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THE MACROECONOMICS OF EPIDEMICS

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ABSTRACT

We extend the canonical epidemiology model to study the interaction between economic decisions and epidemics. Our model implies that people cut back on consumption and work to reduce the chances of being infected. These decisions reduce the severity of the epidemic but exacerbate the size of the associated recession. The competitive equilibrium is not socially optimal because infected people do not fully internalize the effect of their economic decisions on the spread of the virus. In our benchmark model, the best simple containment policy increases the severity of the recession but saves roughly half a million lives in the United States.

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A Matlab replication codes can be downloaded directly from the URL: https://tinyurl.com/ERTcode

1 Introduction

As COVID-19 spreads throughout the world, governments are struggling with how to understand and manage the epidemic. Epidemiology models have been widely used to predict the course of the epidemic (e.g., Ferguson et al. 2020). While these models are very useful, they do have a significant shortcoming: they do not allow for the interaction between economic decisions and rates of infection.

Policy makers certainly appreciate this interaction. For example, in an op-ed piece, Ben Bernanke and Janet Yellen (2020) write: "In the near term, public health objectives necessitate people staying home from shopping and work, especially if they are sick or at risk. So production and spending must inevitably decline for a time."

In this paper, we extend the canonical SIR model proposed by Kermack and McKendrick (1927) to study the equilibrium interaction between economic decisions and epidemic dynamics.[1] Our model features a two-way interaction between the epidemic and the economy. People's decisions to cut back on consumption and work reduce the severity of the epidemic, as measured by total deaths. These same decisions exacerbate the size of the recession caused by the epidemic.

In our model, an epidemic has both aggregate demand and aggregate supply effects. The supply effect arises because the epidemic exposes workers to the virus. Workers react to that risk by reducing their labor supply. The demand effect arises because the epidemic exposes consumers to the virus. Consumers react to that risk by reducing consumption. The supply and demand effects work together to generate a large, persistent recession.

The competitive equilibrium is not Pareto optimal because people infected with the virus do not fully internalize the effect of their consumption and work decisions on the spread of the virus. To be clear, this market failure does not reflect a lack of good intentions or irrationality on the part of infected people. It simply reflects the fact that each infected person takes economywide infection rates as given. But collectively, their behavior does change infection rates, thereby imposing unpriced costs on susceptible people.[2]

A natural question is: what policies should the government pursue to deal with the infection externality? We focus on simple containment policies that reduce consumption and

[1] SIR is an acronym for susceptible, infected, recovered, and removed.

[2] The behavior of susceptible people is also different in the competitive equilibrium and the Pareto optimum. This difference can also be interpreted as an externality because it influences the dynamics of the epidemic and thus affects the number of people killed by the virus. See Rachel (2020) and Garibaldi, Moen, and Pissarides (2020) for a discussion of this effect.

hours worked. By reducing economic interactions among people, these policies exacerbate the recession but raise welfare by reducing the death toll caused by the epidemic. We find that it is optimal to introduce large-scale containment measures that result in a sharp, sustained drop in aggregate output. In our benchmark model, when health care capacity is limited and vaccines and treatments don't arrive before the epidemic is over, containment policy saves roughly half a million lives in the United States.

To make the intuition for our results as transparent as possible, we use a relatively simple model. A cost of that simplicity is that we cannot study many important policy issues related to the epidemic. For example, we do not consider policies that mitigate the economic hardships suffered by households and businesses. Such policies include fiscal transfers to households and loans to keep firms from going bankrupt. We also do not study policies aimed at maintaining the liquidity and health of financial markets.

Finally, we abstract from nominal rigidities that could play an important role in determining the short-run response of the economy to an epidemic. For example, if prices are sticky, a given fall in the demand for consumption would generate a larger recession. Other things equal, a larger recession would mitigate the spread of the infection.[3] But we are confident that the central message from our current analysis will be robust: there is an inevitable trade-off between the severity of the recession and the health consequences of the epidemic.[4]

Our point of departure is the canonical SIR model proposed by Kermack and McKendrick (1927). In this model, the transition probabilities between health states are exogenous parameters. We modify the model by assuming that purchasing consumption goods and working brings people in contact with each other. These activities raise the probability that the infection spreads. We refer to the resultant framework as the *SIR-macro* model.

We choose parameters so that the Kermack-McKendrick SIR model is consistent with the scenario outlined by Chancellor Angela Merkel in her speech on March 11, 2020 (Bennhold and Eddy 2020). According to this scenario, "60% to 70% of the population will be infected as long as this remains the situation." Using 60% as our benchmark value, the SIR model implies that the share of the initial population infected peaks at 6.8%. Applying this scenario to the United States implies that roughly 200 million Americans will eventually become infected and 1 million people will die. An obvious shortcoming of the SIR model is that people do not take any actions to reduce the chances of becoming infected and infection dynamics are

[3] In a follow-up to this paper, Eichenbaum, Rebelo, and Trabandt (2020b) incorporate nominal rigidities and physical investment into the model.

[4] In an interesting essay, Gourinchas (2020) makes a similar point.

not influenced by the level of economic activity.

The interaction between economic activity and transition probabilities in the SIR-macro model substantially changes the dynamics of the epidemic and its economic impact. One way to assess this impact is to focus on the simplest version of the SIR-macro model that abstracts from the possibility of vaccinations, medical treatments, and limited health care capacity. Relative to the SIR model, this simple SIR-macro model implies a sharper recession and fewer deaths. The average fall in aggregate consumption in the first year of the epidemic is roughly seven times larger than in the SIR model (4.7% vs. 0.7%). This larger decline in economic activity reduces the infection peak (5.2% vs. 6.8%) as well as the percentage of the population who becomes infected (54% vs. 60%). Critically, the total number of U.S. deaths caused by the epidemic falls from 1 million to $890,000$.

To design optimal policy, one must understand how epidemics end. In both the SIR and SIR-macro models, epidemics end when a sufficiently high fraction of the population acquires immunity so that the number of infections no longer rises (i.e., the population achieves "herd immunity"). Absent vaccines, the only way to acquire immunity is to become infected and recover. Sadly, without effective medical treatments, this process involves the death of many people. In all versions of our model, it is optimal for policy makers to avoid recurrent epidemics. So a key question for policy is: what is the optimal way to reach herd immunity?

