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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 relaxation 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. The ultimate goal is to study a multi-good multi-region economy model to capture the interdependencies across geographic and sectoral lines to better understand the consequences of policies that treat sectors and regions in a heterogeneous manner in the context of a highly contagious 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 decreases (less contagion) and hence the marginal cost in terms of output should decrease as well. This last step requires a liberalization (more contact among individuals) that, in turn, that pushes up the contagion rate.

We assume that individual preferences depend on individual consumption—ignoring the private value of life— and that social preferences take into ac-

¹Acemoglu et. al. study optimal lockdown for heterogenous 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 on special cases in order to find closed form solutions.

count the utility loss associated with deaths. We discuss different approaches to valuing life and 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 assume that this is a random event and we take the probability of a vaccine arriving at a given time 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 —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. Policies based on infections (or deaths) are suboptimal. Implementation of the optimal policy requires random testing.²

²The case for random testing has been made by many. Among the economists a good

2. In most of our simulations the optimal stay-at-home policy in Phase I (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. It 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 curve lowers consumption at any point but 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.

discussion is in Chari and Phelan (2020).

- (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 testing³. 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 discuss some theoretical results. Section 3 describes the one region model. In section 4 we present our quantitative findings. Section 5 briefly discusses work on ongoing 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. This variable already highlights the tradeoff between current utility and the value of having fewer infections in

³However, 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 too far off the actual values.

the future. The second policy that we consider is the rate at which individuals can be vaccinated when a vaccine becomes available. This 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 Poisson 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 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 in 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

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

and we make standard assumption about the utility function u . We take L as a measure of the available labor supply. In the simple model this is equal to 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

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

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

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

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

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

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

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, v , of annual output, then we can approximate the utility loss associated with one death is

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

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 $\hat{L} = 1$. 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 baseline⁴. 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.⁵ The implication is that the extra cost to society is associated with deaths that potentially could have been prevented if hospital capacity was higher. 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.

⁴It is not straightforward to assume 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 one of our robustness exercises we report the results corresponding to the case in which all deaths are valued equally

⁵We 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.

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.⁶ Here, we 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

$$L = S + R + (1 - \zeta)I. \quad (2)$$

Since we normalized the population to one this is

$$L = 1 - \zeta I. \quad (3)$$

Then the fraction of susceptibles and infectious in the population is ϕS and $\phi(1 - \zeta)I$.⁷

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

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

⁶One 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 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)

⁷In 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)

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

The stock of infectious evolves according to

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

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) it is possible and desirable using a more disaggregate 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

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

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

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

By comparing G_T and O_T we can estimate the output cost per death averted.⁸

⁸It 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

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

subject to equations (4) and (5) and $S_0 = S$ and $I_0 = I$ and subject 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.⁹

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)

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

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. The sign of the other derivative, F_S , is not determined (although we show it is negative at the steady state) However, in all cases, $F_S - F_I > 0$ since $F_I < F_S$ 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

⁹Although 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

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

Proof. See Appendix ■

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 (Poisson rate) η . The planner's problem is

$$V(S, I) = \max E \left[\int_0^{T_\eta} e^{-\rho t} [u(\phi_t w L_t) - \Delta(D_t)] dt + e^{-\rho T_\eta} F(S_{T_\eta}, I_{T_\eta}) \right],$$

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

The key difference 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 $t \rightarrow \infty$ 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 ■

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 $t \rightarrow \infty$. 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 2. Here, we describe the more significant assumptions underlying our baseline case:

- \mathcal{R}_0 is 2.8. We also report results in the case $\mathcal{R}_0 = 4$.
- 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 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)¹⁰. 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 $\lim_{t \rightarrow \infty} \mu_t = 0$).¹¹

¹⁰This is $\eta = 1/50$.

¹¹We 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 4)

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

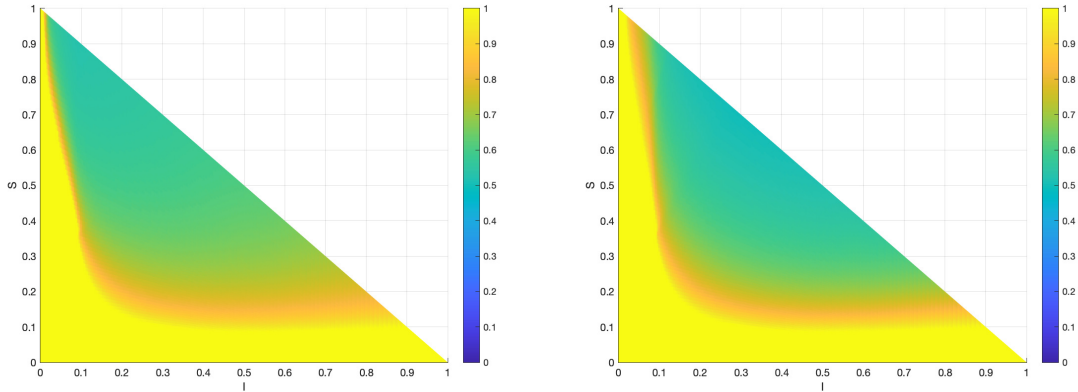


Figure 1. Optimal Policy (ϕ) in Phase I Figure 2. Optimal Policy (ϕ) in Phase II

As expected, for low levels of I , the optimal policy calls for no intervention (the yellow area corresponds to $\phi = 1$). Comparing the optimal policy in the two phases there are two important observations:

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. 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 populations has developed immunity.
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).

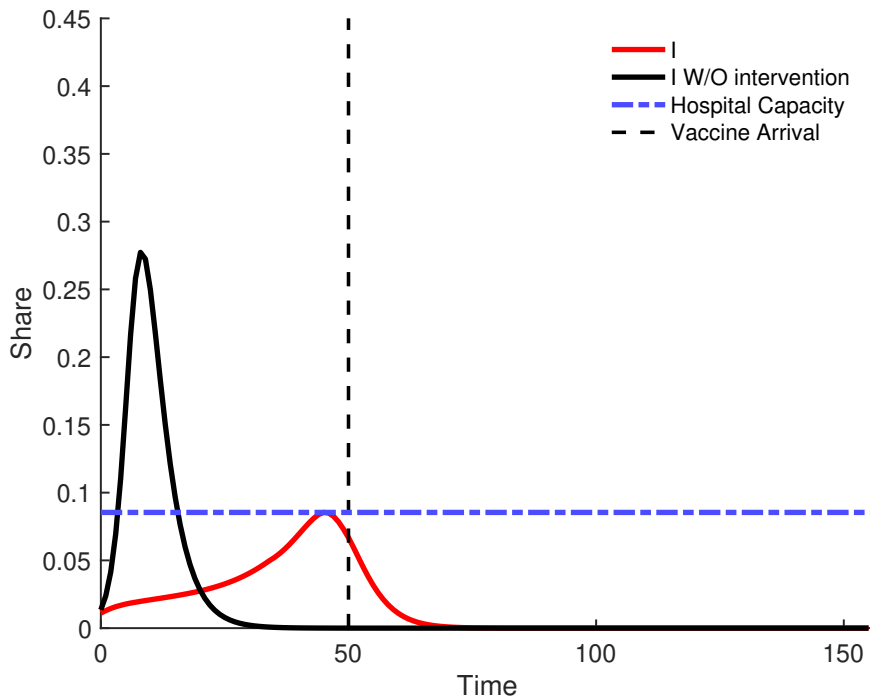


Figure 3. Time Path of I_t : Uncontrolled and Optimal.

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 .

