"Optimal Management of an Epidemic: An Application to COVID-19. A Progress Report" *

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Abstract

We study a dynamic macro model that is well suited to understanding what are the key factors to determine the severity and duration of stay-at-home policies. We present a variety of scenarios and we find that optimal policies can sometimes produce counterintuitive results. For example, optimally chosen liberalization of employment restrictions do result in many cases in an increase in the spread of the virus. Even though there is a significant uncertainty about which is the more reasonable scenario we find that, across the options that we study, output losses under the optimal policy are large in the first year of the epidemic.

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1 Introduction

The objective of this research is to understand how the features of the economy 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 first stage of the program analyzes a multiregion economy. The output in each region uses as inputs goods produced in every other region. As a first pass, we assume that goods can move but people can't. In this setting the planner can decide the severity of the "stay-at-home" policies for each region.

This progress report contains some preliminary results for a one sector economy. The setting is related to the recent work by Alvarez, Argente, and Lippi (2020) and Gonzalez-Eiras and Neipelt (2020). The major differences with Alvarez et. al. is that we consider more general preferences and a more realistic model of the effect of the arrival of a vaccine. Given our setting we can also discuss the implications for asset prices. Gonzalez-Eiras and Neipelt (2020) present a general model but they concentrate of special cases in order to find closed form solutions. There is a large literature (summarized in Appendix 3) that has looked at the economic dimensions of a pandemic but that does not investigate optimal policies.

We assume that individual preferences depend on individual consumption and we assume that social preferences also take into account the losses associated with deaths. We consider different weights of how the value of death averted in a region can influence the optimal policy in another region. On the epidemiological dimension we use a standard SIR model with (endogenously chosen) vaccination rates in some states.

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 reduce the 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, in this report, 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. In this report we assume that the cost is sufficiently low (zero in our quantitative exercises) that the optimal policy is to vaccinate at the highest feasible rate. However, we view this cost as an important element that can affect the speed of vaccination in less developed countries and plan to incorporate a more realistic version in the next iteration.

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.

We calibrate the model using standard estimates of the epidemiological parameters and we find that:

1. *Baseline Simulation* (vaccine arrives after 50 weeks):

   (a) Under the optimal policy the epidemic curve is flattened at the cost of a large output cost. Initially output is reduced by almost 30%.

   (b) The optimal policy —even if there is no arrival of a vaccine— calls for a gradual liberalization of the stay-at-home policy significantly before the peak of deaths.

   (c) The arrival of a vaccine results in an almost complete elimination of all restrictions and this is accompanied, under the optimal policy, by an increase in the spread of the epidemic.

2. *The Impact of the Timing of the Arrival of the Vaccine*:

   (a) A vaccine that arrives "late" (in our case on week 100) has a small impact on the optimal policy relative to the no vaccine case. Compared with the baseline the number of deaths averted is significantly smaller but the output cost is very similar. The cost per death averted is very high.

   (b) A vaccine that arrives "early" (in our case on week 25) has a large impact on the optimal policy. It results in an almost full liberalization and a fairly large increase in the spread of the epidemic.

The number of lives saved over the course of the epidemic is large and the output cost significantly smaller.

3. *Robustness*

   (a) *Higher Infectiousness.* ($\mathcal{R}_0 = 4$ vs $\mathcal{R}_0 = 2.8$) implies more severe employment restrictions, a delay in the peak of the epidemic and fairly large increase in the number of deaths averted at the cost of a large decrease in output.

   (b) *Optimistic Scenario.* Our optimistic scenario assumes, a higher hospital capacity (more ICU beds), higher probability of an early arrival of a vaccine (about a year after the beginning of the epidemic), and a fairly large vaccination rate. We find that the optimal policy does not restrict employment when the level of infection is low but, when it reaches a threshold level it call for a sharp and short lived reduction in employment. The long run output costs and the costs per death averted are small.

   (c) *Pessimistic Scenario.* This is characterized by lower ICU beds availability, longer expected time until a vaccine becomes available (about three years after the beginning of the epidemic), and a low vaccination capacity per week. We find that the optimal policy call for a much more restrictive, and longer lasting, stay-at-home requirement. Output during the first year is about 30% lower and even after two years it does not reach 90% of capacity.

   (d) *Higher Value of Life.* In the baseline we assume that only deaths that exceed a certain "normal" level cause the planner to adjust policy. We also present the results when all deaths are counted equally. Not surprisingly we find that with the higher utility associated with averting deaths the planner is willing to sacrifice more output. In this case even the availability of a vaccine does not imply that the economy reverts back to normal.

4. *Asset Prices*: Our model has implications for interest rates and some measures of total output. For our baseline simulation we find that real interest rates are initially very negative and then turn significantly positive as the employment restrictions are eased.

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[1]. 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. In the simplest version we assume that there is one region and one good that is produced exclusively with labor. In the multi-region version each region consumes its own good but production requires inputs from other regions. Agents are long lived utility is additively separable.

There are two policy variables that we study. A type of "stay-at-home" restriction on the utilization rate of the labor force which has two impacts: It decreases output and, simultaneously, it 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 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.

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

We assume that there is a representative agent that cares about consumption. Social preferences are simply individual preferences adjusted (downward) by the utility cost of excess 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 utility cost of deaths that exceed the normal levels. The details of how we model excess deaths 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,

As mentioned above, we study two different models that vary in their complexity. The simple case —for which we provide some preliminary quantitative results— assumes a single good and, effectively, a single region. The regional model allows for a regional input output structure that is suitable to understanding the forces that would drive region specific changes in stay-at-home policies, both in terms of their intensity and duration.

2.1 The Economic Model: One Region

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 is terms of output of vaccinating a population of size $S + (1-\zeta)I$. This is the population that includes susceptibles and

infected individuals who are asymptomatic. Of course, this term is operative only in Phase II when a vaccine is available.

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 of "excess deaths." We view this term as capturing society's disutility associated with deaths over and above some baseline[2]. Thus, if deaths (per person) are labeled D, and we denote by D^+ deaths in excess of some socially acceptable level, that is, $D^+ = \max\{D - \bar{H}, 0\}$, we assume in our baseline that only excess deaths enter the social utility function. 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.[3] 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 trade-off 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."

The static social payoff is

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

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.

[2]It 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

[3]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.

We assume that

$$\Delta(D^+) = \begin{cases} 0 & \text{if } D^+ = 0 \\ > 0 & \text{if } D^+ > 0 \end{cases} .$$

Moreover the function Δ is strictly increasing and convex in D^+.

Society's preferences are then a function of consumption and net excess 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

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

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

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

$$N \ln\left(w\phi L - \underline{c}\right) - M_0\left[N D^+\right].$$

In this formulation $\underline{c}$ is the minimal level of consumption and N is population size. ND^+ is the total number of excess deaths. Thus, the cost to society of one additional excess 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 $\hat{L} = 1$.

The *per capita* social payoff function —which we take to be the planner's objective function— is then

$$\ln\left(w\phi L - \underline{c}\right) - \ln\left(\upsilon w\hat{\phi}\hat{L} - \underline{c}\right)\frac{1-e^{-\rho T}}{\rho} D^+.$$

An alternative that allows for a social cost associated with total (not just excess) deaths is the following formulation

$$\ln\left(w\phi L - \underline{c}\right) - \ln\left(v w\hat{\phi}\hat{L} - \underline{c}\right)\frac{1-e^{-\rho T}}{\rho}\left[k\min\{D,\bar{H}\} + \max\{D-\bar{H},0\}\right].$$

The baseline case has $k = 0$. If $k = 1$ then all deaths are valued equally. We report the results in those two cases and in the appendix we also show the implications of $k = 0.5$ which captures the idea that it is more costly to society deaths that, potentially, could have been prevented.

