered individuals at time } t. \]

The SIR state equations are:

\[
\dot{S}(t) = \nu N(t) - \beta \frac{S(t)I(t)}{N(t)} - \mu S(t) \tag{1a}
\]

\[
\dot{I}(t) = \beta \frac{S(t)I(t)}{N(t)} - (\alpha + \mu + \mu_I) I(t) \tag{1b}
\]

\[
\dot{R}(t) = \alpha I(t) - \mu R(t) \tag{1c}
\]

\[ \beta := R_{\text{eff}}(t, \tau_1, \tau_2) \tag{1d} \]

\[ N(t) := S(t) + I(t) + R(t). \tag{1e} \]

We set \( \alpha \) equal to \( \frac{1}{15} \) per day, corresponding to an average dwell time in the infected state of fifteen days for those who do not die, and then choose \( \beta \) to make the epidemic’s reproduction rate \( R_{\text{eff}}(t, \tau_1, \tau_2) \) have the appropriate value. The lockdown directly affects the rate of social interaction, \( \beta \), but \( R_{\text{eff}}(t, \tau_1, \tau_2) \) is more readily interpretable, so we describe the lockdown phases in terms of effects on \( R_{\text{eff}}(t, \tau_1, \tau_2) \) and adjust \( \beta \) accordingly.

2.2. Lockdown’s effect on epidemic spread

We distinguish three periods: before, during and after the lockdown, denoted by the subscripts 1, 2, and 3, respectively. The decision maker gets to choose the times, \( \tau_1 \) and \( \tau_2 \), at which the lockdown is initiated and ended.

The epidemic dynamics are identical during those three periods except that the lockdown alters \( R_{\text{eff}}(t, \tau_1, \tau_2) \) as follows.

Before the lockdown, \( R^1_0 = 2.5 \). During the lockdown, the reproductive rate if everyone were susceptible is \( R^2_0 = 0.8 \). After a sustained lockdown, that reproductive number would only bounce back to \( R^3_0 = 2.0 \), because some aspects of social distancing will be maintained indefinitely, or at least until a reliable vaccine is available, even though others will only be maintained during the lockdown. However, if the lockdown is short, then the post lock-down reproductive number would return to its original value of \( R^1_0 \). In particular, the gap between realized and potential value for \( R^3_0 \) decays exponentially in the length of the lockdown.

\(^1\)We introduce the birth and background death rate parameters for completeness. For the qualitative results these are of no importance since the time horizon for lockdowns is relatively short.
2.3. COVID-19 Deaths

For some people, COVID-19 is relatively mild, perhaps akin to a bad seasonal flu. We ignore those cases, apart from recognizing that they cannot work while sick. Instead we focus on those who require hospitalization, particularly, those who require critical care.

If \( p \) denotes the proportion of infected people who need critical care, and \( \xi \) denotes the probability of death for those needing and receiving critical care, then \( \mu = p \xi \alpha \).

That level of modeling precision is adequate for the state dynamics, because deaths from COVID-19 are not common enough to appreciably alter population size, but within the objective function greater precision is needed.

A central challenge of COVID-19 is the surges in demand for care, so we distinguish between those who need critical care and receive it from those who need it when hospitals are full and so cannot be treated properly. That means health harms are driven not only by the number of people who get infected, but also by how peaked the epidemic is; flattening the curve lets more people receive appropriate care.

In particular, we distinguish two components to the flow of deaths from COVID-19. In addition to deaths that are proportional to \( (pI(t)) \) there is an extra penalty term proportional to \( \max(\{0, pI - H_{\text{max}}\}) \) where \( H_{\text{max}} \) is the number of critical care hospital beds available. The first term captures deaths that would occur even if there were no constraints on hospital capacity; the second captures the incremental risk of death if one needs critical care but does not receive it.

The max function is not differentiable. Furthermore, some costs arise when \( pI \) is smaller than but close to \( H_{\text{max}} \). For example, medical personnel (doctors, nurses) cannot give the usual care to individual patients if the intensive care unit is almost fully occupied. So we choose the following smooth function \( \max_s(\cdot, \zeta) \) that is increasing and approximately linear in \( pI(t) - H_{\text{max}} \):

\[
\max_s(\{0, pI - H_{\text{max}}\}, \zeta) := \frac{1}{\zeta} \log \left(1 + e^{\zeta(pI(t) - H_{\text{max}})}\right), \quad \zeta \gg 1.
\]

Fig. 1 shows this is an extremely close approximation when \( \zeta \) is large. Hence, deaths from COVID-19 in the objective function can be written as

\[
\xi_1 pI + \xi_2 \max_s(\{0, pI - H_{\text{max}}\}, \zeta),
\]

where \( \xi_1 \) is the death rate from COVID-19 of infected people who need and receive critical care, and \( \xi_2 \) is the additional, incremental death rate when such individuals do not receive that care.
2.4. Objective function

Deaths from COVID-19 are quick compared to those from chronic diseases or even cancer. The average time spent in hospital is about 10 days for those admitted to critical care and half that for others (CDC, 2020). Since fatality rates are 10-15% for all who are hospitalized and perhaps 45% for those receiving critical care, for any reasonable valuation of the social cost of a premature death, the cost of deaths is much greater than the cost of the hospitalization per se. Hence, health costs are represented by \( M \), the cost per COVID-19 death, times the number of deaths.