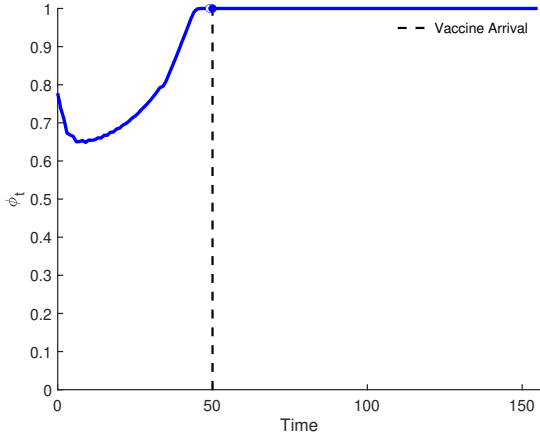


Figure 4. Optimal Policy (ϕ)

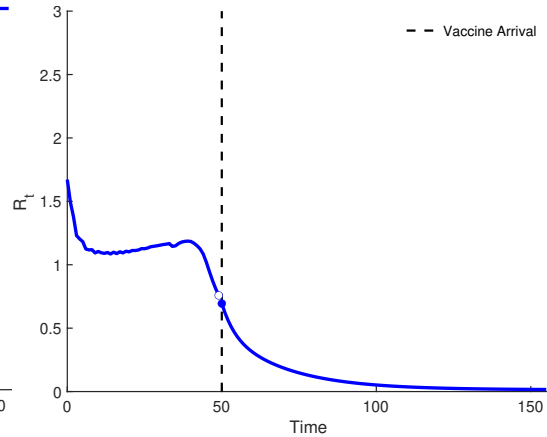


Figure 5. Optimally Managed \mathcal{R}_t

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.¹²

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.

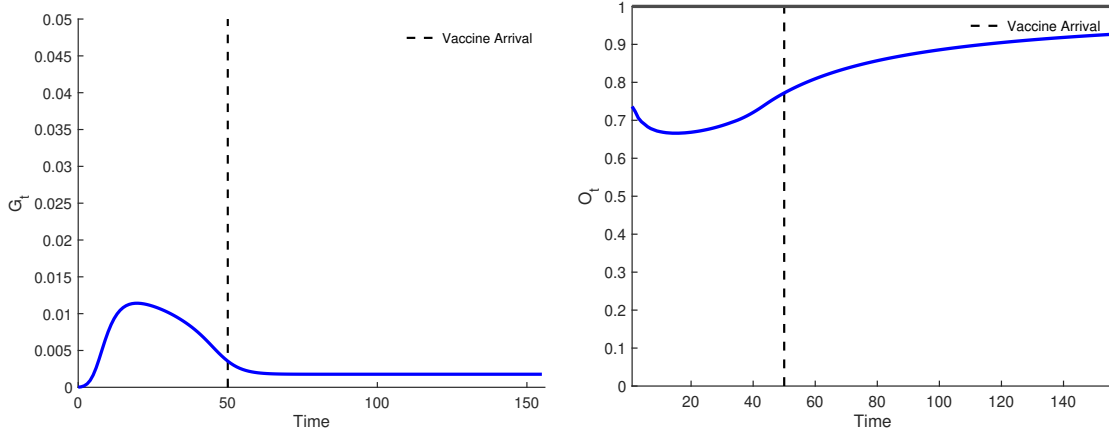


Figure 6. Deaths Averted (left panel) and Output Loss (right panel)

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 lives saved decreases. The reason is simple: under the optimal policy flattening the curve has no impact (under our assumptions) on the true fatality rate of the epidemic 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 0.17%.

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 smaller than what is found in other studies. In the long-run (about 150 weeks)

¹²We present the time path for \mathcal{R}_t for several cases in Appendix 5.

the output loss average over that 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.¹³

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

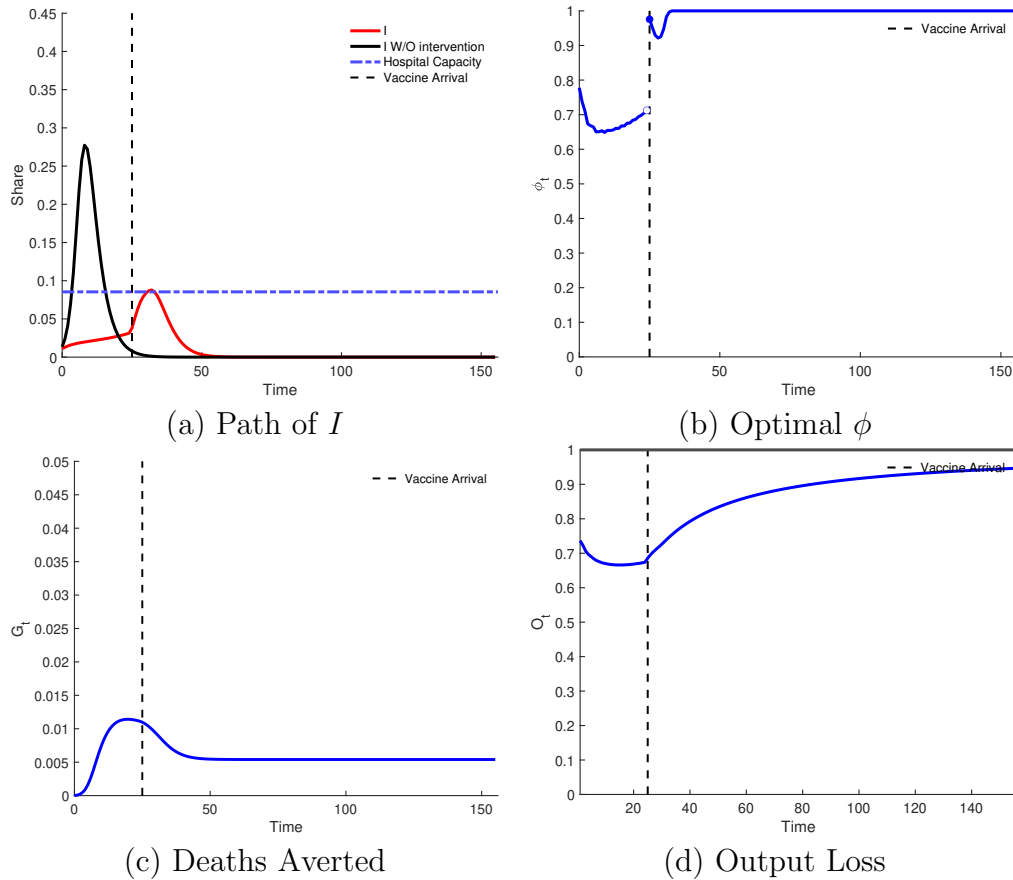


Figure 7: Early Arrival of a Vaccine ($T_\eta = 25$)

¹³In Appendix 5 we present the time path of the output cost per death averted for a variety of scenarios

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 **increases 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 0.18%) 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.

In Appendix 5 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:

- Higher \mathcal{R}_0 .
- “Optimistic” and “Pessimistic” scenarios that simultaneously change several parameters.

- Alternative valuation of human life.¹⁴

4.3.1 Higher \mathcal{R}_0

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.