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.[4] 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

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

Since we normalized the population to one this is

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

[4]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 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)

Then the fraction of susceptibles and infectious in the population is ϕS and $\phi(1-\zeta)I$.[5]

Finally we assume that a certain fraction of the resistant lose their immunity. 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 resistant individuals lose their immunity and the entrance of new susceptible individuals in the population.

The stock of infected evolves according to

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

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) 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

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

[5]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)

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.[6]

2.3 The Multi Region Model

Our second model assumes that the country can be viewed as consisting of J interconnected regions. The value of J will depend on the availability of regional input-output data. To simplify the exposition we describe a two region version.

Output in region j is given by

$$y_j = A_j \left(L_j\right)^{1-\alpha_{j1}-\alpha_{j2}} y_{j1}^{\alpha_{j1}} y_{j2}^{\alpha_{ij2}}, j = 1, 2,$$

where y_{ji} is the amount of output produced in region i that is used as an input in region j. Consumption in region j is given by

$$c_j = y_j - y_{jj} - y_{ij},$$

for $i \neq j$.

To capture size differences we will assume that the productivity factors capture that full employment-no epidemic labor supply.

The flow utility in region j is simply

$$u_j(c_j - c_{V,j}(\mu_j(S_j + (1-\zeta)I_j))) - \Delta_j(D_j).$$

Note that the model allows us to capture differences in the cost of vaccination (for example because the populations differ in terms of density and socio-economic factors) as well as differences in the social cost of excess deaths that depends on the availability of health infrastructure in each region. We plan to study alternative regional differences in social preferences.

Moreover, we allow for different epidemiological models. For example, different densities can affect the value of β. The relevant system is

$$\begin{aligned} \dot{S}_j &= -\beta_j(\phi_j S_j)(\phi_j(1-\zeta)I_j) - \mu_j S_j + \gamma(1 - S_j - I_j) \\ &= -\beta\phi_j^2(1-\zeta)S_j I_j - \mu S_j + \gamma(1 - S_j - I_j). \end{aligned}$$

[6]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.

$$\dot{I}_j = \beta\phi_j^2(1-\zeta)S_jI_j - \kappa I_j.$$

We assume that the purely biological factors, κ and γ, are constant across regions.

3 Analysis of the One Region 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

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

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.[7]

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. 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.

[7] 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.

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 reinfection rate) the steady state displays no output loss ($\phi^* = 1$) and no vaccination ($\mu^* = 0$). We formally summarize this result in the following proposition

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 ■

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

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

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 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 $t \to \infty$ 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 ■

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 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 it takes about 6 weeks for an infected individual to transition either to resistant or to die.
- We assume that, in expectation, it takes about 18 months for a vaccine to become available (Phase II). 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 $\lim_{t\to\infty} \mu_t = 0$).

To compute the results we first discretized the continuous time HJB equation and then 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 $0 \leq S + I \leq 1$. (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.

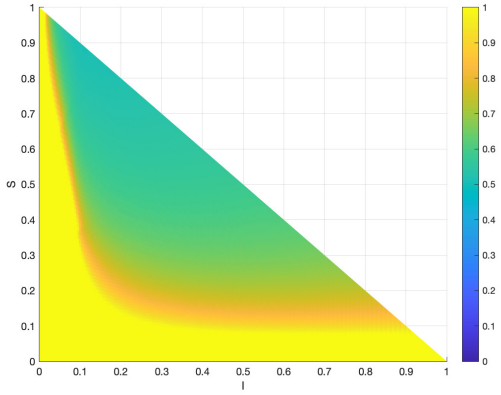


Figure 1. Optimal Policy (ϕ) in Phase I

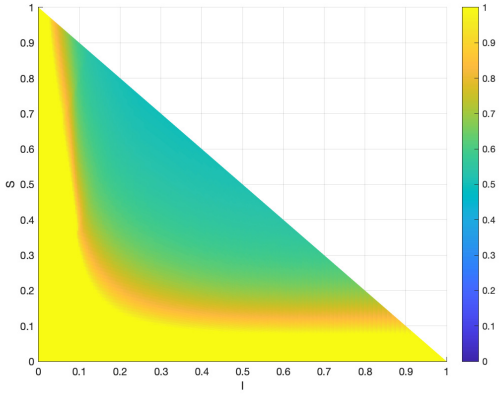


Figure 2. Optimal Policy (ϕ) in Phase II

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. The optimal policy in Phase II is "shifted to the right" relative to Phase I reflecting the fact that the availability of a vaccine implies less severe "stay-at-home" restrictions.

2. There are large subsets of the state space that even in Phase II 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.

Figure 3 shows that path of the stock of infectious individuals, I, in two cases: uncontrolled epidemic (black) and optimally managed epidemic (red). It assumes that the vaccine becomes available after about 50 weeks.

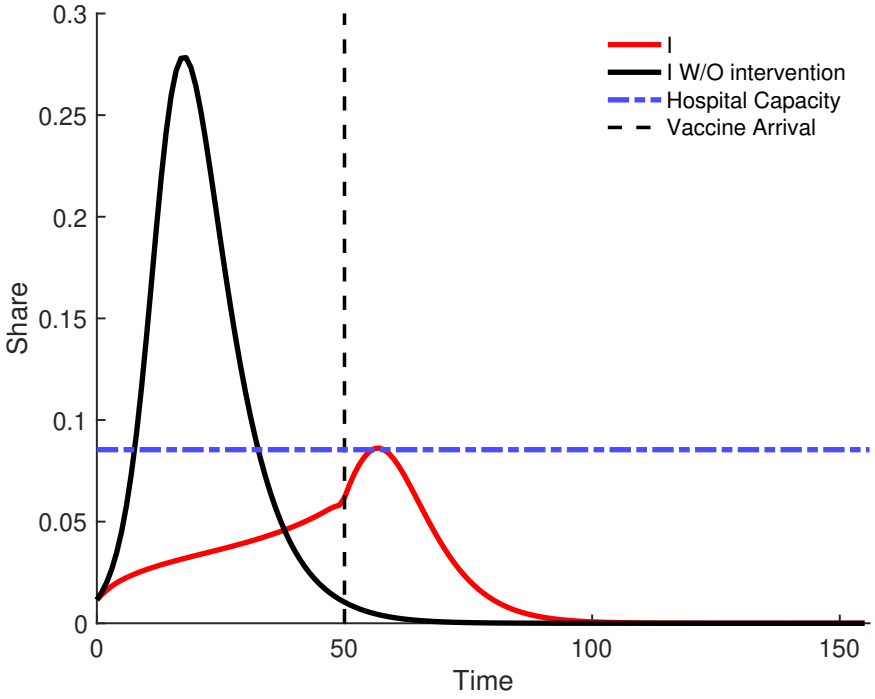


Figure 3. Time Path of I_t : Uncontrolled and Optimal.

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 would have reached about 28% of the population. It also imply 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 over a year for the epidemic to peak (58 weeks) which is almost double

the time in the absence of a policy. At the time of the peak I is 8.6% slightly above the threshold $\bar{H}$ ($\bar{H} = 0.0854$ indicated by the dashed blue lines).