In the SIR-macro model, it is possible to prevent the infection from spreading by adopting large, permanent containment measures. This approach has two problems. First, the permanent containment measures create a persistent economic depression. Second, the population never reaches herd immunity. So, infections would recur if containment was ever relaxed.

The best policy in this world is to curtail consumption when externalities are large, that is, when the number of infected people is high. Such a policy involves gradually ramping up containment measures as infections rise and slowly relaxing them as new infections wane and the population approaches herd immunity.

An important concern in many countries is that the health care system can be overwhelmed by a large number of infected people. To analyze this scenario, we extend the simple SIR-macro model so that the case fatality rate (the probability of dying conditional on being infected) is an increasing function of the number of people infected. We find that the competitive equilibrium involves a much larger recession as people internalize the higher

case fatality rates. People cut back more aggressively on consumption and work to reduce the probability of being infected. As a result, fewer people are infected in the competitive equilibrium, but more people die. The optimal policy involves a much more aggressive response than in the simple SIR-macro economy. The reason is that the cost of the externality is much larger since a larger fraction of the infected population dies.

How does the possibility of an effective treatment being discovered change our results? The qualitative implications are clear: people become more willing to engage in market activities because the expected cost of being infected is smaller. So, along a path in which treatment is not actually discovered, the recession induced by the epidemic is less severe. Sadly, along such a path, the total number of infected people and the death toll rise relative to the baseline SIR-macro model. That said, the quantitative difference between this model and the baseline SIR-macro model is quite small, with respect to both the competitive equilibrium and the best containment policy.

How does the possibility of a vaccine being discovered change our results? Vaccines don't cure infected people, but they do prevent susceptible people from becoming infected. In contrast, treatments cure infected people but do not prevent future infections. Given our benchmark calibration, these differences are not very important for the competitive equilibrium. But they are very important for the design of optimal policy. With vaccination as a possibility, it is optimal to *immediately* introduce severe containment measures to minimize deaths. Those measures cause a large recession. But this recession is worth incurring in the hope that a vaccine arrives before many people get infected.

The most general version of our model, discussed in Section 6, incorporates the probabilistic development of vaccines and treatments, as well as a case fatality rate that rises with the number of infected people. The latter feature reflects capacity constraints in the health care system. We refer to this version of the model as the benchmark SIR-macro model.

In this model, it is optimal to immediately introduce severe containment measures and increase those measures as more of the population is infected. The best containment policy dramatically increases the magnitude of the recession. Absent containment measures, average consumption falls by about 7% in the first year of the epidemic. With optimal containment, average consumption falls by 22%. Notably, the size of the recession is smaller than in the medical preparedness model. The reason is that the prospect of vaccinations and treatments reduces the magnitude of the externality associated with the medical preparedness problem.

The benefit of the large recession associated with optimal containment in the benchmark model is a less severe epidemic. Compared to the competitive equilibrium, the peak infection rate drops from 4.7% to 2.5% of the initial population. The optimal policy reduces the death toll as a percentage of the initial population from 0.40% to 0.26%. For the United States, this reduction amounts to about half a million lives.

We emphasize that these numbers pertain to a worst-case scenario in which vaccines and treatments never arrive. If they do arrive, many more lives would be saved. Thankfully, they would be saved by medicine rather than by containment policies.

Finally, we quantify the effects of delaying or prematurely ending optimal containment policies. Abandoning containment policies prematurely leads to an initial economic recovery. But it also leads to a large rise in infection rates. That rise causes a new, persistent recession. Tragically, the overall death toll rises because optimal policy was abandoned.

Suppose that containment policies are designed and implemented well into an infection episode. At that point, it is optimal to adopt extreme containment measures that cause a large recession. The reason is simple: the longer is the delay, the larger is the number of infections and the externalities associated with economic activity. Optimal policy then involves draconian containment to offset those externalities. Even so, the overall death toll is much larger than if containment had been implemented without delay.

The simple containment strategy that we study mimics a key feature of existing policies: containment applies equally to everyone, regardless of their health status. A natural question is: how much better could a benevolent government do if it could directly choose the consumption and hours worked of susceptible, infected, and recovered people?

We answer this question by solving the relevant social-planning problem. This solution, which we call "smart containment," requires that infected people don't work unless they recover. This isolation policy means that susceptible people can work without the risk of becoming infected. The amount that susceptible and recovered people consume is the same as in the pre-epidemic steady state. Consumption of infected people depends on whether it is feasible to deliver goods to them without the risk of infecting other people. In any event, the economy does not suffer in any meaningful way from a recession. Moreover, the overall death toll of the epidemic is very small, with the number of infected people declining monotonically from its initial level to zero.

The previous results point to the importance of antigen and antibody tests that would allow health care professionals to quickly ascertain people's health status. The social returns

to gathering this information and acting on it are enormous. These actions reduce both the death toll and the size of the economic contraction relative to the outcomes associated with the best simple containment policy.

2 The SIR-Macro Model

In this section, we describe the economy before the start of the epidemic. We then present the SIR-macro model.

2.1 The preinfection economy

The economy is populated by a continuum of ex ante identical people with measure one. Prior to the start of the epidemic, everybody is identical and maximizes the objective function

$$U = \sum_{t=0}^{\infty} \beta^t u(c_t, n_t).$$

Here, $\beta \in (0, 1)$ denotes the discount factor and c_t and n_t denote consumption and hours worked, respectively. For simplicity, we assume that momentary utility takes the form

$$u(c_t, n_t) = \ln c_t - \frac{\theta}{2} n_t^2.$$

The budget constraint of the representative person is

$$(1 + \mu_t)c_t = w_t n_t + \Gamma_t.$$

Here, w_t denotes the real wage rate, μ_t is a Pigouvian tax rate on consumption, and Γ_t denotes lump-sum transfers from the government. As discussed below, we think of μ_t as a proxy for containment measures aimed at reducing social interactions. For this reason, we refer to μ_t as the containment rate. In Section 5, we study an alternative way to model containment that does not involve taxation but yields very similar results.

