COVID-19 AND THE WELFARE EFFECTS OF REDUCING CONTAGION*

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Abstract: I use a simple SIR model, augmented to include deaths, to elucidate how pandemic progression is affected by the control of contagion, and examine the key trade-offs that underlie policy design. I illustrate how the cost of reducing the "reproduction number" R_0 depends on how it changes the infection rate, the total and incremental number of deaths, the duration of the pandemic, and the possibility and impact of a second wave. Reducing R_0 reduces the number of deaths, but extends the duration (and hence economic cost) of the pandemic, and it increases the fraction of the population still susceptible at the end, raising the possibility of a second wave. The benefit of reducing R_0 is largely lives saved, and the incremental number of lives saved rises as R_0 is reduced. But using a VSL estimate to value those lives is problematic.

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1 Introduction.

The COVID-19 virus is being fought largely by policies to reduce contagion. These policies, which have been referred to broadly as "social distancing," include forced closures of businesses and restrictions (either mandatory or recommended) on travel and social gatherings. Research has accelerated on the development of anti-viral drugs to treat the disease and a vaccine to reduce susceptibility, but is unlikely to affect the spread of the virus in the near term. At this point, reducing contagion is the only effective policy tool, but it is extremely expensive in terms of its impact on the economy. So one would naturally ask to what extent and for how long should governments impose social distancing in order to reduce the spread of COVID-19?

Several recent papers have addressed this question using off-the-shelf epidemiological models to conduct cost-benefit analyses of alternative social distancing policies. The cost of social distancing is largely unemployment and lost GDP; firms shut down, some go out of business, and workers lose jobs. The benefit is the value of lives saved and avoided medical treatments. Scherbina (2020), for example, uses an epidemiological model from Ferguson et al. (2020) to estimate deaths and hospitalizations under alternative durations of enhanced social distancing, and uses assumptions regarding weekly employment impacts to estimate lost GDP for each duration. Using "value of a statistical life" (VSL) estimates to monetize deaths, she finds the policy duration that maximizes the benefit-cost ratio.[1] Greenstone and Nigam (2020) use the same Ferguson et al. (2020) model but focus only on the benefits — lived saved and medical expenses avoided — of alternative policies. Using age-adjusted VSL estimates, they find the benefit to the U.S. of social distancing to be about $8 trillion. (Later I explain why using VSL estimates might not make sense in this context.)

Others have calibrated the basic Susceptible-Infected-Removed (SIR) epidemic model to COVID-19 and used it to study potential effects of policy-based variations in contagion.[2]

[1]Medical expenses are included (but far outweighed by the value of lost lives), and lost GDP is augmented by assumptions regarding direct sectoral output losses. The epidemiological model in Ferguson et al. (2020) is an updated version of one developed in Ferguson et al. (2006). The VSL is the marginal rate of substitution between wealth (or discounted lifetime consumption) and the probability of survival. For its use to value the *prevention* (as opposed to treatment) of pandemics, see Martin and Pindyck (2019).

[2]The "R" in SIR is often referred to as *recovered*, but that ignores deaths, i.e., assumes that everyone removed from the susceptible pool recovers.

An advantage of this model is that contagion can be embodied in a single parameter (as discussed below). Stock (2020) focuses on how limited testing (asymptomatics are generally not tested) affects our ability to calibrate the model and evaluate the economic costs of a policy. Atkeson (2020*b*) and Anderson et al. (2020) explore how alternative dynamic social distancing policies (e.g., a year of fixed social distancing versus an initial period of intense social distancing followed by a relaxation of the policy) can affect the spread of the disease. And Thunström et al. (2020) and Alvarez, Argente and Lippi (2020) used the SIR model, combined with assumptions about mortality rates and policy-induced losses of GDP, for cost-benefit analyses of social distancing policies.[3]

So how long should governments limit social interactions? I do not try to answer this question. Both costs and benefits are very difficult to estimate, as are the parameters that go into the epidemiological models, and this limits the value of any point estimates. Instead, I use the simple SIR model, augmented to include deaths (D), to show how pandemic progression is affected by the intensity and duration of a social distancing policy, and to elucidate the key factors that underlie the evolution of a pandemic and the key trade-offs that underlie policy design. This SIRD model has three free parameters, which I calibrate to roughly fit the COVID-19 pandemic, and I then use the model to address the following:

(1) Holding death and recovery rates fixed, how does the maximum fraction of the population that becomes infected, $I_{\max}$, depend on the degree of contagion? (2) As the epidemic ends, what fraction of the population will have died, what fraction will have recovered, and what fraction will have avoided the disease and remain susceptible? (3) How does the duration of the pandemic (the number of days until significant numbers of new infections end) depend on the degree of contagion? (4) Given the fraction of susceptibles at the end, how stringent must social distancing be to avert a second cycle of infections? (5) If a vaccine is developed, what fraction of the population must be vaccinated to prevent more infections, and how does it depend on the fraction of susceptibles? (6) What are the key trade-offs that underlie the costs and benefits of a social distancing policy? (7) How should we monetize the value of lives saved? The use of a VSL estimate is convenient, but is it warranted?

[3]In related work, Eichenbaum, Rebelo and Trabandt (2020) and Jones, Philippon and Venkateswaran (2020) embed the SIR model in macroeconomic models of consumption and production, with economic activity affecting contagion and the spread of the disease. Also, Barro, Ursúa and Weng (2020) use mortality and GDP data from the 1918-1919 Spanish Flu to estimate bounds on possible COVID-19 outcomes.

2 Disease Dynamics in the SIRD Model.

I take the basic SIR model and add an equation to account for deaths. Setting the initial population to $N_0 = 1$ (so that the state variables are measured as fractions of the population), the model can be written as:[4]

$$dS/dt = -\beta S_t I_t \tag{1}$$

$$dI/dt = \beta S_t I_t - (\gamma_r + \gamma_d) I_t \tag{2}$$

$$dR/dt = \gamma_r I_t \tag{3}$$

$$dD/dt = \gamma_d I_t \tag{4}$$

Here S_t is the fraction of the population that is susceptible, I_t the fraction infected, R_t the fraction that have recovered, and D_t the fraction that have died.[5] Note that at $t = 0$, $R_t = D_t = 0$, so $S_0 + I_0 = N_0 = 1$. However, we need $I_0 > 0$ or else the epidemic doesn't begin, so to apply this to COVID-19 we will take I_0 to be very small. An important assumption in this model is that a person who recovers from an infection becomes immune, i.e., is no longer susceptible. (Whether this is realistic for COVID-19 is an open question.)

The parameters of this model can be interpreted as follows. First, β is usually referred to as the *contact rate*, but it can also be thought of as the degree of contagion. It measures how the interaction between susceptibles and infectives causes more susceptibles to become infected (reducing S_t and increasing I_t). It is this parameter that social distancing and related policies seek to control.

Next, $\gamma \equiv \gamma_r + \gamma_d$ is the *removal rate*, i.e., the rate at which people leave the pool of infectives either by recovering ($\gamma_r I_t$) or dying ($\gamma_d I_t$). As is usually done, I treat γ and its components as constants, although successful research on COVID-19 treatments would raise γ_r and lower γ_d. The ratio $\rho = \gamma/\beta$ is referred to as the *relative removal rate*, and $1/\rho$ is referred to as the *reproduction number* or reproduction rate, and is (unfortunately) denoted

[4]The SIR model was proposed by Kermack and McKendrick (1927), and is discussed in detail, along with numerous deterministic and stochastic variations and extensions, along with applications, in Bailey (1975) and Anderson and May (1992). Allen (2017) describes a stochastic version of the basic model. Avery et al. (2020) provide a critical review of this and other models in the context of COVID-19.