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

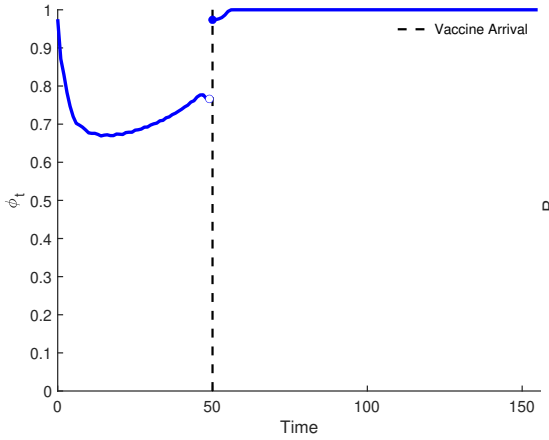


Figure 4. Optimal Policy (ϕ)

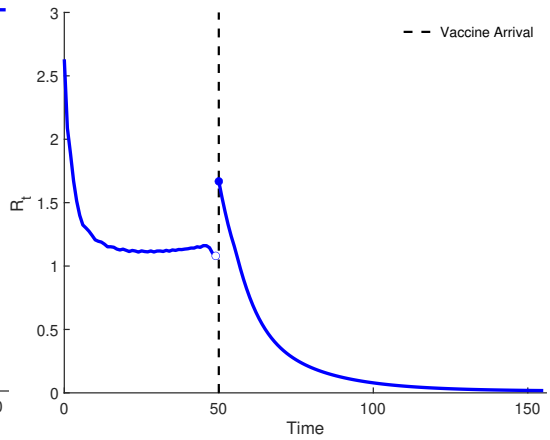


Figure 5. Optimally Managed $\mathcal{R}_t$

Two features of our solution are worth emphasizing. First, initial "stay-at-home" policy is fairly aggressive and output falls more than 30% from its potential level after 20 weeks. At that time there is a slow liberalization and even at $t = 50$ the economy is operating at less than 80% of its maximum capacity. This happens even though at that point infections —and in our simplified version deaths— are increasing. Thus, the optimal policy does not imply that relaxation of stay-at-home restrictions should occur after the peak. The initial aggressive policy keeps the number of severe cases —in our case $\chi\zeta I$— below our measure of hospital capacity (except for two weeks). This implies that even though the number of severe cases —which in our simplified framework is proportional to I— is far from hitting the hospital capacity constraint the liberalization policy in Phase I is gradual: ϕ increases from 0.67 in week 21 to 0.76 in week 49 (just before the vaccine becomes available). The message is clear: fewer than expected (relative to doing nothing) fatalities does not imply complete liberalization.

Second, at the time a vaccine becomes available (at $T_\eta = 50$ in this simulation) the optimal policy calls for a significant increase in ϕ and, after seven weeks, for the complete elimination of distortions. This liberalization induces an uptick in the number of infectious individuals (which corresponds to an increase in $\mathcal{R}_t$) . Thus, consistent with a popular view, eliminating re-

strictions on employment (and, in our model, this is equivalent to eliminating stay-at-home restrictions) is associated with higher infections. This, however is the optimal balance between the costs of forgone output and the disutility associated with the epidemic. The reason why the arrival of a vaccine results in fewer employment restrictions is that it increases the downward drift in the number of susceptible individuals, and with fewer susceptibles even the same number of infected result in a smaller spread of the epidemic.

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.

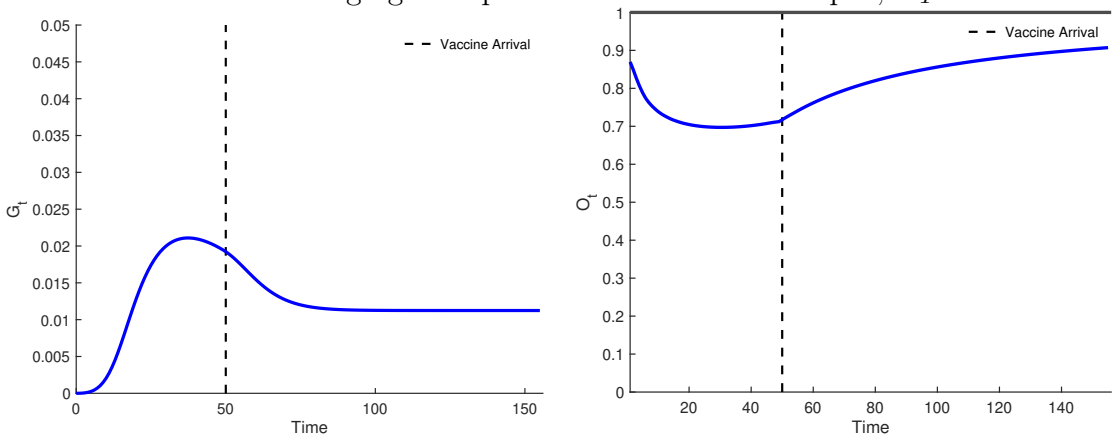


Figure 6. Lives Saved (left panel) and Output Loss (right panel)

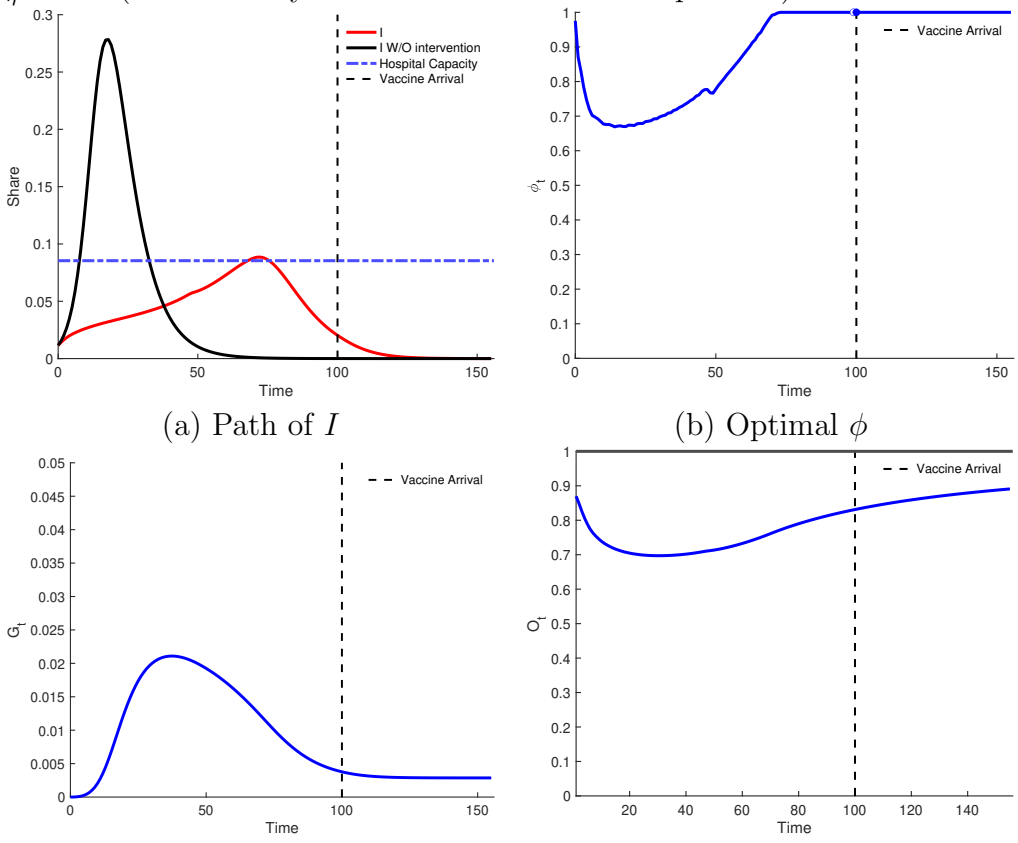
The cumulative number of lives saved is not a monotonically increasing function. It peaks about week 40 at 2.1% of the population when the optimally managed I crosses the uncontrolled case. From then on, the number of lives saved decreases. The reason is simple: under the optimal policy flattening the 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 number of averted deaths converges to 1.12%

The output cost of the optimal policy is significant. After 50 weeks output has been on average about 72% of full capacity. This estimate is much larger than what is found in other studies but it is a consequence of our optimal policy. The model predicts that the decline in output bottoms around week

30, and then it slowly increases over time. In the long-run (about 150 weeks) the output loss average over that period is 10%. 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 1.5 million.

