Optimal Management of an Epidemic: Lockdown, Vaccine and Value of Life*

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Abstract

We study a dynamic macro model to capture the trade-off between policies that simultaneously decrease output and the rate of infection transmission. We find that, in many cases, optimal policies require sharp initial decreases in employment followed by a partial liberalization that occurs before the peak of the epidemic. The arrival of a vaccine (even if only a small fraction of the population is initially vaccinated) requires a significant loosening of stay-at-home policies and, in some cases, results in an increase in the speed of infection. The model implies that the monetary value of producing a vaccine is high at the beginning of the epidemic but it decreases rapidly as time passes. We find that the value that society assigns to averting deaths is a major determinant of the optimal policy.

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1 Introduction

The objective of this research is to understand how features of the economy and the parameters describing an epidemic influence the choice of policy during an epidemic. We consider a government that can impose restrictions on employment along the lines of "stay-at-home" policies, and can allocate resources to attain a certain level of vaccination when a vaccine becomes available.

The macro literature on the impact of pandemics is large and growing. This paper is closest to the recent work of Alvarez, Argente, and Lippi (2020), Acemoglu et. al (2020) and Gonzalez-Eiras and Neipelt (2020). The major differences with Alvarez et. al. is that we take a different approach to analyzing the impact of the availability of a vaccine and this allows us to evaluate the consequences of different arrival times. We also present a more detailed analysis of how the value that society puts on averting deaths during a pandemic influences the optimal stay-at-home policy. We also discuss how the value of a vaccine changes as the epidemic progresses.[1]

The details of how a vaccine interacts with other policies is novel and interesting. We find that unless 100% of the population can be instantaneously vaccinated —a patently unrealistic case— arrival of a vaccine does not imply —depending on the state of the epidemic— that all restrictions on employment should be lifted. Moreover, availability of a vaccine may result in an *increase* in the spread of the epidemic. These counterintuitive results can be easily explained: availability of a vaccine increases the rate at which the susceptible population shrinks and this reduces the future contagion rate. This implies that the cost of the epidemic in terms of future deaths and consumption decreases (less future contagion) and, consequently, the marginal cost in terms of current output should decrease as well. This last step requires a liberalization (more contact among individuals) that, in turn, pushes up the contagion rate.

We posit that individual preferences depend on individual consumption —ignoring the private value of life— and that social preferences take into account the utility loss associated with deaths. We discuss different approaches

[1] Acemoglu et. al. study optimal lockdown for heterogeneous agents and find that the optimal policy call for different lockdown strategies for different individuals. Gonzalez-Eiras and Neipelt present a general model but they concentrate of special cases in order to find closed form solutions. Appendix 5 has a partial list of the rapidly growing literature on the economic effects of COVID-19.

to valuing life since this turns out to be important in our quantitative exercises. On the epidemiological dimension, we use a standard SIR model with (endogenously chosen) vaccination rates in some states and less than perfect immunity.

We assume that at the beginning of the epidemic —what we label Phase I— the only policy available to the planner is a stylized version of "stay-at-home" policies that, simultaneously, restrict employment and lower the rate of transmission of the virus. Phase I ends when a vaccine becomes available and the economy enters Phase II. We view the arrival of a vaccine as a random event and we take the probability distribution as exogenous. At this point, the planner has a second tool to control the epidemic: the speed at which the population can be vaccinated. For a developed country like the U.S. we find that it is in general optimal to vaccinate at the highest possible rate. For that reason, in the quantitative section we simply set the vaccination cost to zero (which implies that the optimal policy is to vaccinate at the highest feasible rate). In the case of less developed countries the cost of vaccination is not trivial and, in those cases, our theoretical model provides guidance.

On the theoretical side, we show that the model has a steady state and, more interesting, that along a path in which a vaccine never becomes available (Phase I) —although optimal policies take into account that the probability is positive— the economy converges to the *same steady state* as another economy that has access to a vaccine. This implies that the economic value of a vaccine (ignoring recurrences) decreases over time. To the extent that the private value of a vaccine moves with the social value, the model predicts that fewer resources will be allocated by the private sector to finding a vaccine as the epidemic progresses.

We calibrate the model using standard estimates of the epidemiological parameters and we find that optimal policies are very sensitive to the details of the model, about which there is significant uncertainty. Some of our more interesting findings include:

1. The optimal policy depends on both the number of infected **and** susceptible individuals. Implementation of the optimal policy requires random testing. [2]

2. In most of our simulations the optimal stay-at-home policy in Phase I

[2]The case for random testing has been made by many. Among the economists a good discussion is in Chari and Phelan (2020).

(no vaccination available) implies:

(a) A **sharp decrease** in employment that ranges, depending on the particular case, between 20 and 35 percent.

(b) A gradual liberalization (e.g. allowing more economic activity to take place) that occurs **always** before the epidemic reaches a peak.

(c) A fairly **wide range** in the estimated lockdown time (that is time until all restrictions are eliminated). It ranges from about 20 weeks in the most optimistic case to over 70 weeks in the pessimistic scenario. Moreover, the duration is highly dependent on the realization of the key random variable: The time when an effective vaccine becomes available.

(d) If a vaccine arrives early (economy enters Phase II), the optimal response is a significant reduction of the restrictions on employment even though a small proportion of the population is vaccinated in the first week. This is often accompanied by an **increase** in the rate at which the virus spreads.

3. Concavity of preferences implies that individuals prefer relatively constant consumption over time. A managed epidemic that flattens the infection curve lowers consumption at some points in time but it avoids the more pronounced peaks that would occur if no policy is put in place. However, this preference for smoothness has a small quantitative impact on the optimal solution. It is the additional value of society puts on averting deaths —another concave function— that drives the severity of the restrictions.

4. We experiment with many alternative ways of valuing human life. We find that varying the implicit value of a life but keeping everything else constant

   (a) The number of deaths averted as a fraction of the population can range from a low of 0.03% to a high of 0.87%.

   (b) The cost per death averted if a vaccine becomes available in about a year, is in the range of 2.5 to 7.85 million. Moreover the policies that put the highest value to human life are also the policies that result in the lowest cost per death averted.

(c) The actual (ex-post) cost per death averted depends on the time at which a vaccine becomes available: in the case that an effective vaccine can be administered after 25 weeks, the cost in many cases decreases by two thirds.

5. The social market value of a vaccine depends on the specific scenario. However, in most cases the estimated value of a vaccine that arrives after a year is significantly lower than a vaccine that is available after six months.

Even though we find our quantitative results useful and suggestive of the implications of following optimal policies we are fully aware that their quality is no better than the quality of the data that we use. At this point there is significant uncertainty about many of the key parameters, both those corresponding to the economic model as well as those implicit in the epidemiological model. In addition, the model assumes that the planner knows the fraction of susceptibles and infected, which is not the case at this moment in the absence of random testing[3]. We view this report as a first attempt to understand optimal policies and hope that better data will allow us to improve our results.

In section 2 we present the model and in section 3 we discuss some theoretical results. In section 4 we present our quantitative findings. Section 5 briefly discusses ongoing work on extensions and section 6 offers some preliminary concluding comments.

2 Model

We study a standard continuous time macro model. We assume that there is one good that is produced exclusively with labor. There are two policy variables that we study. First, a type of "stay-at-home" restriction on the utilization rate of the labor force which has two impacts: It decreases output and, simultaneously, reduces the rate of transmission of a virus since fewer individuals enter in contact with others. The second policy is the rate at which individuals can be vaccinated when a vaccine becomes available. This

[3]However, in the simulations we start from the case in which almost 100% of the population is susceptible. Thus, conditional on the epidemiological parameters, our estimates should not be to far off the actual values.

rate is also subject to an institutional constraint that captures both delays in producing a viable vaccine in large quantities (even after one has been discovered) and the logistical arrangements associated with mass vaccination.

We assume that there is a representative agent that cares about consumption. Social preferences are simply individual preferences adjusted (downward) by the disutility cost of deaths. Thus, from society's perspective there are two reasons to control an epidemic: the direct loss of output associated with lower labor force availability and, in our baseline, the additional disutility cost of deaths associated with the epidemic. The details of how we model this disutility are spelled out below.

We consider two phases that differ on the availability of a vaccine.

- *Phase I*: This is the period in which there is no vaccine available. The only available tool is "stay-at-home" type of policies that reduce employment. We use a single variable to capture a variety of interventions that affect both the rate of transmission of the virus and the level of employment. We leave for future work the analysis of policies that are likely to vary in their impact like social distancing, age-related limitations and complete lockdown, among others.

- *Phase II*: We assume that the availability of a vaccine arrives at an exogenous rate. Unlike Alvarez et. al. (2020) we do not assume that the population can be treated in a very short period of time. We model the speed of vaccination as a Poison process. We assume that the planner can control —at a cost— the rate at which the population is vaccinated,

2.1 The Economic Model

We assume that there is only one good that is produced linearly using labor. If the available labor force is denoted L and only a fraction $\phi \in [0,1]$ is utilized in production, then utility is simply $u(\phi wL - c_V(\mu(S+(1-\zeta)I)))$, where the second term captures the cost is terms of output of vaccinating a population of size $S + (1-\zeta)I$. This is the population that includes susceptible and infected individuals who are asymptomatic. Of course, this term is operative only in Phase II when a vaccine is available, together with a bound on the speed at which the population can be vaccinated.

Social preferences depend on the utility derived from consumption (we abstract away from leisure at this stage) and an additional term that captures

the disutility associated with the loss of life. The static social payoff is

$$u(\phi wL - c_V(\mu(S + (1-\zeta)I))) - \Delta(D).$$

We make standard assumptions about the utility function u. In the simple model L equals the (fixed) labor force minus those infected individuals who have been identified as such. In general we assume that that the function $\Delta(D)$ is increasing and convex.

Society's preferences are then a function of consumption and deaths. Let T_η be the (random) time at which the economy transitions to Phase II (that is, when vaccination becomes available). Formally, preferences are given by

$$U = E\{\int_0^{T_\eta} e^{-\rho t}\left[u(\phi_t wL_t) - \Delta(D_t)\right] dt \tag{1}$$

$$+e^{-\rho T_\eta}\int_0^{\infty} e^{-\rho t}\left[u(\phi_{T_\eta+t} wL_{T_\eta+t} - c_V(\mu_{T_\eta+t}\left(S_{T_\eta+t} + (1-\zeta)\, I_{T_\eta+t}\right)) - \Delta(D_{T_\eta+t})\right] dt\}$$

where the expectation is taken over the realization of T_η.

2.1.1 Special Case

The special case assumes that the instantaneous payoff is

$$N\ln\left(w\phi L - \underline{c}\right) - N\Delta(D).$$

In this formulation $\underline{c}$ is the minimal level of consumption and N is population size. We assume that the function $\Delta(D)$ has the following form

$$\Delta(D) = M_0\left[k_A \min\{D, \bar{H}\} + k_E \max\{D - \bar{H}, 0\}\right].$$

The cost to society of one additional death is M_0. If we assume that this is equal to the utility of the remaining lifetime T, and the value of an additional year is a multiple, υ, of annual output, then we can approximate the utility loss associated with one death is

$$M_0 = \ln\left(\upsilon w\hat{\phi}\hat{L} - \underline{c}\right)\frac{1 - e^{-\rho T}}{\rho},$$

where $(\hat{\phi}, \hat{L})$ are the values of the "stay-at-home" parameter and the size of the available workforce that are used for the calibration. In a steady state with no epidemic $\hat{\phi} = 1$, and $\hat{L} = 1$.[4]

[4]There are different approaches to identify and measure the valuation of life. Examples of some of the options in the context of the analysis of epidemics are Greenstone and Nigam (2020) and Hall, Jones and Klenow (2020).

We study different cases indexed by the values of (k_A, k_E). Our baseline assumes $k_A = 0$ and $k_E = 1$. This corresponds to the case in which society is willing to spend resources only to avert "excess deaths" that is, deaths over and above some baseline[5]. In this case, the only term that matters in the $\Delta(D)$ function is $D^+ = \max\{D - \bar{H}, 0\}$.

This approach requires that we specify what the acceptable level is, $\bar{H}$ in our notation, and this is not easy to do. As a first approximation we will consider these excess deaths as deaths caused by lack of hospital capacity. Thus, in our calibration for $\bar{H}$ we use the number of available ICU beds as a measure of acceptable deaths.[6] The interpretation is that it is the extra cost to society associated with deaths that potentially could have been prevented if hospital capacity was higher that enters the social utility function. Our objective is to capture the tradeoff between the relatively fixed, in the short run, health infrastructure and the output cost of restricting employment. This is one of the aspects of the model that implies that there are benefits from "flattening the curve."

Since there is considerable uncertainty about the social value of averting deaths we explore different formulations. The case $k_A = k_E = 1$ corresponds to the situation in which **all deaths** are valued equally. We report detailed results for those two cases and summaries of the predictions of the model for a variety of (k_A, k_E) combinations.

2.2 The Epidemiological Model

Following the literature, we assume that the dynamics of an epidemic can be reasonably approximated by a version of the standard SIR model.[7] Here, we

[5]It is not obvious that the right approach is to posit that total rather than "excess" deaths should enter social preferences. For example, a large number of individuals die every year due to simple influenza. At the same time, there are relatively simple policies that could potentially avert many of those deaths (e.g. free vaccination, creating "vaccination stations" in convenient places (e.g. supermarkets, public transportation hubs) to reach a large fraction of the population including those that do not have ready access to healthcare). We view the absence of those policies as a revealed preference type of argument against including all deaths in the baseline.

[6]We are aware that as more information about the COVID-19 virus becomes available it is far from obvious that ICU beds or respirators is the appropriate limiting variable. There are reports that suggest that many COVID-19 patients develop renal problems and that dialysis machines might be another limiting factor.

[7]One of the most widely cited epidemiological studies of the COVID-19 epidemic is the Imperial College model in Ferguson et.al. that uses the SIR model. Economic analyses of

present a simple version although more general formulations (e.g. hospitalizations as a separate state with its own law of motion, alternative matching function to replace the canonical βSI in the SIR model) are relatively easy to incorporate.

In the model I is the total number of infectious individuals. This includes both symptomatic and asymptomatic. We assume that only a certain fraction, ζ, is identified as infected. These individuals do not contribute to the labor supply and we assume that they do not infect susceptible agents. The number of infected individuals who are asymptomatic is then $(1-\zeta)I$.

Let S be the number of susceptible individuals and R the population of resistant individuals. Then the potential labor force, L, is given by

$$L = S + R + (1-\zeta)I. \tag{2}$$

Since we normalized the population to one this is

$$L = 1 - \zeta I. \tag{3}$$

Then given a value of the stay-at-home policy ϕ, the fraction of susceptibles and infectious in the population is ϕS and $\phi(1-\zeta)I$ respectively.[8]

Finally we assume that a certain fraction of the resistant/recovered lose their immunity (at rate γ). The simple model is then given by

$$\begin{aligned} \dot{S} &= -\beta(\phi S)(\phi(1-\zeta)I) - \mu S + \gamma(1-S-I) \\ &= -\beta\phi^2(1-\zeta)SI - \mu S + \gamma(1-S-I). \end{aligned} \tag{4}$$

The first term is the standard matching function of the SIR model, while the second term, μS is the population that becomes resistant as a result of vaccination. The last term, $\gamma(1-S-I)$ captures both the rate at which

the COVID-19 epidemic from an economic point of view relying on the SIR model include Alvarez et. al. (2020), Atkeson (2020), Fernandez-Villaverde and Jones (2020).