[5]Some studies, e.g., Atkeson (2020*b*), include an exposed group, E_t, only some of which become infected. This adds a state variable and a parameter, but the disease dynamics remains the same.

by R_0. With γ constant, changing R_0 changes β, the degree of contagion, and it is R_0 that is usually treated as the key policy variable. If $R_0 \leq 1$, removals from the pool of infectives (as infected people recover or die) exceeds entry into the pool, so the pandemic cannot take off.[6] This can be seen from eqn. (2); $dI_0/dt > 0$ requires the initial fraction of susceptibles, S_0, to exceed $1/R_0$. So if $S_0 < 1$, i.e., not everyone is susceptible, a greater degree of contagion is needed ($R_0 > 1/S_0$) for the epidemic to take off.

This SIRD model is extremely simple and ignores several aspects of COVID-19 and the design of polices to control it. Perhaps most important, it treats the epidemic as occurring within one large mass of homogeneous individuals, whereas in fact outbreaks are regional, with each region consisting of heterogeneous individuals, and with new outbreaks igniting as regions interact with each other. Nonetheless, the model can help elucidate the dynamics of COVID-19 and provide rough answers to several interesting questions.

2.1 Some Basic Analytics.

Assuming that we start with a fraction of infectives I_0 close (but not equal) to zero, and thus a fraction of susceptibles close to 1, the speed, duration and intensity of the epidemic depend on the values of β and γ. We want to address the following questions: (1) What is the maximum fraction of the population that will become infected, $I_{\max}$, and taking γ_r and γ_d as fixed, how does it depend on the contact rate β? (2) How does the duration of the epidemic depend on β? (3) As the epidemic ends, what fraction of the population will have died, what fraction will have recovered, and what fraction will have avoided the disease and remain susceptible? (4) If the fraction of susceptibles at the end is large, would a relaxation of the social distancing policy generate another cycle of infections and deaths? (5) Suppose a vaccine is developed. What fraction of the population must be vaccinated to prevent more infections, and how does it depend on the fraction of susceptibles at the time?

The Pool of Infectives.

To find the behavior of I_t and $I_{\max}$, divide eqn. (2) by eqn. (1):

$$dI/dS = -1 + \rho/S_t \ , \tag{5}$$

[6]This was roughly the case for the Ebola pandemic: Infectives were contagious only when very sick (or dead), and the fatality rate was very high, so β was low and γ was high, making $R_0 < 1$.

so

$$I_t = \int_0^t [-1 + \rho/S] dS = S_0 + I_0 - S_t + \rho \log(S_t/S_0) = 1 - S_t + \rho \log(S_t/S_0) \, .$$

I_t will reach a maximum when $dI/dS = 0$, i.e., at the point where $S^* = \rho$. Then $dI/dt >$ $(<)$ 0 when $S_t > (<)$ ρ. Thus the maximum number of infectives is

$$I_{\max} = 1 - \rho + \rho \log(\rho/S_0) \approx 1 - \rho + \rho \log \rho \tag{6}$$

Recall that $\rho = \gamma/\beta$, and note that $\partial I_{\max}/\partial \rho = \log \rho$. So as long as $\rho < 1$, i.e., the reproduction number $R_0 = 1/\rho > 1$, a decrease in the contact rate β will reduce the maximum number of infectives. (If $R_0 = 1$, $I_{\max} = 0$, and the pandemic cannot take off.)

The Dead and the Susceptibles.

As the epidemic (asymptotically) ends, the total number of deaths (denoted by D_∞) depends on the number of infectives at each moment in time, and the rate at which those infectives recover or die (i.e., the parameters γ_r and γ_d). But the total number of deaths is also a simple function of the remaining number of susceptibles, S_∞, which we can determine as follows.

Dividing eqn. (1) by eqn. (3), $d \log S_t / dR_t = -\beta/\gamma_r$, so $\log(S_\infty/S_0) = (-\beta/\gamma_r) R_\infty$. But $R_\infty = N_0 - D_\infty - S_\infty = 1 - D_\infty - S_\infty$, so

$$\log(S_\infty/S_0) = -(\beta/\gamma_r) S_\infty - \beta/\gamma_r - (\beta/\gamma_r) D_\infty \, .$$

Dividing (1) by (4), $d \log S_t / dD_t = -\beta/\gamma_d$, so $D_\infty = -(\gamma_d/\beta) \log(S_\infty/S_0)$. Substituting above for D_∞ gives us the fundamental equation for the final number of susceptibles, S_∞:

$$(\gamma/\beta) \log(S_\infty/S_0) - S_\infty + 1 = 0 \, . \tag{7}$$

Then S_∞ is the root of this equation (which can be solved numerically). This equation lets us determine the fraction of the population still susceptible when the epidemic ends. Note that reducing $R_0 = \beta/\gamma$ raises S_∞, and $S_\infty \to S_0$ as $R_0 \to 1$.

Since S_0 is close to 1, and using (7), we can write the total number of deaths as

$$D_\infty = (\gamma_d/\gamma)(1 - S_\infty) \, . \tag{8}$$

How does the final number of susceptibles and total number of deaths depend on the contact rate β? From (8), $dD_\infty/d\beta = (-\gamma_d/\gamma)dS_\infty/d\beta$. Taking the total differential of eqn. (7) with respect to S_∞ and β,

$$\frac{dS_\infty}{d\beta} \;=\; \frac{S_\infty \log S_\infty}{\beta(1-S_\infty)} \;\leq 0$$

A higher contact rate means more people are infected during the course of the epidemic, making the final number of susceptibles, S_∞, lower.

Since policies to reduce the contact rate are usually expressed in terms of the reproduction number $R_0 = \beta/\gamma$, and $dD_\infty/dR_0 = \gamma dD_\infty/d\beta$, we have

$$\frac{dD_\infty}{dR_0} \;=\; -\frac{\gamma_d S_\infty \log S_\infty}{\gamma R_0(1-S_\infty)} \;\geq 0\;. \tag{9}$$

Once we solve for S_∞, we can use eqn. (9) to determine how many deaths are averted if R_0 is reduced by an incremental amount.

Given a value for lives saved, eqn. (9) can be used to calculate a "willingness to pay" (WTP) for reductions in R_0. After scaling up by the actual population, it gives the social demand curve for "quantities" of R_0. Of course to determine the optimal value of R_0, we also need a supply curve, i.e., the incremental cost of reducing R_0 as a function of R_0. That incremental cost might be a measure of lost GDP, as in some of the cost-benefit studies cited in the Introduction.

A Possible Second Wave.

The solution to eqn. (7) is $S_\infty > 0$, i.e., at the end not everyone will have been infected and thus (by assumption) immune. Furthermore, the lower the reproductive number R_0 the larger will be S_∞. Suppose we have reached S_∞, i.e., the epidemic has ended, but now some new infectives are introduced into the population. Will a new cycle of infections take off?

The answer depends on what happens to the reproduction number. Suppose that because of a stringent social distancing policy, R_0 has been kept at a low value (say 1.5) throughout the course of the epidemic. If R_0 continues to be kept at this low value, and there is no significant change in the size of the population, there can be no second wave of infections. This is because the system of equations (1) to (4) has a unique steady-state equilibrium; the solution to eqn. (7) depends only on $\gamma/\beta = 1/R_0$. Given R_0, whatever the value of S_∞, it will be too small to sustain an increase in the number of infectives.

But suppose instead that the social distancing policy is relaxed, so that R_0 increases. Will a second wave of infections take place? The answer depends on the size of the increase in R_0. If the increase in R_0 is small, no new infections will occur.[7] But if the increase in R_0 is sufficiently large, a second wave will occur.