The first-order condition for the representative person's problem is

$$(1 + \mu_t)\theta n_t = c_t^{-1} w_t.$$

There is a continuum of competitive representative firms of unit measure that produce consumption goods (C_t) using hours worked (N_t) according to the technology

$$C_t = AN_t.$$

The firm chooses hours worked to maximize its time-t profits Π_t

$$\Pi_t = AN_t - w_t N_t.$$

The government's budget constraint is given by

$$\mu_t c_t = \Gamma_t.$$

In equilibrium, $n_t = N_t$ and $c_t = C_t$.

2.2 The outbreak of an epidemic

Epidemiology models generally assume that the probabilities governing the transition between different states of health are exogenous with respect to economic decisions. We modify the canonical SIR model proposed by Kermack and McKendrick (1927) so that these transition probabilities depend on people's economic decisions. Since purchasing consumption goods or working brings people into contact with each other, we assume that the probability of becoming infected depends on these activities.

The population is divided into four groups: susceptible (people who have not yet been exposed to the disease), infected (people who contracted the disease), recovered (people who survived the disease and acquired immunity), and deceased (people who died from the disease). The fractions of people in these four groups are denoted by S_t, I_t, R_t, and D_t, respectively. The number of newly infected people is denoted by T_t.[5]

Susceptible people can become infected in three ways. First, they can meet infected people while purchasing consumption goods. The number of newly infected people that results from these interactions is given by $\pi_1(S_t C_t^s)(I_t C_t^i)$. The terms $S_t C_t^s$ and $I_t C_t^i$ represent total consumption expenditures by susceptible and infected people, respectively. The parameter π_1 reflects both the amount of time spent shopping and the probability of becoming infected as a result of that activity. In reality, different types of consumption involve different amounts of contact with other people. For example, attending a rock concert is much more

[5] We assume that people know their current health status. In subsequent work, Eichenbaum, Rebelo, and Trabandt (2020a), we develop a model in which people do not know their health status unless they are tested.

contact intensive than going to a grocery store. For simplicity, we abstract from this type of heterogeneity.

Second, susceptible and infected people can meet at work. The number of newly infected people that results from interactions at work is given by $\pi_2(S_t N_t^s)(I_t N_t^i)$. The terms $S_t N_t^s$ and $I_t N_t^i$ represent total hours worked by susceptible and infected people, respectively. The parameter π_2 reflects the probability of becoming infected as a result of work interactions. We recognize that different jobs involve different levels of social contact. For example, working as a dentist or a waiter is much more contact intensive than writing software. Again, for simplicity, we abstract from this source of heterogeneity.

Third, susceptible and infected people can meet in ways not directly related to consuming or working, for example, meeting a neighbor or riding an elevator. The number of random meetings between infected and susceptible people is $S_t I_t$. These meetings result in $\pi_3 S_t I_t$ newly infected people.

The total number of newly infected people is given by[6]

$$T_t = \pi_1 \left(S_t C_t^s\right)\left(I_t C_t^i\right) + \pi_2 \left(S_t N_t^s\right)\left(I_t N_t^i\right) + \pi_3 S_t I_t. \tag{1}$$

Kermack and McKendrick's (1927) SIR model is a special case of our model in which the propagation of the disease is unrelated to economic activity ($\pi_1 = 0$, $\pi_2 = 0$).

The number of susceptible people at time $t+1$ is equal to the number of susceptible people at time t minus the number of susceptible people who got infected at time t:

$$S_{t+1} = S_t - T_t. \tag{2}$$

The number of infected people at time $t+1$ is equal to the number of infected people at time t plus the number of newly infected (T_t) minus the number of infected people who recovered ($\pi_r I_t$) and the number of infected people who died ($\pi_d I_t$):

$$I_{t+1} = I_t + T_t - (\pi_r + \pi_d) I_t. \tag{3}$$

Here, π_r is the rate at which infected people recover from the infection and π_d is the case fatality rate, that is, the probability that an infected person dies.

The timing convention implicit in Equation (3) is as follows. Social interactions happen in the beginning of the period (infected and susceptible people meet). Then, changes in

[6] To simplify, we assume that the probability of a given person being infected through more than one form of social interactions is zero. In addition, we do not explicitly incorporate the constraint that T_t must be between zero and the size of population. This constraint is satisfied in all our simulations.

health status unrelated to social interactions (recovery or death) occur. At the end of the period, the consequences of social interactions materialize: T_t susceptible people become infected.

The number of recovered people at time $t+1$ is the number of recovered people at time t plus the number of infected people who just recovered ($\pi_r I_t$),

$$R_{t+1} = R_t + \pi_r I_t. \tag{4}$$

Finally, the number of deceased people at time $t+1$ is the number of deceased people at time t plus the number of new deaths ($\pi_d I_t$),

$$D_{t+1} = D_t + \pi_d I_t. \tag{5}$$

Total population, Pop_{t+1}, evolves according to

$$\text{Pop}_{t+1} = \text{Pop}_t - \pi_d I_t,$$

with $\text{Pop}_0 = 1$.

We assume that at time zero, a fraction ε of susceptible people is infected by a virus through zoonotic exposure, that is, the virus is directly transmitted from animals to humans:

$$I_0 = \varepsilon,$$

$$S_0 = 1 - \varepsilon.$$

Everybody is aware of the initial infection and understands the laws of motion governing population health dynamics. Critically, people take as given aggregate variables, such as $I_t C_t^i$ and $I_t N_t^i$.

We now describe the optimization problem of different types of people in the economy. The variable U_t^j denotes the time-t lifetime utility of a type-j person ($j = s, i, r$). The budget constraint of a type-j person is

$$(1+\mu_t)c_t^j = w_t \phi^j n_t^j + \Gamma_t, \tag{6}$$

where c_t^j and n_t^j denote the consumption and hours worked of a type-j person, respectively. The parameter governing labor productivity, ϕ^j, is equal to one for susceptible and recovered people ($\phi^s = \phi^r = 1$) and less than one for infected people ($\phi^i < 1$).

The budget constraint (6) embodies the assumption that people cannot pool risk associated with the infection. Going to the opposite extreme and assuming complete markets

considerably complicates the analysis without necessarily making the model more realistic.