We are aware of the limitations of the model. See Korolev (2020) for example. An alternative forecasting model, the IHME model also appears to have serious limitations. See Marchant et. al. (2020)

[8]In this setting, ϕ is a summary of the effects of a variety of different policies like lockdown, social distancing, school closure, mask wearing, travel restrictions and centralized quarantine. There is some evidence (see Chen and Qiu (2020)) that the effects of these NPIs is quite heterogeneous in terms of consequences of the epidemic. However, at the level of aggregation in this model they correspond to an average of feasible combinations. Future work will deal with heterogeneity in policies (different ϕ)

resistant individuals lose their immunity and the entrance of new susceptible individuals in the population.

The stock of infectious evolves according to

$$\dot{I} = \beta\phi^2(1-\zeta)SI - \kappa I.^9 \tag{5}$$

At this aggregate level this simple model suffices. However, keeping track of hospitalizations, deaths and individuals who have immunity (recovered if it turns out that infection provides immunity) is possible and desirable using a more disaggregated model.

In the simple model, we do not keep separate track of deaths associated with the epidemic. However, they play an important role determining the optimal policy. To keep the model simple —and ignoring the obvious lags— we will assume that a fixed fraction of those individuals who are identified as infected, ζI, die. We denote this fraction by χ. Then, the flow of deaths at time t is $D_t = \chi\zeta I_t$, and excess deaths, D_t^+ is simply

$$D_t^+ = \max\{0, \chi\zeta I_t - \bar{H}\},$$

Finally if we denote the path of the epidemic in the absence of a policy —what we label the uncontrolled case— by $(\hat{S}, \hat{I})$ the number of **deaths averted** up until time T, G_T, under a policy $\{\phi_s, \mu_s\}$ is

$$G_T = \int_0^T \chi\zeta\left(\hat{I}_s - I_s\right) ds,$$

and the **cumulative output cost** (relative to the full employment case) is

$$O_T = \left(\frac{1}{T}\right)\int_0^T \phi_s\left(1-\zeta I_s\right) ds.$$

By comparing G_T and O_T we can estimate the output cost per death averted.[10]

[9] It is interesting to note that in this simple version of the SIR model knowledge of I_t (the level of infections) and the rate of change over time (dI_t/dt) suffices to inform a planner that knows ϕ what S_t is.

[10] It is clear that our measure of output cost ignores many other consequences of drastic reductions in economic activity. For example all the managerial human capital that is lost (or reallocated to less profitable activities) is not included in our measure.

3 Analysis of the Model

Since the problem faced by the planner in Phases I and II is different, we start by discussing the optimal policy contingent on the economy having switched to Phase II first. We then discuss Phase I.

3.1 Phase II

In this Phase vaccination is available and the planner's objective function is

$$F(S,I) = \max_{\{\phi_t\}\{\mu_t\}} \int_0^\infty e^{-\rho t} u(\phi_t w(1-\zeta I_t) - c_V(\mu_t\left(S_t + (1-\zeta)I_t\right))) - \Delta\left[D_t\right] dt, \tag{6}$$

subject to equations (4) and (5) and $S_0 = S$ and $I_0 = I$ and subject to to $0 \leq \phi_t \leq 1$ and $0 \leq \mu_t \leq \bar{\mu}$, where $\bar{\mu}$ is a measure of the economy's speed to vaccinate the population.[11]

The optimal stay at home policy depends on the difference of the marginal shadow values of infectious and susceptibles. Formally, in the interior case, that is when $\phi \in (0,1)$, the optimal ϕ solves (details in Appendix 1)

$$\frac{u'(\phi w(1-\zeta I) - c_V(\mu(S+(1-\zeta)I)))(1-\zeta I)}{2\beta\phi(1-\zeta)SI} = \left(F_S - F_I\right). \tag{7}$$

For a given state (S,I) the left hand side is decreasing in ϕ. F_I measures the contribution of an additional infected individual to the value of the problem and it is negative, and so is F_S. However, in all cases, $F_S - F_I > 0$ since 100% of the susceptibles are in the labor force but only $1-\zeta$ of the infected. Increases in the marginal welfare cost of infected over susceptibles —for example when I is large and S is small— increase the right hand side of equation (7) and it results in a decrease of ϕ. Thus, optimal stay-at-home policy depends negatively on the excess welfare loss of an additional infected over an additional susceptible.

The model has a steady state. For sufficiently small γ (the rate at which immunity is lost in the population) the steady state displays no output loss ($\phi^* = 1$) and no vaccination ($\mu^* = 0$). We formally summarize this result in the following proposition

[11] Although we assume that the planner knows the aggregate fractions of S and I, we do not assume knowledge of which individual is infected. Thus, the planner has to vaccinate all the asymptomatic individuals even though the vaccine is "wasted" on those infected.

Proposition 1 (Phase II: Steady State) *Assume that the utility function is strictly increasing and strictly concave and that the marginal cost of vaccination is positive even at zero (that is, $c_V'(0) > 0$) then, for a small enough γ, there exists a steady state characterized by $\phi^* = 1$ and $\mu^* = 0$ and the epidemiological variables are*

$$S^* = \frac{\kappa}{\beta(1-\zeta)},$$

and

$$I^* = \frac{\gamma}{\kappa+\gamma}\left(1 - S^*\right).$$

Proof. See Appendix 1 ■

3.2 Phase I

In this Phase there is no vaccine. We assume (as in Alvarez et. al. (2020)) that vaccines become available at the (Poison rate) η. The planner's problem is

$$V(S,I) = \max_{\{\phi_t\}} E\left[\int_o^{T_\eta} e^{-\rho t}\left[u(\phi_t w L_t) - \Delta(D_t)\right]dt + e^{-\rho T_\eta} F(S_{T_\eta}, I_{T_\eta})\right],$$

where the expectation is taken over the distribution of the stopping time T_η which gives the first time that the Poison process jumps. The expected time until a vaccine is discovered is $1/\eta$.

The key differences between phases I and II are the non-availability of a vaccine (in Phase I) and a higher discount factor (also in Phase I).

It is interesting to study what happens in Phase I as $t \to \infty$ and there is no switch. The following proposition summarizes this case

Proposition 2 (Phase I: Pseudo Steady State) *The Phase I model has a steady state that coincides with the steady state in Phase II.*

Proof. See Appendix 1 ■

This result says that after a long enough period of time the availability of a vaccine does not have a large impact on the optimal policy. Thus, the social value of a vaccine —measured as the impact on the continuation value— decreases to zero as $t \to \infty$. This, of course, ignores future epidemics that can be averted using vaccines.

This result has some implications for how to finance a vaccine. If the winner receives a patent, the economic value of that patent —again in the case of one epidemic— goes to zero as time goes by because the epidemic is being controlled. Specifically, the convergence result implies that, for T_η large the change in optimal policy is small. One consequence of this is that firms that have to allocate resources to produce a patent see their potential payoff decreasing as time goes by and, intuitively, this should result in fewer resources allocate to discovering a vaccine as time goes by.

This suggests that financing a vaccine with a prize with a fixed value can potentially be a more efficient mechanism relative to a patent —at least in terms of inducing resources to be allocated— to produce a vaccine in a shorter period.

4 Quantitative Results

In this section we present some results for the baseline. We take our time unit to be a week. We report the complete list of parameters used in Appendix 3. Here, we describe the more significant assumptions underlying our baseline case:

- $\mathcal{R}_0$ is 2.8.
- Our measure of excess deaths uses hospital capacity as the threshold. We assume that 140,000 ICU beds would be available for COVID-19. We also consider both an optimistic and a pessimistic option.
- We assume that the infectious period lasts 3 weeks.
- We assume that, in expectation, it takes about 50 weeks for a vaccine to become available (Phase II)[12]. We also assume that it is costless to administer a vaccine and that the upper bound of the speed at which the population can be vaccinated is $\bar{\mu} = 0.05$. We also experiment with optimistic and pessimistic bounds. This implies that $\mu_t = \bar{\mu}$ in Phase II which differs from the optimal policy (that has $\lim_{t\to\infty} \mu_t = 0$).[13]

[12]This is $\eta = 1/50$.

[13]We solved a number of cases with endogenous μ but for low values of reinfection, γ, the solution is always ban-bang and, hence, setting it at the upper bound at zero cost is a very good approximation.

To compute the results we first discretized the continuous time HJB equation and then solved the weekly model using value function iteration. Given the model is highly non-linear, we solve the problem over a fine non-uniform grid and restrict the space to $0 \leq S + I \leq 1$. (See details in Appendix 2)

The optimal policy ϕ is a function of (S, I) is displayed (for Phases I and II) in the next two figures.

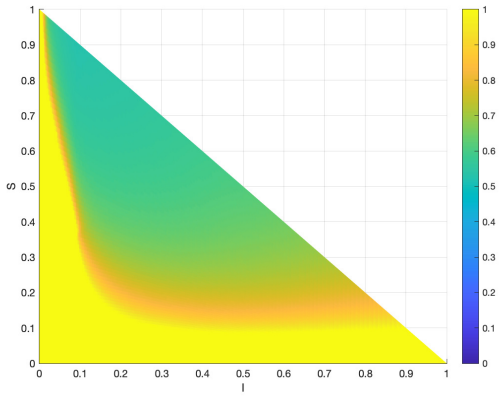


Figure 1. Optimal Policy (ϕ) in Phase I

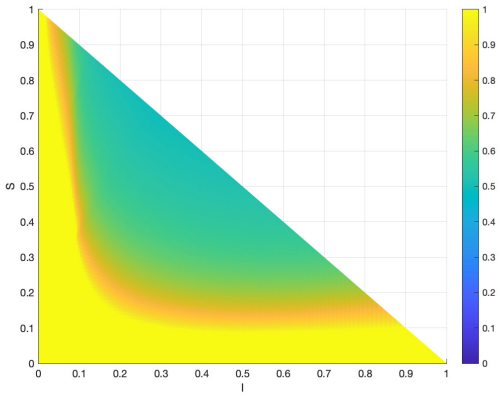


Figure 2. Optimal Policy (ϕ) in Phase II

As expected, for low levels of I, the optimal policy calls for no intervention (the yellow area corresponds to $\phi = 1$). Some interesting results are:

1. By construction the optimal policy is a function of the state (S, I) and it is such that for regions of the state space the optimal policy is not to restrict output (bright yellow area).

2. As expected, any policy that chooses the severity of the stay-at-home policy considering only infectious (or deaths) is bound to be suboptimal[14]. For example, in Phase I (no vaccine) and for $I = 0.1$, the optimal ϕ ranges —depending on susceptibles— from slightly below 0.6 to 1. Even though many of those points would not be observed in an epidemic starting from the natural initial condition ($S = 1$ and $I = 0$), the situation is quite different in a second (or higher) wave when, presumably, a certain fraction of the population has developed immunity.

[14]As indicated before, using information on I_t and dI/dt it is possible to infer S_t.

3. Availability of a vaccine has a significant impact on the optimal policy (compare Phase II with Phase I). The optimal policy in Phase II is "shifted to the right" relative to Phase I and it implies that, for all states, Phase II imposes less severe "stay-at-home" restrictions.

4. There are large subsets of the state space that even if a vaccine is available it is optimal to restrict employment. In the model arrival of a vaccine is not equivalent to lifting restrictions. It depends on the state of the economy.

4.1 Baseline Simulation

Any simulation must make an assumption about the realization of T_η, the time at which Phase II (vaccine) arrives. In our baseline we assume that $T_\eta = 50$, that is, that a vaccine becomes available after about 50 weeks, which is also the expected time of arrival.

Figure 3 shows that path of the stock of infectious individuals, I, in two cases: uncontrolled epidemic (black) and optimally managed epidemic (red).

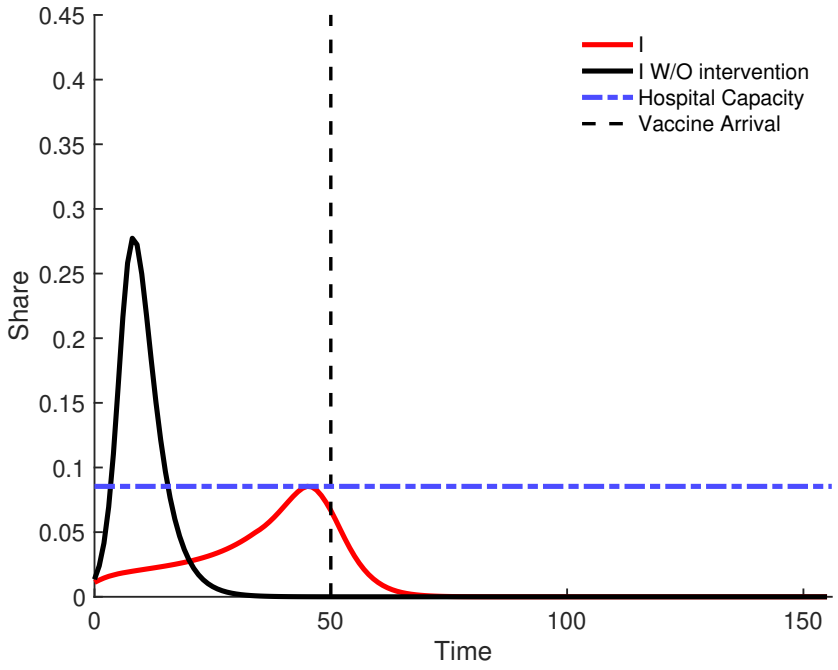


Figure 3. Time Path of I_t : Uncontrolled and Optimal.

The results in the uncontrolled case are independent of the economic model and are driven by the assumptions embedded in the epidemiological model. In the absence of controls, the epidemic would peak at about 20 weeks and about 28% of the population would be infectious at the time. In the absence of a policy (i.e. $\phi = 1$) there is a significant number of excess deaths (the area below the black curve above the blue dashed line).

Under the optimal policy the infectiousness curve is indeed flattened, and it takes slightly less than a year for the epidemic to peak (45 weeks) which is almost double the time in the absence of a policy. At the time of the peak I is 8.54% which is exactly our estimate of hospital capacity $\bar{H}$ ($\bar{H} = 0.0854$ indicated by the dashed blue lines).

Figure 4 shows the optimal policy and Figure 5 shows the implied $\mathcal{R}_t$.

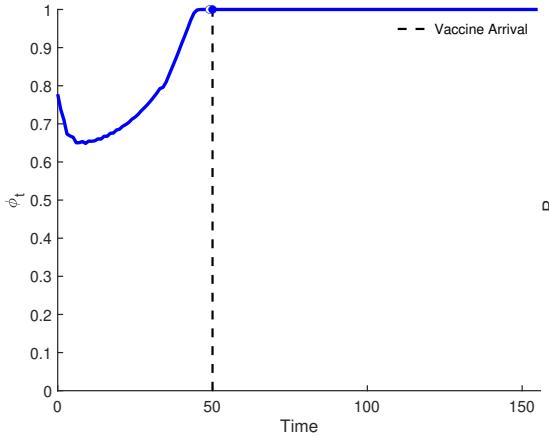


Figure 4. Optimal Policy (ϕ)

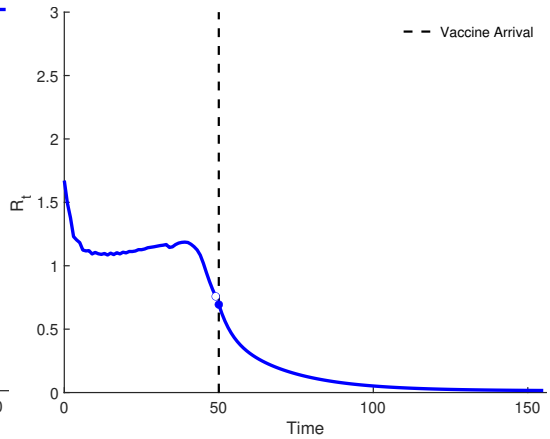


Figure 5. Optimally Managed $\mathcal{R}_t$

Two features of our solution are worth emphasizing. First, the initial "stay-at-home" policy is fairly aggressive and employment (and output in our linear model) falls approximately 22% from its full employment level and it bottoms out after 9 weeks at which time output is 35% below its pre-epidemic level. Full liberalization in this particular realization happens when the number of infectious individuals peak. At that point $\phi = 1$ and it stays at that level.