How large must the increase in R_0 be to generate a second wave? From eqns. (5) and (7), we know that $S_\infty < \gamma/\beta = 1/R_0$., and from eqn. (2), for $dI/dt > 0$ we need $S_t > \gamma/\beta$. But now we are at a new starting point, $S_0' = S_\infty$, so for a new wave of infections to start, we need $S_\infty > \gamma/\beta$. In other words, the contact rate β (and thus the reproductive number R_0) must increase sufficiently so that $\gamma/\beta' < S_\infty$.

The start, end, and possible restart of the epidemic are illustrated in the phase diagram of Figure 1. The epidemic starts with a very small number of infectives, and thus a number of susceptibles S_0 (as a fraction of the population) just under 1. The reproduction rate $R_0 = \beta/\gamma$ is assumed to be only 1.5, so the number of infectives reaches its maximum value of 0.21 at $S = \gamma/\beta = 1/R_0 = 0.67$. (Note that I_t is increasing as long as $S_t > 1/R_0$ and is decreasing when $S_t < 1/R_0$.) In this example, the epidemic stops when S_t falls to $S_\infty = 0.5$.

Now suppose the social distancing policy is relaxed somewhat, so that R_0 increases to 1.8. Can a second wave begin? The answer is no, because although R_0 is now larger, the number of susceptibles is too small to sustain a growing number of new infections. (Note that had R_0 been 1.8 instead of 1.5 at the beginning, the maximum number of infections in the first wave would have been higher and S_∞ would be lower.) For a second wave to begin, we would need $S_\infty > 1/R_0 = .55$, but as Figure 1 shows, S_∞ is only .50. But suppose instead that the social distancing policy is completely relaxed, so that R_0 increases to 3.4, and $1/R_0 = .29 < .50$. Now a second wave will occur, starting at $S_0' = 0.50$, reaching a peak fraction of infectives of about .05, and (as shown in the figure) ending as S_t falls to $S_\infty' = 0.2$.

In the example illustrated in Figure 1, the second wave is much less intense than the first wave (the maximum number of infections is lower and the number of deaths will be lower), because the pool of susceptibles is only half of what it was at the beginning of the first wave. In general, the intensity of the second wave will depend on how many susceptibles remain after the first wave, and on how much larger is the reproduction rate R_0. From eqn. (7), the

[7]This might not be the case in a more complex (and realistic) model. The SIRD model assumes a closed and homogeneous population with random mixing, which is not the case for the U.S. or most other countries.

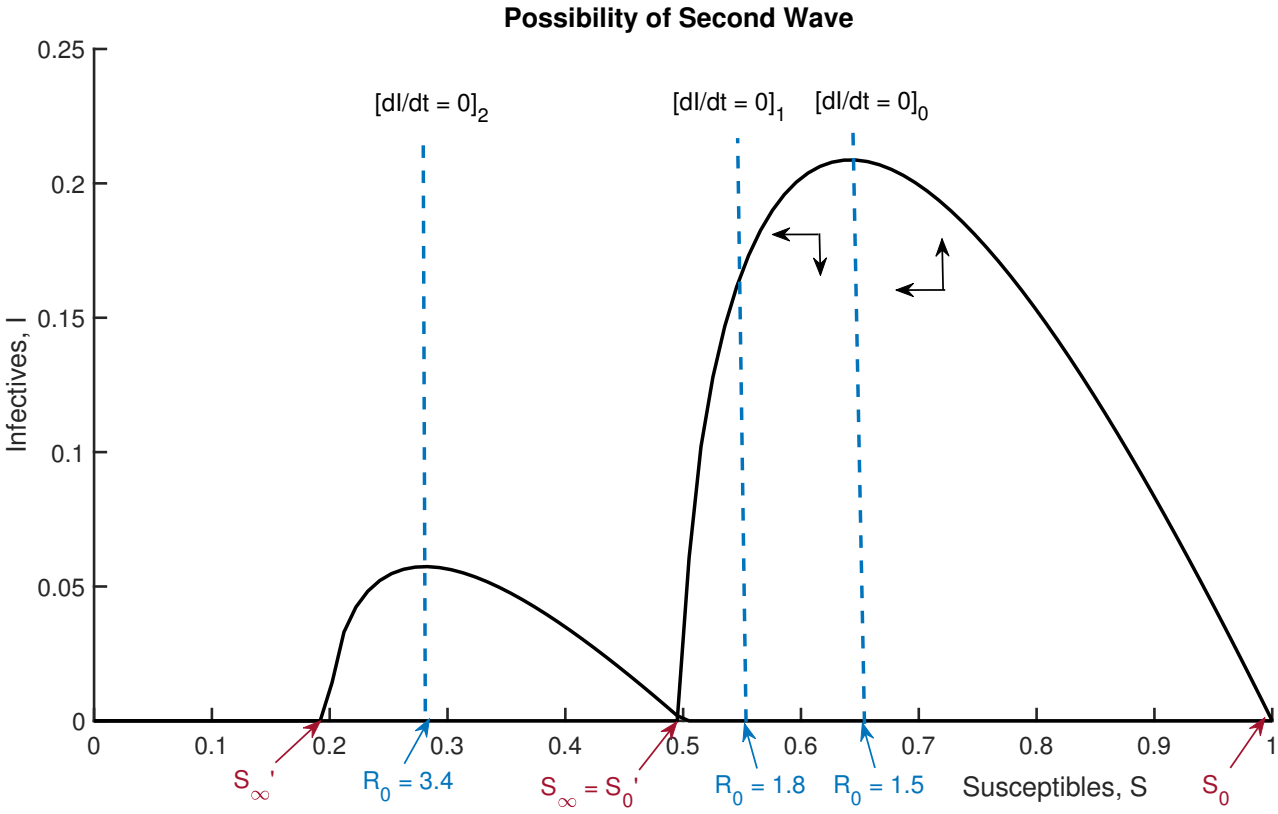

Figure 1: Possibility of a Second Wave. First wave starts at S_0 close to 1. With $R_0 = 1.5$ infections peak when S_t reaches $1/R_0 = .67$, and wave ends when S_t falls to $S_\infty = .50$. An increase in R_0 to 1.8 is insufficient to start a second wave, because the number of susceptibles is too small. A second wave requires $R_0 > 1/S_\infty = 2$. In the figure, R_0 is increased to 3.4, so a second wave begins and ends when S_t falls to $S'_\infty = .20$.

number of susceptibles at the end of the second wave, S'_∞, is the solution of

$$(\gamma/\beta')\log(S'_\infty/S_\infty) - S'_\infty + 1 = 0\,. \tag{10}$$

The total number of deaths from both the first and second waves is $D'_\infty = (\gamma_d/\gamma)(1 - S'_\infty)$, so the additional number of deaths is

$$\Delta D = D'_\infty - D_\infty = (\gamma_d/\gamma)(S_\infty - S'_\infty)\,. \tag{11}$$

The larger is the new contact rate β', the smaller will be S'_∞ and the larger will be ΔD. So given a sufficiently large increase in R_0, a second wave can occur, and it can be severe.

Herd Immunity.

Herd immunity is often described in terms of a critical fraction of susceptibles S^* that is low enough to prevent an epidemic from taking off. But in fact herd immunity depends on the product of two numbers — the fraction of susceptibles S and the reproduction number R_0. Growth in the fraction of infectives requires $SR_0 < 1$. Thus herd immunity is meaningful only in the context of the reproduction number likely to prevail when there is no policy in place to reduce contagion.