Susceptible people. The lifetime utility of a susceptible person, U_t^s, is

$$U_t^s = u(c_t^s, n_t^s) + \beta \left[(1 - \tau_t) U_{t+1}^s + \tau_t U_{t+1}^i \right]. \tag{7}$$

Here, the variable τ_t represents the probability that a susceptible person becomes infected

$$\tau_t = \pi_1 c_t^s \left(I_t C_t^i \right) + \pi_2 n_t^s \left(I_t N_t^i \right) + \pi_3 I_t. \tag{8}$$

Critically, susceptible people understand that consuming less and working less reduce the probability of becoming infected.

The first-order conditions for consumption and hours worked are

$$u_1(c_t^s, n_t^s) - (1 + \mu_t)\lambda_{bt}^s + \lambda_{\tau t}\pi_1 \left(I_t C_t^i \right) = 0,$$

$$u_2(c_t^s, n_t^s) + w_t \lambda_{bt}^s + \lambda_{\tau t}\pi_2 \left(I_t N_t^i \right) = 0.$$

Here, λ_{bt}^s and $\lambda_{\tau t}$ are the Lagrange multipliers associated with constraints (6) and (8), respectively.

The first-order condition for τ_t is

$$\beta \left(U_{t+1}^i - U_{t+1}^s \right) - \lambda_{\tau t} = 0. \tag{9}$$

Infected people. The lifetime utility of an infected person, U_t^i, is

$$U_t^i = u(c_t^i, n_t^i) + \beta \left[(1 - \pi_r - \pi_d) U_{t+1}^i + \pi_r U_{t+1}^r \right]. \tag{10}$$

The expression for U_t^i embodies a common assumption in macro and health economics that the cost of death is the forgone utility of life.

The first-order conditions for consumption and hours worked are given by

$$u_1(c_t^i, n_t^i) = \lambda_{bt}^i (1 + \mu_t),$$

$$u_2(c_t^i, n_t^i) = -\phi^i w_t \lambda_{bt}^i,$$

where λ_{bt}^i is the Lagrange multiplier associated with constraint (6).

Recovered people. The lifetime utility of a recovered person, U_t^r, is

$$U_t^r = u(c_t^r, n_t^r) + \beta U_{t+1}^r. \tag{11}$$

The first-order conditions for consumption and hours worked are

$$u_1(c_t^r, n_t^r) = \lambda_{bt}^r(1 + \mu_t),$$

$$u_2(c_t^r, n_t^r) = -w_t \lambda_{bt}^r,$$

where λ_{bt}^r is the Lagrange multiplier associated with constraint (6).

Government budget constraint. The government budget constraint is

$$\mu_t \left(S_t c_t^s + I_t c_t^i + R_t c_t^r \right) = \Gamma_t \left(S_t + I_t + R_t \right).$$

Equilibrium. In equilibrium, each person solves his or her maximization problem and the government budget constraint is satisfied. In addition, the goods and labor markets clear:

$$S_t C_t^s + I_t C_t^i + R_t C_t^r = A N_t,$$

$$S_t N_t^s + I_t N_t^i \phi^i + R_t N_t^r = N_t.$$

In addition, $c_t^j = C_t^j$ and $n_t^j = N_t^j$ for $j = s$, i, and r. In the appendix, we describe our algorithm for computing the equilibrium.[7]

3 Medical Preparedness, Treatments, and Vaccination

In this section, we extend the SIR-macro model in three ways. First, we allow for the possibility that the case fatality rate increases with the number of infections. Second, we

[7] Matlab replication codes can be downloaded from the authors' websites or directly from `https://tinyurl.com/ERTcode`.

allow for the probabilistic development of a cure for the disease. Third, we allow for the probabilistic development of a vaccine that inoculates susceptible people against the virus.

3.1 The medical preparedness model

In our basic SIR-macro model, we abstract from the possibility that the efficacy of the health care system deteriorates if a substantial fraction of the population becomes infected. A simple way to model this scenario is to assume that the case fatality rate depends on the number of infected people, I_t:

$$\pi_{dt} = \pi_d + \kappa I_t^2,$$

where $\kappa > 0$. This functional form implies that the case fatality rate is a convex function of the fraction of the population who becomes infected.[8] The basic SIR-macro model corresponds to the special case of $\kappa = 0$.

3.2 The treatment model

The basic SIR-macro model abstracts from the possibility that an effective treatment against the virus will be developed. Suppose instead that an effective treatment that cures infected people is discovered with probability δ_c each period. Once discovered, treatment is provided to all infected people in the period of discovery and in all subsequent periods, thereby transforming them into recovered people. As a result, the number of new deaths from the disease goes to zero.

The lifetime utility of an infected person before the treatment becomes available is

$$U_t^i = u(c_t^i, n_t^i) + (1 - \delta_c)\left[(1 - \pi_r - \pi_d)\,\beta U_{t+1}^i + \pi_r \beta U_{t+1}^r\right] + \beta \delta_c U_{t+1}^r. \tag{12}$$

This expression reflects the fact that with probability $1-\delta_c$, a person who is infected at time t remains so at time $t+1$. With probability δ_c, this person receives treatment and becomes recovered.

We now discuss the impact of an effective treatment on population dynamics. Before the treatment is discovered, population dynamics evolve according to Equations (1), (2), (3), (4), and (5). Suppose that the treatment is discovered at the beginning of time t^*. Then, all

[8] We do not explicitly impose the constraint that $\pi_{dt} < 1$, but this constraint is satisfied in all our simulations.

infected people become recovered. The number of the deceased stabilizes once the treatment arrives, so for $t \geq t^*$,

$$D_t = D_{t^*}.$$

All infected people are instantly cured. Since no one dies from the disease, we normalize the number of susceptible people to zero for $t > t^*$. The number of recovered people is given by

$$R_t = 1 - D_t.$$

3.3 The vaccination model

The basic SIR-macro model abstracts from the possibility that a vaccine against the virus will be developed. Suppose instead that a vaccine is discovered with probability δ_v per period. Once discovered, the vaccine is immediately provided to all susceptible people.