In this realization, the arrival of a vaccine occurs shortly after it is optimal to set $\phi = 1$ and has little effect on the optimal policy.

The time path of the optimal $\mathcal{R}_t$ does not follow the smoothly decaying path that is assumed in many analysis. In our calibration $\mathcal{R}_0 = 2.8$ but the

first observed $\mathcal{R}_t$ is 1.7 due to the aggressive restrictions on output that are implemented in the first week. From week 6 to week 39 the path is almost flat and just slightly above one. This path for $\mathcal{R}_t$ is consistent with controlling the epidemic to attain a sustained but relatively flat increase in infections. The peak occurs at week 39, and after that time the optimally managed $\mathcal{R}_t$ monotonically decreases and, in the long run, reaches zero.[15]

We defined averted deaths as the integral of the difference between the deaths that the model predicts would have occurred in the absence of a policy (i.e. $\phi = 1$) and the deaths under the optimal policy. We also defined the economic cost of managing the epidemic as the loss of output, O_T. Figure 6 shows the time paths of these two variables for this simulation.

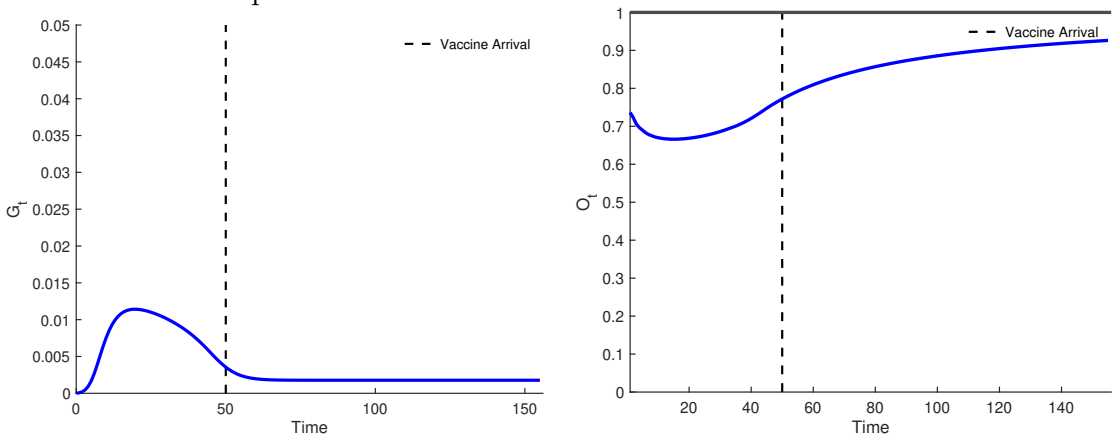


Figure 6. Deaths Averted (left panel) and Output Loss (right panel)

The cumulative number of lives saved is not a monotonically increasing function. It peaks about week 20 at 1.1% of the population when the optimally managed I crosses the uncontrolled case. From then on, the number of deaths averted decreases. The reason is simple: under the optimal policy flattening the curve has no impact (under our assumptions) on the true fatality rate —other than what is accounted for the arrival of a vaccine— but it spreads deaths over time. Thus, stay-at-home policies have a large impact in terms of saving lives early in the epidemic but that advantage turns negative as time goes by because an epidemic that would have extinguished itself persists in the population. In the long run the fraction of averted deaths converges to 0.177%.

The output cost of the optimal policy is significant. After 50 weeks output has been on average about 77% of full capacity. This estimate is substantially

[15]We present the time path for $\mathcal{R}_t$ for several cases in Appendix 4.

smaller than what is found in other studies. In the long-run (about 150 weeks) the average output loss over the whole period is 7.5%. This is large. The model implies that the cost —foregone output— per life saved is not a constant —as expected in any model with slowly moving state variables— and, in the long run, it is slightly above 7.5 million.[16]

The only random element in the model is the time at which a vaccine becomes available. It is not practical to report a large number of realizations but it is interesting to discuss how the optimal policy should react if a vaccine is available earlier than expected. In the next section we describe one such realization.

4.2 Early Arrival of a Vaccine

In Figure 7 we display the outcome of our simulation when the vaccine arrives fairly early, at about week 25.

By construction the first 25 weeks display an optimal policy that is identical to the baseline. However, at that point the optimal policy (and the outcomes) differ significantly (the economy enters Phase II) relative to the baseline. The most salient changes are:

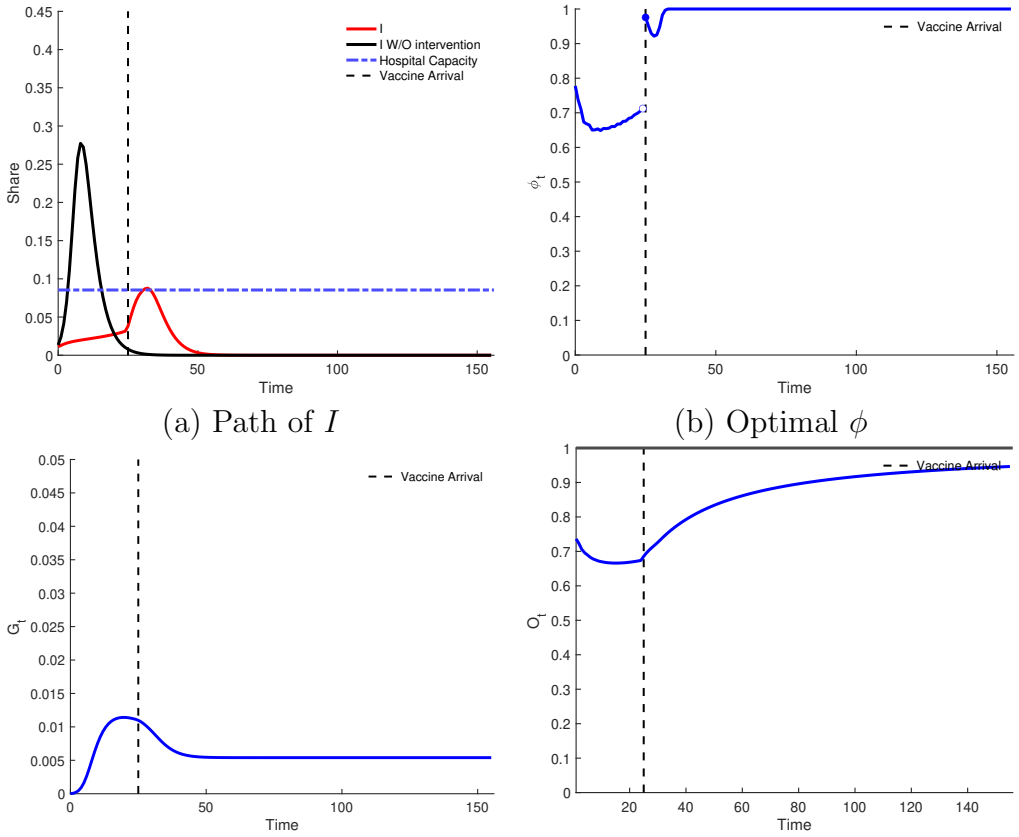
1. At the time the vaccine arrives the optimal stay-at-home policy discontinuously jumps. Employment goes from 71% to 97%, an almost complete liberalization.

2. This optimal policy liberalizes **before** the epidemic reaches a peak of infectious individuals.

3. A somewhat surprising consequence is that following the arrival of the vaccine the number of infectious individuals actually **grows faster** than before. The reason for this is that the availability of a vaccine increases the downward drift in the number of susceptible individuals (they are getting vaccinated) and, hence, it lowers the rate of contagion going forward.

4. Relative to the baseline the number of deaths averted triples (0.54% vs 0.177%) and, on average, the loss of output is about 5.5%. The output cost per life saved is substantially smaller (but still large) at 1.8 million.

[16]In Appendix 4 we present the time path of the output cost per death averted for a variety of scenarios



(a) Path of I (b) Optimal ϕ

(c) Deaths Averted (d) Output Loss

Figure 7: Early Arrival of a Vaccine ($T_\eta = 25$)

In Appendix 4 we show the results if the vaccine arrives late. It turns out that this late arrival has a small impact on the optimal policy, but a sizable effect on the realized output cost per death averted which is over 10 million.

4.3 Alternative Scenarios

Since there is a huge amount of uncertainty about the appropriate values of key parameters we discuss how the results of the model change when we modify some of our baseline assumptions. We consider the following variations:

- "Optimistic" and "Pessimistic" scenarios that simultaneously change several parameters.

- Alternative valuation of human life.[17]

4.3.1 Optimistic and Pessimistic Scenarios

In this section we describe two scenarios that are designed to provide bounds for a number of aspects of the model about which there is significant uncertainty:

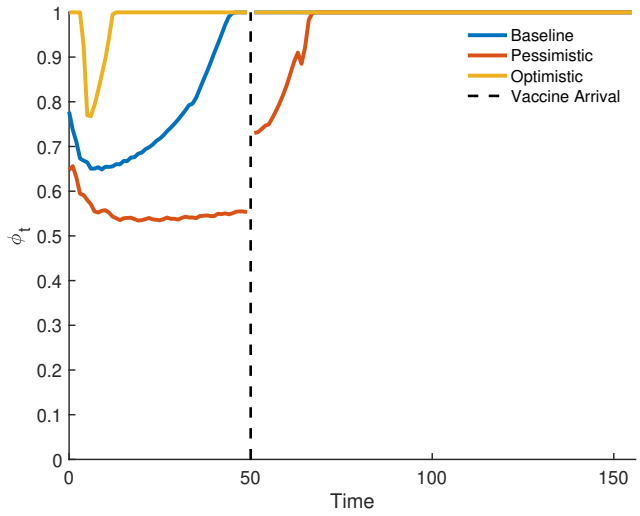


Figure 8: Time Path Optimal ϕ

- **Optimistic Scenario**: We assume that the upper bound of the vaccination rate is six times as high as in the baseline which implies about 30% of the vaccinable population[18] is vaccinated in one week. [19] We also assume that the expected time until a vaccine arrives is 24 weeks (50 in the baseline) and the actual hospital capacity is twice the value in the baseline (280,000 ICU beds for COVID-19 patients).

- **Pessimistic Scenario**: In this case we assumed that the hospital capacity rate is 1/2 of our baseline (about 70,000 ICU beds for COVID-

[17] Appendix 4 also includes the results from a number of other robustness experiments.
[18] This includes susceptibles and asymptomatic infectious.
[19] Note that this is also the proportion of all people that have to be vaccinated if it is not possible to determine who are the susceptible. In Appendix 4 we display the time paths of the relevant variables.

19), that the infection rate is higher, $\mathcal{R}_0 = 4$, and that the maximum vaccination capacity is one fifth of the baseline case[20].

In Figure 8 we show the path (to provide a comparison, we also report the same statistic for the baseline) of the optimal stay-at-home policy assuming that a vaccine arrives in week 50.

The differences are striking: In the optimistic case the stay-at-home restriction is severe (over 20% of a reduction in employment) but short lived. The economy goes back to full employment in 5 months. At the other end, the pessimistic scenario results in a deeper and prolonged recession: employment during the first year is below 60% of normal and the arrival of a vaccine does not result in a complete liberalization. It takes 1 1/2 year to get back to full employment. The difference between the optimal policy in the post-vaccine era between the two extreme scenarios illustrates well our view that ignoring the post-vaccine optimal policy will result in a misleading characterization of the optimal policy.

Table 1 displays the results for some indicators.

Table 1**: Scenario Comparison**

Indicator	*Baseline*	*Optimistic*	*Pessimistic*
Y loss (1Y) (%)	22%	3%	45%
Y loss (3Y) (%)	7.5%	1%	16.4%
Full Recovery (months)	13	5	18
Deaths Averted (%)	0.177%	0.05%	1.83%
Cost per Death Averted ($)	7.56M	3.2M	1.61M

The main takeaways from this exercise are:

1. In most of our indicators the possible range is large, and this illustrates how much uncertainty about the appropriate model of the world impacts the optimal policy.

2. Both the depth and the duration of the recession are highly dependent on the scenario. In the optimistic case, the first year drop in output is significant but the economy quickly recovers. In the pessimistic case the economy does not return to full employment for 18 months, and the average yearly loss of output is over 15% at the three year mark.

[20]The time paths for this case are in Appendix 4 labeled Pessimistic Scenario I. There is an alternative pessimistic scenario (Pessimistic Scenario II) that gives similar results.

3. The estimate of the number of deaths averted and the unit cost displays large bounds. There is no simple association between number of deaths averted and unit cost. For example, the largest number of deaths averted correspond to the pessimistic scenario and the unit cost is the lowest. The unit costs depend on the elasticities of the two relevant variables (averted deaths and output) with respect to the optimal policy. The former, in turn, also depends on the shape of the epidemiological model.

4.3.2 Alternative Valuation of Deaths Averted

The implicit value of life in the model depends on two elements about which there is significant uncertainty:

1. Which deaths are counted?
2. What is the shadow value of forgone consumption for individuals who die?

With respect to the first question, our baseline assumes that only deaths that exceed a threshold matter. This is meant to capture scenarios in which society cares about extraordinary deaths associated with the epidemic. For example, it allows for a higher valuation of "possibly preventable" deaths that occur because of insufficient health infrastructure.