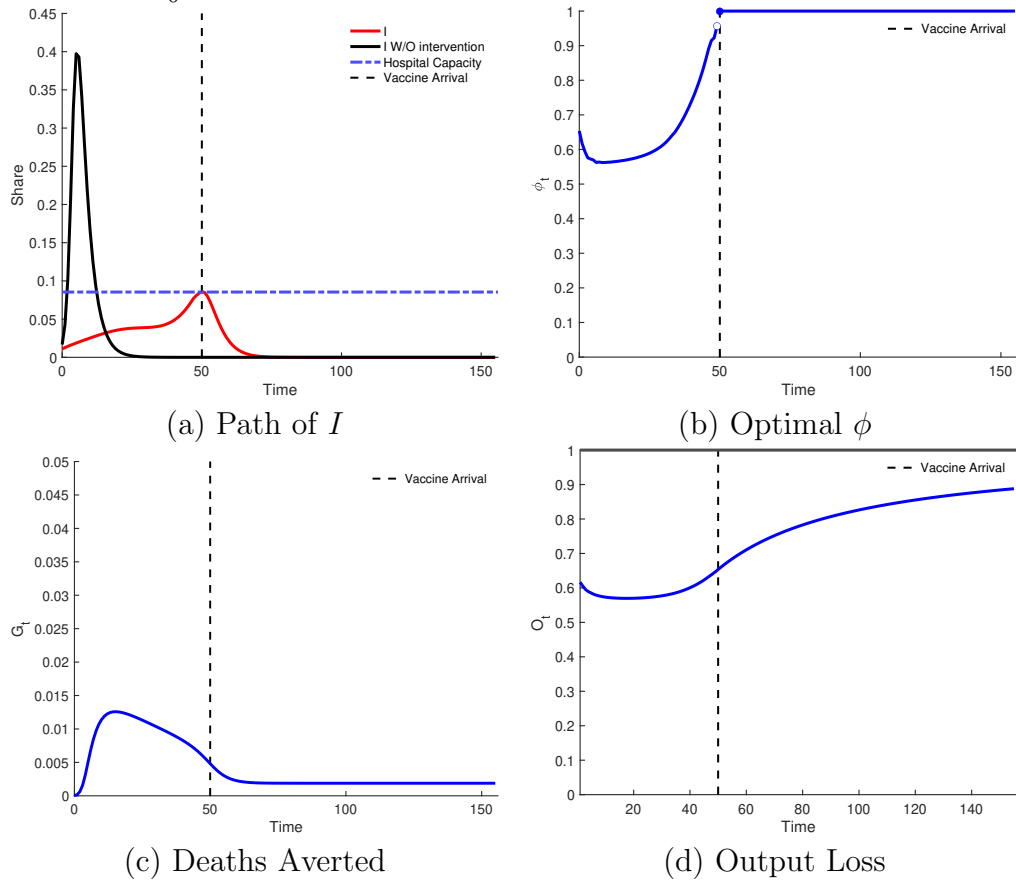


Figure 8: Higher Infectiousness ($\mathcal{R}_0 = 4$)

The most important differences with the baseline are:

1. In this case the uncontrolled epidemic moves faster and the number of infectious individuals peaks around week 5, and it is basically zero

¹⁴Appendix 5 also includes the results from assuming that $\zeta = 0.2$ (instead of $\zeta = 0.1$).

after 35 weeks. This is a deadly but short lived epidemic.

2. The optimal policy flattens the curve and is significantly more restrictive than in the baseline. As in the baseline, the path of ϕ displays a partial and smooth liberalization —which basically keeps the \mathcal{R}_t slightly above one— and it displays a small upward jump when the vaccine becomes available.¹⁵
3. In the long run, the number of deaths averted is slightly higher than in the baseline (0.19% vs. 0.17% of the population) and the loss of output over the three year period is over 11%. This implies a very high cost per death averted: 10.8 million.

Although there is no firm consensus, there is some very limited evidence that suggests that the value of \mathcal{R}_0 is not independent of features of a region such that the type of economic activity, the population density (urban vs. rural) and the age distribution of the population. If this is the case, our results suggest that centralized policies of the type one size fits all are bound to be suboptimal. High \mathcal{R}_0 regions should implement more restrictive policies and, in expectation, will bear higher cost per death averted.

4.3.2 Optimistic Scenario

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 vaccinateable population¹⁶ is vaccinated in one week.¹⁷ 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.

¹⁵If the vaccine arrives early (week 25), then employment —as a fraction of full employment— jumps from 59% to 80% but it takes over two months after the arrival of the vaccine to eliminate all restrictions on employment.

¹⁶This includes susceptibles and asymptomatic infectious.

¹⁷Note 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.

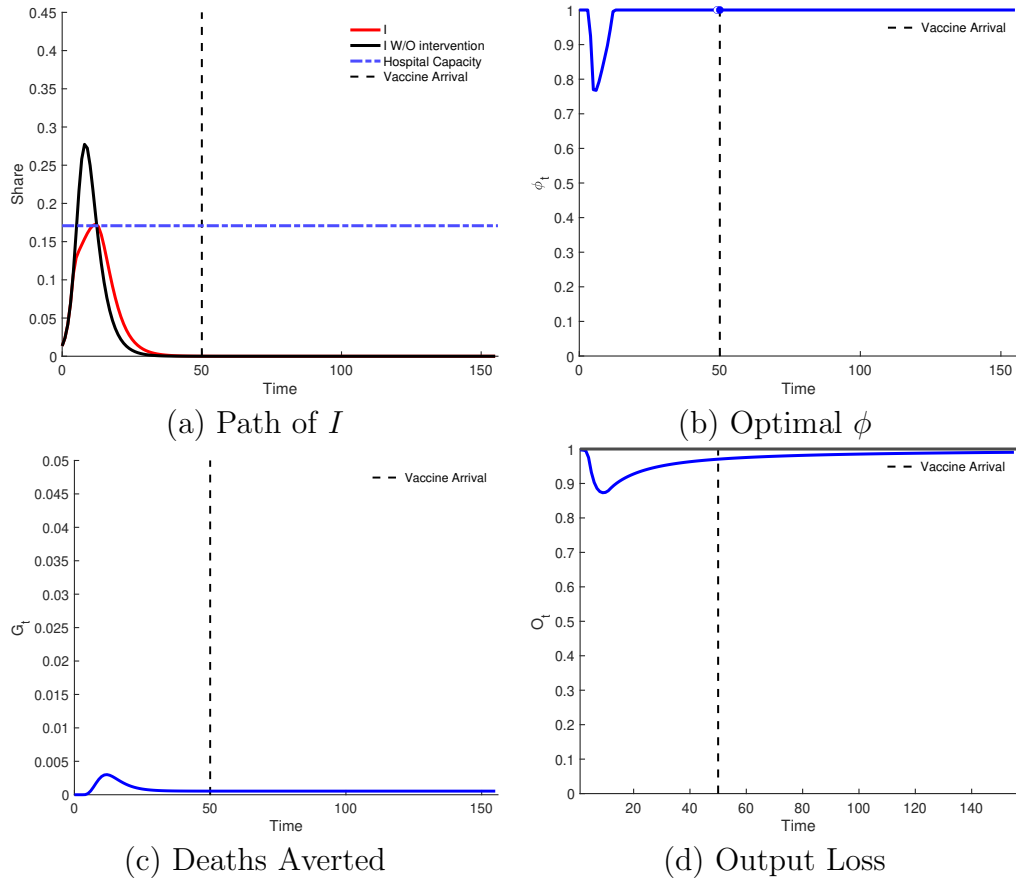


Figure 9: Optimistic Scenario

The main takeaways from this alternative are:

1. The optimal policy does not respond until the virus spreads significantly in the population (in our case this is when 10% have been infected, which occurs in week 4). At that point there is a large decrease in employment (output is about 77% of capacity) that is short lived. By week 12 the economy is at full capacity.
2. The number of deaths averted is about 1/3 the level of the baseline and the cost per death averted is about 3.2 million, a slightly less than half of the baseline.

4.3.3 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-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). However, we still assume that $T_\eta = 50$.¹⁸