Let R_0^m denote the maximum value of R_0 we can expect if the social distancing policy is completely removed. Estimates of R_0^m vary widely (see Atkeson (2020*b*)), and depend on local living conditions and social mores. Given an estimate, the critical fraction of susceptibles is $S^* = 1/R_0^m$. In Figure 1, the second wave ends with $S'_\infty = 0.2$, and I assumed that $R_0^m = 3.4$. So in that hypothetical case, $S'_\infty R_0^m = 0.68$, and there is herd immunity.

A Vaccine Is Developed.

Suppose a vaccine is developed that provides perfect long-term immunity to the virus. How does the evolution of the epidemic depend on the fraction of the population that is vaccinated? How does it depend on the number of susceptibles at the time the vaccine arrives, and on the reproduction number R_0?

A vaccine is subject to the same externality that exists for social distancing: I benefit if you are vaccinated, just as I benefit if you stay home and practice social distancing. This complicates optimal pricing, whether vaccinations should be required, and the estimation of vaccine effectiveness, and there is a large literature that deals with these issues. There is likewise a literature (smaller and more recent) on optimal policies for vaccine development. I will abstract away from these issues and simply assume that once a vaccine is available, a random fraction of the susceptible population is vaccinated at no cost. I consider two cases: (1) The vaccine is available at the beginning of the epidemic; and (2) the vaccine becomes available after the first wave ends, but before any second wave begins.

Vaccine Available at the Beginning. Suppose that before the epidemic starts to take off, so that everyone is susceptible, a fraction v_0 of the population is vaccinated. How does v_0 affect the number of deaths and maximum number of infections, and how large must v_0 be to prevent the epidemic from taking off at all? The answers are best understood in the

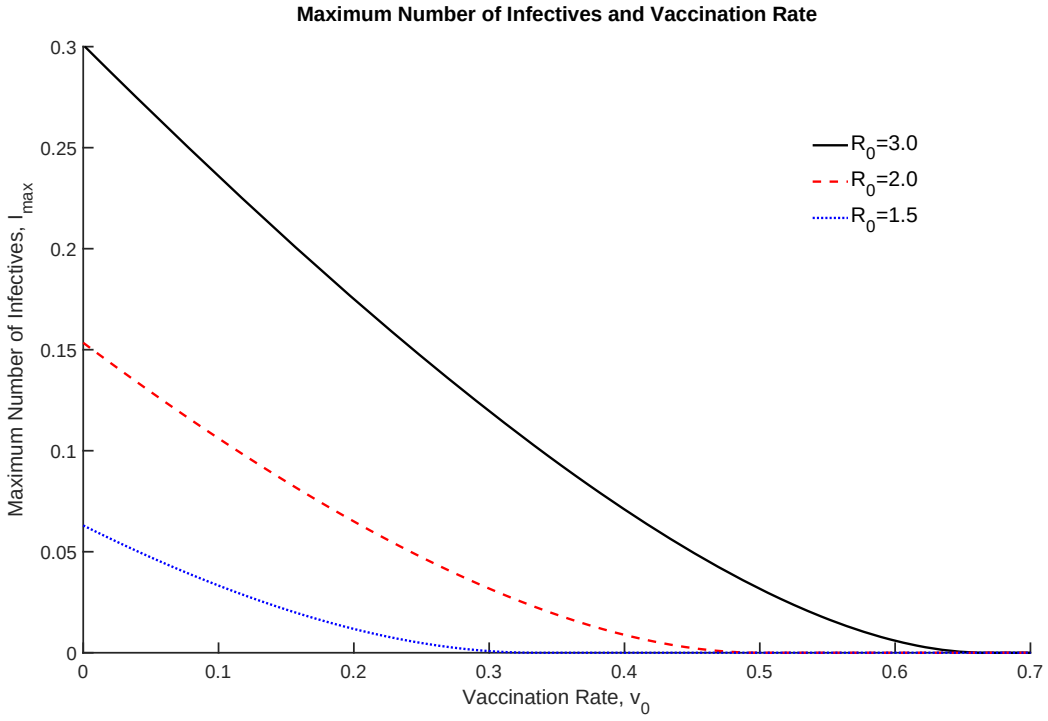

Figure 2: Maximum Infection Rate with Vaccine. Without a vaccine, the starting number of susceptibles is $S_0 = 1$. If the vaccination rate v_0 exceeds $\rho = 1/R_0$, the remaining number of susceptibles, $(1 - v_0)$, is too small to sustain an epidemic.

context of the "second wave" analysis presented above. The number of initial susceptibles is reduced from S_0 to $(1 - v_0)S_0 \approx (1 - v_0)$. If, for example, $v_0 = .50$, Figure 1 would apply, but we would be starting at $S_0 = (1 - v_0) = 0.50$, and the epidemic could only take off if the reproductive number R_0 is above 2.0. If $R_0 < 2$, the number of susceptibles would be too small to sustain a growing number of new infections.

If $R_0 > 1/(1 - v_0)$, the epidemic can take off, and the larger is R_0, the larger will be the maximum number of infectives and the number of deaths. From eqn. (5), I_t again reaches a maximum when $S_t = \rho = 1/R_0$, but (6) now becomes:

$$I_{\max} = (1 - v_0) - \rho + \rho \log(\rho/(1 - v_0)) \ , \quad v_0 < 1 - \rho \tag{12}$$

This dependence of I_{max} on v_0 is illustrated in Figure 2.

From (10), the final number of susceptibles, S_∞, is the solution to

$$(\gamma/\beta) \log(S_\infty/(1 - v_0)) - S_\infty + 1 = 0 \, ,$$

so $\partial S_\infty/\partial v_0 > 0$. The total number of deaths is, as before, $D_\infty = (\gamma_d/\gamma)(1 - S_\infty)$.

Vaccine Available After First Wave. Now suppose a first wave of infections has ended when the vaccine becomes available, and only those who are still susceptible are vaccinated. Because the number of susceptibles is smaller than at the outset, the vaccine can more readily substitute for social distancing.

Suppose that social distancing kept R_0 at only 1.5 during the first wave, as in Figure 1. We saw that with no vaccine, we could avoid a second wave by keeping R_0 below $1/S_\infty$ (in Figure 1, $1/S_\infty = 2.0$). But R_0 could rise well above this level if we vaccinate a sufficient fraction of the remaining susceptibles. Denoting the vaccination rate by v_0, a second wave now requires $R_0 > 1/(1 - v_0)S_\infty$. In Figure 1, a vaccination rate of .33 would allow R_0 to increase to 3.0 without a second wave occurring.

2.2 Rough Calibration to COVID-19.

Calibrating the SIRD model involves only three parameters: β, γ_r and γ_d. Unfortunately, in the case of COVID-19 we lack the necessary data to estimate these parameters in any precise way. For example, we don't know the true number of infectives (in the U.S. or anywhere else), because testing has been very limited, and many people infected show mild or no symptoms. Likewise, we don't know the true number of deaths from the virus; with limited testing and almost no autopsies, the cause of death for many COVID-19 victims is recorded as something else. Some implications of this lack of data have been explored by others, e.g., Atkeson (2020*a*), Manski and Molinari (2020), Stock (2020), and Avery et al. (2020). Here I simply stress that any calibration of this (or any other epidemiological) model to COVID-19 must be be viewed as extremely rough, and any projections from a calibrated model should be taken with a grain of salt.

With that caveat, I will select values for β, γ_r and γ_d based on the limited information we have for the U.S., and on calibration exercises done recently by Atkeson (2020*b*), Eichenbaum, Rebelo and Trabandt (2020), and Stock (2020). I will then use the calibrated model to further illustrate some of the analytical results described above.