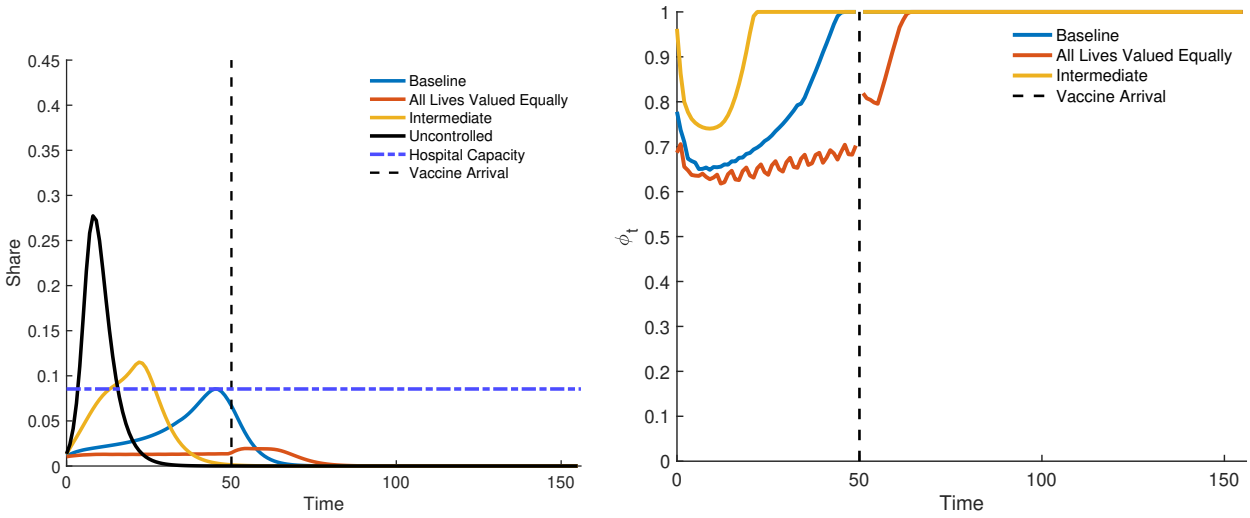
With respect to the second question there is also considerable uncertainty. Our baseline assumes that $\upsilon = 3$ (see Hall et. al (2020) for an application to the COVID-19 epidemic). In this section we report the results of two very different scenarios:

- **All Lives Matter**: In this case we assume that all deaths (and not just those exceeding our measure of hospital capacity) enter in the social utility function. In this case $\Delta(D) = M_0 D$.[21]
- **Lower Consumption Value**: This alternative —which corresponds to a lower value of life— assumes that society only cares about those deaths that exceed our measure of health capacity, $\bar{H}$, and that it values

[21] We emphasize that this social disutility of deaths is **over and above** the disutility that society endures from deaths caused by other somewhat preventable activities (e.g. driving, drinking) and diseases (influenza, drug addiction).

the foregone consumption at 1/2 its market value (the baseline assumes that the relevant factor is three). In this case $\Delta(D) = (M_0/3)\, D^+$.

The results for the optimal policy and the loss of output are in Figure 9 (where the lower consumption value is labeled Intermediate)



Path of I_t Path of ϕ_t

Figure 9: Curve Flattening and Optimal Policies

The results are striking: The social utility associated with averting deaths plays a major role in determining how much the time path of infections is flattened and the restrictiveness of the optimal policy. When the value of consumption is low, the epidemic disappears before week 50. The cost of this, is a number of deaths that exceed the hospital capacity. At the other end, when society cares about all lives equally the curve is flattened, the number of people who are infectious (and, in this model, this is proportional to those who require hospitalization) does not come even close to the hospital constraint. The cost is large. The economy loses a large amount of output in the first year. Interestingly, even in this conservative case the optimal policy starts lifting restrictions on employment (the ϕ_t path is upward sloping) a long time before the epidemic peaks.

In Table 2 we report some statistics about the time path of output in these three cases.

Table 2: **Output Loss (%) and Recovery (in months)**			
(k_A, k_E)	Initial Y	Beginning of Recovery	Full Recovery
All Lives matter ($\upsilon = 3$)	32%	4 (37%)	17
Excess Deaths ($\upsilon = 3$)	27%	3 (37%)	14
Excess Deaths ($\upsilon = 0.5$)	16%	3 (25%)	7

The value that society puts on averting deaths has a large impact on the initial (one year) drop in output that ranges from 16% to 32% and the length of the time that the economy faces restrictions on employment: When all lives are counted equally it takes almost 1 1/2 year to completely eliminate stay-at-home restrictions, while at the other extreme the economy is back to normal in about 7 months. Interestingly, in all three cases the lockdown is partially lifted after 3-4 months. The difference resides in the speed at which it is lifted.

We conclude that the value of life —about which we have considerable uncertainty— plays a major role in determining the optimal policy. In particular, it shapes the time series properties of output and employment and has a large effect on the speed of recovery. In the next section we expand the discussion of the role that the value of life has on other outcomes.

4.3.3 More on the Value of Life and the Cost of Averting Death

Our basic equation for the social value of a life is

$$\Delta(D) = M_0 \left[k_A \min\{D, \bar{H}\} + k_E \max\{D - \bar{H}, 0\} \right].$$

In this section we present the consequences in terms of deaths averted and output cost per death averted of different combinations of (k_A, k_E). The results are in Table 3.

Table 3: **Deaths Averted (%) and Unit Cost (millions)**				
	Expected Arrival		Early Arrival	
(k_A, k_E)	Deaths Averted	Cost	Deaths Averted	Cost
(1,1)	0.88	2.50	1.01	1.30
(0.5,1)	0.68	2.94	0.88	1.37
(0.1,1)	0.22	6.58	0.58	1.73
(0,1)	0.18	7.56	0.54	1.81
(0,.79)	0.16	7.87	0.48	1.93
(0,.72)	0.14	7.71	0.45	1.98
(0,.33)	0.10	5.11	0.16	3.28
(0,.20)	0.03	5.34	0.03	4.95

The table is such that as one moves down the rows the value of life —either in terms of who counts or how much is valued— decreases. For reference, the "All Lives Matter" case is the (1,1) case, while our baseline is (0,1)[22]. There are several interesting results:

1. As expected the smaller the value of life the smaller the number of deaths averted. However, in the base case (a vaccine arrives when expected, that is, at $T_\eta = 50$) the price tag is not a monotone function of the social value of life. It actually peaks for intermediate values but even in the case of the lowest valuation the cost is twice as high as in the all lives matter.

2. In the baseline the costs are fairly high but not out of line with some standard estimates of the monetary value of a life.

3. The costs per death averted are very sensitive to the arrival time of a vaccine, especially when lives are very valuable.

What is driving these findings? From a formal point of view our baseline (0,1) is a more concave function of deaths than the all lives matter (1,1), this extra curvature implies that the optimal policy puts a lot of weight on not exceeding the threshold and the cost in terms of output is very high. This depends on the non-linearities in the model: In the all lives matter output is kept low for a long time but the flattening of the curve results in a large number of deaths averted. In the baseline (and when lives are not very

[22]The case (0,.79) corresponds to $\upsilon = 1$ and (0,.72) to $\upsilon = .8$.

valuable) the improvement in output is large but less than proportional to the fewer deaths averted.

4.4 The Social Value of A Vaccine

For any given state of the economy —a pair (S, I)— the value of a vaccine is given by the difference in the value of the problem between Phase II and Phase I. Proposition 2 shows that even if a vaccine never arrives the economy in a "permanent" Phase I converges to the same steady state as in Phase II. Thus, if the difference between the total utility in each Phase is computed at a long enough horizon, our theory predicts that the value of a vaccine is small. The interesting quantitative question is to assess how much this social value changes for relatively short time horizons.

Table 4 reports the dollar equivalent of the gains associated with the availability of a vaccine for several of our scenarios (some values are in trillions, T, and some in billions, B). We include the gains at four points in time: at the beginning of the epidemic, a month into the epidemic, 6 months into the epidemic and a year after the beginning of the epidemic.

Table 4: Value of a Vaccine

	Arrival Time			
Scenarios	1	4	25	50
Baseline	1.05T	1.10T	430B	4B
Optimistic	2B	2B	2B	2B
Pessimistic	4.79T	4.94T	4.30T	3.11T

As expected, the different scenarios imply very different values of a vaccine. In the optimistic case the key determinant of the low market value of a vaccine is the assumption that even if employment is not severely restricted, the ensuing demand for hospitalizations can be accommodated given the assumption of a better (larger) health infrastructure. The pessimistic case has two elements that account for the high value of the vaccine: much higher infection rate —which generates a more severe peak demand for hospitalizations— and a lower hospital capacity. In all these cases the value of life used is the baseline, that is, only excess deaths are counted. Moreover, along all the paths that we consider, the actual excess deaths are basically zero. Thus, the value of the vaccine reflects mostly the changes in

consumption associated with the availability of a vaccine. Averting excess deaths plays a large role —it dramatically influences the choice of ϕ— but there are very few actual excess deaths.

Figure 10 reports the value of the vaccine as a function of the state. It is interesting to note that the value of a vaccine after a month is higher than at the outset of the epidemic. The reason is that the state of the economy after one month has moved in the direction the yellowish area in Figure 10 that corresponds to the highest valuation of a vaccine. However, along a path that starts close to the case where 100% of the population is susceptible (the red line) the trend is to move away from the high value region.

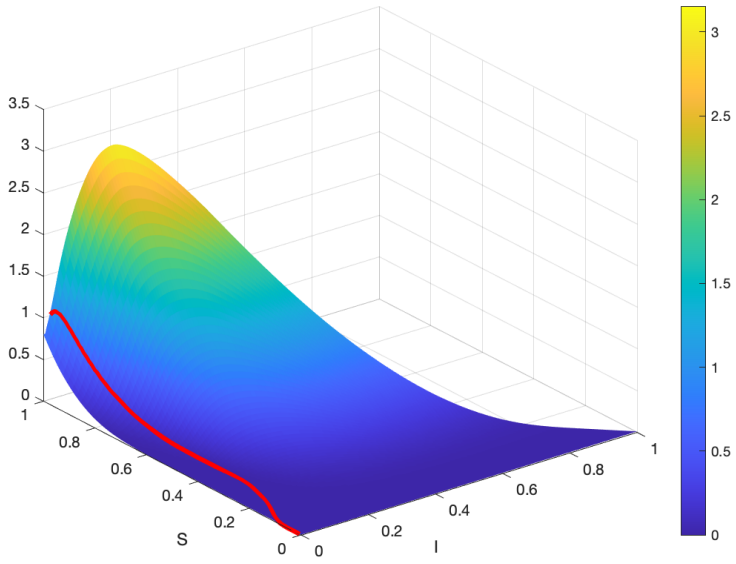


Figure 10. The Value of a Vaccine (in Trillions USD)

It follows that there are some states (in yellow) in which the vaccine would be very valuable. However, those are not reached from reasonable initial conditions. For an epidemic that starts with a large number of susceptible individuals and a small number of infectious (which is our case) the time path of the state stays away from the high valuation area.

We view these results as giving an upper bound on the value that society puts on a vaccine. The values are large and there is a wide range of estimates.

If we ignore the "Optimistic" scenario, a vaccine available after one month is worth somewhere between 1 and 5 trillion. However, after a year the estimates are below 5 billion (except in the "Pessimistic" case). To the extent that the sharp drop in value that we find matches the slope of the private payoff, our results suggest that the private sector will face decreasing incentives to allocate resources to produce a vaccine as time passes.

The key driver of these results is the dynamics of the epidemic as embedded in the epidemiological model. Highly infectious viruses spread rapidly and, in the case of COVID-19, can be quite deadly but they also extinguish themselves in a short period. For example the major wave of the 1918 pandemic (starting roughly in October 1918) lasted about 2 1/2 months. The value of a vaccine by the mid 1920s was essentially zero. This is important because in the model early availability of a vaccine has two effects on social welfare: it prevents deaths (fewer susceptible individuals in the population) and this, in turn, allows for more liberalization of employment restrictions and higher output.

As we discussed before, the different alternatives that we consider about how to determine the value of averting deaths have a large impact on policy and, hence, on the value of a vaccine. Table 5 reports the same statistics for different implicit value of life formulas as discussed in section 4.3. The effective value of life decreases as one moves down the rows of the table.

Table 5: $\Delta(D)$ **and the Value of a Vaccine**

	Arrival Time			
(k_A, k_E)	1	4	25	50
(1,1)	3.19T	3.16T	2.34T	1.64T
(0.5,1)	1.78T	1.78T	1.07T	0.51T
(0.1,1)	1.13T	1.18T	500B	61B
(0,1)	1.05T	1.10T	431B	4B
(0,.79)	758B	834B	241B	4B
(0,.72)	666B	747B	182B	4B
(0,.33)	148B	215B	4B	4B
(0,.20)	220B	410B	4B	4B

Most of the social value of a vaccine is driven by the valuation of human life. The differences are large. The first row corresponds to the "all lives matter" and the value of producing a vaccine after 6 months is roughly 6

times higher than the value in our baseline (the (0,1) case).[23]

5 Extensions

In this section we describe some extensions that are part of our ongoing work in this area.

Recurrent Epidemics In our formal model we have assumed away the possibility of a recurrence of the epidemic. However, this is feature is relatively easy to accommodate within our structure by adding a Phase III (that also arrives as a Poisson shock) that either increases temporarily the rate at which the population lose immunity or, more directly, increases the number of people who are susceptible.

Within the context of our model an approximation to this is to consider an MIT type of shock. Specifically if we assume that the economy is in Phase II and there is a one time (small) increase in the number of susceptible individuals starting from the steady state (that is $S = 34\%$ and $I = 1\%$), the optimal policy has $\phi = 1$ when the upper bound of the vaccination rate is $\bar{\mu} = 0.05$. This implies that the number of infected monotonically decreases to zero is about 6 months.

More formally, we are extending the model to allow a random increase in either the number of susceptibles (and an equivalent decrease in the number of resistant) or a temporary increase in γ as two alternative ways to capture the possibility of a second wave or the arrival of a new version of the same virus in the case that a fraction of the population already have immunity. We expect this version of the model to give more realistic estimates of the value of a vaccine.

Short vs. Long Run The current version of the model is not well equipped to seriously consider the possibility that impact effects are quite different from long run effects. In particular, it cannot capture differences between short and long run. For example, it is possible that individuals are willing to comply with "stay-at-home" policies in the short run but that, as the restriction remains in effect for a prolonged period, more individuals are

[23]These calculations ignore the value of a vaccine in the case of a second outbreak. We discuss some options in the section on extensions.

willing to violate the policy. Similarly on the supply side of the economy, closing down some activities may not have a large impact in the short run but it may be impossible to sustain over long periods of time. To capture this we will allow past values of the control variable ϕ to influence productivity[24]. Thus, for an economy that starts from $\phi = 1$ it may be easy (and not very costly) to lower ϕ to say 0.5, but after a long period at 0.5 the productivity will be decreasing (more details needed).

An example of this type of adjustment is to allow productivity to vary over time. We assume that productivity at time t is

$$w_t = w(1 - x_t),$$

where x_t evolves according to

$$\dot{x}_t = -\lambda x_t + (1 - \phi_t), \text{ for } \lambda > 1.$$

This formulation captures the idea that of $\phi = 1$ all the time then if $x_0 = 0$ then $x_t = 0$ and productivity is constant. If, on the other hand x_t is positive and the planner switched to $\phi = 1$ then x_t will converge (the speed depends on λ) to zero, and productivity will be increasing.

The solution for x_t is

$$x_t = \int_0^t e^{-\lambda(t-s)} (1 - \phi_s) \, ds.$$

Development The model can be used to ascertain how Phase I policies should be chosen depending on the ability to vaccinate the population rapidly. Our preliminary results suggest that poor healthcare facilities —as proxied by a low $\bar{H}$— and low feasible vaccination rate —as captured by a low $\bar{\mu}$— imply that a country with a more precarious public health (for example a very poor country) infrastructure should have a more aggressive policy —conditional on the state (S, I)— compared with a country with a good health sector.

To consider differences across the development spectrum we are extending the model to better capture the role of healthcare infrastructure and the output cost of mass vaccination.

In addition we will move to a CRS utility function since the log function implies that income and substitution effects cancel.

[24]We can also use the same type to model the potential loss of effectiveness of social distancing policies as a function of how long they have been in effect.