Our baseline assumes a slightly optimistic arrival time of 50 weeks (which would correspond to February 2021). Next we analyze the impact of bad (late arrival) and good (early arrival) news.

Late Arrival of a Vaccine How does the availability of a vaccine change the optimal policy? Figure 7 (panel (a)) displays the path of I under both the no-policy case —which is the same in all realizations— and the implied path by the optimal policy (right panel) if the vaccine becomes available at $T_\eta = 100$ (almost two years after the start of the epidemic).



(a) Path of I

(b) Optimal ϕ

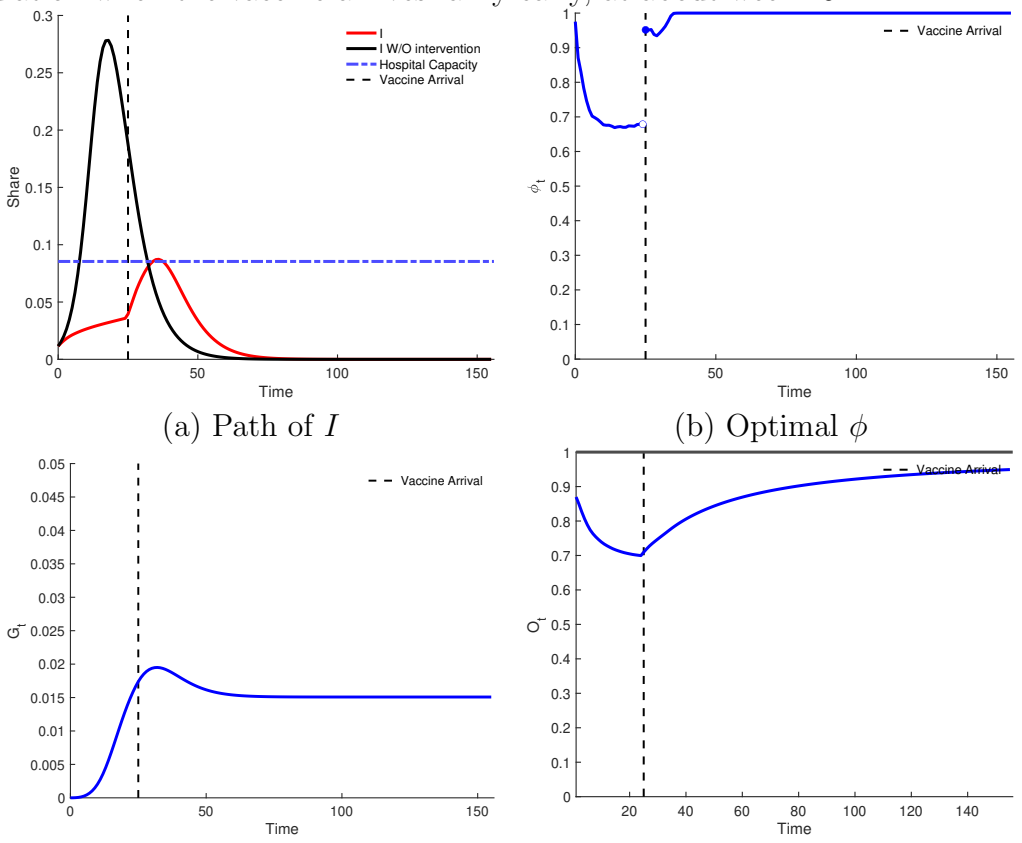
(c) Deaths Averted

(d) Output Loss

Figure 7: Late Arrival of a Vaccine ($T_\eta = 100$)

Relative to the case in which the vaccine becomes available after 50 weeks the infection peaks at a later date and, at its peak, it induces additional losses of life since it exceeds our capacity measure. The optimal policy is just the continuation of the policy in Phase I: Increasing and large restrictions on employment for the first 10 weeks followed by a slow but steady relaxation. The restrictions are completely eliminated shortly after the peak. The late arrival of the vaccine implies hardly noticeable changes in the optimal policy. The number of averted deaths is significantly smaller (about 1/10th in the long run) but the costs in terms of foregone output are very similar. The output cost per life averted in this case is close to 7 million.

Early Arrival of a Vaccine In Figure 8 we show the results of our simulation when the vaccine arrives fairly early, at about week 25.



(a) Path of I (b) Optimal ϕ

(c) Deaths Averted (d) Output Loss

Figure 8: Early Arrival of a Vaccine ($T_\eta = 100$)

The results illustrate how critical is this uncertainty. As in the baseline,

the arrival of the vaccine implies a significant relaxation of the employment restrictions and a *fairly large* increase in I. The optimal policy actually increases $\mathcal{R}_t$ (Shown in Appendix 5) quite a bit and this speeds up the peak of the epidemic. Even though using that metric early arrival might appear as bad news this is misleading: the number of averted deaths increases (relative to the baseline) and in the long run is about 1.5%. The long-run output cost is much lower at about 5% of capacity, and the cost per death averted (long run) is significantly smaller than in the baseline (0.6 million)

4.2 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.