Taking the population to be $N_0 = 1$, I assume that the initial number of infectives is $I_0 = 6 \times 10^{-6}$, and given a U.S. population of about 330 million, this would correspond to about 2,000 people infected at the outset.[8] This may seem high, but many thousands of

[8]This illustrates an important aspect of the simple SIRD model that is unrealistic: Infections in fact took

people entered the U.S. from China and other infected areas during January and February of 2020, and some of them were likely to be infected but asymptomatic. The initial number of susceptibles is $S_0 = 1 - I_0$.

I take the time interval Δt to be one day. I set the total removal rate γ at .07, which is an average of the estimates used by Atkeson (2020*b*) and Stock (2020).[9] Assuming the average illness duration is about the same whether the patient recovers or dies, γ_d depends only on the fraction of patients that die. But determining that fraction is difficult. Apart from age dependence (γ_d is much higher for older people), there is a strong dependence on the quality and availability of critical medical care. Thus we see enormous variation across countries (and across states in the U.S.) in the ratio of deaths to confirmed cases.[10] This variation does not mean that countries or states with high ratios have poor medical care; instead they had high congestion, i.e., hospitals were overwhelmed by a sudden surge of cases.

Whatever the ratio of deaths to confirmed cases, it is probably overestimates the true death rate by a factor of two or more, because the denominator is an underestimate of the actual number of cases. In the U.S., for example, the death rate is probably well below 4 or 5 percent. But how much below is unclear. So what number should we use for γ_d?

Eichenbaum, Rebelo and Trabandt (2020) cites a March 16, 2020 WHO estimate of about 1%, based on data from South Korea. The ratio of deaths to confirmed cases was about .02 for South Korea, and compared to other countries, there was little or no congestion in the health care system, so 1% seems reasonable.[11] But as discussed in Jones, Philippon and Venkateswaran (2020), if there is congestion, that should be taken into account as part of the death rate. (They estimate the death rate to be 1% with no congestion, but significantly higher with congestion.) In many areas of the U.S. there is indeed congestion, so I will assume a death rate of 2%. Thus $\gamma_d = (.02)(.07) = .0014$ (and $\gamma_r = .0686$).

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off at specific points in the U.S., not from a pool of people spread out evenly across the country.

[9] Assuming the half-life of an infection is 6 days, Stock (2020) sets γ to 0.55 on a weekly basis, which corresponds to 0.08 for $\Delta t = 1$ day. Atkeson (2020*b*) sets γ (daily) at about .06, based on an average illness duration of 18 days.

[10] On April 15, 2020, that ratio was below .03 in Israel, South Korea, Austria and Germany, .045 in the U.S., and above .13 in Italy, France and the U.K.

[11] Alvarez, Argente and Lippi (2020) sets $\gamma_d = .01\gamma$, and argues that this is consistent with the 1% age-adjusted fatality rate from the Diamond Princess cruise ship. But Hortaçsu, Liu and Schwieg (2020), using regional epicenter data, obtains much lower estimates of the fatality rate.

Given γ and its components, we are left with the contact rate, β, or equivalently, the reproduction number $R_0 = \beta/\gamma$, which is a function of the social distancing policy that is in place. What is a reasonable value for the reproduction number R_0 at the outset, i.e., before any social distancing policy has been applied? Atkeson (2020*b*) surveys estimates from about 8 studies, based on data from China, Italy, the U.S., and the Princess Cruise ship; those estimates suggest a range between 2.2 and 3.3. Of course R_0 will depend on social mores and living conditions, and is likely to be higher for Italy, New York City, or a cruise ship than for rural areas of the U.S. I take the base value of R_0, with no social distancing policy, to be 3.0, and then explore what happens when R_0 is reduced.

2.3 Contagion and Disease Dynamics in the Calibrated Model.

Figure 3 shows solutions of eqns. (1) to (4), with $\gamma_d = .0014$ and $\gamma_r = .0686$, and $R_0 = 3.0, 2.5, 2.0$ and 1.5 (corresponding to $\beta = \gamma R_0 = .210$, .175, .140 and .105), and with starting value $I_0 = 6 \times 10^{-6}$. The figure suggests that new infections (and deaths) begin and end at specific points in time, but in fact new infections begin on day 1 and drop to zero only asymptotically.[12] So to measure the *duration* of the epidemic, I will (arbitrarily) take its onset (end) to be the date at which I_t first reaches (falls back to) 1% of its maximum value. So for $R_0 = 3.0$, the epidemic runs from day 49 to day 187, for a duration of 138 days. For $R_0 = 2.5$, 2.0, and 1.5, the durations are 166, 189, and 374 days respectively.

Figure 3 illustrates some fundamental characteristics of the model and their implications for social distancing policies. First, as R_0 and thus β are lowered, the epidemic takes off (i.e., the fraction of infectives becomes significant) later and then evolves more slowly and lasts longer. Using my definitions of the onset and end dates, new infections begin, peak, and end later. For $R_0 = 3.0$, 2.5, 2.0, and 1.5, the onset is on Day 49, Day 61, Day 118, and Day 136 respectively, and as shown above, the durations run from 138 to 374 days. Furthermore, the "duration" that matters for policy is the period of time the policy must be in place, i.e., from Day 1 until the end date. For $R_0 = 3.0$, 2.5, 2.0, and 1.5, these durations are 187, 227, 307 and 510 days respectively. This creates a policy problem: Even if the per-day economic cost of a social distancing policy is the same no matter how strict it is, the total cost will be

[12]Suppose $R_0 = 3$ so $\beta = .21$ (the black solid line in each panel). Then for the U.S. (population 330 million), on Day 1 there are 277 new infections, and over the first week there are about 2,400 new infections and 6 deaths. On day 250 there are about 23,000 people infected, and on day 400 about 3 people infected.