Asset Prices In Phase I the interest rate satisfies

$$r_t^I = \rho + \left(\frac{\dot{\phi}_t^I}{\phi_t^I} - \frac{\zeta \dot{I}_t}{1 - \zeta I_t} \right) + \eta \left(\frac{\phi_t^{II}}{\phi_t^I} - 1 \right).$$

Once the economy switches to Phase II the interest rate is given by

$$r_t^{II} = \rho + \left(\frac{\dot{\phi}_t^{II}}{\phi_t^{II}} - \frac{\zeta \dot{I}_t}{1 - \zeta I_t} \right)$$

There are three forces that impact the interest rate in Phase I. During periods in which the economy is contracting due to the stay-at-home policies, $\dot{\phi}_t^I < 0$, and the number of infectious cases is increasing, $\dot{I}_t > 0$, the interest rate is low and it falls below the discount factor if the degree of liberalization —as measured by the term ϕ_t^{II}/ϕ_t^I which is always greater than one— is not large. As time passes, the term $\dot{\phi}_t^I/\phi_t^I$ turns positive and the interest rate over shoots its long run value (ρ).

We expect the term $\dot{\phi}_t^I/\phi_t^I$ will dominate in most of our cases and hence we view the model as predicting low interest rates until the ϕ function reaches its lowest level and high interest rates after that. In the cases in which the arrival of a vaccine induces a large jump in ϕ, the interest rate is close to the discount factor.

What is the impact of the pandemic on the value of output? It is useful to first describe the valuation formula once the economy has switched to Phase II. It is given by

$$A_t^{II} = \int_0^\infty e^{-\int_t^{t+s} r_u^{II} du} \left(w \phi_s^{II} (1 - \zeta I_s) \right) ds.$$

The corresponding value during Phase I is

$$A_t^I = \int_0^\infty e^{-\int_t^{t+s} r_u^I du} \left(w \phi_s (1 - \zeta I_s) + \eta A_s^{II} \right) ds.$$

We are currently computing the results for both interest rates and asset prices for a variety of scenarios.

Multi-Region Model We are extending the model to capture regional differences. We assume that each region produces two goods: one of the

goods is produced with local inputs and the other uses inputs from other regions. In the first stage, we study the no-migration case and hence, in the epidemiological dimension we have N independent SIR models. We view the planner as choosing stay-at-home policies and vaccination rates (when available) for each region separately. We also compute the one-size-fits-all solution and study the welfare losses associated with that.

6 Concluding Comments

Here we offer some tentative comments about what we have learned from the exercise (in addition to the large impact that model uncertainty has on our predictions):

1. **Stylized features of optimal policies**.
   (a) Optimal stay-at-home policies policy depends on both the fraction infected and the fraction susceptible.
   (b) They have a shocks treatment aspect to them: initially they are all severe but the duration depends on details of the economy.
   (c) The liberalization starts before the epidemic reaches its peak, and this typically results in an increase in the rate of infection.
2. **Stylized features of suboptimal policies**.
   (a) Policies that posit that liberalization must start after the epidemic peaks are suboptimal.
   (b) Policies that uniformly respond to increase in the rate of infection by tightening stay-at-home rules are suboptimal.
3. **Vaccines**.
   (a) Optimal pre-vaccine policies are not independent of the probability distribution of the arrival of a vaccine and the feasible vaccination rate.
   (b) The market value of a vaccine decreases rapidly (especially if the infection curve cannot be flattened)

4. **The Value of Life**.

   (a) The particular details how society puts a value on averting deaths has a first order effect on the optimal stay-at-home policy. This, in turn, determines the severity of the recession associated with the epidemic and the duration.

   (b) In many of our scenarios, the ex-post cost of averting a death is large and exceeds 2 million.

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Appendix: List of Contents

Appendix 1: Proofs

Discussion of Phase II Optimal Policy Let the value function of this problem satisfy the following HJB equation

$$\rho F(S,I) = \max_{(0\leq\phi\leq 1\ ,\ 0\leq\mu\leq\bar{\mu})} \{u(\phi w(1-\zeta I) - c_V(\mu(S+(1-\zeta)I))) - \Delta\left[(\chi\zeta I_t - \bar{H})^+\right])$$
$$+F_S\left[-\beta\phi^2(1-\zeta)SI - \mu S + \gamma(1-S-I)\right] + F_I\left[\beta\phi^2(1-\zeta)SI - \kappa I\right]\}.$$

The associated Hamiltonian is

$$H^{II} = \max_{(0\leq\phi\leq 1\ ,\ 0\leq\mu\leq\bar{\mu})} u(\phi w(1-\zeta I) - c_V(\mu(S+(1-\zeta)I))) - \Delta\left[(\chi\zeta I_t - \bar{H})^+\right]$$
$$-\lambda_S\left[\beta\phi^2(1-\zeta)SI + \mu S - \gamma(1-S-I)\right] + \lambda_I\left[\beta\phi^2(1-\zeta)SI - \kappa I\right]$$
$$+\hat{\gamma}^{\phi}(1-\phi) + \hat{\gamma}^I_+(\bar{\mu}-\mu) + \hat{\gamma}^I_+(\bar{\mu}-\mu) = 0.$$

The FOC are standard and given by the static conditions

$$\begin{aligned} u'()w(1-\zeta I) &= 2\beta\phi(1-\zeta)SI\left(\lambda_S - \lambda_I\right) + \hat{\gamma}^{\phi}, \\ \hat{\gamma}^{\phi}(1-\phi) &= 0 \end{aligned} \tag{8}$$

and

$$-u'()c'_V(\mu(S+(1-\zeta)I))(S+(1-\zeta)I) = -\lambda_S S + \hat{\gamma}^I_- - \hat{\gamma}^I_+, \tag{9}$$

where we omit the argument in the utility function to keep the expression simple. The constraints imply that $\hat{\gamma}^I_+(\bar{\mu}-\mu) = 0$, and $\hat{\gamma}^I_-\mu = 0$,

In the interior case, that is when $\phi \in (0,1)$, equation (8) can be written as, given that $\lambda_S = F_S$ and $\lambda_I = F_I$,

$$\frac{u'(\phi w(1-\zeta I) - c_V(\mu(S+(1-\zeta)I)))w(1-\zeta I)}{2\beta\phi(1-\zeta)SI} = \left(F_S - F_I\right),$$

which corresponds to equation (7) in the text.

Proof. Consider first Phase II. The relevant co-state variables evolve according to the following differential equations

$$\begin{aligned} \dot{\lambda}_S &= u'(\phi w(1-\zeta I) - c_V(\mu(S+(1-\zeta)I)))c'_V(\mu(S+(1-\zeta)I))\mu \\ &+ \left(\rho+\mu+\gamma\right)\lambda_S + \left(\lambda_S - \lambda_I\right)\beta\phi^2(1-\zeta)I \end{aligned} \tag{10}$$

$$\dot{\lambda}_I = u'(\phi w(1-\zeta I) - c_V(\mu(S+(1-\zeta)I)))\left[w\phi\zeta + c_V'(\mu(S+(1-\zeta)I))(1-\zeta)\mu\right] \tag{11}$$
$$+\Delta'[(\chi\zeta I - \bar{H})^+]\chi\zeta + (\rho+\kappa)\,\lambda_I + (\lambda_S - \lambda_I)\,\beta\phi^2(1-\zeta)S + \lambda_S\gamma.$$

The static first order conditions are equations (8) and (9).

We conjecture that there is a steady state such that $\phi^* = 1$ and $\mu^* = 0$. At this steady state the epidemiological variables satisfy

$$\beta(1-\zeta)S^* = \kappa, \tag{12}$$

and

$$I^* = \frac{\gamma}{\gamma+\kappa}(1-S^*). \tag{13}$$

It suffices to show that the system of equations that is implied by $\dot{\lambda}_S = \dot{\lambda}_I = 0$ has a solution evaluated at the candidate steady state and that satisfies equations (8) and (9).

Simple calculations show that $\dot{\lambda}_S = \dot{\lambda}_I = 0$ imply

$$\lambda_S^* = -\frac{\beta(1-\zeta)I^*u'(w(1-\zeta I^*)\zeta w}{\Lambda} \tag{14}$$

and

$$\lambda_I^* = -\frac{(\gamma+\rho+\beta(1-\zeta)I^*)\,u'(w(1-\zeta I^*)\zeta w}{\Lambda}, \tag{15}$$

where

$$\Lambda = \rho\,(\gamma+\rho+\beta(1-\zeta)I^*) + \beta(1-\zeta)I^*\,(\gamma+\kappa)\,.$$

To complete the argument it suffices to show that equations (8) and (9) hold as inequalities (ignoring the Lagrange multipliers). Some standard manipulations show that this is equivalent to (in the case of equation (8)) to

$$(1-\zeta I^*) > 2\kappa I^*\left[\frac{(\rho+\gamma)\,\zeta}{\Lambda}\right].$$

Since the left hand side is decreasing in I^* and it converges to 1 as $I^* \to 0$, while the right hand side converges to zero as $I^* \to 0$ then equation (8) is satisfied. To check that equation (9) holds as well, it suffices to show that

$$u'(w(1-\zeta I^*)c_V'(0) \geq -\lambda_S^*\frac{S^*}{S^*+I^*} = \frac{S^*}{S^*+I^*}\frac{\beta(1-\zeta)I^*u'(w(1-\zeta I^*)\zeta w}{\Lambda},$$

and this holds for I^* sufficiently small since $\lim_{I^*\to 0}\Lambda = \rho(\rho+\gamma) > 0$.

Now we want to show that same steady state is a rest point of the dynamical system associated with the optimal solution in Phase I. The Hamiltonian in this case is

$$H^I = u(\phi wL) - \Delta\left[(\chi\zeta I_t - \bar{H})^+\right] + \eta F(S,I) - \pi_S\left[\beta\phi^2(1-\zeta)SI + \gamma(1-S-I)\right] \\ +\pi_I\left[\beta\phi^2(1-\zeta)SI - \kappa I\right],$$

where μ is exogenously set equal to zero and that the relevant discount factor is $\rho+\eta$ during Phase I is $\rho+\eta$

We assume that the function $F(S,I)$ is differentiable (to be proved later) and we look at the limiting behavior of the relevant dynamical system along a path in which the Poisson counter never goes off under the assumption that the limiting $\phi = 1$. The steady state is such that

$$\pi_S^* = \frac{\eta(\rho+\eta)F_S^* + \beta(1-\zeta)I^*\left(\eta F_S^* - u'(w(1-\zeta I^*))w\zeta\right)}{\tilde{\Lambda}},$$

$$\pi_I^* = \frac{(\rho+\eta+\gamma+\beta(1-\zeta)I^*)\left(\eta F_S^* - u'(w(1-\zeta I^*))w\zeta\right) + \eta F_S^*\left(\beta(1-\zeta)I^*\right)}{\tilde{\Lambda}},$$

where

$$\tilde{\Lambda} = (\rho+\eta+\gamma+\beta(1-\zeta)I^*)(\rho+\eta) + \beta(1-\zeta)I^*\left(\gamma+\kappa\right),$$

and

$$F_S^* = \lambda_S^* \text{ and } F_I^* = \lambda_I^*.$$

It follows that $\lim_{I^*\to 0}\pi_S^* = 0$ (details omitted but just brute force) and $lim_{I^*\to 0}\pi_I^* < 0$ (and finite)

The relevant first order condition to guarantee that the solution to the static condition is $\phi = 1$ is

$$(1-\zeta I^*) > 2\kappa I^*\left[\pi_S^* - \pi_I^*\right],$$

and it is clearly satisfied for small I^*.

To summarize if

$$I^* = \frac{\gamma}{\gamma+\kappa}(1-S^*)$$

is sufficiently small (that is, if γ —the rate at which the population of susceptibles is replenish) is small) then the long run behavior with and without vaccines is exactly the same. ■

Appendix 2: Computational Appendix

Writing the HJB equations for the two phases,

$$\rho F(S,I) = \max_{(0\leq\phi\leq 1)} \{u(\phi w(1-\zeta I) - c_V(\mu(S+(1-\zeta)I))) - \Delta\left[(\chi\zeta I_t - \bar{H})^+\right])$$
$$+F_S\left[-\beta\phi^2(1-\zeta)SI - \mu S + \gamma(1-S-I)\right] + F_I\left[\beta\phi^2(1-\zeta)SI - \kappa I\right]\}$$
$$\rho V(S,I) = \max_{(0\leq\phi\leq 1)} \{u(\phi w(1-\zeta I) - c_V(\mu(S+(1-\zeta)I))) - \Delta\left[(\chi\zeta I_t - \bar{H})^+\right])$$
$$+V_S\left[-\beta\phi^2(1-\zeta)SI + \gamma(1-S-I)\right] + V_I\left[\beta\phi^2(1-\zeta)SI - \kappa I\right]\} + \eta(F(S,I) - V(S,I))$$

Discrete Version of HJB

We discretize the above HJB equations and solve the weekly model using value function iteration. Given the model is highly non-linear, we solve the problem over a fine non-uniform grid and restrict the space to $0 \leq S+I \leq 1$.

Phase II

$$F(S_t,I_t) = \max_{(0\leq\phi\leq 1)} \left\{\frac{(1-e^{-\rho\Delta})}{\rho}\left(u(\phi w(1-\zeta I) - c_V(\mu(S+(1-\zeta)I))) - \Delta\left[D_t\right]\right) + e^{-\rho\Delta}F(S_{t+\Delta},I_{t+\Delta})\right\}$$

$$S_{t+\Delta} = S_t + \left[-\beta\phi^2(1-\zeta)SI - \mu S + \gamma(1-S-I)\right]\Delta$$
$$I_{t+\Delta} = I_t + \left[\beta\phi^2(1-\zeta)SI - \kappa I\right]\Delta$$

FOC:

$$\frac{(1-e^{-\rho\Delta})}{\rho}\frac{(1-\zeta I)}{\phi(1-\zeta I)-(1-c_0)} = e^{-\rho\Delta}[F_S(S_{t+\Delta},I_{t+\Delta}) - F_I(S_{t+\Delta},I_{t+\Delta})]\left[2\beta\phi(1-\zeta)SI\right]\Delta$$

$$\phi[\phi(1-\zeta I)-(1-c_0)] = \frac{(1-e^{-\rho\Delta})}{\Delta\rho e^{-\rho\Delta}}\frac{(1-\zeta I)}{\left[2\beta(1-\zeta)SI\right]\left[F_S(S_{t+\Delta},I_{t+\Delta}) - F_I(S_{t+\Delta},I_{t+\Delta})\right]}$$

$$\phi^2(1-\zeta I) - \phi(1-c_0) - \frac{(1-e^{-\rho\Delta})}{\Delta\rho e^{-\rho\Delta}}\frac{(1-\zeta I)}{\left[2\beta(1-\zeta)SI\right]\left[F_S(S_{t+\Delta},I_{t+\Delta}) - F_I(S_{t+\Delta},I_{t+\Delta})\right]} = 0$$

Define:

$$d \equiv \frac{(1-e^{-\rho\Delta})}{\Delta\rho e^{-\rho\Delta}}\frac{1}{\left[2\beta(1-\zeta)SI\right]\left[F_S(S_{t+\Delta},I_{t+\Delta}) - F_I(S_{t+\Delta},I_{t+\Delta})\right]}$$

$$\phi^2(1-\zeta I) - \phi(1-c_0) - d(1-\zeta I) = 0$$
$$\phi = \frac{(1-c_0) \pm \sqrt{(1-c_0)^2 + 4(1-\zeta I)^2 d}}{2(1-\zeta I)}$$

Given that we want $c \geq (1-c_0)w$, we can ignore the lower root. Thus,

$$\phi = \frac{(1-c_0) + \sqrt{(1-c_0)^2 + 4(1-\zeta I)^2 d}}{2(1-\zeta I)}$$

Phase I

$\mu = 0$

$$V(S_t, I_t) = \max_{(0 \leq \phi \leq 1)} \left\{ \frac{(1-(e^{-(\rho+\eta)\Delta})}{\rho+\eta} \Big(u(\phi w(1-\zeta I) - c_V(\mu S)) - \Delta\left[D_t\right] + \eta F(S_t, I_t) \Big) + e^{-(\rho+\eta)\Delta} V(S_{t+\Delta}, I_{t+\Delta}) \right\}$$
$$S_{t+\Delta} = S_t + \left[-\beta\phi^2(1-\zeta)SI - \mu S + \gamma(1-S-I)\right]\Delta$$
$$I_{t+\Delta} = I_t + \left[\beta\phi^2(1-\zeta)SI - \kappa I\right]\Delta$$

FOC:

$$\phi = \frac{(1-c_0) + \sqrt{(1-c_0)^2 + 4(1-\zeta I)^2 d}}{2(1-\zeta I)}$$

Where,

$$d \equiv \frac{(1-e^{-(\rho+\eta)\Delta})}{\Delta(\rho+\eta)e^{-(\rho+\eta)\Delta}} \frac{1}{\left[2\beta(1-\zeta)SI\right]\left[V_S(S_{t+\Delta}, I_{t+\Delta}) - V_I(S_{t+\Delta}, I_{t+\Delta})\right]}$$

Appendix 3: Calibration

1. **Utility**. We consider log utility. To be precise we assume that

   $$u(\phi wL - c_V(\mu(S+(1-\zeta)I))) = \ln\left[\phi wL - c_V(\mu(S+(1-\zeta)I)) - (1-c_0)w\right].$$

   Since in the steady state there is no vaccination and $\phi = L = 1$, $1 - c_0$ is the fraction of steady state output that captures the minimal level of consumption.