There are instances of long-lasting morbidity associated with severe cases of COVID-19, with persistent damage to lungs or kidneys, for example. Should those turn out to be large compared to the costs of death, they could be accommodated through this same expression, just by using a larger value of \( M \), if they are also driven by infections and unmet need for care.

Note this makes the health part of the objective function effectively piecewise linear in \( I \). It is common to make cost be convex in an outcome, e.g., to use a quadratic function. However, that would imply the marginal cost of the 100th person who cannot receive needed care is greater than that of the 50th person. Making costs be (effectively) linear in the amount of unmet need values all people equally.

Economic activity is modeled as being proportional to the number of employed people raised to a power, as in a classic Cobb-Douglas model, with that exponent set to \( \sigma = \frac{2}{3} \) (Acemoglu, 2009). Since the model’s time horizon is so short, capital is presumed to be fixed and so is subsumed into the objective function coefficient \( K \) for economic activity.

Susceptible and recovered individuals are eligible to work (infected individuals are assumed to be too sick to work, or are in quarantine), but because of the lockdown the actual number working is only a proportion \( \gamma(t) \) of those eligible to work. Before the lockdown \( \gamma(t) = 1.0 \), during it is reduced to 0.25, and afterwards it bounces back but only partially. The longer the lockdown, the more jobs are lost semi-permanently because firms go out of business. That recovery is modeled as decaying exponentially in the length of the lockdown with a time constant of 0.001 per day, so that if a lockdown ended after six months, 17% of jobs suspended during the lockdown would not reappear, at least until a vaccine became available.

It is presumed that after a vaccine has been widely deployed, there will not again be many deaths from COVID-19, but the economy will not necessarily snap back to full employment instantly, so the objective function includes a salvage function to capture that. In particular, the salvage value function measures the reduction in economic activity between time 0, before COVID-19, and time \( T \) when a vaccine is deployed. That is a rate or flow and needs to be multiplied by a factor \( \Gamma \) reflecting how long it takes for the economy to recover from that underemployment. No one knows the duration or shape of that recovery; which has variously been discussed as being V-shaped, U-shaped or W-shaped. For simplicity we set \( \Gamma = 365 \), as it would be for example if the recovery were linear but took two years, so the area is triangle-shaped.

Note. Omitting deaths after time \( T \) is a slight simplification, because some people who have an active infection at time \( T \) might still die after time \( T \). However, because of the speed of the infection relative to the time horizon, it will be shown that the number of people infected at time \( T \) is not so large, so this is not a major concern.
2.5. Full model

The decision variables are \(\tau_1\) and \(\tau_2\), the times when the lockdown begins and ends, and the full model can be written as:

\[
V(X_0, \tau_1, \tau_2) := V_h(X_0, \tau_1, \tau_2, M) + V_l(X_0, \tau_1, \tau_2, K) + V_s(T, \tau_1, \tau_2, K) \tag{2a}
\]

\[
V^*(X_0) := \min_{\tau_1, \tau_2} V(X_0, \tau_1, \tau_2), \quad X := (S, I, R), \quad L := S + R. \tag{2b}
\]

\[
\dot{X}(t) = \begin{cases} 
SIR_1(X(t), \tau_1, \tau_2) & 0 \leq t < \tau_1 \\
SIR_2(X(t), \tau_1, \tau_2) & \tau_1 \leq t \leq \tau_2 \\
SIR_3(X(t), \tau_1, \tau_2) & \tau_2 < t \leq T 
\end{cases} \tag{2c}
\]

\[
X(0) = X_0 \geq 0 \tag{2d}
\]

\[
\gamma(t, \tau_1, \tau_2) := \begin{cases} 
\gamma_1 & 0 \leq t < \tau_1 \\
\gamma_2 & \tau_1 \leq t \leq \tau_2 \\
\gamma_3(\tau_1, \tau_2) := \gamma_2 + (\gamma_1 - \gamma_2) e^{\gamma_2(\tau_1 - \tau_2)} & \tau_2 < t \leq T 
\end{cases} \tag{2e}
\]

\[
\text{Reff}(t, \tau_1, \tau_2) := \begin{cases} 
R_0^1 & 0 \leq t < \tau_1 \\
R_0^2 & \tau_1 \leq t \leq \tau_2 \\
R_0^3(\tau_1, \tau_2) := R_0^3 + (R_0^1 - R_0^2) e^{\gamma_2(\tau_1 - \tau_2)} & \tau_2 < t \leq T 
\end{cases} \tag{2f}
\]

with

\[
R_0^2 \leq R_0^3 \leq R_0^1. \tag{2g}
\]