The lifetime utility of a susceptible person is given by

$$U_t^s = u(c_t^s, n_t^s) + (1 - \delta_v)(1 - \tau_t)\beta U_{t+1}^s + \delta_v (1 - \tau_t)\beta U_{t+1}^r + \tau_t \beta U_{t+1}^i. \tag{13}$$

This expression reflects the fact that with probability $1 - \delta_v$, a person who is susceptible at time t and did not get infected remains susceptible at time $t + 1$. With probability δ_v, this person is vaccinated and becomes immune to the disease. So, at time $t + 1$, this person's health situation is identical to that of a recovered person. The vaccine does not affect the health status of people who are infected or recovered. The lifetime utilities of infected and recovered people are given by (10) and (11), respectively.

We now discuss the impact of vaccinations on population dynamics. Before the vaccine is discovered, these dynamics evolve according to Equations (1), (2), (3), (4), and (5). Suppose that the vaccine is discovered at the beginning of time t^*. Then, all susceptible people become recovered. Since no one is susceptible, there are no new infections.

Denote the number of susceptible and recovered people right after a vaccine is introduced at time t^* by S'_{t^*} and R'_{t^*}. The values of these variables are

$$S'_{t^*} = 0$$

$$R'_{t^*} = R_{t^*} + S_{t^*}.$$

For $t \geq t^*$, we have

$$R_{t+1} = \begin{cases} R'_t + \pi_r I_t & \text{for } t = t^*, \\ R_t + \pi_r I_t & \text{for } t > t^*. \end{cases}$$

The laws of motion for I_t and D_t are given by (3) and (5).

4 Competitive Equilibrium

In this section, we discuss the properties of the competitive equilibrium via a series of numerical exercises. In the first subsection, we describe our parameter values. In the second and third subsections, we discuss how the economy responds to an epidemic in the SIR and SIR-macro models, respectively. In the fourth subsection, we discuss the implications of medical preparedness. In the fifth subsection, we discuss the effects of treatments and vaccines. Finally, in the sixth subsection, we discuss the robustness of our results.

4.1 Parameter values

In this subsection we report our choice of parameters. We are conscious of the considerable uncertainty about the true values of these parameters. Below, we report the robustness of our results to using different parameter configurations.

Each time period corresponds to a week. To choose the case fatality rate, π_d, we use data from the South Korean Ministry of Health and Welfare from March 16, 2020.[9] These estimates are relatively reliable because, as of late March, South Korea had the world's highest per capita test rates for COVID-19 (Pueyo 2020). Estimates of case fatality rates based on data from other countries are probably biased upward because the number of infected people is likely to be underestimated. We compute the weighted average of the case fatality rates using the percentage of the U.S. population in different age groups as weights. If we exclude people that are 70 and older because their labor force participation rate is very low, we obtain an average case fatality rate of 0.4%. If we exclude people that are 75 and older, we obtain an average case fatality rate of 0.7%. Based on these estimates, we set the case fatality rate equal to 0.5% and report robustness results below. Our baseline case fatality rate is consistent with the estimates reported in Salje (2020).

[9] This estimate is roughly eight times greater than the average influenza case fatality rate in the United States.

As in Atkeson (2020), we assume that it takes 18 days on average to either recover or die from the infection. Since our model is weekly, we set $\pi_r + \pi_d = 7/18$. A 0.5% case fatality rate for infected people implies $\pi_d = 7 \times 0.005/18$.

We now discuss our calibration procedure to choose the values of π_1, π_2, and π_3. It is common in epidemiology to assume that the relative importance of different modes of transmission is similar across viruses that cause respiratory diseases. Ferguson et al. (2006) argue that, in the case of influenza, 30% of transmissions occur in the household, 33% in the general community, and 37% in schools and workplaces.

To map these estimates into our transmission parameters, we proceed as follows. We use the Bureau of Labor Statistics 2018 American Time Use Survey (ATUS) to estimate the percentage of time spent on "general community activities" that is devoted to consumption. We compute the latter as the fraction of time spent on "purchasing goods and services" or "eating and drinking outside the home." To estimate the time spent "eating and drinking outside the home," we multiply the time spent "eating and drinking" by the fraction of total food expenditures on "food away from home" in 2018 (54% according to the U.S. Department of Agriculture).[10] These considerations imply that the fraction of time spent on general community activities related to consumption activities is 48%. Since 33% of transmissions occur in the general community, we estimate that 16% of transmissions are related to consumption (0.33×0.48, which is roughly one-sixth).

Turning to work, recall that 37% of transmissions occur in schools and workplaces. To compute the fraction of transmissions that occur in the workplace, we weight the number of students by 10 and the number of workers by 4. These weights are the average number of contacts per day at school and work reported by Lee et al. (2010). According to the Bureau of Labor Statistics, the number of students and workers in the population in 2018 is 76.6 million and 162.1 million, respectively. These considerations imply that the fraction of transmissions occurring in the workplace is 46% ($162.1 \times 4/(162.1 \times 4 + 76.6 \times 10)$). Since 37% of transmissions occur in schools and workplaces, 17% of transmissions are related to work (0.37×0.46, which is roughly one-sixth).

We assume that virus transmission unrelated to consumption or work activities belongs to the exogenous category ($\pi_3 S_t I_t$) emphasized in the SIR model. The values of π_1, π_2, and π_3 are chosen to satisfy

[10] We classify the following entries in the ATUS survey as general community activities: purchasing goods and services; eating and drinking outside the home; organizational, civic, and religious activities; socializing and communicating; sports, exercise, and recreation; and caring for and helping nonhousehold members.

$$\frac{\pi_1 C^2}{\pi_1 C^2 + \pi_2 N^2 + \pi_3} = 1/6,$$
$$\frac{\pi_2 N^2}{\pi_1 C^2 + \pi_2 N^2 + \pi_3} = 1/6.$$

Here, C and N are consumption and hours worked in the preinfection steady state. In addition, we assume that at the limit of the simple SIR model, 60% of the population either recovers from the infection or dies. This assumption corresponds to the Merkel scenario discussed in the introduction. The implied values for π_1, π_2, and π_3 are 7.8408×10^{-8}, 1.2442×10^{-4}, and 0.3901, respectively.