   We assume that $c_0 = 0.4$, and, hence, that output cannot fall below 60% of its steady state value

2. **Vaccination**.

   (a) The *cost of vaccination*:

   $$c_V(\mu(S+(1-\zeta)I)) = c_V^0(\mu(S+(1-\zeta)I))^{1+c_V^1}.$$

   The value of c_V^0 depends on the units in the function u.
   Baseline: $c_V^0 = 0$.

   (b) *Vaccination capacity.* We set $\bar{\mu} = 0.05$

3. **Epidemiological Parameters**.

   (a) The *Fraction* ζ. A difficulty estimating ζ is the lack of random testing at this point and the as-hoc assumptions about mortality that have to be made to produce estimates. Hortacsu et. al. estimate a range for ζ. Their results —based on data prior to the institution of stay-at-home policies in many states in early March 2020— imply that $\zeta \in [0.4, 0.25]$. Li et. al. (2020) using a different approach and relying on Chinese data estimate $\zeta = 0.04$.

   What are the implied fatality rates? Given that the case fatality rate is $\chi\zeta I$, the true fatality rate is $\chi\zeta$. Thus, we need an estimate of χ which corresponds to the ratio of fatalities/diagnosed cases. In the US at the time of this writing, the measured death rate is about 5.3%, while for the world as a whole it exceeds 6%. At the same time there is a large number of countries —including many European countries— in which the case fatality rate is below 5%. Thus, it seems that $\chi = 0.05$ is a reasonable estimate?

The implied fatality rate is $0.05 \times \zeta$. If we assume that $\zeta = 0.1$ the fatality rate is 0.5%, which is in the range of estimates. The lower bound of the estimates of ζ (around 0.04) implied a fatality rate of 0.2% which is slightly higher than the influenza fatality rate. The upper bound of the estimates ($\zeta = 0.2$) implies a true fatality rate equal to 1%.

Baseline: $\zeta = 0.1$

Alternative: $\zeta = 0.2$

(b) *The Recovery Rate* κ. If on average individuals exit the infected category (either resistant or deceased) recover in 3 weeks, then $\kappa = 1/3 = 0.33$

(c) *The Gross Transmission Rate* β. We view estimates of $\mathcal{R}_0$ as more reliable than estimates of β. Our strategy is to use estimates of $\mathcal{R}_0$ to estimate β. In our base case $\mathcal{R}_0 = 2.8$. Then given

$$\frac{\beta(1-\zeta)}{\kappa} = \mathcal{R}_0,$$

we estimate β as

$$\beta = \frac{\kappa \times \mathcal{R}_0}{1-\zeta} = \frac{0.16 \times 2.8}{0.9} = 0.497.$$

There is significant uncertainty about the relevant value of $\mathcal{R}_0$. Many studies put the range of $\mathcal{R}_0$ between 1.5 and 4.0. A recent study by Fernandez-Villaverde and Jones (2020) that matches the evidence with the SIR model —but that imposes an arbitrary sequence ϕ_t— estimates that $\mathcal{R}_0 = 4.2$ and even higher in some European countries.

Baseline: $\beta = 0.497$

Alternative: $\beta = 0.71$

4. **Economic/Institutional Parameters**.

(a) *Output per worker*. Our unit of analysis is an individual. We assume that there are 328×10^6 individuals, and GDP of 20 trillion/year. Thus, output per worker per week is 1,173

$$w = 1,173.$$

(b) *Discount factor.* We assume that the annual discount factor ρ is somewhere between 1 and 3%. The base case (for sentimental reasons) is $\rho = 0.0122$ on an annual basis. Since the model is weekly we have that

$$\rho = 0.000233.$$

This value has the "property" that the present discounted value of weekly output of the average worker (who earns twice as much as the average person since only 50% of us work) satisfies

$$\frac{2,331}{0.000233} = 10,000,000$$

which is not an unreasonable number.

5. **The Δ function.**

(a) *Estimation of M_0.* Since the constant is given by

$$\ln\left(\upsilon w \phi(1-\zeta I) - \underline{c}\right) \frac{1-e^{-\rho T}}{\rho},$$

and we assume that $\upsilon = 3$ which is standard in the literature. We also take $\phi = 0.8$ and $I = 0.1$ but our estimate is not very sensitive to changes in these two parameters. We find that

$$M_0 = 7.75 \times 489 = 3790$$

(b) *Estimation of $\bar{H}$.* The U.S. has about 1,000,000 hospital beds (actually a little less than that). A reasonable estimate is that no more than 20% are ICU beds. If we assume (rather generously) that 70% of the capacity will be available for COVID-19 patients this gives 140,000 ICU beds. Then availability per worker is

$$\frac{140,000}{328,000,000} = 0.000427$$

Thus, our (somewhat optimistic) estimate of $\bar{H}$ is 0.000427.

A pessimistic estimate uses the actual fraction of ICU beds which is about 14% and assumes a 50% availability rate. Thus the pessimistic estimate is $\bar{H} = 0.000214$.

Baseline: $\bar{H} = 0.000427$.

Alternative: $\bar{H} = 0.000214$.

6. **Definition:** $\mathcal{R}_t$

$$\mathcal{R}_t = \frac{\beta(1-\zeta)\phi_t^2 S_t}{\kappa}.$$

7. Next we pick γ. Fix the steady state I^*, then (modulo algebraic error) I get that

$$\gamma = \frac{\kappa I^*}{(1 - I^* - \frac{1}{\mathcal{R}_0})}.$$

If $I^* = 0.001$ then I get that $\gamma = .00025$.

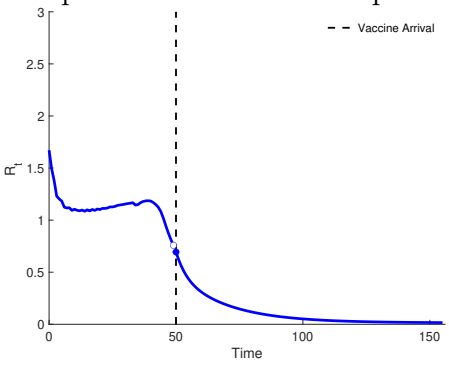
Table 1: Baseline Parameters

Meaning	Parameter	Value
Fraction Diagnosed among Infected	ζ	0.1
Immunity Loss Rate	γ	0.00025
Basic Reproduction Number	R_0	2.8
Recovery Rate	κ	0.33
Discount Rate	ρ	0.000233
Time Step	Δ	1/15
Loss function	M_0	3797
Output per Worker	w	1173
Case Fatality Rate	χ	0.05
Hospital Capacity	$\bar{H}$	0.000427
Vaccine Cost	c_v	0
Vaccination capacity	$\bar{\mu}$	0.05
Minimum Consumption (1-c_0)	c_0	0.60
Vaccine Arrival Poisson	η	1/50
Initial I_0	I_0	0.01
Initial S_0	S_0	0.99

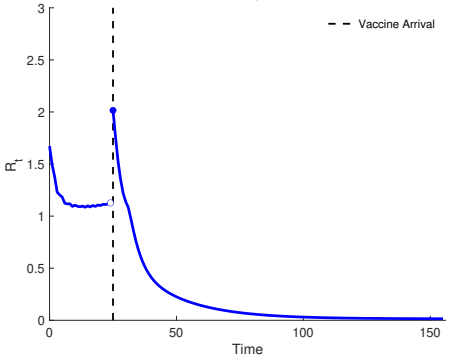
Appendix 4: Supplementary Graphs

Implied Contagion Factor $\mathcal{R}_t$

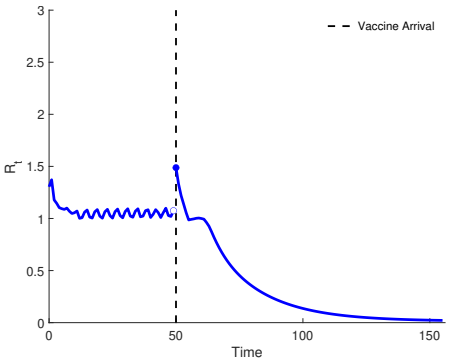
Here we present the model's implications for measured $\mathcal{R}_t$ in several cases.



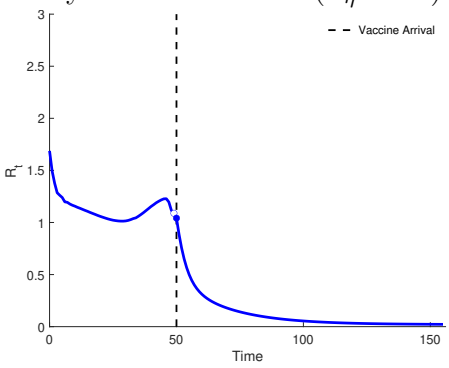
Baseline



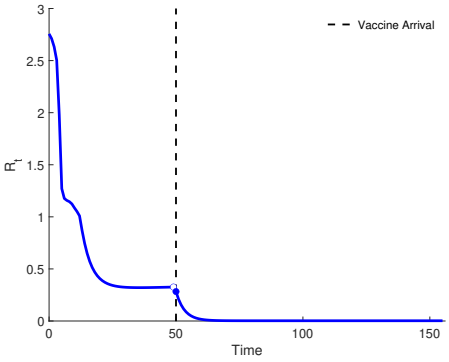
Early Arrival Vaccine ($T_\eta = 25$)



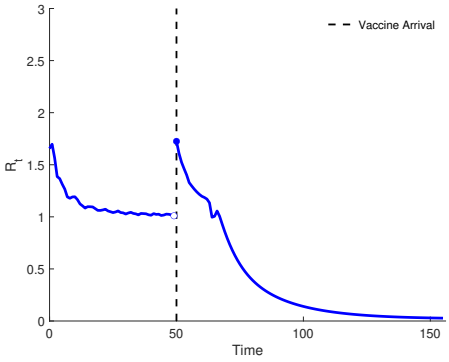
All Lives Matter



$\mathcal{R}_0 = 4$



Optimistic Scenario

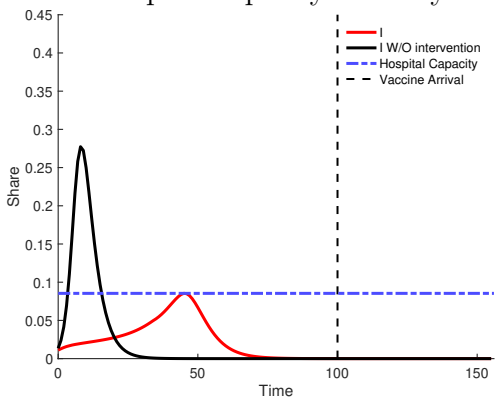


Pessimistic Scenario

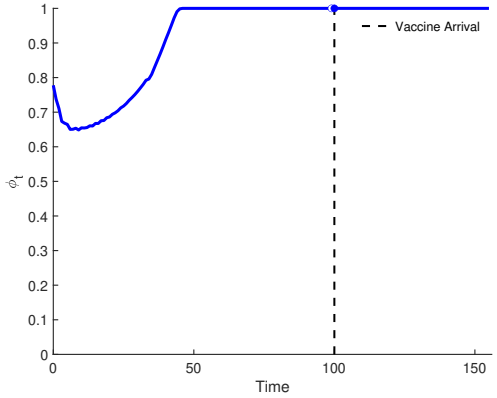
Time Path for $\mathcal{R}_t$

Late Arrival of a Vaccine: Time Paths

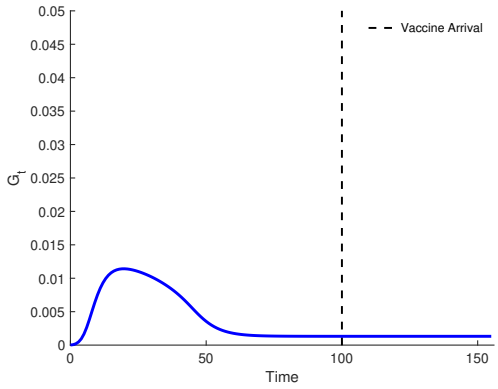
This realization assumes that a vaccine arrives after 75 weeks. The results about the optimal policy are very similar to the baseline.