To refer to the health care term, the economic (labor) term, and the salvage functions the objective value Eq. (2a) we shortly write

\[
V_h(X_0, \tau_1, \tau_2, M) := M \int_0^T (\xi p I + \xi \max_{\{0, p I - H_{\text{max}}\}}) \, dt \tag{2h}
\]

and

\[
V_l(X_0, \tau_1, \tau_2, K) := \Gamma KL(0)^\sigma \gamma(0, \tau_1, \tau_2)^\sigma - K \int_0^T \gamma(t, \tau_1, \tau_2)^\sigma L(t)^\sigma \, dt \tag{2i}
\]

\[
V_s(T, \tau_1, \tau_2, K) := \Gamma KL(0)^\sigma \gamma(0, \tau_1, \tau_2)^\sigma - \Gamma KL(T)^\sigma \gamma(T, \tau_1, \tau_2). \tag{2j}
\]

The derivation of the necessary optimality conditions for this problem can be found in Appendix A. For the numerical calculations the Matlab toolbox OCMat\(^2\) is used.

2.6. Parameterization

The initial population is normalized to 1.0. Optimization begins with \(I(0) = 0.1\%\) of the population infected and continues over a finite time horizon of \(T = 365\) days, representing the time until a vaccine is hoped to be widely available.

The qualitative results are similar with \(T = 730\) days.

Table 1 summarizes the base case parameters, several of which have already been discussed. The others are addressed here.

There is not truly consensus in the literature about any of the key parameters, but the two for which the widest range of values seem plausible are the probability an infected individual needs critical care, \(p\), and the social cost of a death, \(M\), so we discuss them at length.

Our sense is that the probability of needing hospitalization given a detected infection has been around \(15\%\), about \(30\%\) of those entering the hospital require critical care beds, and about \(45\%\) of those needing critical care die even if they receive that care.

\(^2\)See http://orcos.tuwien.ac.at/research/ocmat_software
There is though enormous uncertainty as to the proper value of \( p \) because there is no agreement as to how many infections remain undetected. At one point it appeared that about half of all infections were detected, implying that the probability of needing a critical care bed given infection, \( p \), might be about \( 50\% \times 15\% \times 30\% = 2.25\% \) and the probability of death given infection and receiving suitable care is about \( 45\% \), making the probability of death given infection a little over \( 1\% \).

That value of \( p \) is only a little smaller than Piguillem and Shi (2020) value of \( p = 1.32\% \), but there have recently though been community-wide antibody tests in several European countries, in New York State, Santa Clara and Los Angeles Counties in California, and in a number of prisons, all suggesting that there may be far more undetected infections than was previously thought. For example, the Santa Clara study concluded that the actual number infected could be 50 to 85 times more than the number of confirmed cases (Bendavid et al., 2020).

If those initial results are confirmed, that would suggest a substantially lower level of \( p \) may be appropriate. Delius et al. (2020), considering data from multiple European countries, suggest that infections could be ten times more common than previously supposed, and so the fatality rate given infection could be an order of magnitude lower, so we also consider \( p = 0.00225 \) in other runs of the model.

In late May, the U.S. Centers for Disease Control (2020) released new guidance for parameters in COVID-19 planning models. Their best estimate scenario had a 65% probability of symptoms given infection, a 3.4% probability of hospitalization given symptoms, and a roughly 25% probability of needing ICU care given hospitalization, suggesting that \( p \) might be about \( 65\% \times 3.4\% \times 25\% = 0.5525\% \).

Because of the uncertainty concerning \( p \), the results below include sensitivity analysis with respect to this key parameter. \( \xi_1 \) is the death rate per day for infected people who need critical care and receive it. If the death rate for such individuals over an entire infection is 45% and the average dwell time in the \( I \) state is 15 days, then the death rate per day is \( \xi_1 = \alpha 45\% \), or about 3%.

\( \xi_2 \) is the additional, incremental death rate per day for infected people who need critical care but do not receive it. If the death rate for such individuals over an entire infection is 100% and the average dwell time in the \( I \) state is 15 days, then the incremental death rate per day is \( \xi_2 = \alpha (1 - 45\%) \), or about 3.67%.

Since the average length of stay is shorter for regular vs. critical care patients, about 30% of hospital patients require critical care, and fewer than 30% of all hospital beds are critical care beds, the constraint will be on critical care beds, not total hospital beds. So we make them the basis for \( H_{\text{max}} \).