Our calibration procedure requires various judgment calls. For example, we had to choose which categories to include in "general community activities."[11] For this reason, we report robustness results below.

The initial population is normalized to one. The number of people who are initially infected, ε, is 0.001. We choose $A = 39.835$ and $\theta = 0.001275$ so that in the pre-epidemic steady state, the representative person works 28 hours per week and earns a weekly income of $\$58,000/52$. We obtain the per capita income estimate for 2019 from the U.S. Bureau of Economic Analysis and the average number of hours worked from the Bureau of Labor Statistics 2018 ATUS. We set $\beta = 0.96^{1/52}$ so that the value of a life is 9.3 million 2019 dollars in the pre-epidemic steady state. This value is consistent with the economic value of life used by U.S. government agencies in their decision process.[12] We understand there is considerable uncertainty in the literature about this value. We find that our conclusions are robust to reasonable perturbations of this value.

We set ϕ^i, the parameter that controls for the relative productivity of infected people, at 0.8. This value is consistent with the notion that symptomatic people don't work and the assumption that 80% of infected people are asymptomatic, according to the China Center for Disease Control and Prevention. In the baseline SIR-macro model, the containment rate, μ_t, is equal to zero.

In the medical preparedness model, we set κ equal to 0.9, which implies a peak case fatality rate of 1%, two times higher than that in the basic SIR-macro model. We obtain

[11] We chose to focus on the connection between market activities and the epidemic. This choice led us to abstract from the response of nonmarket activities (e.g., "organizational, civic, and religious activities") to the outbreak of an epidemic.

[12] See U.S. Environmental Protection Agency (2010) and Moran (2016). See Viscusi and Aldy (2003) for a review of the literature on the value of a statistical life.

this higher case fatality rate by computing the weighted average of the case fatality rates in Italy with weights equal to the percentage of different age groups in the U.S. population, excluding people that are 70 and older. In both the treatment and vaccination models, we set $\delta_c = \delta_v = 1/52$, which implies that it takes 52 weeks on average for these medical discoveries to become available.

The model's basic reproduction number A statistic widely used to diagnose the severity of an epidemic is the "basic reproduction number," $\mathcal{R}_0$. This statistic is the total number of infections caused by one infected person (with measure zero) in his or her lifetime in a population in which everybody is susceptible ($S_0 = 1$). The higher is the value of $\mathcal{R}_0$, the faster is the spread of the virus.

The average rate of infection, which we denote by γ, is the ratio of the number of newly infected people to the total number of infected people at the beginning of an epidemic (T_0/I_0). The expected number of infections caused by a single infected person is

$$\mathcal{R}_0 = \gamma + (1 - \pi_r - \pi_d)\gamma + (1 - \pi_r - \pi_d)^2\gamma + ... = \frac{\gamma}{\pi_r + \pi_d}.$$

In this expression, $(1-\pi_r-\pi_d)^t$ is the probability that the infected person reaches period t without recovering or dying.

In the epidemiology literature, the value of $\mathcal{R}_0$ is generally estimated using one of two methods (see, e.g., Breban, Vardavas, and Blower 2007). The first method uses individual-level data collected by contact tracing at the beginning of the epidemic to estimate the number of secondary infections produced by an infected person. The second method involves choosing a value of $\mathcal{R}_0$ so that a given model matches aggregate data on the number of infections and deaths during an epidemic episode. The implied estimates of $\mathcal{R}_0$ depend on the features of the model, including parametric assumptions. So, reported standard errors in any given study understate the true uncertainty about $\mathcal{R}_0$.[13]

We proceed in the spirit of the second method and choose parameters that imply a value of $\mathcal{R}_0 = 1.45$ because they produce plausible implications for the dynamics of the epidemic. This value is at the low end of available estimates for $\mathcal{R}_0$ but consistent with the evidence taking sampling uncertainty into account (see, e.g., Riou and Althaus 2020). In Subsection

[13] The difficulty in estimating $\mathcal{R}_0$ is reflected in the broad range of estimates obtained for widely studied diseases, such as measles. The literature on the measles epidemics often cites values of $\mathcal{R}_0$ from 12 to 18. In a recent survey, Guerra et al. (2017) find an even wider range of $\mathcal{R}_0$ estimates, from 4 to 60.

4.6, we discuss the robustness of our results to different values of $\mathcal{R}_0$.

4.2 The SIR model

The dashed pink lines in Figure 1 represent the equilibrium population dynamics implied by the SIR model. The share of the initial population who is infected peaks at 6.8% in week 31. Thereafter, this share falls because fewer people are susceptible to infection. Eventually, 60% of the population becomes infected. Assuming a U.S. population of 330 million people, this scenario implies that roughly 200 million Americans eventually become infected. A case fatality rate of 0.5% implies that the virus kills roughly 1 million people in the United States.

Figure 1 shows that the epidemic induces a recession: aggregate consumption falls by roughly 1.5% from peak to trough. This fall reflects two factors. First and foremost, the virus causes infected people to be less productive at work ($\phi^i = 0.8$). The associated negative income effect lowers the consumption of those who are infected. The dynamic behavior of aggregate consumption mimics the share of infected people in the overall population. Second, the death toll caused by the epidemic permanently reduces the size of the workforce.

Since the production function has constant returns to scale, per capita income is the same in the pre- and post-epidemic steady states. In the post-epidemic steady state, the population and real gross domestic product (GDP) are both 0.3% lower than in the initial steady state.

4.3 The SIR-macro model

In the SIR model, economic decisions about consumption and work don't influence the dynamics of the epidemic. In contrast, in the SIR-macro model, susceptible people can lower the probability of being infected by reducing their consumption and hours worked. The solid blue lines in Figure 1 represent how the epidemic unfolds in this model.

The share of the initial population who is infected peaks at 5.3% in week 33. This peak is substantially smaller and occurs a little later than the corresponding peak in the SIR model. Eventually, 54% of the population becomes infected. So, for the United States, roughly 180 million people eventually become infected and $890,000$ people die.