4.2.1 Higher $\mathcal{R}_0$

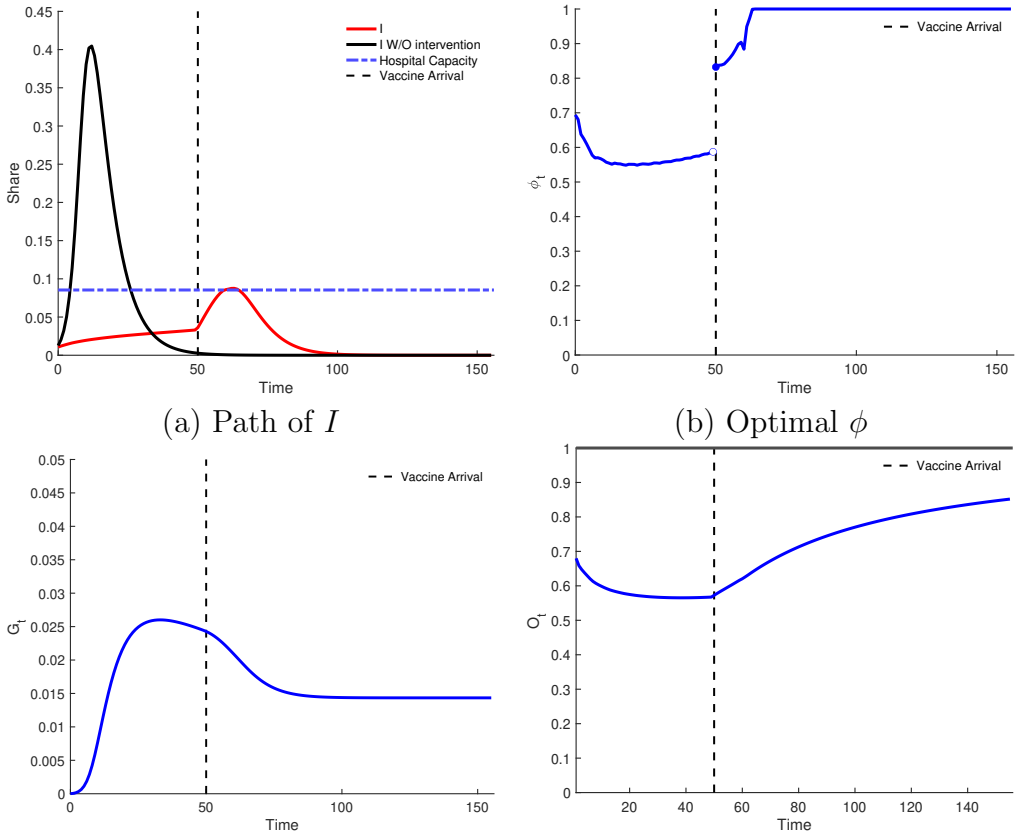
Figure 9 reports the simulation (with $T_\eta = 50$) if the epidemiological parameter $\mathcal{R}_0$ is 4.2 (it was assumed 2.8 in the baseline) as suggested by the estimates of Fernandez-Villaverde and Jones (2020).

The most important differences with the baseline are:

1. The optimal policy implies more stringent employment restrictions. When the vaccine arrives —even though the spread of the epidemic, I, is relatively low— the stay-at-home policy is relaxed but full liberalization takes more than two months. More precisely, at $t = 0$, the optimal policy is such that employment is only 70% of the labor force. This implies that the "measured" $\mathcal{R}_0$ is about 2.

2. Following the arrival of the vaccine there is a sharp increase in the number of infected individuals associated with the partial lifting of restrictions. This increase is much large than in the baseline. The reason is that, relative to the baseline, there are fewer susceptible individuals at $t = 50$.

3. In the long run the number of deaths averted is slightly higher than in the baseline (1.5% vs. 1.2%) and so is the output cost which is about 15% over the 150 weeks that we simulate. This implies that the cost per death averted is about 1.9 million.



(a) Path of I (b) Optimal ϕ

(c) Deaths Averted (d) Output Loss

Figure 9: Higher Infectiousness ($\mathcal{R}_0 = 4$)

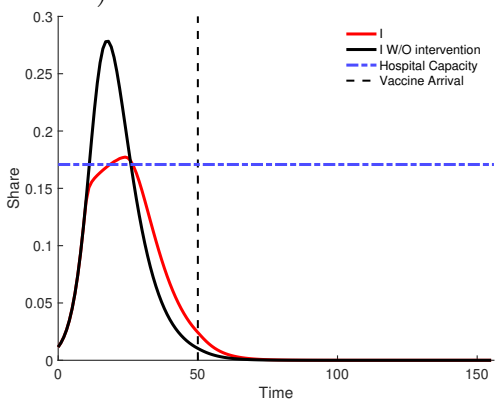
The estimates of $\mathcal{R}_0$ seem to vary significantly across location (see Fernandez-Villaverde and Jones (2020)). To the extent that similar regions differ in terms of this epidemiological parameter the model implies differences in the optimal policies that we hope to more fully capture in the multi-region version of the model.

4.2.2 Optimistic Scenario

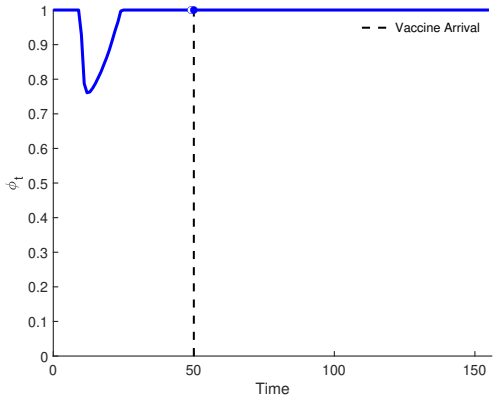
In this scenario we assume that the upper bound of the vaccination rate is six times as high as in the baseline which implies about 30% of susceptibles is vaccinated in one week. [8] We also assume that the expected time until a vaccine arrives is 52 weeks (78 in the baseline) and the actual hospital

[8]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.

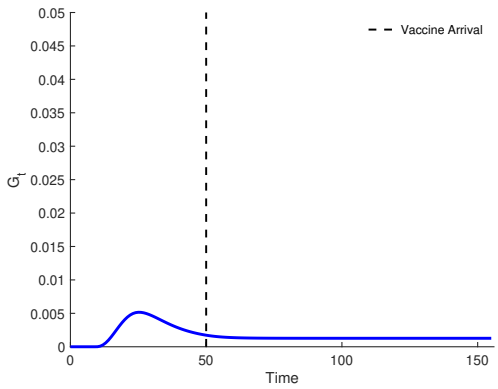
capacity is twice the value in the baseline (280,000 ICU beds for COVID-19 patients)