Tsai et al. (2020) suggest that in the U.S., 58,166 of the existing 84,750 ICU beds could be made available for treating COVID-19 patients. Given the U.S. population is about 330 million, that is 0.176 per 1,000 people. The model acts as if patients who need critical care at some point need that care throughout their 15-day infection, but the average dwell time in critical care is shorter than that. So we double that value and set \( H_{\text{max}} = 0.35 \) per 1,000 or 0.00035. There are roughly ten times as many hospital beds as critical care beds, so sensitivity analysis with larger values of \( H_{\text{max}} \) may be appropriate, as some regular beds could be converted over to critical care beds.

Note that if \( p = 2.25\% \) and \( H_{\text{max}} = 0.35 \) per 1,000 then a crisis occurs if more than about 1.5% of the population is infected at any one time, roughly consistent with Atkeson (2020) warning that problems could arise if more than 1% of the population is infected at any one time. However, if \( p = 0.5525\% \) or \( 0.225\% \), then a much larger proportion of the population can be infected simultaneously before the healthcare system becomes overwhelmed.

Estimating the relative value of lost work vs. lost lives is tricky, to say the least, so without loss of generality we set \( K = 1 \) and consider a very wide range of values for \( M \).

Still, it is helpful to determine at least roughly the size of \( M \) relative to \( K \). Alvarez et al. (2020) value a premature death at 20\times GDP per capita, while noting that Kniesner et al. (2012) use a much greater value of 150\times GDP per capita. Lower values would apply if one focused on years-of-life-lost,
since most deaths are among the elderly, especially those with other pre-existing conditions. E.g., Richardson et al. (2020) report that the vast majority of those hospitalized for COVID-19 had prior serious comorbidities such as hypertension, obesity, and diabetes, to the extent that their estimated 10-year survival rate absent COVID-19 was only 53%. So we consider a range from 10× to 150×GDP per capita.

\[ K(\gamma L)^{2/3} \] measures GDP per day - \( K \) is the constant that we assume to capture everything except labour, so 365\( K(\gamma L)^{2/3} \) equals the nation’s GDP. Since the population size is normalized to 1.0, that implies values of a premature death, \( M \), somewhere in the range from 3,650 to 54,750. We set \( M = 16,255 \) for analyses with a fixed \( M \).

\[
\begin{array}{|c|c|c|c|c|c|c|c|}
\hline
\alpha & R_{01}^{1,2,3} & H_{\text{max}} & p & M & K & \gamma_{1,2,3} & \Gamma \\
\hline
\frac{1}{15} & 2.5, 0.8, 2.0 & 3.5 \times 10^{-4} & 2.25 \times 10^{-2} & 16255.8 & 1 & 1, 0.25, 0.75 & 365 \\
\hline
\kappa_2 & \sigma & \mu & \nu & \mu_f & \zeta & \xi_1 & \xi_2 \\
\hline
10^{-3} & \frac{1}{5} & \frac{1}{365} & \frac{1}{365} & \frac{1}{15} & 5000 & 0.03 & \frac{0.35}{15} \\
\hline
\end{array}
\]

Table 1: Base case parameter values.

3. Results

3.1. Optimizing only over the end time, \( \tau_2 \)

Most countries have already started their lockdown, so we start by optimizing only over \( \tau_2 \), but for various values of \( \tau_1 \) because different places started their lockdowns at different times. Fig. 2 plots the resulting optimized value of \( \tau_2 \) (blue curve) vs. \( \tau_1 \). For convenience, the value of \( \tau_1 \) is also shown, by the black line, so it is easy to visualize the duration of the lockdown as the height of the gap between the blue and black lines.

As \( \tau_1 \) increases from 0 to a little more than 25, \( \tau_2 \) increases by about the same amount, keeping the duration of the lockdown roughly constant. That makes intuitive sense. The later one begins the lockdown, the later it should be relaxed.

However, as \( \tau_1 \) increases further, from about 25 to 55, the optimal \( \tau_2 \) decreases. Hence, over that range, countries that started their lockdown later should end it sooner, something that is perhaps surprising. It may be that the timing of the ending of the lockdown is related to the accumulation of infections, which happens rapidly when the lockdown is not initiated early.

The solid red vertical line indicates the value of \( \tau_1 \) that produces the lowest total cost, when \( \tau_2 \) is optimized, and that is for \( \tau_1 = 13.3 \).

That this optimal value of \( \tau_1 \) is greater than zero is perhaps a second surprising result. If lockdowns are costly, in this case because they reduce work, then it is possible to start a lockdown too soon, and for a later starting date to be preferred. Since the epidemic grows explosively, even exponentially, before the lockdown, some might have thought that earlier was always better.