Figure 1 shows that the infection is less severe in the SIR-macro model than in the SIR model. The reason is that in the SIR-macro model, susceptible people severely reduce their consumption and hours worked to lower the probability of being infected. Figure 2 shows

that no offsetting effects arise from the behavior of recovered and infected people because they behave as in the SIR model.

Consistent with these observations, the recession is much more severe in the SIR-macro model: average aggregate consumption in the first year of the epidemic falls by 4.7%, a fall seven times larger than in the SIR model.

For similar reasons, the dynamics and magnitude of the drop in hours work are very different in the two models. In the SIR model, hours worked decline smoothly, falling by 0.30% in the post-epidemic steady state. This decline entirely reflects the impact of the death toll on the workforce.

In the SIR-macro model, hours worked follow a U-shaped pattern. The peak decline of 9.8% occurs in week 33. Thereafter, aggregate hours rise, converging to a new steady state from below. These dynamics are driven by the labor supply decisions of susceptible people. Interestingly, the long-run decline in hours worked is slightly lower in the SIR-macro model (0.27%) than in the SIR model (0.30%). The reason is that fewer people die in the epidemic, so the population falls by less in the SIR-macro model than in the SIR model.

Figure 3 shows the competitive equilibrium and the optimal containment policy in the SIR-macro model. We return to this figure in the next section.

4.4 Medical preparedness model

The dashed dotted red lines in Figure 4 show that the competitive equilibrium with an endogenous case fatality rate involves a much larger recession than that in the basic SIR-macro model (solid blue lines). The reason is that people internalize the higher case fatality rates associated with a health care system that can become overburdened with infected people. Since the costs of becoming infected are much higher, people cut back more on consumption and work to reduce the probability of becoming infected. The net result is that fewer people are infected but more people die.

4.5 The treatment and vaccination models

As discussed in the introduction, the possibility of treatments being discovered makes people become more willing to engage in market activities. The reason is that the expected costs of being infected are smaller. Because of this change in behavior, the recession is less severe. In Figure 5, the solid blue and dashed dotted red lines virtually coincide. So, in practice, the quantitative effect of the possibility of treatments on the competitive equilibrium is quite

small. As discussed in Section 5.4, the possibility of treatments does not substantively affect the design of optimal policy.

Vaccines don't cure infected people, but they do prevent susceptible people from becoming infected. So the possibility of future vaccination induces people to reduce their market activities before the vaccine actually arrives. Given our calibration, this effect is small in the competitive equilibrium (see the solid blue and dashed dotted red lines in Figure 6). But, as discussed in Section 5.4, the possibility of vaccinations substantively affects the design of optimal policy. It becomes optimal to immediately introduce severe containment measures to minimize deaths.

4.6 Robustness

Table 1 reports results of a series of robustness exercises in which we vary key parameters of the basic SIR-macro model. Consider first the parameter ϕ^i, which controls the productivity of infected workers. The lower is ϕ^i, the smaller is the average consumption drop, the peak infection rate, the cumulative mortality rate, and the total number of U.S. deaths. The behavior of aggregate consumption reflects two opposing forces. On the one hand, a lower ϕ^i makes it more costly to become infected. So, susceptible people reduce their consumption by more. On the other hand, cautious behavior by susceptible people reduces the total number of people infected. Since infected people consume much less than susceptible people (see Figure 2), this effect increases average consumption in the population. In our model, the first force is somewhat stronger than the second.

Table 1 also reports the results for different parameters of the infection transmission function (Equation (1)). Recall that in the benchmark model, we choose our baseline parameters so that, at the beginning of the infection episode, economic decisions account for one-third of the infection rate. Table 1 summarizes results for the case in which economic decisions account for one-sixth of the initial infection rate. In this scenario, the drop in consumption is smaller. The reason is that people understand that economic activity has less of an impact on infection rates. The peak infection rate, the cumulative mortality rate, and the total number of U.S. deaths is larger. Table 1 also reports the case in which economic decisions account for two-thirds of the initial infection rate. In this scenario, the drop in consumption is larger, and the peak infection rate and cumulative mortality rate are smaller. The reason is that people cut back more on economic activities because they have a larger impact on infection rates.

Table 1: Robustness in Basic SIR-Macro Model without Containment[a]

	Consumption %[b]	Infection rate %[c]	Mortality rate %[d]	U.S. deaths millions[e]
Productivity of infected people, ϕ^i				
0.7	−4.61	4.85	0.26	0.85
0.8 (baseline)	−4.66	5.23	0.27	0.88
Share of initial infections due to consumption, work, and general contacts				
$1/12, 1/12, 5/6$	−2.77	6.15	0.287	0.94
$1/6, 1/6, 2/3$ (baseline)	−4.66	5.23	0.267	0.88
$1/3, 1/3, 1/3$	−7.24	3.25	0.218	0.72
Mortality rate, π_d				
$0.005 \times 7/18$ (baseline)	−4.66	5.23	0.26	0.88
$0.01 \times 7/18$	−8.25	4.74	0.51	1.69
Limited health care capacity parameter, κ (slope of endogenous mortality rate)				
0 (baseline)	−4.66	5.23	0.26	0.88
0.9	−6.83	4.71	0.39	1.31
Household discount factor, β				
$0.96^{1/52}$ (baseline)	−4.66	5.23	0.26	0.88
$0.94^{1/52}$	−3.37	5.42	0.27	0.89

[a] See Section 4.6 for a discussion of the results provided in this table.

[b] Average drop in consumption in first year relative to preinfection steady state.

[c] Peak infection rate relative to pre-epidemic population.

[d] Cumulative mortality rate at the end of the epidemic.

[e] Total number of deaths in the United States at the end of the epidemic.

Next, we increase the case fatality rate from 0.5% to 1%. This change increases the severity of the recession as people cut back on their consumption and work to reduce the chances of being infected. Despite the concomitant fall in peak infection rates, the cumulative mortality rate and the number of U.S. deaths rise.

Table 1 reports the impact of a change in the medical preparedness parameter, κ. The lower is κ, the higher is the degree of medical preparedness. We consider a value of $\kappa = 0.9$ such that the case fatality rate in the medical preparedness model peaks at 1%. Table 1 shows that this higher value of κ is associated with a more severe recession as people curtail their economic activity in response to higher case fatality rates. While the peak level of infections falls, the cumulative death rate and the total number of U.S. deaths rise.