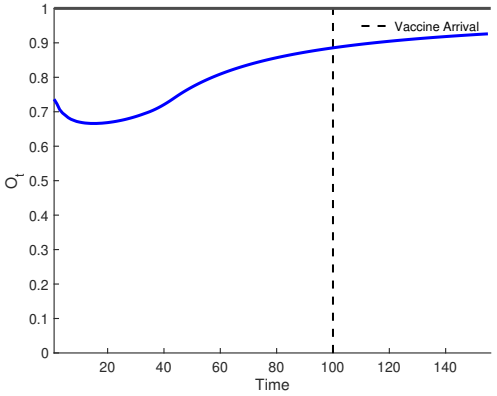
(a) Path of I



(b) Optimal ϕ



(c) Deaths Averted

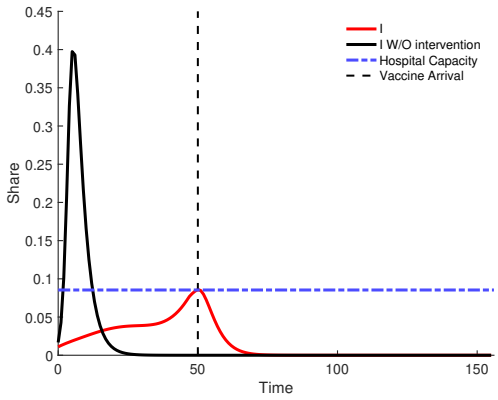


(d) Output Loss

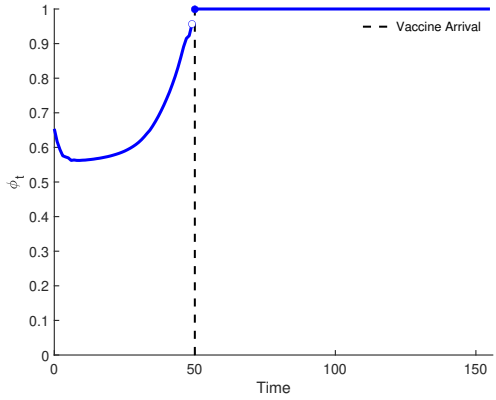
Figure 7: Early Arrival of a Vaccine ($T_\eta = 25$)

Higher Infectiousness

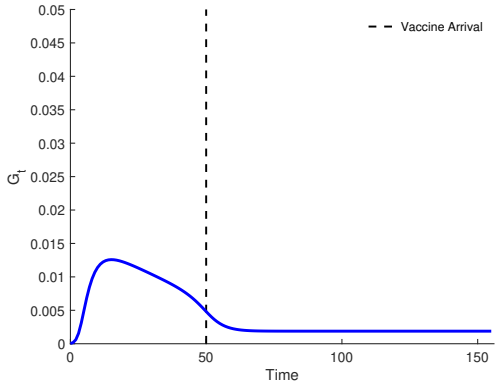
Our baseline assumes that $\mathcal{R}_0$ is 2.8 but there is not clear consensus in the epidemiology literature about the correct value. Some recent estimates (see Fernandez-Villaverde and Jones (2020)) suggest that $\mathcal{R}_0$ is substantially higher. Figure 8 reports the simulation (with $T_\eta = 50$) if the epidemiological parameter $\mathcal{R}_0$ is 4.0.



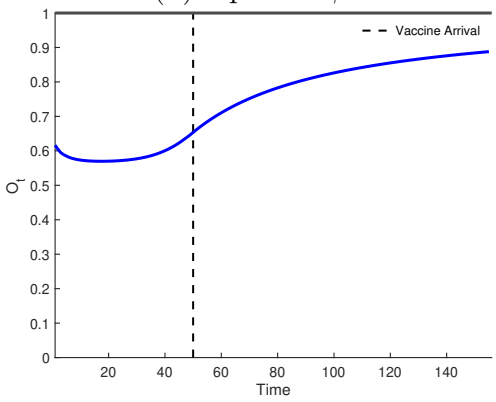
(a) Path of I



(b) Optimal ϕ



(c) Deaths Averted

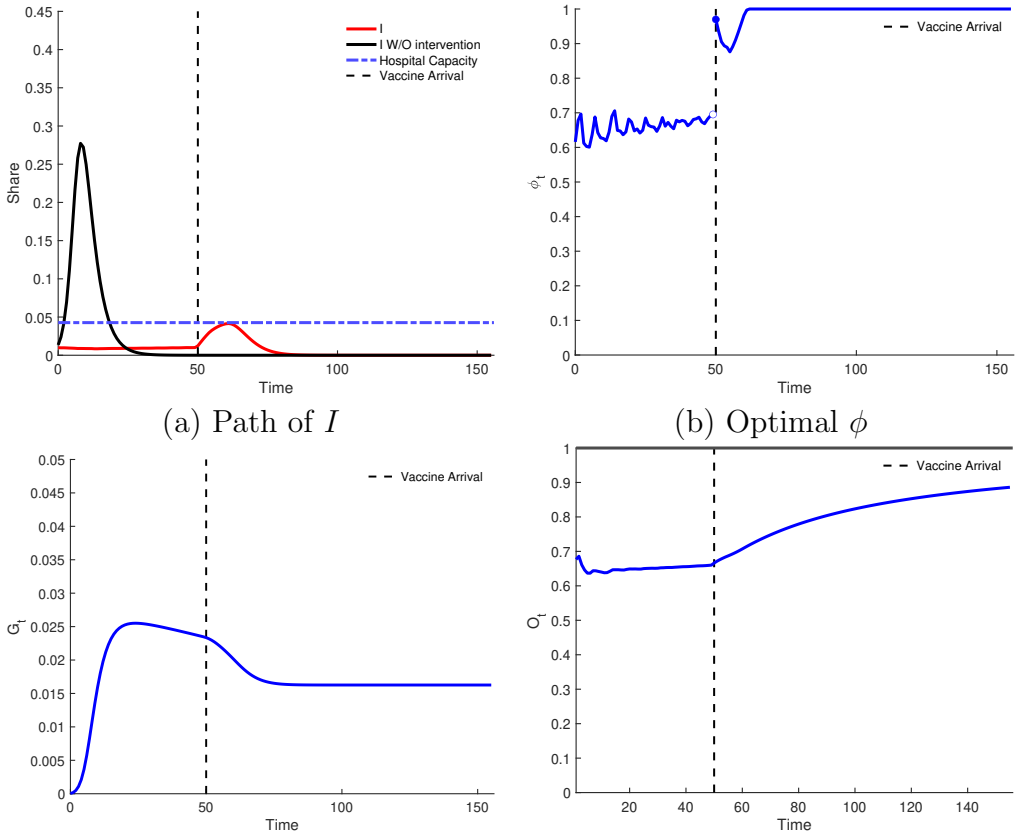


(d) Output Loss

Figure 8: Higher Infectiousness ($\mathcal{R}_0 = 4$)

Higher ζ

Basic data if $\zeta = 0.20$.



(a) Path of I (b) Optimal ϕ

(c) Deaths Averted (d) Output Loss

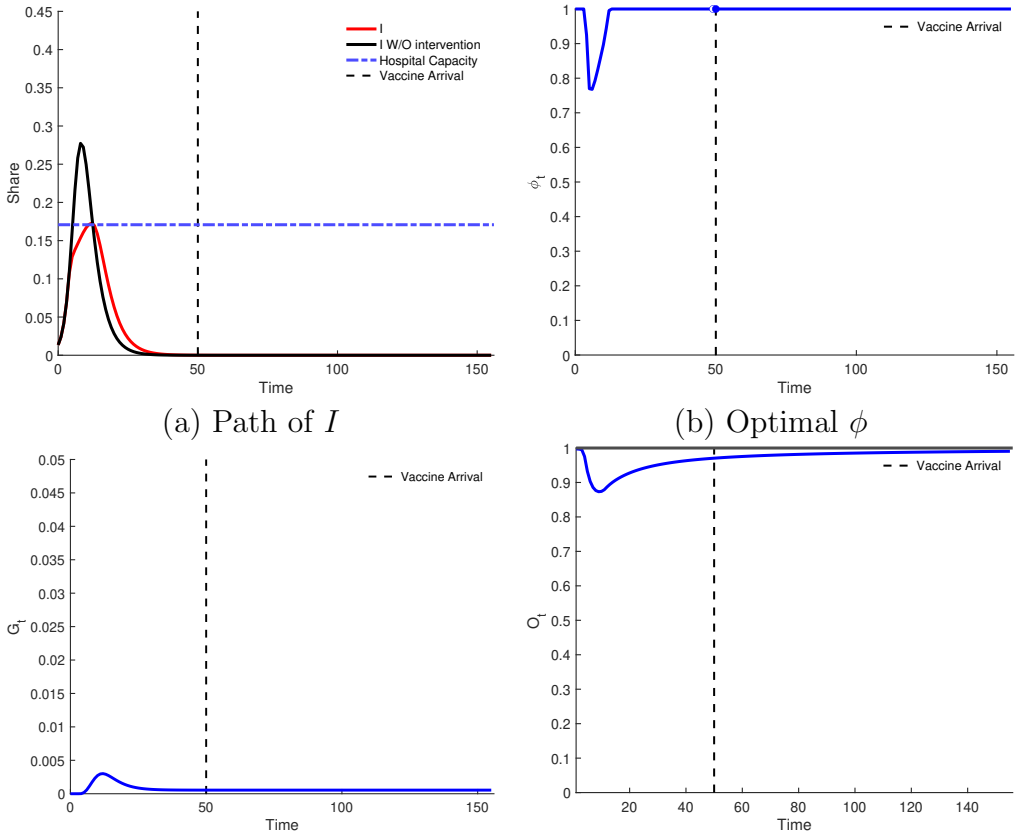
Lower Asymptomatic Cases ($\zeta = 0.2$)

Optimistic Scenario: Time Paths

In this scenario we assume that the upper bound of the vaccination rate is six times as high as in the baseline which implies about 30% of the vaccinable population[1] is vaccinated in one week. [2] We also assume that the expected time until a vaccine arrives is 24 weeks (50 in the baseline) and the actual hospital capacity is twice the value in the baseline (280,000 ICU beds for COVID-19 patients). To keep realizations comparable to the baseline, we still look at the case in which the vaccine arrives in week 50.

[1] This includes susceptibles and asymptomatic infectious.

[2] Note that this is also the proportion of all people that have to be vaccinated if it is not possible to determine who are the susceptible.



(a) Path of I (b) Optimal ϕ

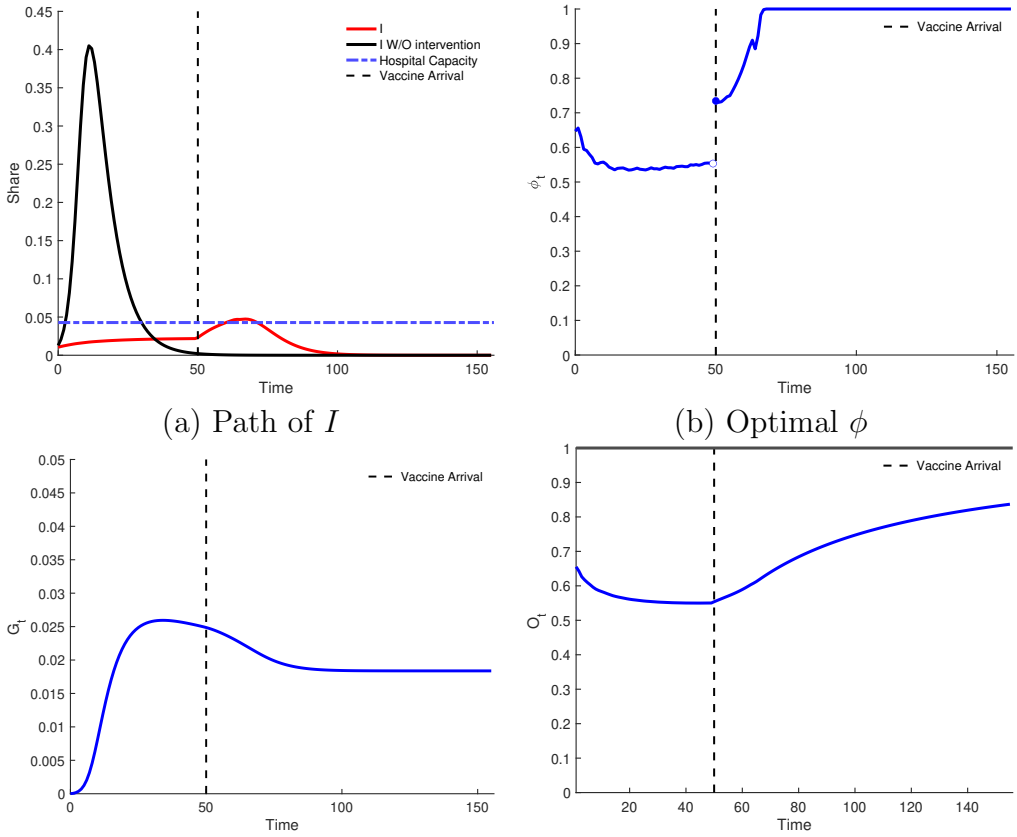
(c) Deaths Averted (d) Output Loss

Figure 9: Optimistic Scenario

Pessimistic Scenario - I: Time Paths

In this case we assumed that the hospital capacity rate is 1/2 of our baseline (about 70,000 ICU beds for COVID-19) and that the vaccine capacity is 1/3 of the baseline ($\bar{\mu} = 0.01$). We also assume that the expected time until a vaccine is available is 156 weeks (twice the baseline) and that the infectiousness is higher ($\mathcal{R}_0$ is 4.0). However, we still assume that $T_\eta = 50$.[3]

[3]In Appendix 5 we present an alternative pessimistic scenario. There we assume that $\mathcal{R}_0 = 4$ and that hospital capacity is half of the baseline.



(a) Path of I

(b) Optimal ϕ

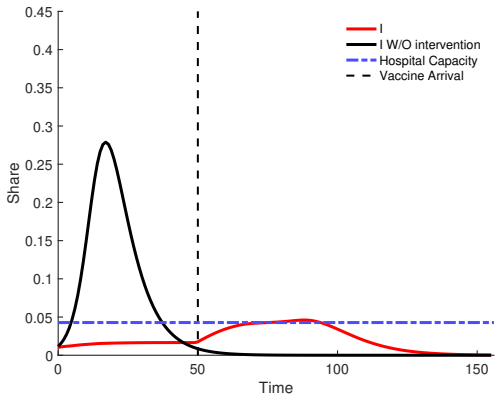
(c) Deaths Averted

(d) Output Loss

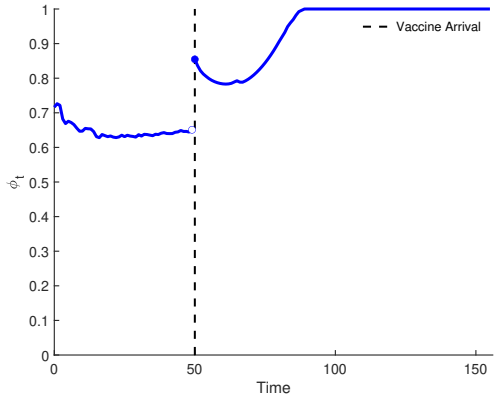
Figure 10: Pessimistic Scenario

Pessimistic Scenario - II: Time Paths

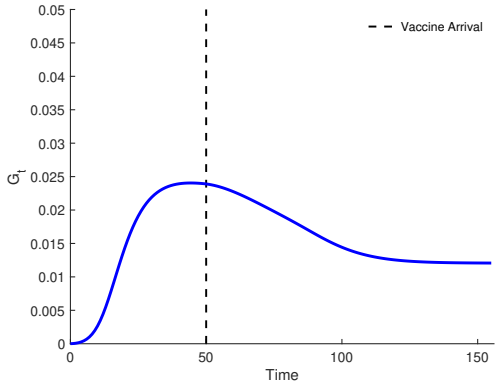
Assumes $\mathcal{R}_0 = 4$ and $\bar{H}' = (1/2)\bar{H}$