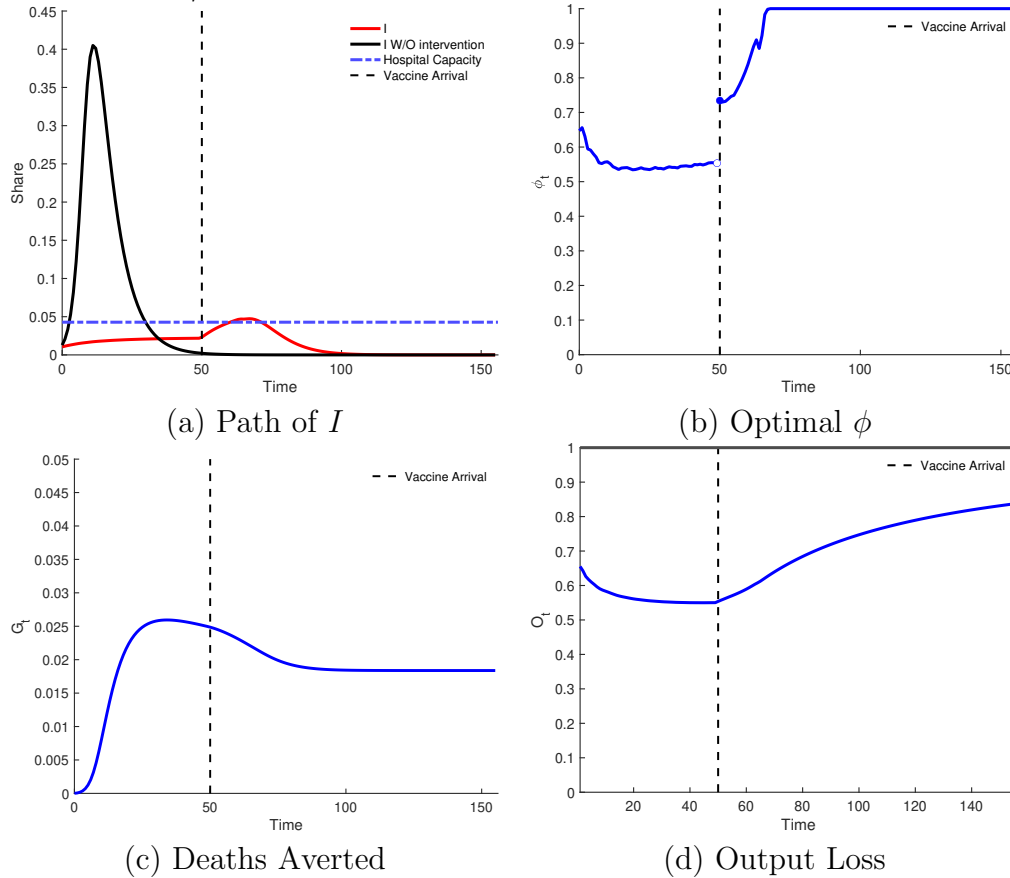


Figure 10: Pessimistic Scenario

In this case:

1. The epidemic peaks much later relative to the baseline and there are more excess deaths.
2. The optimal policy is more restrictive in Phase I but quite different in

¹⁸In 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.

Phase II. In particular, the arrival of a vaccine results in an upward jump in employment but the level is significantly below full employment (73%) and it takes the economy about 16 months to get output back to normal.

3. In this pessimistic scenario, the restrictive policy results in a large number of deaths averted (more than 10 times the baseline) and a relatively small output cost per death averted (1.6 million). However, there is a large welfare loss since over a three year period output more than 16% below capacity.

4.3.4 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 $v = 3$, but we report other values below.

All Lives Valued Equally. First, we report the results from assuming that the Δ function is simply

$$\Delta(D) = M_0 D,$$

where, as before, D captures **all** deaths associated with the epidemic. Our findings are in Figure 11.

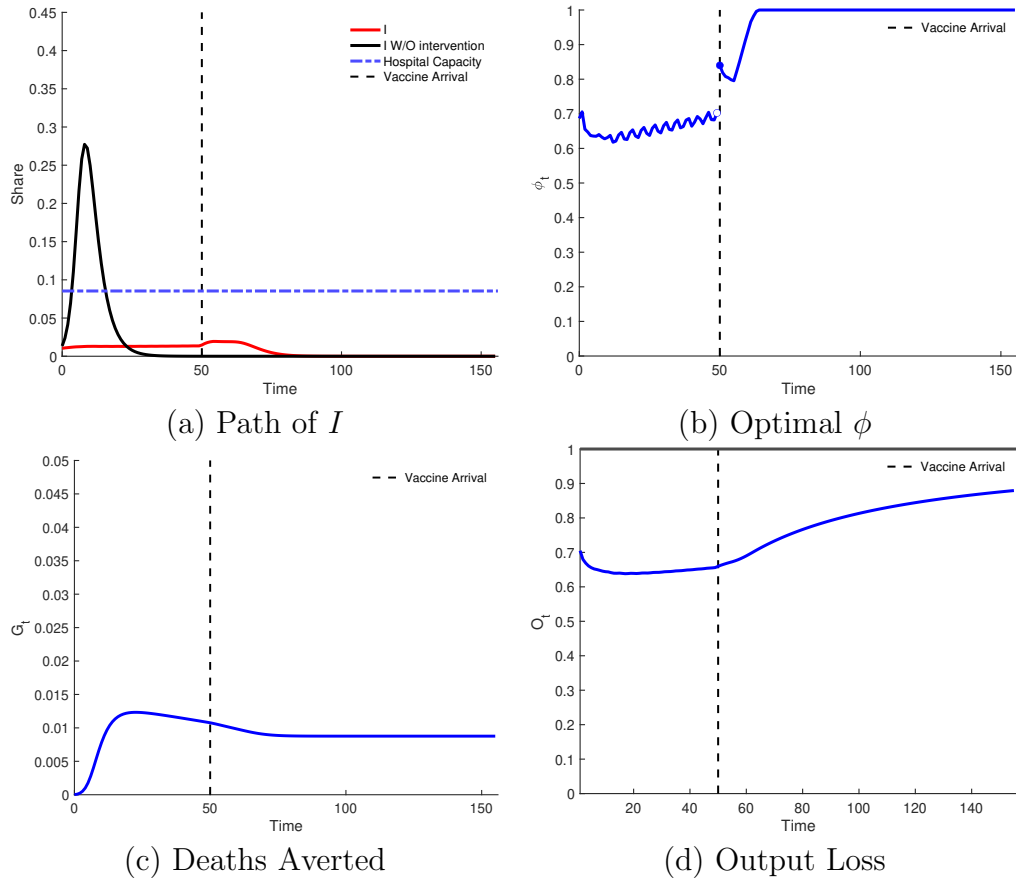


Figure 11: All Lives Valued Equally

1. Not surprisingly, when society puts higher value on averting deaths it results in more stringent employment restrictions. During Phase I the economy is operating below 70% of capacity and even the arrival of a vaccine does not result in a complete liberalization.
2. As in many other cases when a vaccine becomes available at the time that stay-at-home restrictions are severe, the optimal policy results in an uptick in infections.
3. The epidemic in this case has a fairly long duration (almost two years) and a large output cost: Over a three year period the loss of output is about 12%.
4. Somewhat surprisingly the number of deaths averted is only 0.87% of the population and the unit output cost is 2.5 million

The Valuation of Foregone Consumption The second element in our valuation formula is a multiplier applied to consumption to evaluate forgone utility associated with death. We followed the literature (see Hall et. al (2020) for an application to the COVID-19 epidemic) and assumed that the shadow value of foregone consumption is three times actual consumption. (In our notation $v = 3$.) However, if we assume that $v = 0.51$ (that is, we value the utility of individuals who die at 1/2 the market value) we do find that the optimal policy is substantially different. In terms of our constant M_0 it implies that the new value is 1/3 of the baseline.

A summary of the results is in Figure 12.

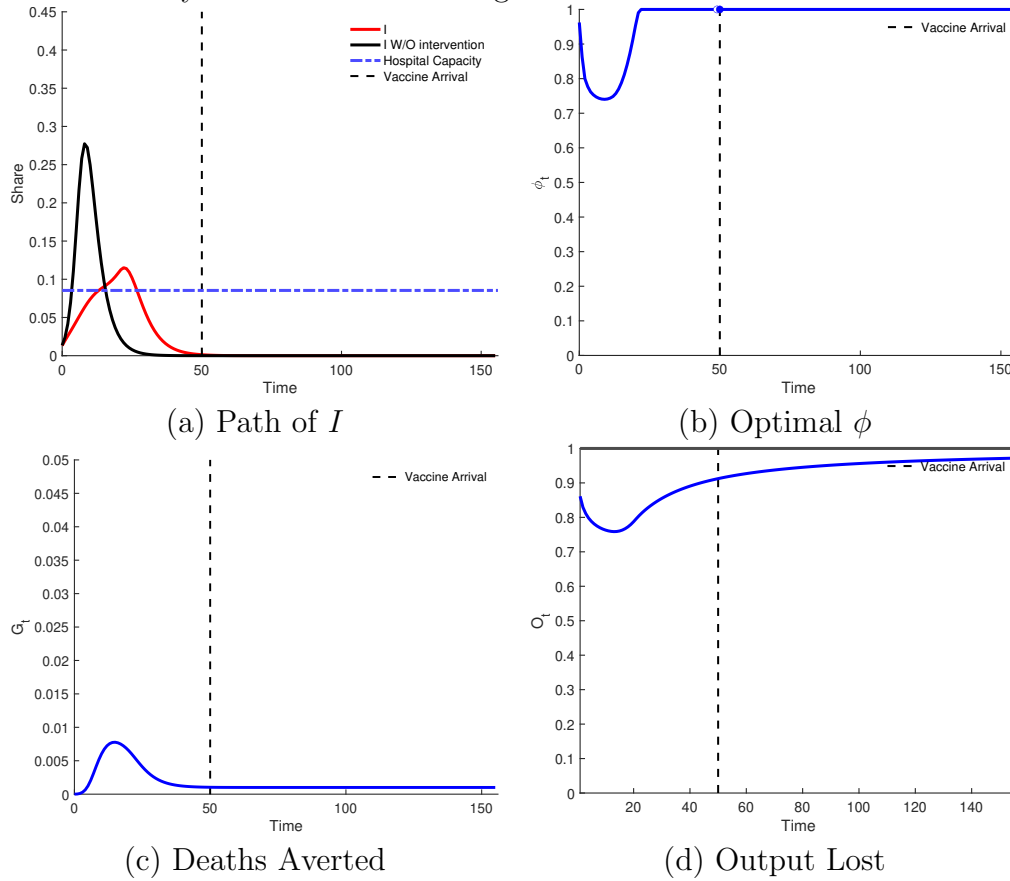


Figure 12: Low Consumption Multiplier
Relative to the baseline we find that:

1. There is an initial contraction of employment but it is relatively short lived. Partial liberalization starts in week 12, and the stay-at-home

restriction is lifted around week 24, before the vaccine arrives (on week 50).

2. Under the optimal policy the hospital capacity is exceeded. The peak is over 11.5% (our hospital capacity constraint is 8.5%) and even at this point the optimal policy has $\phi = 1$.
3. Not surprisingly, very few deaths are averted but the cost per averted deaths is very high, 5.1 million, as output is almost 3% below capacity on average over a three year period.

We view the results of this section as a powerful reminder of the degree of uncertainty associated with this type of planning exercise. We recognize the our optimistic and pessimistic scenarios are undoubtedly “too optimistic” and “too pessimistic” but the differences in optimal policies and in outcomes is vary large.

The optimal policy in the model is very dependent on society’s valuation of human life. It is not clear to us what is the appropriate criterion but we recognize that it has a large impact on employment and the utility of those who survive.

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

Our basic equation for the social value of a life is

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

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 the following Table.

Deaths Averted (%) and Unit Cost (millions)				
Scenarios	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

Table 1. Value of Life and Deaths Averted

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)¹⁹. 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 nonlinearities 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

¹⁹The case (0,.79) corresponds to $v = 1$ and (0,.72) to $v = .8$.

number of deaths averted. In the baseline (and when lives are not very valuable) the improvement in output is large but less than proportional to the fewer deaths averted.

4.5 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 2 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.

Value of a Vaccine				
Scenarios	Arrival Time			
	1	4	25	50
Baseline	1.05T	1.10T	430B	4B
Higher \mathcal{R}_0	2.82T	2.90T	1.48T	4B
Optimistic	2B	2B	2B	2B
Pessimistic	4.79T	4.94T	4.30T	3.11T

Table 2. Value of a Vaccine

As expected the different scenarios imply very different values of a vaccine. In three of the four scenarios the value converges to 4 billion or less after a year. 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.

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 to the yellowish area in Figure 13 that displays the value of a vaccine.

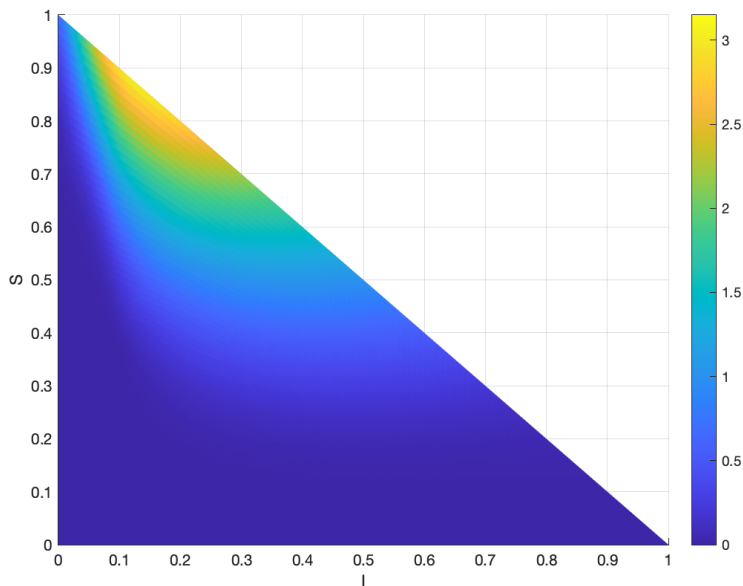


Figure 13. Value of a Vaccine as a function of the State

We view these results as giving an upper bound on the value that society puts on a vaccine. The numbers 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.

As we discussed before, the different alternatives that we consider about how to determine the value of averting deaths has a large impact on policy and, hence, on the value of a vaccine. Table 3 reports the same statistics for different implicit value of life formulas as discussed in section 4.4. The effective value of life decreases as one moves down the rows of the table.

Value of Life and the Value of a Vaccine				
Scenarios	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

Table 3. Value of Life and Value of a Vaccine

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).²⁰

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 infected.

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 infected 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

²⁰These calculations ignore the value of a vaccine in the case of a second outbreak. We discuss some options in the section on extensions.

$\bar{\mu} = 0.05$. 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 infectious (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 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 productivity²¹. 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 if $\phi = 1$ 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.

²¹We 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.

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.

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 overshoots 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

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

The corresponding value during Phase I is

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

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:

1. In the model the optimal policy depends on both the fraction infected and the fraction susceptible. Simple policies that use only information on the number of infectious individuals (or deaths) are bound to be suboptimal. In addition to the state, many other features of the environment —about which there is significant uncertainty— play a large role in determining the optimal policy.

2. Across a variety of scenarios and realizations partial liberalization of the stay-at-home policy occurs before the peak in the number of infectious individuals (and consequently in the number of deaths in a setting that allows for delay).
3. Optimal policies also imply—in several of the cases that we study—that a relaxation of stay-at-home restrictions will be accompanied by an *increase* in the spread of the virus. Policies that respond *uniformly* to an increase in the rate of infection by imposing employment restrictions are suboptimal.
4. Ignoring the role of a vaccine can lead to mistakes in designing policy. In particular, changes in the probability of developing a vaccine and differences in the rate at which the population can be vaccinated have a significant effect on the optimal policy. Moreover, the realization of the arrival time has a first order impact on the economy. An early arriving vaccine significantly lowers the costs in terms of output and lives lost while, in our simulation relative to the baseline, a late arriving vaccine does not have a large impact on policies but is very costly in terms of deaths averted.
5. Across many of the scenarios that we have looked at the optimal policy implies a large decrease in employment—it ranges from 20% to over 35%— and, in many cases average output after one year is less than 80% of potential.
6. The optimal policy depends on the value of averting deaths and the disutility associated with deaths related to the epidemic:
 - (a) If all deaths matter, then the optimal policy is very restrictive and, in our simulation, it takes over 15 months for the economy to lift all restrictions. The loss of output is large.
 - (b) If the shadow value of consumption of individuals who die is low (about 50% of average consumption) then the optimal policy lifts all restrictions after about 21 weeks and it results in a small loss of output.
7. The model implies that, in most scenarios, the output cost per death averted exceed 1.5 million, and can be as high as 10 million.

8. We find that the social value of a vaccine available after 6 months is large, often exceeding one trillion. However, except in the “Pessimistic” scenario this value decreases substantially at a one year mark and it is at most four billion.