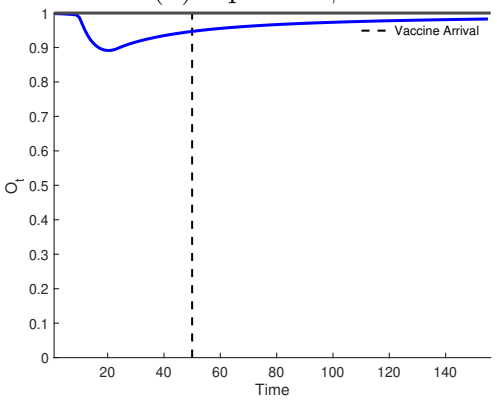
(a) Path of I



(b) Optimal ϕ



(c) Deaths Averted



(d) Output Loss

Figure 10: Optimistic Scenario

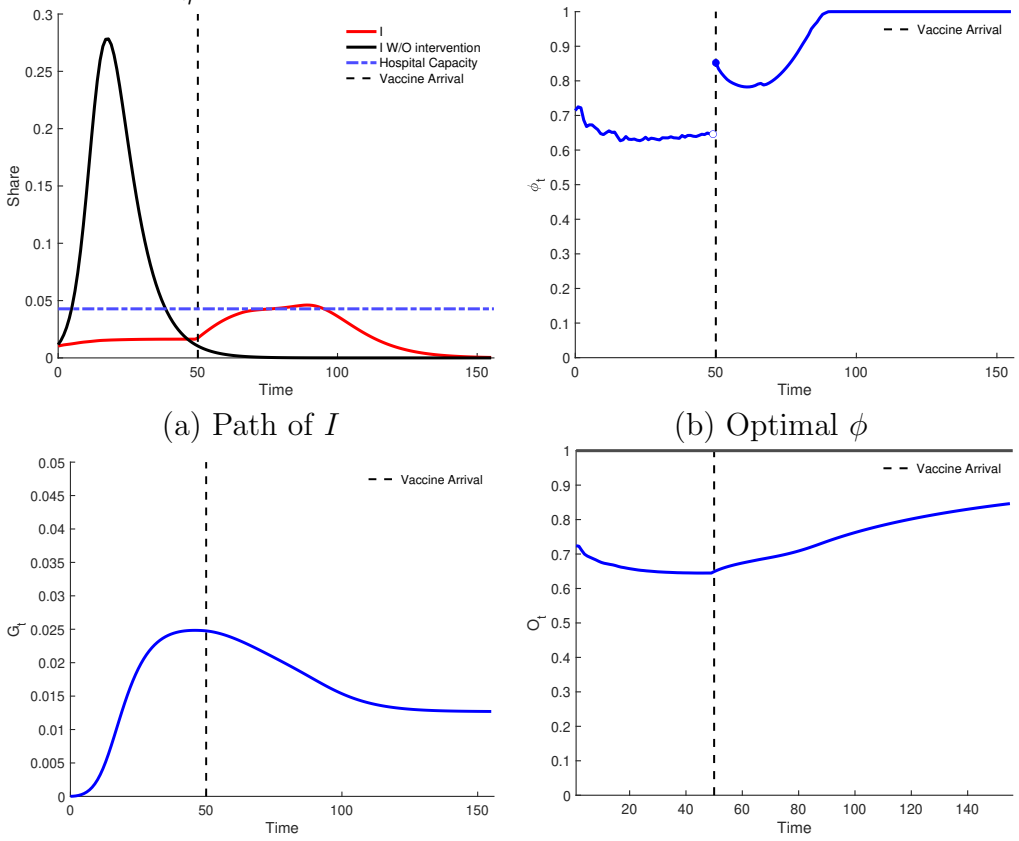
The main takeaways from this alternative are:

1. The optimal policy does not respond until the virus spreads significantly in the population (in our case this when more than 10% have been infected). At that point there is a large decreases in employment (output is about 80% of capacity) that is short lived. By week 25 the economy is at full capacity.

2. The number of deaths averted is small (0.12%) and the output cost per death averted is relatively high (2.5 million)

4.2.3 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 $T_\eta = 50$.[9]



(a) Path of I (b) Optimal ϕ

(c) Deaths Averted (d) Output Loss

Figure 11: 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

[9] 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.

Phase II. In particular, the arrival of a vaccine results in more liberalization of the economy followed by a gradual tightening. In Phase II for the first 20 weeks or so ϕ is around 80%: availability of a vaccine does not imply liberalization. The reason for this is the poorer healthcare infrastructure implicit in this scenario which implies that the value of a vaccine is lower than in an environment with a good health system.

3. The number of deaths averted is similar to the estimate in the baseline and the output cost significantly more elevated: after 50 weeks output does not even reach 70% of capacity. The output cost per life averted is 2.2 million, about twice the value in the baseline.

4.2.4 Higher Social Valuation of Deaths Averted

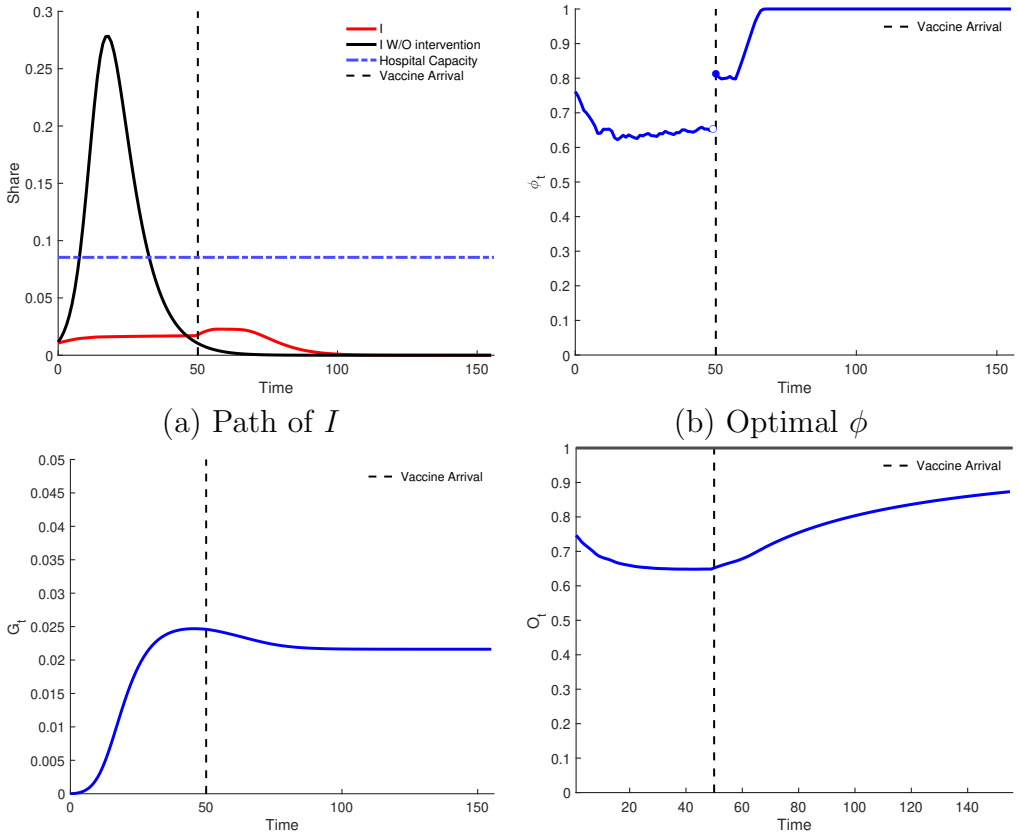
In our baseline we assume that revealed preference arguments suggest that to understand the situations in which society is willing to incur higher than normal costs to avert deaths is useful to focus on higher than normal mortality. However, it is not clear to us what the right societal preferences are in the case of a pandemic. In this section 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.

Figure 12 reports the results. 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.

In this simulation output is about 13% below capacity over the approximately three year period that we report. In the long run, the number of deaths averted exceeds 2% and the cost per death averted is just above 1 million.



(a) Path of I (b) Optimal ϕ

(c) Deaths Averted (d) Output Loss

Figure 12. Higher Social Valuation of Lives Saved

4.3 Interest Rates and Asset Prices

It is straightforward to describe the implications of the model for interest rates and a broad measure of the value of the economy (our proxy to the value of the stock market).

In Phase I the interest rate satisfies

$$r_t^I = \rho + \frac{\dot{c}_t^I}{c_t^I} + \eta \left(\frac{c_t^{II}}{c_t^I} - 1 \right).$$

Since in our one sector model $c_t^j = \phi_t^j w(1 - \zeta I_t)$, for $j = I, II$, it follows that

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

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). In most of our simulations $r_t^{II} > \rho$ when the economy switches to Phase II but it converges to the long run level rather rapidly.

The path followed by the real interest rate in our baseline is in Figure 13

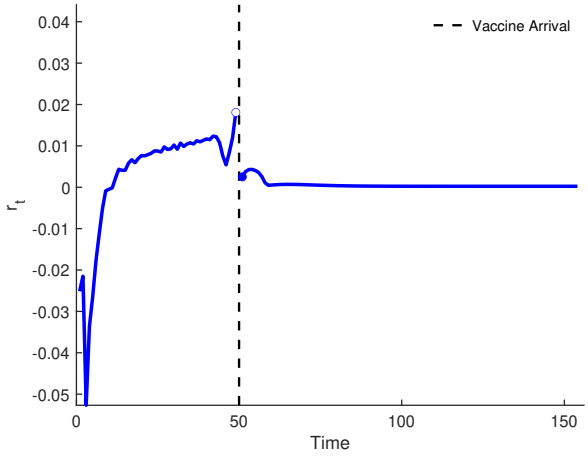


Figure 13. Real Interest Rate (Baseline)

Our optimal policy implies fairly negative interest rates (note that these are weekly rates) for the first few weeks (about 10), and after that it turns significantly positive until a vaccine becomes available. Shortly after that time the interest rate converges to the long run value.