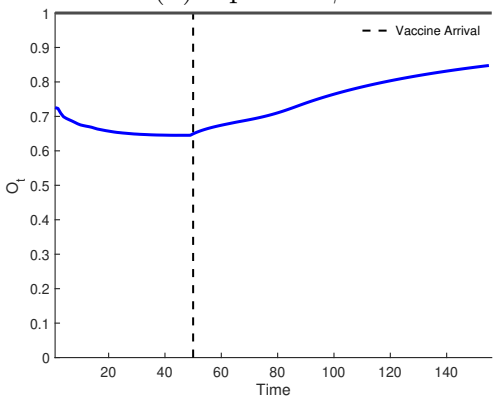
(a) Path of I



(b) Optimal ϕ



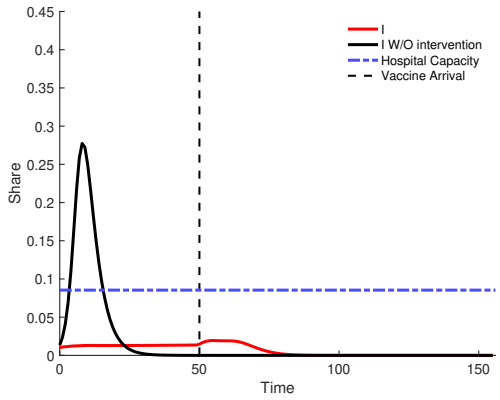
(c) Deaths Averted



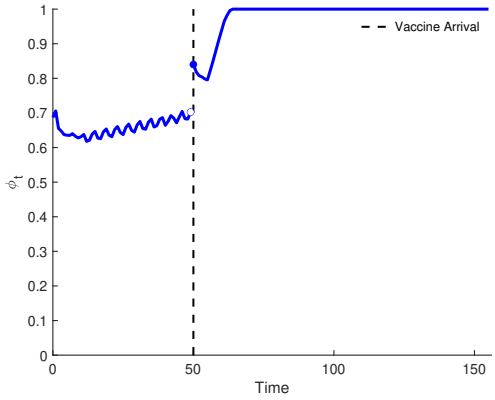
(d) Output Loss

Lower Asymptomatic Cases ($\zeta = 0.2$)

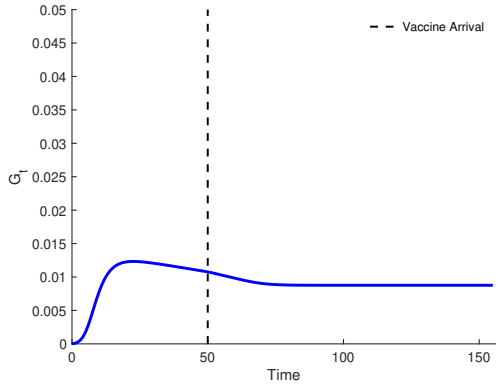
All Lives Matter: Time Paths



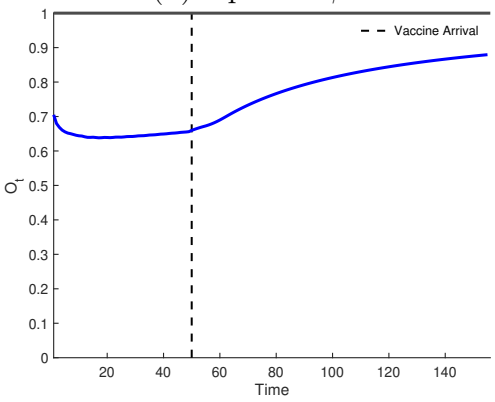
(a) Path of I



(b) Optimal ϕ



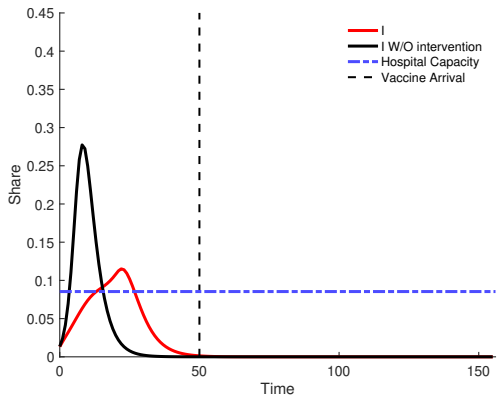
(c) Deaths Averted



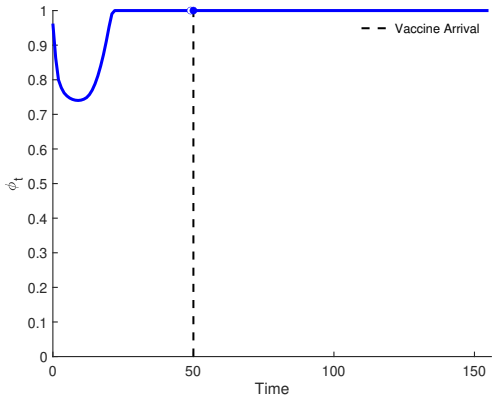
(d) Output Loss

All Lives Valued Equally

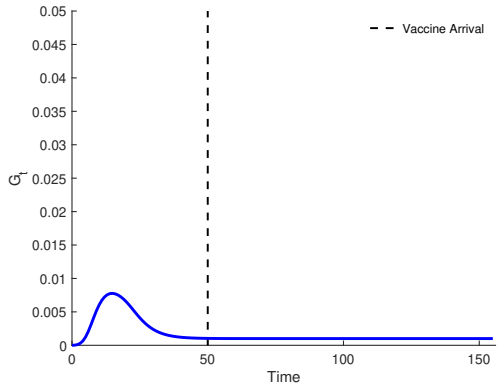
Lower Value of Consumption: Time Paths



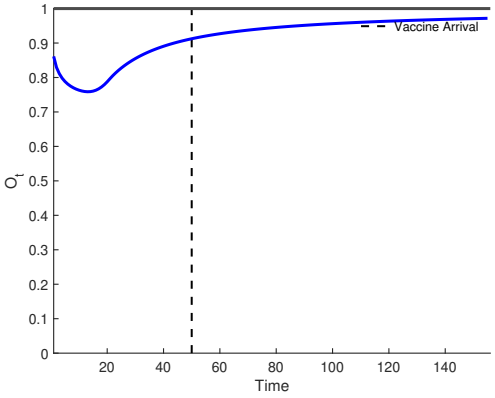
(a) Path of I



(b) Optimal ϕ



(c) Deaths Averted



(d) Output Lost

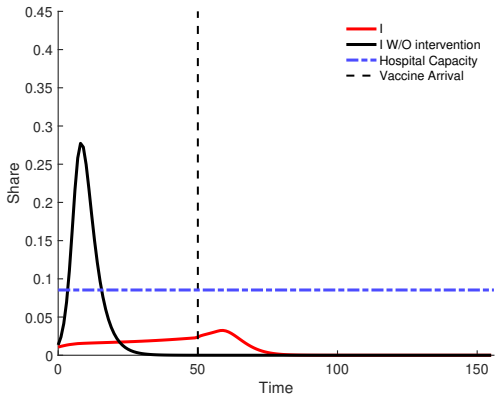
Low Consumption Multiplier

Intermediate Social Valuation of Deaths Averted

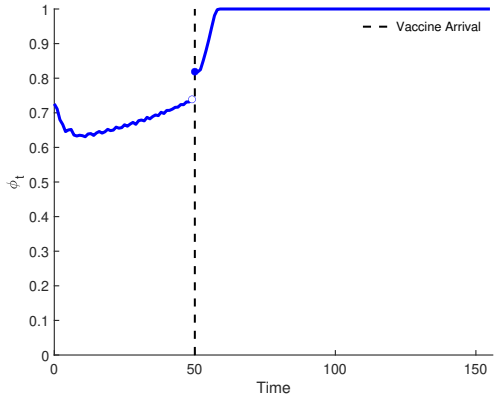
Our valuation of deaths averted function is given by

$$\Delta = M_0 \left[k \min\{D, \bar{H}\} + \max\{D - \bar{H}, 0\} \right].$$

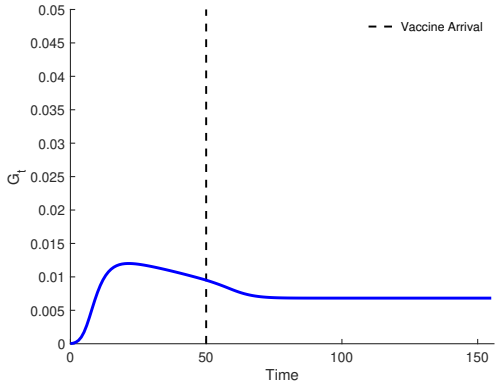
Our baseline assumes that $k = 0$ which implies that only excess deaths enter the social utility function. Our "Higher Social Value of Life" scenario assumes that $k = 1$ which implies that all averted deaths are counted equally. Here we show the intermediate case $k = 0.5$ that counts all deaths but puts a higher weight on excess deaths.



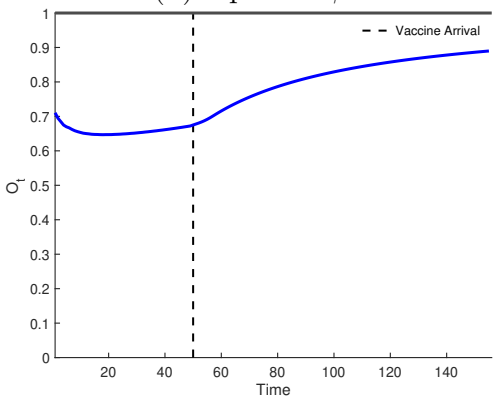
(a) Path of I



(b) Optimal ϕ



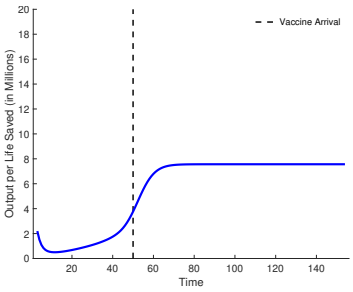
(c) Deaths Averted



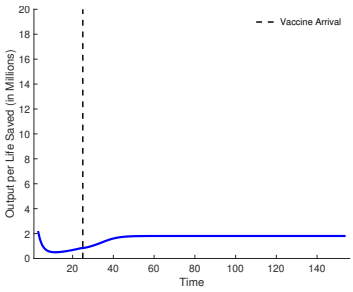
(d) Output Loss

Intermediate Social Valuation of Deaths Averted ($k = 0.5$)

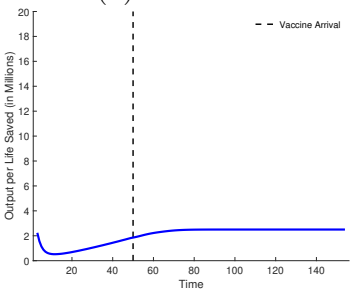
Output Cost per Death Averted



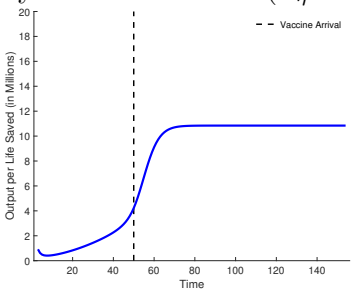
(a) Baseline



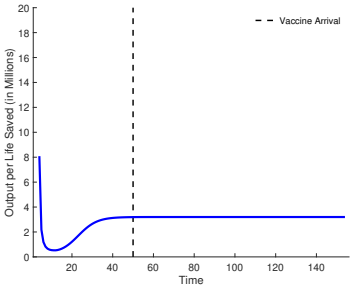
Early Arrival Vaccine ($T_\eta = 25$)



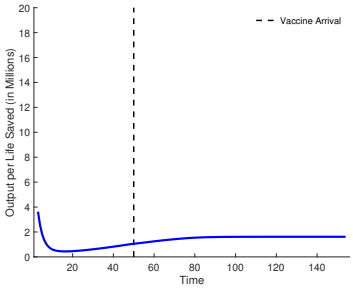
All Lives Matter



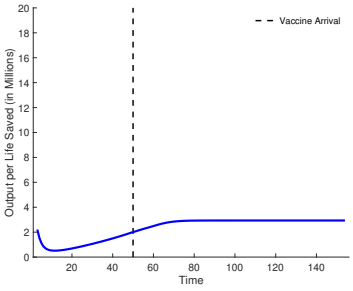
$\mathcal{R}_0 = 4$



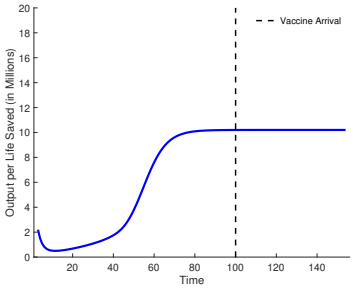
Optimistic Scenario



Pessimistic Scenario



$k = 0.5$



Late Arrival Vaccine

Appendix 5: Literature Review

We are fully aware that this summary is incomplete and that closely related research is probably missing. However, we still want to acknowledge some of the recent work in the topic, and found useful to group difference papers in different categories, with the obvious caveat that these are imperfect and that there exists a substantial amount of overlap in the existing work.

Optimal management of the epidemic in SIR models: These papers use optimal control techniques to explore the management of an epidemic. See Alvarez, Argente, and Lippi (2020), Gonzalez and Niepelt (2020), Acemoglu, Chernozhukov, Werning, and Whinston (2020), Jones, Philippon, and Venkateswaran (2020).

Policies in an SIR models: These papers explore the effects of different policies in the dynamics of the pandemic (i.e. social distancing, lockdown, etc...) in SIR models developed by Kermack and McKendrick (1927). See for example, Atkeson (2020), Berger, Herkenhoff, and Mongey (2020), Neumeyer (2020), Bassetto (2020), Droz and Tavares (2020), Hsiang et al. (2020), Fang, Wang, and Yang (2020), Shao (2020), Wang et al. (2020). Avery, Bossert, Clark, Ellison, and Ellison (2020), Farboodi, Jarosch, and Shimer (2020), Aum, Lee, and Shin (2020). Glover, Heathcote, Krueger, and Ríos-Rull (2020), Pindyck (2020), Chang and Velasco (2020).

Measurement issues: These papers discuss measument issues in the data or key parameters on SIR model (Stock 2020, Korolev, 2020, Kubinec, 2020, Manski and Molinari 2020, Fernández-Villaverde and Jones 2020, Hortacsu, Liu and Schwieg 2020, Harris 2020, Blavin and Arnos, 2020).

Valuation of life and death: These papers use different approaches to measure the valuation of life and deaths based on the observed policies (i.e. Greenstone and Nigam 2020), or try to assess the maximum level of consumption drop necessary to avoid the deaths associated with the covid-19 (Hall, Jones, and Klenow 2020). Early papers included Murphy and Topel (2006).

Macroeconomic implications of epidemics: Some of the research provides a historical perspective by analyzing the economic implications of past pandemics as Barro, Ursúa, and Weng (2020), Correia, Luck, and Verner (2020), Velde (2020). A number of papers explores macro implications of epidemic shocks and policy interventions. See Eichenbaum, Rebelo, and Trabandt (2020a,b), Fornaro and Wolf (2020), Bairoliya and Imrohoroglu (2020), Krüger, Uhlig, and Xie (2020), Kozlowski, Veldkamp, Venkateswaran (2020),

Bodenstein, Corsetti, and Guerreri (2020), Guerrieri, Lorenzoni, Straub, and Werning (2020), Faria-e-Castro (2020), Caballero and Simpsek (2020), as well as implications for different markets (i.e. labor market Bick and Blandin 2020, Kapicka and Rupert 2020, Dingel and Neiman 2020, Kurman, Lale, and Ta 2020, stock market i.e. Alfaro, Chari, Greenland, and Schott 2020, Baker, Bloom, Davis, Kost, Sammon, and Viratyosin 2020, Gormsen, N. and Koijen 2020).