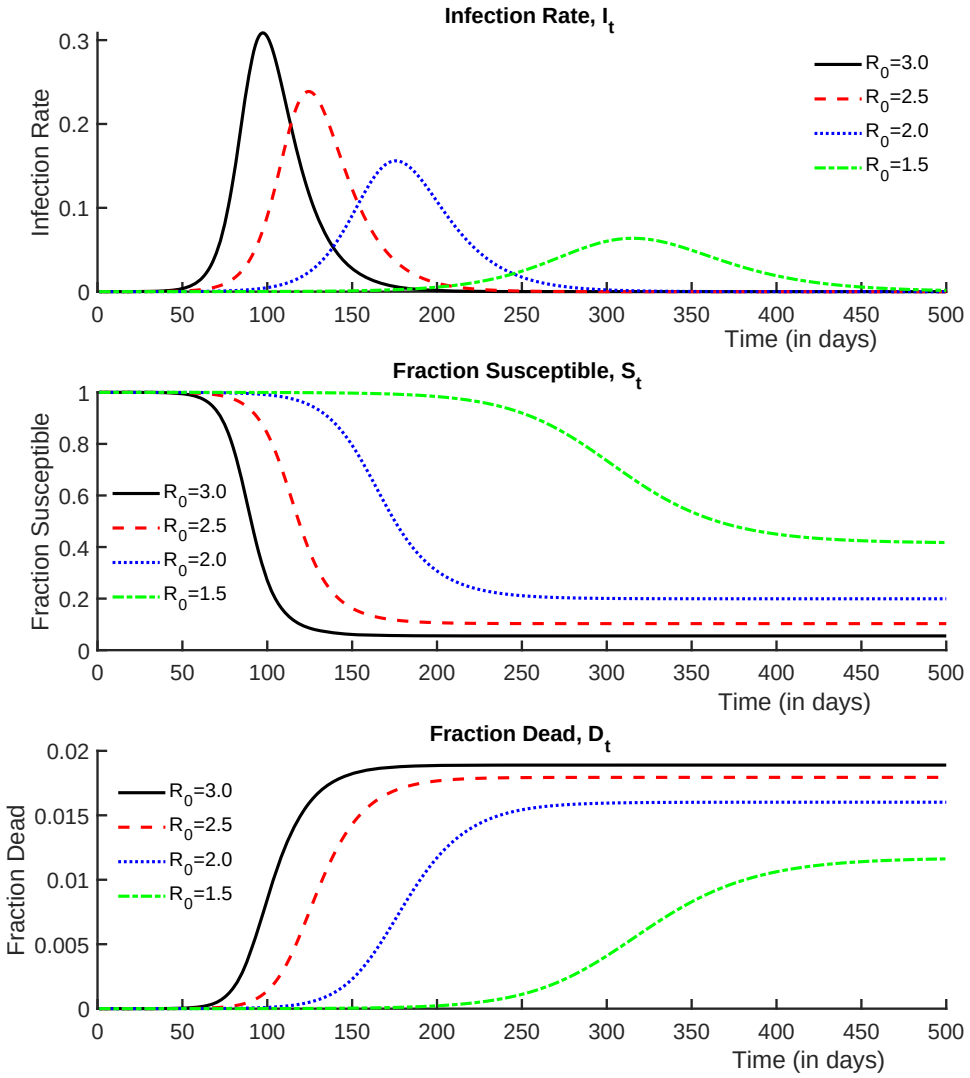

Figure 3: Solution of SIRD Model. The top panel shows the fraction of the population infected over time (in days) for $R_0 = 3.0$, 2.5, 2.0 and 1.5, and with starting value $I_0 = 6 \times 10^{-6}$. The middle and bottom panels show the fraction that is susceptibles and the fraction that have died. The parameter values are $\gamma_d = .0014$ (corresponding to a 2% fatality rate) and $\gamma_r = .0686$, so the total removal rate is $\gamma = .07$, and $\beta = .07R_0$.

greater for a stricter policy because it must be maintained for many more days.

Second, deaths (and recoveries) occur in proportion to the number of infectives on each day. So the total number of deaths is δ_d times the area under the infection rate curve in the top panel of Figure 3 (black curve for $R_0 = 3.0$). As R_0 is reduced, the infection rate curves "spread out," but the areas under them fall, i.e., there are fewer deaths in total.

The area under the infection rate curve is also the fraction of the population no longer susceptible, i.e., the fraction that have been "removed" from the population. Of those "removed," a fraction γ_r/γ will have recovered and a fraction γ_d/γ will have died, which is what eqn. (8) is telling us. As the middle panel of Figure 3 shows, as R_0 is reduced and the total number of infectives falls, the total number removed falls, and the number of susceptibles rises. So if $R_0 = 3.0$, the final fraction of susceptibles is only $S_\infty = .055$, but if $R_0 = 1.5$, that fraction is 0.42.

This creates another policy problem: If a stringent social distancing policy reduces R_0 from, say, 3.0 to 1.5, at the end there will be fewer deaths but a larger pool of people still susceptible. If there is no vaccine, then once the policy is removed (and R_0 returns to 3.0), there will be a greater chance of a second wave of infections (along the lines of Figure 1). Thus if the fatality rate is low, a less strict social distancing policy might be preferable because it reduces the chance of second wave after life returns to normal.

Contagion and the Number of Deaths.

Figure 3 shows the fraction of deaths over time for four values of R_0. But social policies are expensive, so one might ask if there are increasing or decreasing returns to incremental reductions in R_0. Figure 4 shows the total number of deaths at the end (i.e., after 500 days) as a function of R_0. Using eqn. (9), it also shows the incremental number of deaths corresponding to $\Delta R_0 = 0.1$. Both numbers have been scaled to the U.S. population.

As we move from right to left and R_0 falls, the total number of deaths declines, slowly at first and then more rapidly, as the incremental number of avoided deaths from small reductions in R_0 rises increasingly fast. This follows from eqns. (7) and (8); as R_0 is reduced towards 1, S_∞ approaches 1 and D_∞ approaches zero. So if we think of an incremental reduction in R_0 as a unit of policy "output," we see increasing returns. As R_0 is reduced in increments of 0.1, the incremental reduction in deaths becomes larger and larger. Of course the incremental cost of reducing R_0 will probably also become larger, as discussed below.

Fundmental Policy Trade-offs.

I do not attempt a cost-benefit analysis that would lead to a policy recommendation; the SIRD model is too simple, even more complex models have parameters that we can't identify, and we have little data from which to estimate economic impacts. Nonetheless,

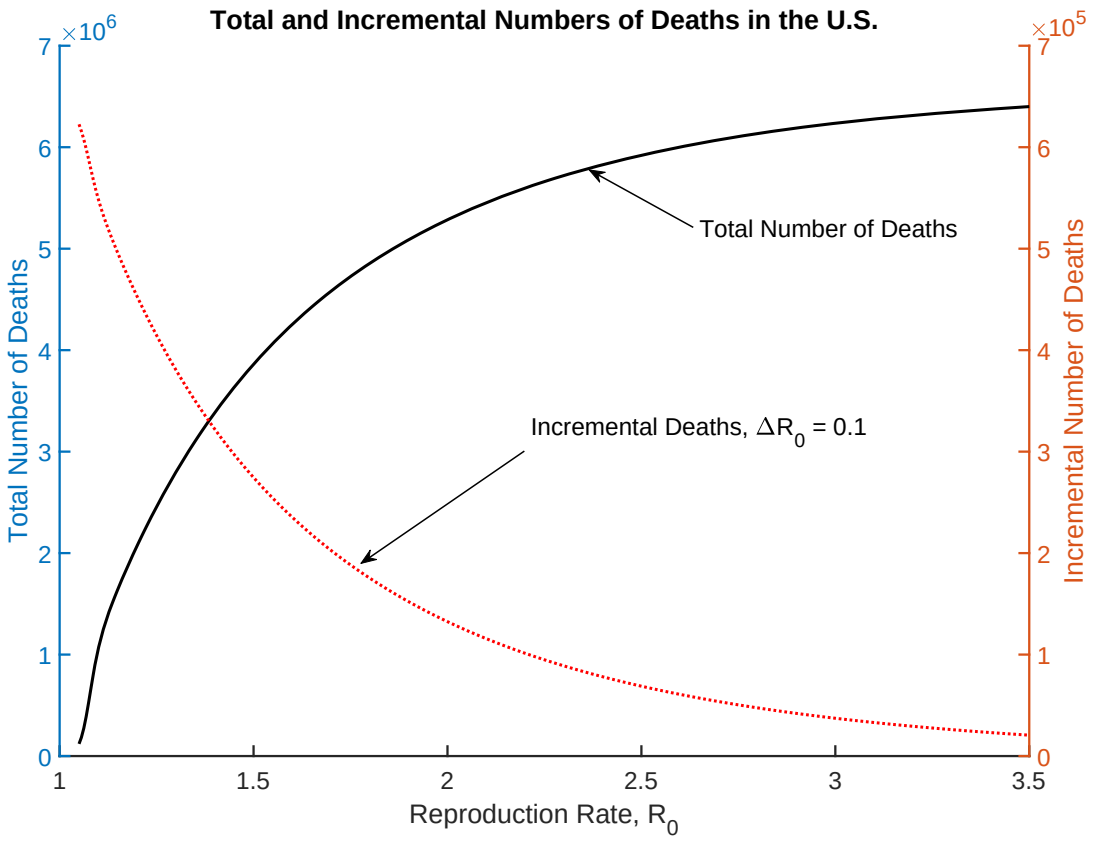

Figure 4: Total and Incremental Numbers of Deaths in the U.S. Deaths are plotted as a function of the reproduction rate R_0, based on a U.S. population of 330 million. The incremental number of lives saved rises as R_0 is reduced.

the calibrated SIRD model can help elucidate some key policy trade-offs, and clarify the parameter values and data that are fundamental to policy design.