Perhaps not surprisingly, if the initial reproductive rate \( R_{01} \) is smaller then this optimal starting time comes later. This may explain some of the tension observed between residents of rural areas and urban areas over lockdown timing. The optimal date for starting the lockdown in a place like New York City, with high population density (so likely higher \( R_{01} \)) and early onset could be sooner than the optimal date for starting the lockdown elsewhere in the country.

When \( \tau_1 \) equals the critical value of 55, indicated by the vertical dashed line, there are two alternative optimal solutions for \( \tau_2 \). Both \( \tau_2 = 220 \) (so lockdown for almost 6 months) and \( \tau_2 = 105 \) (so a lockdown of less than two months) produce the same objective function value. And as \( \tau_1 \) increases beyond 55, the best end time for the lockdown decreases further. Eventually, if the lockdown doesn’t start until day 94, it is optimal not to start a lockdown at all.
Figure 2: Fixed initial lockdown time $\tau_1$ and optimally chosen time $\tau_2$. For $\tau_1 = 55$ there exists a Skiba solution, i.e. there are two different solution paths which deliver the same objective value.

The interpretation for this is that there are two broad strategies that can be pursued. One, might be called an ‘eradication’ strategy. It locks down long enough to push infections down to minimal levels, with just a modest rebound shortly before the end of the time horizon when a vaccine becomes available. The second, which might be called ‘curve flattening’, uses the lockdown to reduce the size of the initial spike in infections, so fewer come when infections exceed hospital capacity. The later the lockdown starts, the harder it is to pull off the eradication strategy, until at some critical point (in this case $\tau = 55$) it becomes optimal to switch to the ‘curve flattening’ strategy which requires a much shorter lockdown.

Optimizing over both $\tau_1$ and $\tau_2$ makes these results easier to understand. Figure 3 shows the optimal values of $\tau_1$ and $\tau_2$ (left panel) and the optimal solution value $V^*$ (right panel) as a function of $M$, the cost of a COVID-19 death. It shows three regions. In region I, on the far left when $M$ is very small, it is optimal to never lockdown and just let the epidemic run its course. Basically, if one does not care about deaths, beyond their effect on economic productivity from reducing work, then the lockdown is not worth it.

Figure 3: Figure showing the different regimes for varying $M$. The other parameter values are taken from Table 1. Mathematical description of the three regimes: Regime I: no lockdown, i.e. $\dot{X}(t) = SIR_1(t), 0 \leq t \leq 365$, Regime II: lockdown in interval $(0, 365)$, i.e. $0 < \tau_1 < \tau_2 < 365$, Regime III, lockdown starts immediately, i.e. $0 = \tau_1 < \tau_2 < 365$. 

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In region III, corresponding to large values of $M$, one should begin the lockdown immediately and keep it in place until shortly before the vaccine becomes available.

In Region II both broad strategies (flattening and eradication) can be considered, with flattening being preferred in Region IIa, eradication being preferred in Region IIb, and either being optimal when $M$ is just exactly at the value separating Regions IIa and IIb. At that $M = 16255.8$, there is a so-called Skiba point.

The existence of the Skiba point implies another surprising result. Both short and long lockdowns can be optimal for the exact same set of parameter values. Exact equality only occurs at that specific value of $M$, but for a range of values in that neighborhood, the two very different strategies perform nearly as well. So a single person can be indifferent, or nearly indifferent, between two very different approaches.

Furthermore, two people with modestly different relative valuations on work and health can favor very different policies, if their modestly different values of $M$ lie on either side of the Skiba threshold.

As mentioned, plausible values of $M$ range from 3,650 to 54,750. The lower end of that range lies within Region I, but increasing $M$ within that range would carry the solution through Regions IIa and IIb, all the way into Region III. As mentioned earlier, we focus on qualitative results, not specific values. So we do not think the model shows that one strategy or another is necessarily the best. Rather, we would say that the model suggests that it is hard to be certain about that. Any one of the strategies could plausibly turn out to be optimal, depending on how uncertainty about the parameter values is resolved.

Figure 4 contrasts the two solutions when $M$ is exactly at its Skiba threshold value of 16255.8. The vertical lines indicate the start ($\tau_1$) and end ($\tau_2$) times of the lockdown for the curve flattening strategy (solid lines; left panel) and the eradication strategy (dashed lines; right panel). The blue lines show the numbers who are infected under those two strategies, while the gray line shows the uncontrolled epidemic with no lockdown.

![Figure 4: Panel (a) and (b) show the two different solutions paths emerging from the Skiba point for the number of infected $I(\cdot)$ (blue lines). The gray line in panel (a) depicts the uncontrolled path without lockdown for comparison. For the total costs and its shares see Table 2. The horizontal red line shows the health care capacity $H_{\max}/\rho$.](image)

Note the very different vertical scales in the two panels. The eradication strategy keeps the number of infections well below the point where hospital capacity becomes binding, whereas the curve flattening strategy (greatly) exceeds that capacity, at times.