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.

5 One Region Model: 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 $\bar{\mu} = 0.05$. This implies that the number of infected monotonically decreases to zero is about 6 months.

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 productivity[10]. 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.

6 Concluding Comments

Here we offer some tentative comments about what we have learned from the exercise:

[10]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.

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. Optimal policies also imply —in the majority 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.

3. 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. A late arriving vaccine is very costly, while an early arriving vaccine significantly lowers the costs in terms of output and lives lost.

4. Across many of the scenarios that we have looked at the optimal policy implies a large decrease in employment —it ranges from 20% to almost 40%— and, in many cases average output after one year is less than 80% of potential.

5. The model implies that, in most scenarios, the output cost per death averted exceed 1.5 million, and can be as high as 7 million.

6. Optimal policies imply that the paths assumed for the observed instantaneous contagion rate, $\mathcal{R}_t$, in many simulation studies are quite different from those implied by optimal policies.

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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

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

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 ommit 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: 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 6 weeks, then $\kappa = 1/6 = 0.16$

(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	0.16
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	$\bar{\mu}$	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 force to shut-down by government policies aimed to reduce the infection in the population. Bodenstein, Corsetti, and Guerroeri (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 Simpsek, 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,

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

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

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

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

$\mu = 0$

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

FOC:

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

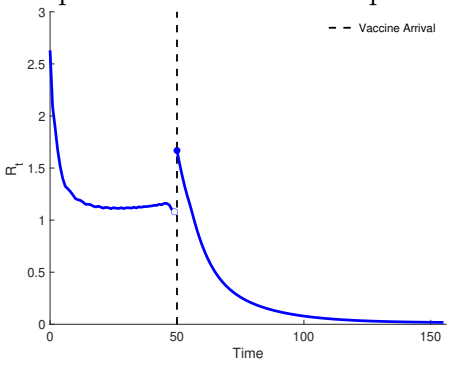
Where,

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

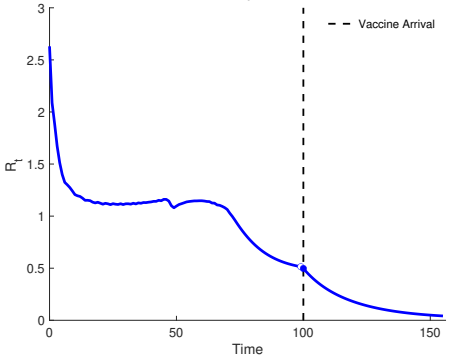
7 Appendix 5: Supplementary Graphs

7.1 Implied Contagion Factor $\mathcal{R}_t$

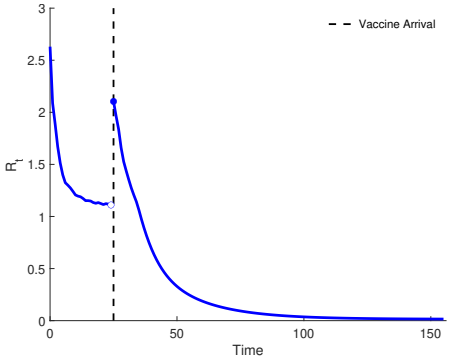
Here we present the model's implications for measured $\mathcal{R}_t$ in several cases.



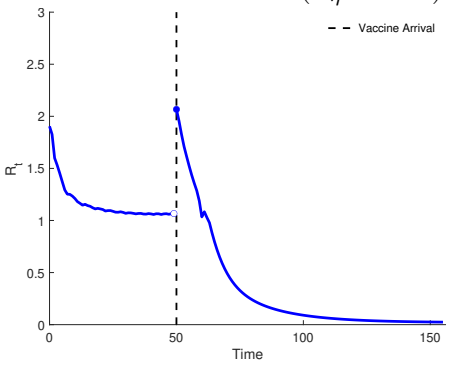
Baseline



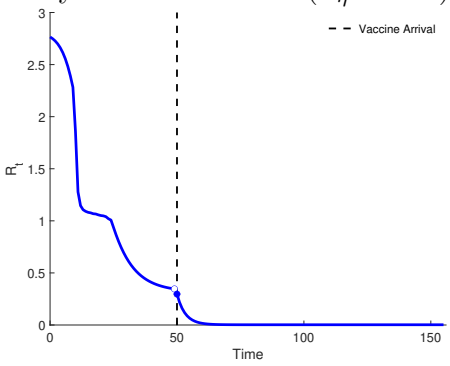
Late Arrival Vaccine ($T_\eta = 100$)



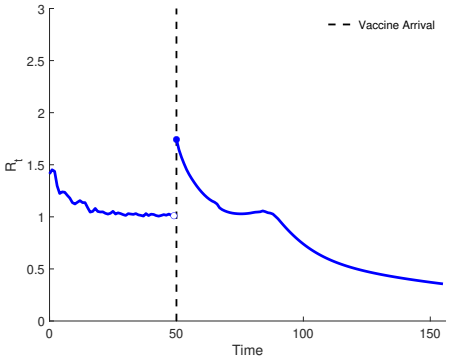
Early Arrival Vaccine ($T_\eta = 100$)



$\mathcal{R}_0 = 4$



Optimistic Scenario

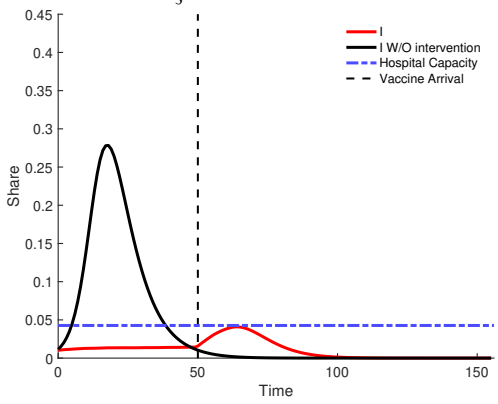


Pessimistic Scenario

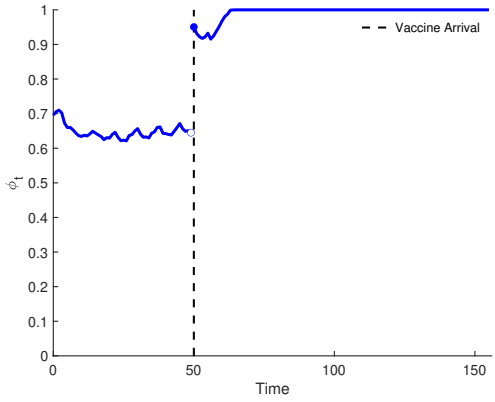
Time Path for $\mathcal{R}_t$

7.2 Higher ζ

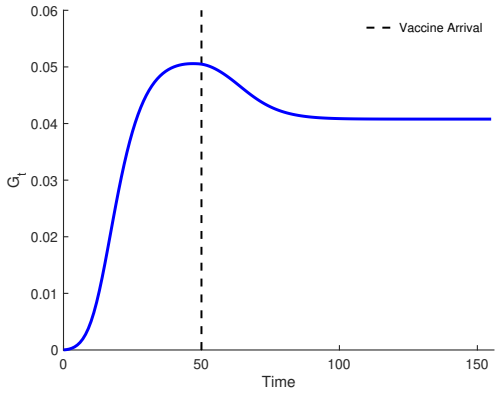
Basic data if $\zeta = 0.20$.