Suppose that with no policy intervention, $R_0 = 3.5$. What are the costs and benefits of reducing R_0 to some number below 3.5? Start with the cost, which for simplicity I will take to be lost GDP.[13] It can be broken into two parts: (1) the cost per day of social distancing, which depends on the size of the reduction in R_0 but also on the number of days the policy is in effect; and (2) the number of days itself, which in turn depends on the reduction in R_0.

Denoting the per-day cost by C and the number of days by N, and ignoring for now a

[13]There are other costs that don't appear directly in GDP, such as unemployment, bankruptcies, losses of homes and businesses, lost education, and increases in inequality. And I ignore the psychological costs of social distancing. Mulligan (2020) estimated the total annual economic cost to be about $7 trillion.

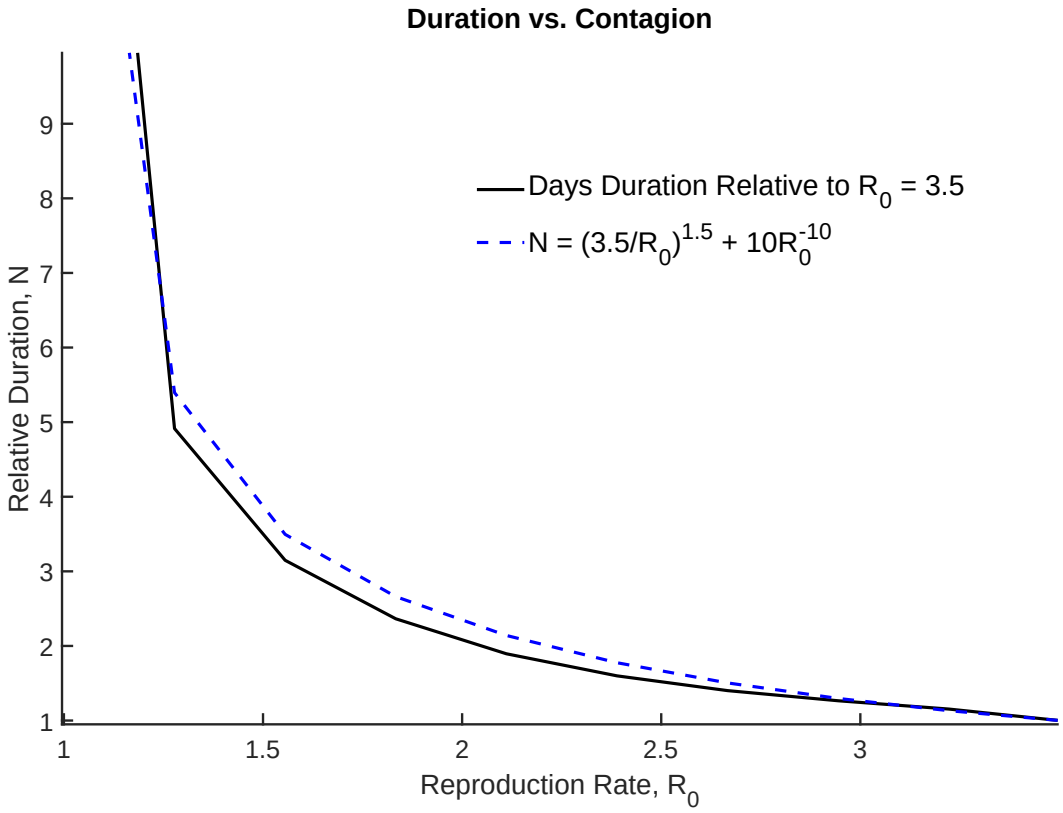

Figure 5: Duration, Relative to $R_0 = 3.5$. Solid line is number of days from start (Day 1) to end of epidemic, relative to 162 days for $R_0 = 3.5$. (Duration $\to \infty$ as $R_0 \to 1$.) Dashed line is a function fitted to the duration curve.

possible second wave, the total cost can be written as:

$$\text{TC} = N(R_0) \times C(R_0, N(R_0))\,, \tag{13}$$

Start with the duration, $N(R_0)$. Clearly $N'(R_0) < 0$, but what about $N''(R_0)$? Figure 5 shows the number of days from the start (Day 1) to the end of epidemic, relative to the 162-day duration for $R_0 = 3.5$. (The relative duration is more informative because the absolute duration depends on I_0, which as a rough guess we set at 6×10^{-6}. Note that duration becomes infinite as $R_0 \to 1$.) Also shown (as a dashed line) is a function fitted to the relative duration curve: $D = (3.5/R_0)^{1.5} + 10R_0^{-10}$. The figure shows that $N''(R_0) > 0$, and it lets us determine how N is affected by incremental reductions in R_0.

As for the per-day cost, by assumption $C(3.5, N) = 0$, and we expect $\partial C(R_0, N)/\partial R_0 < 0$ because larger reductions in R_0 require stricter social distancing rules, which presumably impose a greater cost on the economy. But it is likely that $\partial^2 C(R_0, N)/\partial R_0^2 > 0$; even

weak social distancing (e.g., reducing R_0 to 2.5) requires many businesses to shut down or reduce operations, whereas the additional economic losses from "strict" to "very strict" regulations (e.g., reducing R_0 from 2.0 to 1.5) are likely to be smaller. Finally, we expect $\partial C(R_0, N)/\partial N > 0$; it will be more than twice as costly to keep $R_0 = 2.0$ for 200 days than it will for 100 days, because the longer duration will cause permanent damage due to bankruptcies, layoffs, etc.

The benefit of reducing R_0 is mostly the value of lives saved, but also reduced medical costs. How many lives would be saved? In Figure 4, the total number of U.S. deaths with no social distancing policy ($R_0 = 3.0$ to 3.5) in on the order of 6 million. Moderate social distancing, e.g., reducing R_0 to 2.0 to 2.5, would save about 1 million lives, but strict social distancing, e.g., reducing R_0 to 1.2 to 1.5 would save 3 to 5 million lives. (This is with a fatality rate of 2%, which may be too high, but even 1% implies around 3 million deaths with no social distancing.) Denote the number of deaths by $D(R_0)$, with (as Figure 4 shows) $D'(R_0) > 0$ and $D''(R_0) < 0$, and denote the social value of a life lost by V.

The basic cost-benefit calculation comes down to reducing R_0 up to the point that equates the marginal benefit $VD'(R_0)$ to the marginal cost dTC/dR_0. Using eqn. (13):

$$VD'(R_0) = N(R_0)\left[\frac{\partial C}{\partial R_0} + \frac{\partial C}{\partial N}N'(R_0)\right] + N'(R_0)C(R_0, N) \tag{14}$$

Both $N(R_0)$ and $D(R_0)$ would come from an epidemiological model; for the simple SIRD model, they are shown in Figures 4 and 5. Given the lack of data, specifying $C(R_0, N)$ requires assumptions about employment and output impacts, and will be subject to considerable uncertainty. That leaves the social value of a life, V, which I turn to that next.[14]

Value of Lives Saved.

All of the studies that I have cited use a VSL estimate for V, typically around $11 million per life saved, which is roughly the number used by the EPA, DOT, and other regulatory agencies in the U.S.[15] Then 3 million lives saved would be valued at $33 trillion. (The U.S.