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Appendix 1: Proofs

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

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

The associated Hamiltonian is

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

The FOC are standard and given by the static conditions

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

and

$$-u'(\cdot)c_V'(\mu(S + (1 - \zeta)I))(S + (1 - \zeta)I) = -\lambda_S S + \hat{\gamma}_-^I - \hat{\gamma}_+^I, \quad (9)$$

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 $\lambda_I = F_I$,

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

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

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

$$\begin{aligned} \dot{\lambda}_I = & u'(\phi w(1 - \zeta I) - c_V(\mu(S + (1 - \zeta)I))) [w\phi\zeta + c'_V(\mu(S + (1 - \zeta)I))(1 - \zeta)\mu] \\ & + \Delta'[(\chi\zeta I - \bar{H})^+] \chi\zeta + (\rho + \kappa) \lambda_I + (\lambda_S - \lambda_I) \beta\phi^2(1 - \zeta)S + \lambda_S\gamma. \end{aligned} \quad (11)$$

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

$$\beta(1 - \zeta)S^* = \kappa, \quad (12)$$

and

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

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

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

and

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

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 $I^* \rightarrow 0$, while the right hand side converges to zero as $I^* \rightarrow 0$ 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 $\lim_{I^* \rightarrow 0} \Lambda = \rho(\rho + \gamma) > 0$.

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

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

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

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

where

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

and

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

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

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

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

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: Calibration

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

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

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

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

and we assume that $v = 3$ 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	0.16
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	μ	0.05
Minimum Consumption ($1-c_0$)	c_0	0.60
Vaccine Arrival Poisson	η	1/78
Initial I_0	I_0	0.01
Initial S_0	S_0	0.99

Appendix 3: Literature Review

There is an extensive and fast growing research area interacting models of epidemiology and macroeconomics, but also exploring the effects of covid-19 in the specific sectors (i.e. services vs. manufacturing) and markets (i.e. labor market, stock market). This section attempts to summarize some of the recent work by grouping research in different buckets, with the obvious caveat that this is a very imperfect way to categorize the existing work.

Policies in an SIR models: There a large number of research that explore the effects of different policies (i.e. social distancing, lockdown, etc. . .) in SIR models developed by Kermack and McKendrick (1927). See for example, Atkeson (2020), Neumeyer (2020), Bassetto (2020), Droz and Tavares (2020), Hsiang et al. (2020), Fang, Wang, and Yang (2020), Shao (2020), Wang et al. (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).

Measurement issues: Other paper use some empirical approach to measure key parameters on SIR model (i.e. Stock 2020, Korolev, 2020, Kubinec, 2020), or deal with measurement issues in the data like under reported infection rates (i.e. Hortacsu, Liu and Schwieg 2020, Harris 2020), measuring hospital capacity and healthcare constraints (i.e. Blavin and Arnos, 2020)

Optimal management of the epidemic: Alvarez, Argente, and Lippi (2020), Gonzalez and Niepelt (2020).

Macroeconomic implications of pandemics: 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),

Another strand combines the canonical macro framework with a SIR model to explore the short-term consequences of the pandemic, (i.e. Eichenbaum, Rebelo, and Trabandt 2020 Fornaro and Wolf 2020), whereas other explore the long-run implication (i.e. Kozlowski, Veldkamp, Venkateswaran, 2020). All the economic models in the previous papers use a single sector economy. One of the challenges in the present situation is the asymmetric effect of the pandemic shocks in the different sectors of the economy. Some

essential sectors have remained open whereas non-essential sectors have been forced to shut-down by government policies aimed to reduce the infection in the population. Bodenstein, Corsetti, and Guerrieri (2020) explore the effects of social distancing epidemiological model combined with a multisector model, designed to capture key characteristics of the U.S. Input Output Tables. They argue that the economic cost can be large in the presence of core non-essential sectors that provide key inputs for the essential sectors. Policies should take these links into consideration. Guerrieri, Lorenzoni, Straub, and Werning (2020) also show theoretically that supply shocks are amplified in economies with multiple sectors and incomplete markets.

Fiscal and monetary interventions during a pandemic: Some research explores the effects of fiscal interventions in economies with service and non-service sectors (i.e. Faria-e-Castro 2020), the effect of large scale asset purchases when the monetary policy rate is at the zero lower bound (i.e. Caballero and Simsek, 2020).

Effects across different markets

- Labor market: Explore the effects of pandemics in the labor market trying to identify the evolution of outcomes in real time (i.e. Bick and Blandin, 2020), the implications for wages and unemployment (i.e. Kapicka and Rupert, 2020), the amount of employment that can be completed by workers at home (i.e. Dingel and Neiman, 2020).
- Stock market volatility : Explore the implications in the pandemic in stock market returns using high frequency data. For example Alfaro, Chari, Greenland, and Schott (2020), Baker, Bloom, Davis, Kost, Sammon, and Viratyosin (2020), Gormsen, N. and Koijen (2020)

Appendix 4: Computational Notes

Writing the HJB equations for the two phases,

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

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

$$\begin{aligned}F(S_t, I_t) &= \max_{(0 \leq \phi \leq 1)} \left\{ \frac{(1 - e^{-\rho \Delta})}{\rho} \left(u(\phi w(1 - \zeta I) - c_V(\mu(S + (1 - \zeta)I))) - \right. \right. \\ &\quad \left. \left. \Delta [(\chi \zeta I_t - \bar{H})^+] \right) + e^{-\rho \Delta} F(S_{t+\Delta}, I_{t+\Delta}) \right\} \\ S_{t+\Delta} &= S_t + [-\beta \phi^2(1 - \zeta)SI - \mu S + \gamma(1 - S - I)] \Delta \\ I_{t+\Delta} &= I_t + [\beta \phi^2(1 - \zeta)SI - \kappa I] \Delta\end{aligned}$$

FOC:

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

Define:

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

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

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

$$\mu = 0$$

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

FOC:

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

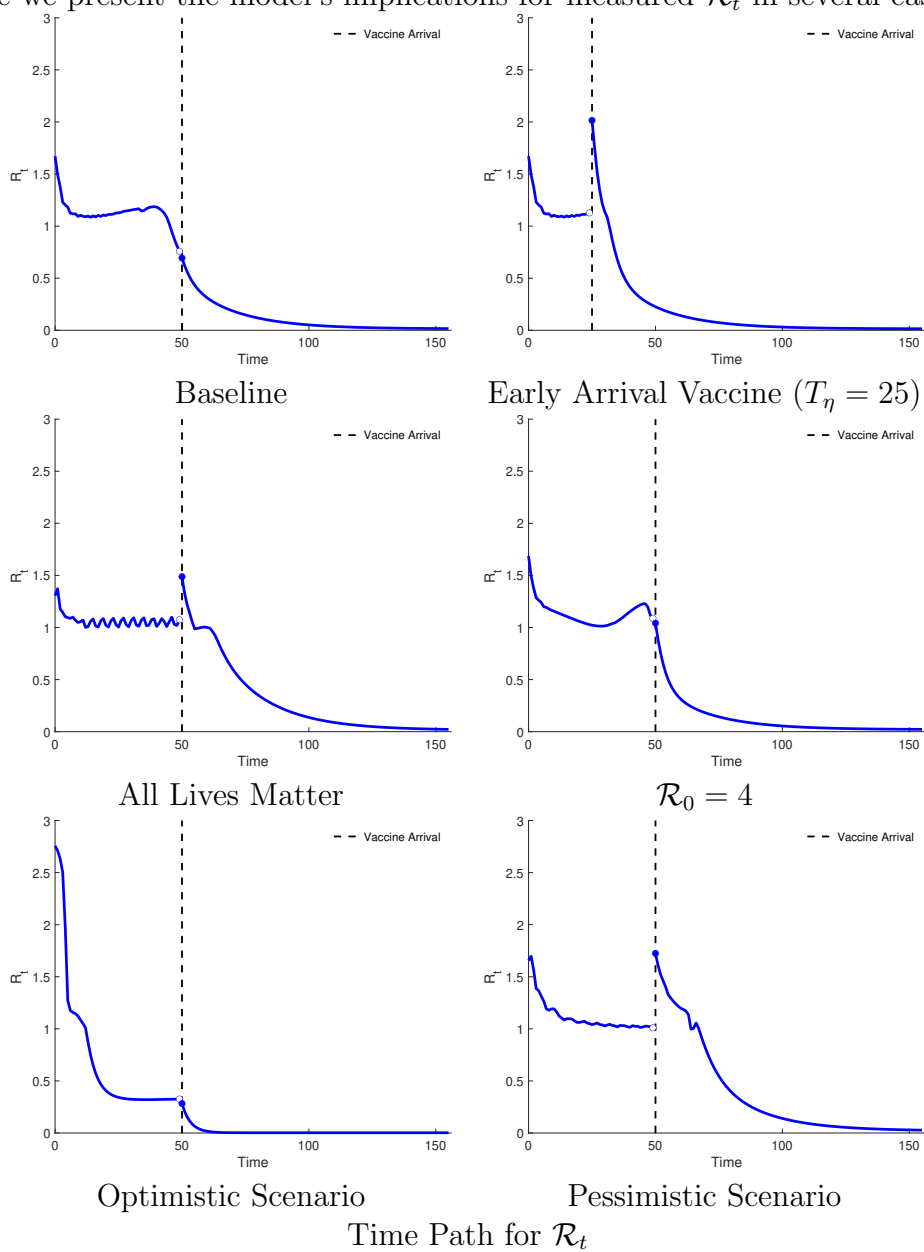
Where,

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

Appendix 5: Supplementary Graphs

Implied Contagion Factor \mathcal{R}_t

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



Late Arrival of a Vaccine

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

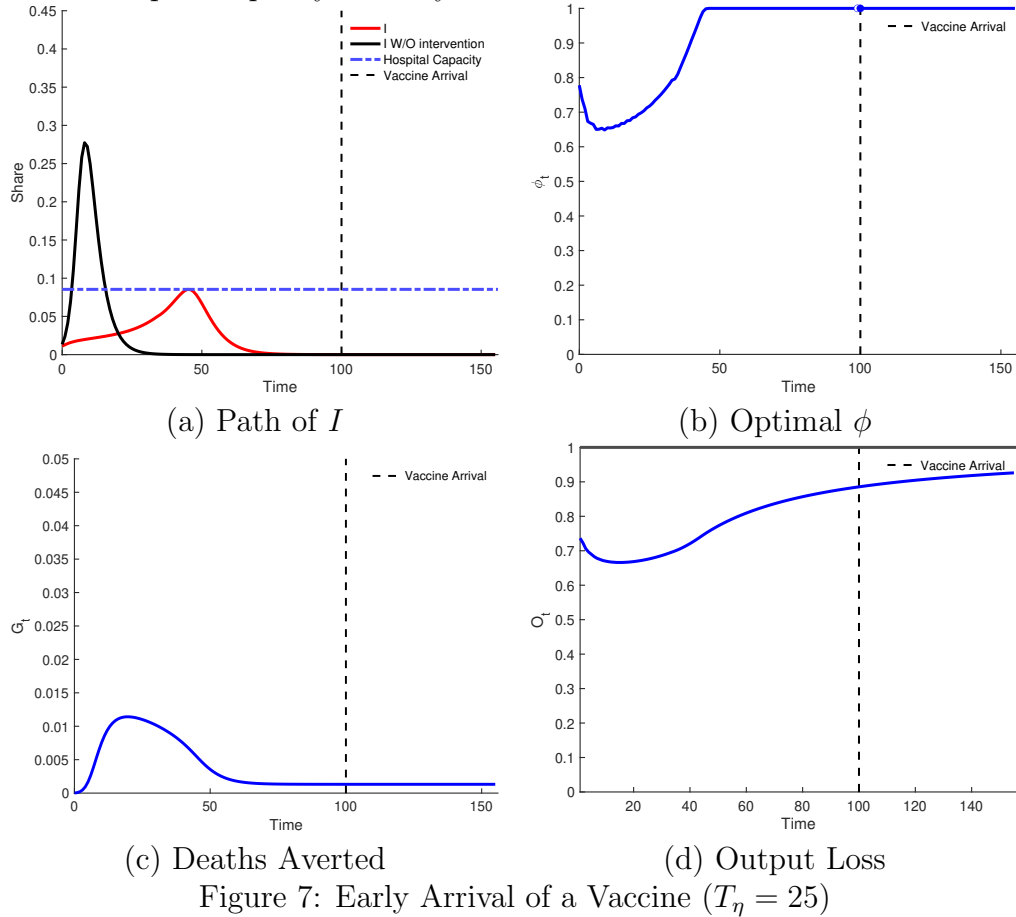
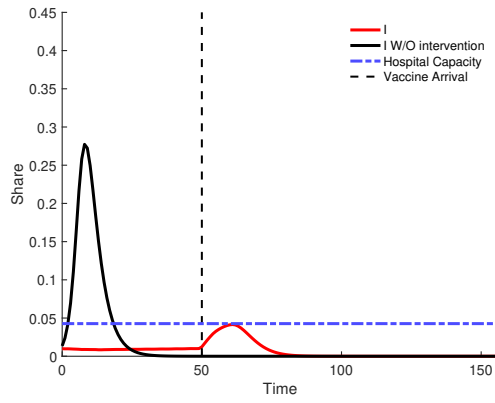


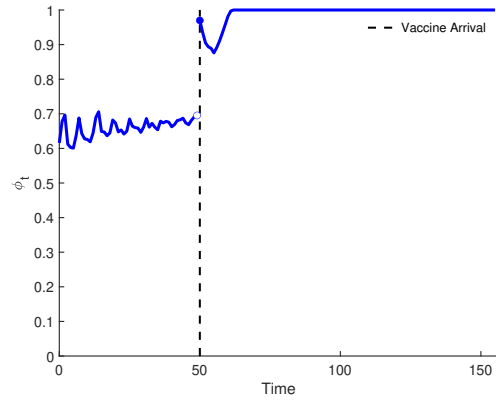
Figure 7: Early Arrival of a Vaccine ($T_\eta = 25$)

Higher ζ

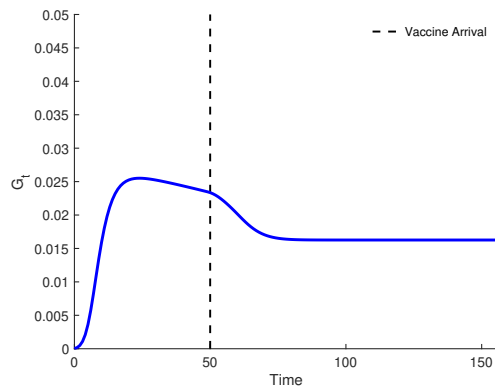
Basic data if $\zeta = 0.20$.