Here the optimal curve flattening strategy experiences a second, much smaller bump, but for other parameter values it can be a second spike. That is, it can be optimal to end the lockdown in a way that does create a resurgence of the epidemic. That the epidemic spikes again after ending a lockdown
does not imply that ending the lockdown was a mistake.

Table 2 contrasts the valuations of the three components of the objective function under the two strategies, and the uncontrolled epidemic as a foil. The values can perhaps best be understood in percentage terms. In the absence of an epidemic, economic output would have been 365, and in the absence of controls the health cost would be 299. So under the shorter lockdown, economic output falls by about 12% for the year, and the costs of premature deaths are reduced by about one-quarter. With the longer lockdown, 95% of the health costs are averted, but economic output falls nearly 50%.

Table 2: The total costs, the share of costs of the health care system, profit from labor and the salvage value for the Skiba solution and the uncontrolled solution of Fig. 4 ($M = 16255.8$, $K = 1$) and the lockdown times.

|                  | Eradication | Flattening | Uncontrolled |
|------------------|-------------|------------|--------------|
| $V^*(X_0)$       | 288.2       | 288.2      | 306.7        |
| $O_h(X_0, \tau_1, \tau_2, M)$ | 228.3       | 15.0       | 299.0        |
| $O_l(X_0, \tau_1, \tau_2, 1)$ | 44.6        | 186.1      | 9.1          |
| $O_s(T, \tau_1, \tau_2, K)$ | 15.3        | 87.1       | -1.4         |
| $[\tau_1, \tau_2]$ | [64.8, 110.9] | [16.8, 300.9] | $\emptyset$ |

3.2. Sensitivity analysis with respect to $M$

Figure 3 is a sensitivity analysis with respect to the value of $M$, i.e., the relative valuation placed on health as opposed to economic outcomes.

The upper right panel shows which type of solution is optimal for various values of the two most uncertain parameters, $p$ and $M$. Varying $p$ between 0.00225 and 0.0225 and $M$ between 0.365 and 5.475 × 10^4 spans regions I, IIa, IIb and III. That indicates that people can disagree about the best broad strategy depending on their judgments about how to balance health and economic outcomes and on the degree to which the new antibody testing results sway judgments about how many infections remain undetected. This shows that two reasonable, intelligent people who agree completely on how to think about the epidemiology, health, and economics of the COVID-19 crisis can still reach very different conclusions about what policy is best, just because of differences concerning these two key parameters, about which there is no scientific agreement, at least at present.

For $p = 0.5525\%$, corresponding to CDC’s (2020) new best parameter guidance, the eradication strategy is not optimal unless $M$ is 507, implying that the lockdown should end before a vaccine is available unless the value on preventing a COVID-19 death is quite high.

That upper right panel is drawn for $H_{max} = 0.00035$. The upper left panel of Figure 5 is a bifurcation diagram showing which strategies are optimal as a function of both $M$ and $H_{max}$, which represents the number of critical care beds, while holding $p$ at the value of 0.0225. That diagram shows that the qualitative results would not change until $H_{max}$ increased to a very large multiple of its base case value of 0.00035. For the most part, the curves in that panel slope up and to the right, meaning that if treatment capacity and the valuation of a life both increase, the structure of the solution remains the same. Eventually those curves turn vertical when hospital capacity is no longer binding.

The exception is that for $M$ small we have, for increasing $H_{max}$, first Region I then IIa and then I again. This curious pattern can be explained as follows. Because $M$ is small, lockdowns will be short, if they exist at all, and so most people will become infected. If $H_{max}$ is very small then almost all of those infections will occur when hospital capacity has been exceeded even if there is a lockdown. Conversely, if $H_{max}$ is very large, then almost all infections will occur when there is adequate hospital capacity, even if there is no lockdown. It is only for intermediate values of $H_{max}$ that the lockdown has
the ability to tip a meaningful share of the infections from times when hospital capacity is exceeded to times when adequate care can be provided.

The lower left panel shows how the optimal solutions depend on both $R_0^2$ and $M$ when $p$ is fixed at the value 0.0225. Not surprisingly, lockdowns are preferred when $M$ is large and/or $R_0^2$ is low, which explains why the boundaries are generally upward sloping.

Within the range of $M$ of greatest interest, the boundaries between no lockdown and short lockdown, and between short and long lockdowns are almost vertical, indicating that the precise value of $R_0^2$ is less important than the value of $M$ for driving what strategy is optimal.

An exception is the curve between regions IIb and III. That is because for very low values of $R_0^2$, the lockdown is so effective that it is not necessary to start the lockdown immediately to fight the epidemic.