[14]I am ignoring costs of hospitalizations and other medical treatment, which most studies show are small relative to the value of lives lost. If medical costs are proportional to deaths, we could account for them by scaling up $VD(R_0)$. Also, note that discounting is irrelevant because the time horizon is less than two years.

[15]See, e.g., Greenstone and Nigam (2020) and Thunström et al. (2020). For an overview of the VSL and some issues with its use, see Viscusi (1993, 2018), Ashenfelter (2006) and Hammitt and Treich (2007). The DOT (EPA) used a $9.6 ($9.9) million VSL in 2016 (2011), which is about $10.4 ($11.5) million in 2020.

GDP in 2019 was about $21 trillion.)

But should we use the VSL? The VSL is the *marginal* rate of substitution between wealth (or future lifetime consumption) and the probability of survival, i.e., minus the slope of the indifference curve between wealth w and survival probability p, measured at a particular point (w, p). It is a local measure that tells us how much wealth or consumption an individual would sacrifice in return for a small increase in the probability of survival. It does *not* tell us how much an individual would sacrifice to avoid a significant probability of death, which might be very different from the VSL. Consistent with its definition, estimates of the VSL often come from data on risk-of-death choices made by individuals, such as the decision to take a riskier but higher-paying job rather than a safer one. And consistent with its definition, the VSL can be applied to cost-benefit analyses of government regulations. An example is the requirement that cars have air bags, which reduces drivers' fatality risk by a small amount, at the cost of a small sacrifice of lifetime consumption.

The VSL has a number of well-recognized problems, but the biggest one is that it reflects individual preferences, not the preferences of society. So it is increasing in a person's wealth level (because a wealthier person has more utility to lose should she die), which need not correspond to social preferences. And because it is a marginal rate of substitution, it does not aggregate consistently; applying an $11 million VSL to the U.S. population yields $3,600 trillion, about 170 times the U.S. GDP, and about 230 times annual U.S. consumption.

But shouldn't the preferences of society reflect individual preferences? Not necessarily, as illustrated by the aggregation problem. The small amount of income I would sacrifice for a safer job might imply a VSL of $11 million, but it could have little to do with the amount I would sacrifice to avoid a substantial risk of death, or the amount society is willing to sacrifice to prevent the deaths of a substantial fraction of the population.

How does this apply to pandemics? Martin and Pindyck (2019) use the VSL to calculate the social willingness to pay (WTP) to avert a low-probability risk to life — the *possibility* of a major pandemic, that if not averted would have an annual probability of around .02 of occurring, and should it occur might kill 2 to 5 percent of the population. The benefit to each member of society from averting the threat is a reduction in their fatality risk of about $(.02)(.05) = .001$, which is indeed a marginal change. But the COVID-19 pandemic is a sure thing, not a potential threat, so the fatality risk is much larger. And the cost of reducing

the risk is much larger, as we see from the economic impact of social distancing policies.

Eliminating or reducing the risk of death from COVID-19 significantly increases the survival probability p. The convexity of the indifference curves means that as p is increased, $-dw/dp$ decreases. Thus the \$11 million VSL figure will overstate the benefit from lives saved, even if we base that benefit on individual preferences toward mortality risk. But to say how much it overstates the benefit we would need to map out the indifference curves, which we can't do. We can, however, consider an extreme case. Suppose instead of asking people how much of their wealth they would give up to avoid a very small probability of death, we ask them how much they would give up to avoid certain death. Presumably they would give up their entire wealth. In 2018, the total net wealth of U.S. households was \$98 trillion, i.e., \$297,000 per person, quite a bit less than \$11 million.[16] (This ignores the extremely unequal distribution of wealth in the US, but the VSL ignores that as well.)

Fortunately, COVID-19 does not imply certain death. There is still a great deal of uncertainty over the actual fatality rate, and it varies enormously across regions. If the fatality rate turns out to be very small, the VSL might be appropriate, but if it is on the order of 2%, the number for V should be much less than \$11 million. We could also look at what societies actually spend to save large numbers of lives. For example, the U.K. National Health Service (NHS) limits what they will pay for a given treatment by using a "Quality-Adjusted Value of a Statistical Life Year" of af about \$38,000, which translates to a VSL of around \$1 million.

A Second Wave.

If we have a number for V along with estimates of $N(R_0)$, $D(R_0)$ and $C(R_0, N)$, we could use eqn. (14) to calculate the optimal value for R_0, and from the SIRD model, determine the final fraction of susceptibles, S_∞. But then we have to ask what happens if at the end we remove the social distancing restrictions so that R_0 returns to its unregulated value. Will we have a second wave of infections, and if so, how many additional deaths?

Suppose R_0 is maintained at an "optimal" value of 1.5 (as in the green line in Figure 3). Then after a duration of 510 days the remaining fraction of susceptibles would be 0.42, so we

[16]Federal Reserve, Financial Assets of the U.S., Table Z.1. Total assets were \$113 trillion and liabilities were \$15 trillion.

could increase R_0 to $1/(0.42) = 2.4$ without creating a second wave. But what if the social distancing policy is lifted entirely and R_0 rises to 3.0? Using eqns. (10) and (11), we can calculate that the fraction of susceptibles will fall further to $S'_\infty = .022$, and the additional fraction of the population that dies is .008, i.e., about 2.6 million additional deaths.

However, this scenario — R_0 kept at 1.5 and then allowed to rise to 3.0 — is probably not optimal. More generally, picking a single number for R_0, or two successive numbers, will always be dominated by a fully dynamic policy in which R_0 is varied over time. That is the rationale for the dynamic optimization problems studied by Jones, Philippon and Venkateswaran (2020) and Alvarez, Argente and Lippi (2020). If we take the epidemiological model at face value, and assume that continuous variation of R_0 if feasible, we could do better than the more limited options examined above. But how much better is an open question.

3 Conclusions.

I have not tried to determine an optimal policy for the control of COVID-19 contagion, or evaluate alternative policies. Others have tried to do this using off-the-shelf epidemiological models, but are limited by our current inability to identify the parameters of these models and estimate the relevant policy costs and benefits. Instead I have used a simple SIRD model to elucidate how pandemic progression is affected by the control of contagion and the key trade-offs that underlie policy design.

Isn't the SIRD model too simple and unrealistic? Yes and no. Yes, because it treats the epidemic as occurring within one large mass of homogeneous individuals, whereas in fact a key element of COVID-19 progression is its outbreak in local epicenters followed by transmission and seeding of new epicenters. And yes, because it assumes that the contact and removal rates β and γ (and hence R_0) are the same for all groups of individuals. Given these limitations, the model is probably not well suited for forecasting and policy design.

But no, insofar as the objective is to get a basic understanding of how contagion affects pandemic progression and policy trade-offs. It illustrates, for example, how R_0 affects the infection rate, the total and incremental number of deaths, the duration of the pandemic, and the possibility and impact of a "second wave." That is the main reason for my use of the model, and why it has been used by other studies (e.g., those cited in the Introduction).

With these caveats, I have used the model to show (1) how the marginal cost of re-

ducing R_0 depends on the marginal duration $N'(R_0)$, and on how the marginal daily cost $C(R_0, N(R_0))$ varies with R_0 and the duration; and (2) how the marginal benefit depends on the marginal number of deaths $D'(R_0)$ and the social value of a life V. In practice, both $N(R_0)$ and $D(R_0)$ would come from an epidemiological model, and $C(R_0, N)$ would require assumptions about employment and output impacts. As for the social value of a life V, most studies use an estimate of the VSL. I have argued that this is problematic. The VSL is a marginal rate of substitution, but reducing the risk of death from COVID-19 implies a significant increase in the survival probability, so the convexity of the indifference curves means that the VSL will overstate V.

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