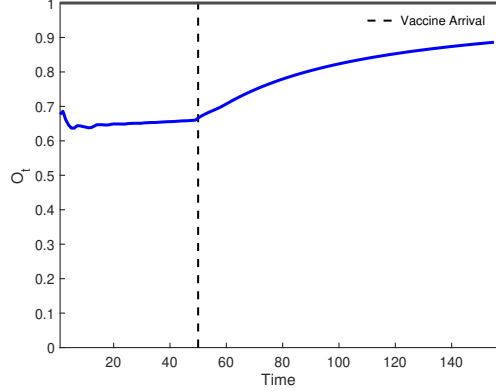
(a) Path of I



(b) Optimal ϕ



(c) Deaths Averted

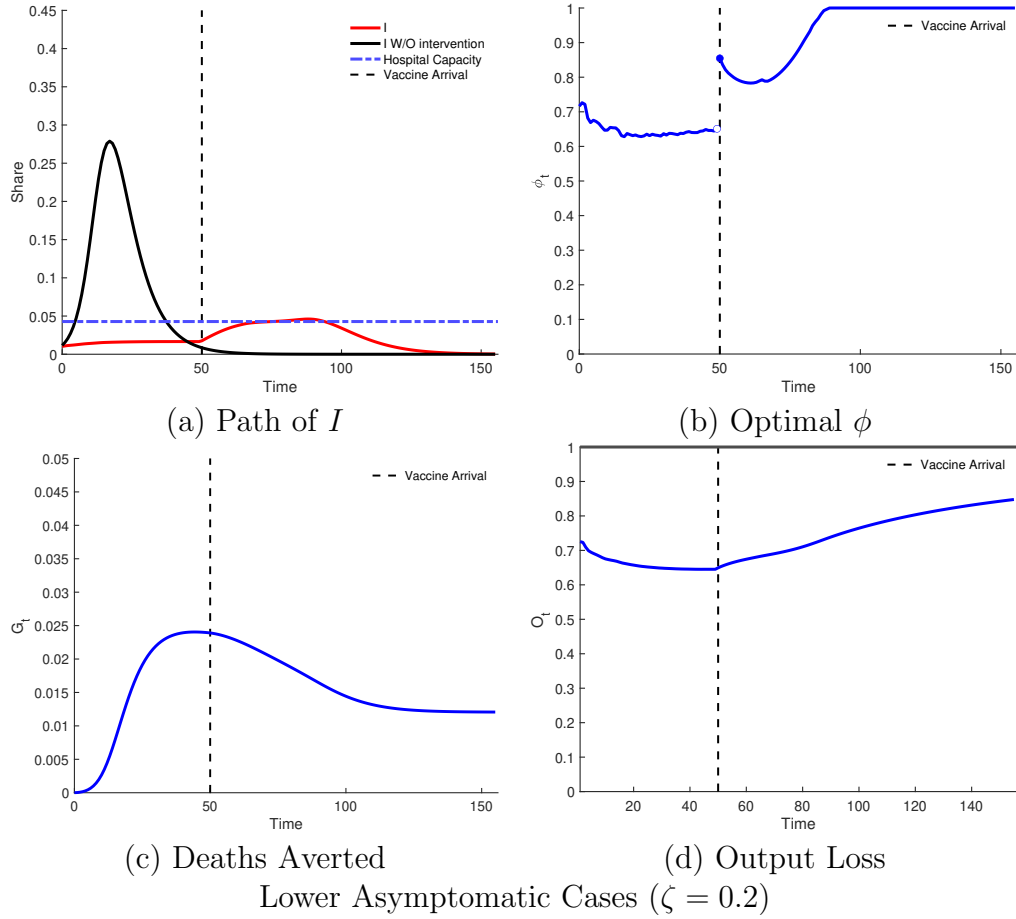


(d) Output Loss

Lower Asymptomatic Cases ($\zeta = 0.2$)

Pessimistic Scenario II

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

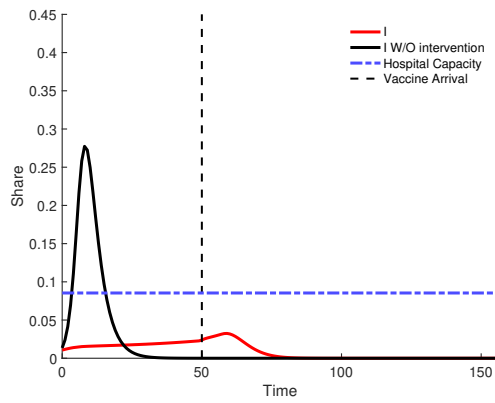


Intermediate Social Valuation of Deaths Averted

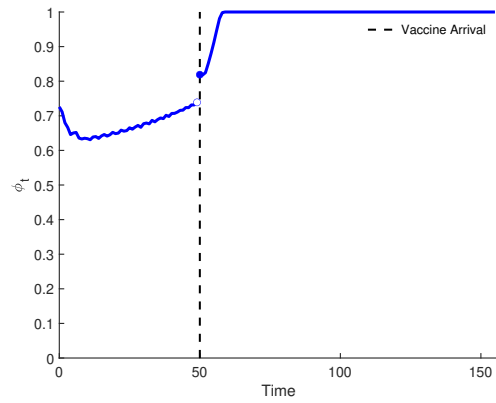
Our valuation of deaths averted function is given by

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

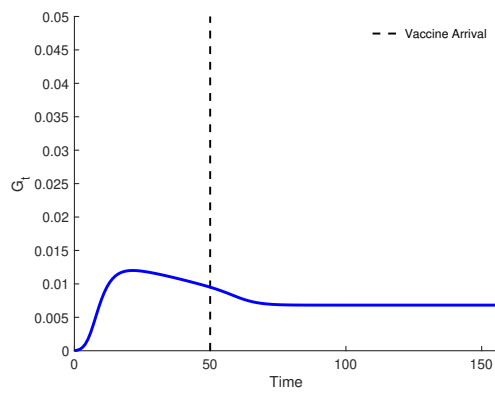
Our baseline assumes that $k = 0$ which implies that only excess deaths enter the social utility function. Our “Higher Social Value of Life” scenario assumes that $k = 1$ which implies that all averted deaths are counted equally. Here we show the intermediate case $k = 0.5$ that counts all deaths but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



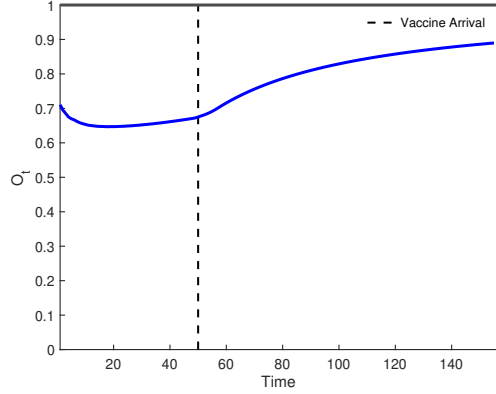
(a) Path of I



(b) Optimal ϕ



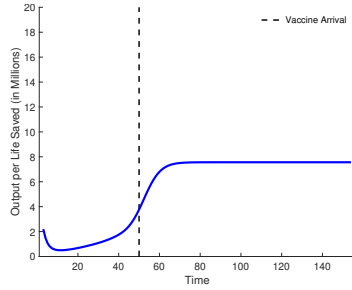
(c) Deaths Averted



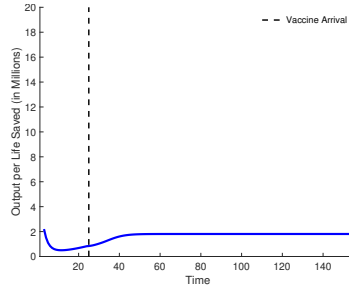
(d) Output Loss

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

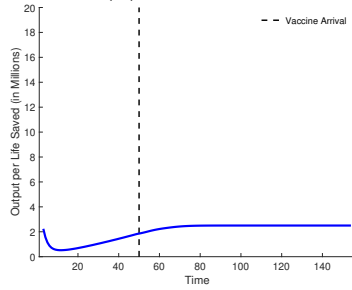
Output Cost per Death Averted



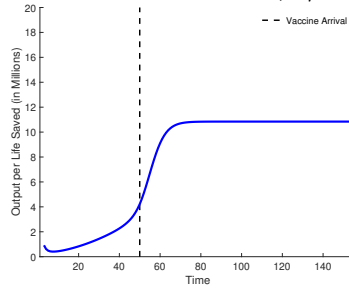
(a) Baseline



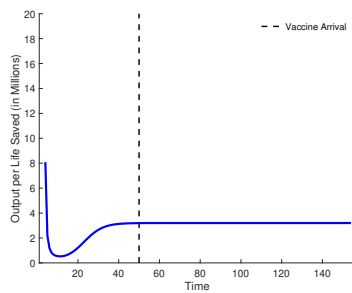
Early Arrival Vaccine ($T_\eta = 25$)



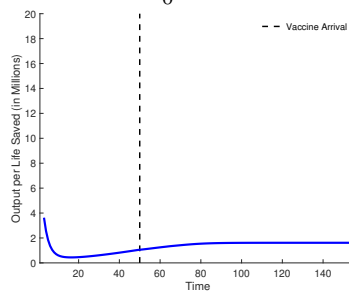
All Lives Matter



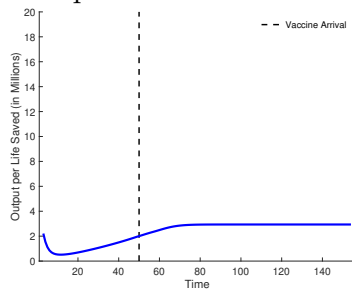
$\mathcal{R}_0 = 4$



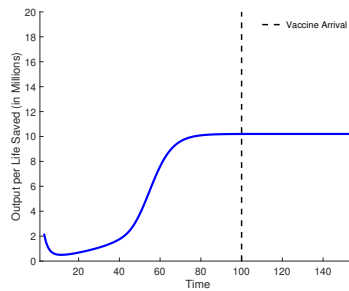
Optimistic Scenario



Pessimistic Scenario



$k = 0.5$



Late Arrival Vaccine