For $R_0^2 = 1.27$ and $M = 26631$ there is a so-called triple Skiba point, where three qualitatively different strategies perform equally well. The lower right panel depicts the time trajectories for $I(t)$ under those three strategies which are: (1) Locking down immediately (dashed line), (2) Locking down around day 25 and holding that nearly until the vaccine arrives (dot-dash lines), and (3) merely flattening the curve with a relatively short lockdown from $\tau_1 = 56$ to $\tau_2 = 136$. Infections increase initially after lockdown under the two eradication strategies because $R_0^2$ is greater than one at that point.

One additional sensitivity analysis (not shown) considered the scenario when treatment is much more effective, so that the probability of dying if one needs and receives critical care is only 4.5% not 45%. That makes the extra penalty for exceeding hospital capacity gets much worse, 95.5% not 55%, but this does not change much about the structure of the solution.

4. Conclusions

4.1. Primary findings

This paper is about planning the lockdown in COVID-19 times. The aim of installing a lockdown is to limit social contact to reduce the number of people getting infected. The drawback is the immediate reduction in economic activity. The latter also has an effect after the lockdown has ended, because it takes time for the economy to recover.

Our model is based on the epidemiological SIR dynamics, which we combine with a basic Cobb-Douglas model of economic activity to develop a framework that can evaluate the above-described tradeoff. In many countries the number of COVID-19 patients needing intensive care treatment came close to or even exceeded the available intensive care capacity, so that aspect is included in our model.

We find that essentially two different solution patterns can be optimal. One is what we call an *eradication strategy*, where installing a long lockdown period not only significantly reduces the bad health effects of the epidemic but also economic activity. The other is a *curve flattening strategy* characterized by a relatively short lockdown period. The idea is to reduce the peak of the number of infected in order to limit the violation of the intensive care capacity constraint, where at the same time economic activity is not harmed as much.

Interesting is that the performance of both strategies can be quite similar. Indeed, there are specific parameter values such that decision makers with the same preferences for health versus the economy can still wisely for completely different lockdown policies.

We note that over the plausible ranges for two key parameters, namely the proportion of infections that require critical care and the valuation placed on preventing a COVID-19 related death, all types of solutions can be optimal. We view that as indicating that a degree of humility and open-mindedness may be appropriate; at least in our model, it is not necessarily clear whether a longer or a shorter lockdown is best.
Figure 5: Bifurcation diagrams. Panel (a) shows the bifurcation diagram for parameter values of Table 1 and varying $H_{\text{max}}$ and $M$. Panel (a) shows the bifurcation diagram for parameter values of Table 1 while varying $p$ and $M$. Panel (c) shows the bifurcation diagram for $M$ and $R_0^2$. In panel (d) a triple Skiba solution is depicted. In that case two “eradication” solutions and one “curve flattening” solutions exist. The dashed solution starts with quarantine immediately. The black horizontal lines depict base case parameter valued. The solid vertical lines in panel (b) delimit the region $M \in [0.365 \times 10^3, 5.475 \times 10^4]$. The dashed vertical line shows the $M$ value of the base case (Skiba case).
4.2. Limitations

This model simplifies in many respects that could bear on the optimal timing and duration of a lockdown. That is why we stress qualitative results, not numerical results. These limitations include:

- Transmission mitigation is modeled crudely as a lockdown being in place or not. The intensity of the lockdown could instead be modeled as a continuous control variable. Also, the most important innovations might be efforts to re-engineer operations to make them safer, rather than either shutting them down or allowing them to operate normally.

- This model does not divide the population by age or pre-existing health condition. Death rates are sharply higher for those who are older or who have pre-existing conditions. Strategies that have longer or more restrictive lockdowns for vulnerable populations may be prudent but cannot be modeled here.

- The technology for treating COVID-19 cases may improve over time. E.g., Remdesivir and convalescent plasma treatments are now being used. Improving technology could favor earlier and longer lockdowns if that defers cases until technology is better.

- The model does not consider seasonality. If virus transmission is slower during warmer weather, that might favor easing lockdowns during summer.

- The model does not consider the possibility of long-term health effects of survivors of COVID-19, although there are reports of lasting harm not only to lungs, but also to the brain and kidney.

4.3. Further work

A variety of extensions of this model are possible, including making the timing of the vaccine’s arrival unknown, allowing for multiple lockdowns separated by periods of relaxation, and altering the epidemic’s dynamics when the number of new infections is small enough that contact tracing can lead to quickly quarantining everyone that an incident case might have infected.

The epidemic model could be enriched in various ways including allowing for multiple regions connected by migration (which might be affected by the lockdown), making the rates of social interaction a distributed parameter that varies across a heterogeneous population, and modeling explicitly the population’s patient with and commitment to lockdown restrictions.

Suppose $\tau_2 - \tau_1$ is fixed, when should one start that fixed-length lockdown?