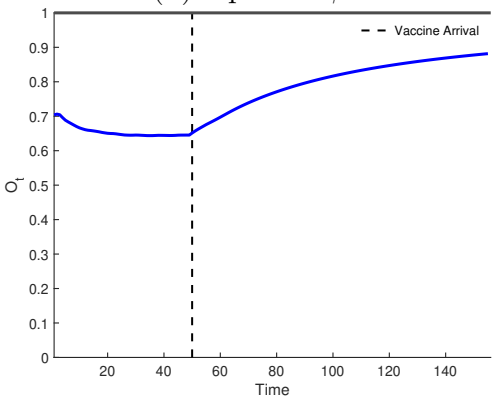
(a) Path of I



(b) Optimal ϕ



(c) Deaths Averted

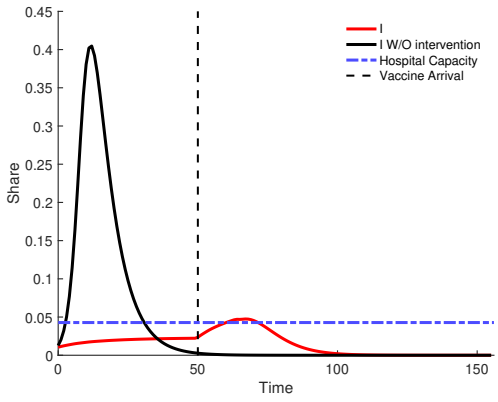


(d) Output Loss

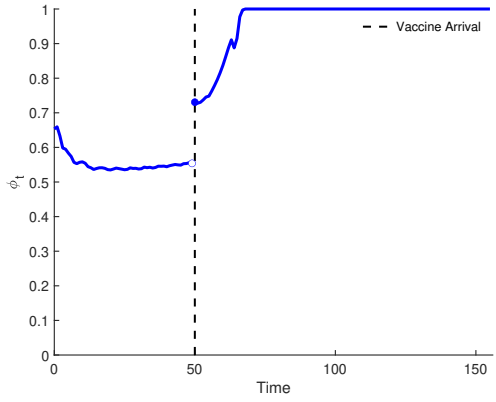
Lower Asymptomatic Cases ($\zeta = 0.2$)

7.3 Pessimistic Scenario II

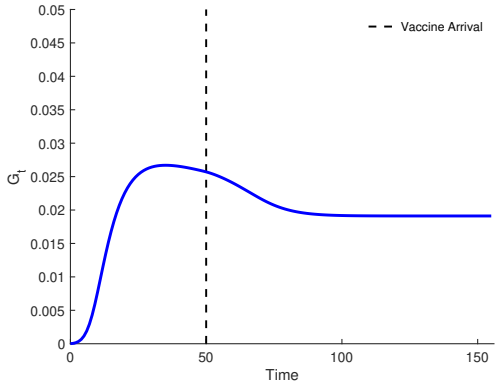
Assumes $\mathcal{R}_0 = 4$ and $\bar{H}' = (1/2)\bar{H}$



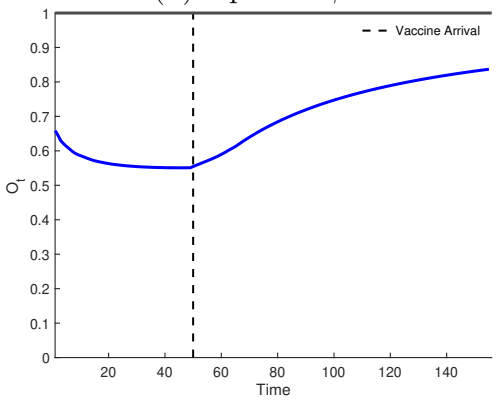
(a) Path of I



(b) Optimal ϕ



(c) Deaths Averted



(d) Output Loss

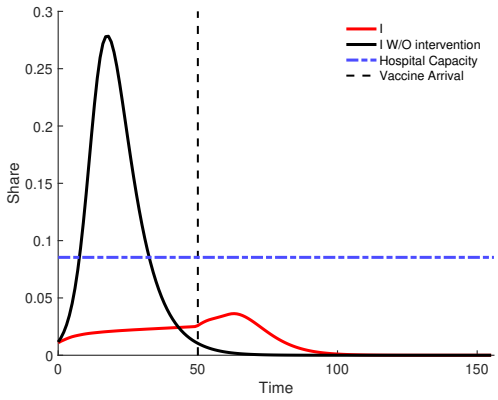
Lower Asymptomatic Cases ($\zeta = 0.2$)

7.4 Intermediate Social Valuation of Deaths Averted

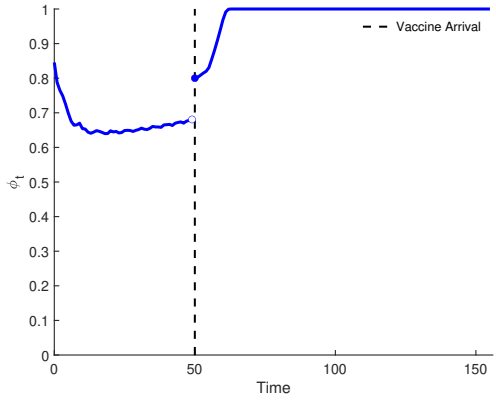
Our valuation of deaths averted function is given by

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

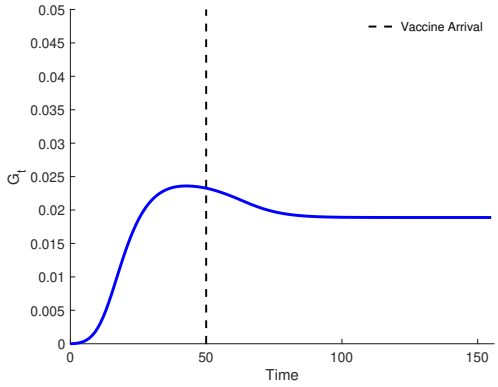
Our baseline assumes that $k = 0$ which implies that only excess deaths enter the social utility function. Our "Higher Social Value of Life" scenario assumes that $k = 1$ which implies that all averted deaths are counted equally. Here we show the intermediate case $k = 1$ that counts all deaths but puts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



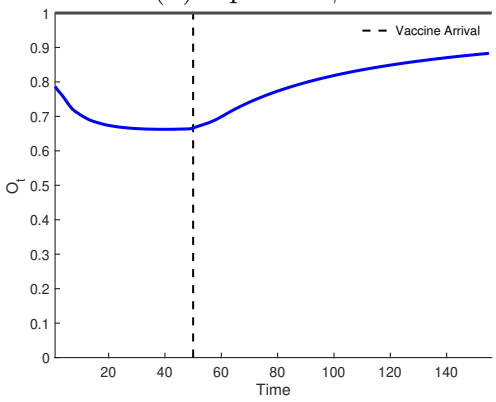
(a) Path of I



(b) Optimal ϕ



(c) Deaths Averted



(d) Output Loss

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