A. Necessary Optimality Conditions

Setting $\tau_0 := 0$ and $\tau_3 := T$ the Hamiltonians for the three stages $i = 1, 2, 3$ are

$$\mathcal{H}_i(X_i, \Lambda_i, \tau_1, \tau_2) := M (\xi_1 p I + \xi_2 \max \{0, p I - H_{\text{max}}\}, \zeta) - K \gamma(t, \tau_1, \tau_2)^\sigma L(t)^\rho + \Lambda'_i \text{SIR}_i(X_i, \tau_1, \tau_2),$$

(A.1)

with

$$X_i(t) := X(t), \quad \tau_{i-1} \leq t \leq \tau_i$$

$$\Lambda_i(t) := (\lambda_1(t), \lambda_2(t), \lambda_3(t)), \quad \tau_{i-1} \leq t \leq \tau_i.$$

The costates satisfy the canonical system

$$\dot{\Lambda}_i(t) = -\frac{\partial}{\partial X_i} \mathcal{H}_i(X_i(t), \Lambda_i(t), \tau_1, \tau_2), \quad \tau_{i-1} < t < \tau_i.$$  

(A.2)
Since the RHS of the ODEs (A.2) are continuously differentiable, the costates can continuously be extended to the left and right side of the interval $\tau_{i-1} < t < \tau_i$. Thus

$$\Lambda_i(\tau_{i-1}) := \lim_{t \to \tau_{i-1}^+} \Lambda_i(t)$$

and

$$\Lambda_i(\tau_i) := \lim_{t \to \tau_i^-} \Lambda_i(t)$$

uniquely exist. For the derivatives of the Hamiltonians with respect to the switching times we find

$$\frac{\partial}{\partial \tau_1} H_i(X_i, \Lambda_i, \tau_1, \tau_2) = \begin{cases} 0 & i = 1, 2 \\ \gamma_3(\tau_1, \tau_2) K \sigma \gamma_3(\tau_1, \tau_2) \sigma^{-1} L^\sigma + \Lambda_i' \frac{\partial}{\partial \tau_1} \text{SIR}_i(X_i, \tau_1, \tau_2) & i = 3 \end{cases}$$

$$\frac{\partial}{\partial \tau_2} H_i(X_i, \Lambda_i, \tau_1, \tau_2) = \begin{cases} 0 & i = 1, 2 \\ -\gamma_3(\tau_1, \tau_2) K \sigma \gamma_3(\tau_1, \tau_2) \sigma^{-1} L^\sigma + \Lambda_i' \frac{\partial}{\partial \tau_2} \text{SIR}_i(X_i, \tau_1, \tau_2) & i = 3 \end{cases}$$

The Hamiltonian in the third stage (after the lockdown) and the salvage value explicitly depend on the switching times $\tau_1$ and $\tau_2$. Thus for $0 < \tau_1 < \tau_2 < T$ the necessary optimality conditions at the switching times $\tau_1$ and $\tau_2$ write as (cf. Tomiyama and Rossana, 1989)

$$H_2(X_2(\tau_1), \Lambda_2(\tau_1), \tau_1, \tau_2) - H_1(X_1(\tau_1), \Lambda_1(\tau_1), \tau_1, \tau_2) = \int_{\tau_2}^{T} \frac{d}{d\tau_1} H_3(X_3(t), \Lambda_3(t), \tau_1, \tau_2) \, dt + K \frac{d}{d\tau_1} \gamma(T, \tau_1, \tau_2)$$

$$H_3(X_3(\tau_2), \Lambda_3(\tau_2), \tau_1, \tau_2) - H_2(X_2(\tau_2), \Lambda_2(\tau_2), \tau_1, \tau_2) = \int_{\tau_2}^{T} \frac{d}{d\tau_2} H_3(X_3(t), \Lambda_3(t), \tau_1, \tau_2) \, dt + K \frac{d}{d\tau_2} \gamma(T, \tau_1, \tau_2)$$

and at $\tau_i$, $i = 1, 2$ the costates satisfy

$$\Lambda_1(\tau_1) = \Lambda_2(\tau_1)$$

$$\Lambda_2(\tau_2) = \Lambda_3(\tau_2).$$

At the endtime $T$ the costates satisfy the transversality condition

$$\Lambda(T) = \begin{pmatrix} \lambda_1(T) \\ \lambda_2(T) \\ \lambda_3(T) \end{pmatrix} = \begin{pmatrix} -\Gamma K \sigma L(T)^{\sigma^{-1}} \gamma(T, \tau_1, \tau_2)^\sigma \\ 0 \\ -\Gamma K \sigma L(T)^{\sigma^{-1}} \gamma(T, \tau_1, \tau_2)^\sigma \end{pmatrix}.$$

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