We also assess the impact of reducing the discount factor from $0.96^{1/52}$ to $0.94^{1/52}$. This parameter change reduces the value of a life from 9.3 million to 6.1 million 2019 dollars. As a result, consumption falls less during the epidemic and infection rates rise. The overall quantitative sensitivity is small.

Overall, Table 1 indicates that the qualitative conclusions of the basic SIR-macro model are very robust and that the quantitative conclusions are robust to the perturbations that we consider.

We now discuss the impact of different values of $\mathcal{R}_0$ on the properties of our model. Recall that in the baseline SIR-macro model, $\mathcal{R}_0$ is equal to 1.45. Table 2 reports results for alternative values of $\mathcal{R}_0$, ranging from 1.33 to 2.95. Three key features emerge from this table. First, high values of $\mathcal{R}_0$ generate implausibly large peak infection rates and mortality rates. Second, high values of $\mathcal{R}_0$ imply that the epidemic runs its course very quickly. For example, for $\mathcal{R}_0 = 2.95$, infections peak in the 13th week of the epidemic. Third, the peak-to-trough drop in consumption is increasing in $\mathcal{R}_0$. This result reflects people's response to the higher probability of becoming infected. Interestingly, the average drop in consumption over the first year of the epidemic is not very sensitive to $\mathcal{R}_0$. This property results from two effects. The first effect is a much larger peak-to-trough drop in consumption for high values of $\mathcal{R}_0$. The second effect is a shorter epidemic, and a shorter recession, for high values of $\mathcal{R}_0$. For example, for $\mathcal{R}_0 = 2.95$, consumption initially drops precipitously but recovers after 13 weeks as infections wane.

In sum, Table 2 shows that the qualitative features of our model are very robust to different values of $\mathcal{R}_0$. But the quantitative properties of the model do depend on $\mathcal{R}_0$. As we discuss above, much of the evidence on $\mathcal{R}_0$ in the literature is model based. Viewed through

the lens of our model, the most plausible value of $\mathcal{R}_0$ is relatively low, around 1.5, a value that is consistent with the literature taking sampling uncertainty into account. If one insists on calibrating the model with a high value of $\mathcal{R}_0$, then the model must be extended to make it consistent with the data.

Table 2: Robustness in basic SIR-macro model without containment with respect to $\mathcal{R}_0^a$

Pop. infected %[b]	$\mathcal{R}_0$	Cons. %[c]	Cons. trough %[d]	Inf. rate %[e]	Mort. rate %[f]	U.S. deaths millions[g]	Time to peak infection week[h]
50	1.33	−3.4	−6.7	3.2	0.21	0.72	42
60 (baseline)	1.45	−4.7	−9.8	5.2	0.26	0.88	34
70	1.62	−5.2	−13.3	8.2	0.31	1.05	28
80	1.86	−5.2	−16.9	12.5	0.37	1.22	23
93	2.49	−4.1	−20.6	23.9	0.44	1.46	16
97	2.95	−3.2	−20.9	31.2	0.47	1.55	13

[a] See Section 4.6 for a discussion of the results provided in this table.

[b] Percentage of the population eventually infected in the canonical SIR model.

[c] Average drop in consumption in the first year relative to the preinfection steady state.

[d] Peak-to-trough drop in weekly consumption.

[e] Peak infection rate relative to pre-epidemic population.

[f] Cumulative mortality rate at the end of the epidemic.

[g] Total number of deaths in the United States at the end of the epidemic.

[h] Week at which the fraction of the infected population as a percentage of initial population peaks.

The model can be extended in at least three ways. The first is to explicitly model the impact of nonpharmaceutical interventions, such as masks and social distancing which slow down the transmission of the virus (see, e.g. Eichenbaum, Rebelo and Trabandt 2020a). The second is to include the possibility of substitution from high- to low-contact forms of consumption and work, so that a given reduction in the transmission rate results in a smaller decline in economic activity than in our benchmark model (see Jones, Philippon, and Venkateswaran 2020 and Krueger, Uhlig, and Xie 2020 for models along these lines).

The third is to endogenize the time that people spend on nonmarket social interactions. In this setting, a decline in these interactions can reduce the rate of virus transmission without producing a drop in measured output (see Farboodi, Jarosch, and Shimer 2020 for a model along these lines). Evaluating the empirical performance of these different approaches is an interesting topic for future research.

5 Economic Policy

The competitive equilibrium of our model economy is not Pareto optimal. A classic externality is associated with the behavior of infected people. Because each person is atomistic, people don't take into account the impact of their actions on the infection and death rates of other people. But collectively, the behavior of infected people imposes an unpriced cost on susceptible people. In this section, we consider a simple Ramsey problem designed to deal with this externality. As it turns out, the solution to the Ramsey problem is quite similar to the solution of a planner's problem in which the planner chooses consumption and labor subject to the constraint that these choices are the same for everybody regardless of health status.

5.1 Ramsey problem

As with any Ramsey problem, we must take a stand on the policy instruments available. In reality, governments can reduce social interactions in many ways. Examples of containment measures include shelter-in-place laws and shutting down restaurants and bars. Analogous to Farhi and Werning's (2012) treatment of capital controls, we model these measures as a tax on consumption, the proceeds of which are rebated lump sum to people in the economy. We refer to this tax as the "containment rate."

We compute the optimal sequence of 250 containment rates $\{\mu_t\}_{t=0}^{249}$ that maximize social welfare, U_0, defined as a weighted average of the lifetime utility of different people. Since at time zero $R_0 = D_0 = 0$, the value of U_0 is

$$U_0 = S_0 U_0^s + I_0 U_0^i. \tag{14}$$

Given the sequence of containment rates, we solve for the competitive equilibrium and evaluate the social welfare function. We iterate on this sequence until we find the optimum.

Figure 3 displays our results. First, the gradual escalation of containment measures over time is optimal. The optimal containment rate rises from 4.5% in week 0 to a peak value of 72% in week 37. The rise in containment rates roughly parallels the dynamics of the infection rate itself. The basic intuition is as follows. Containment measures internalize the externality caused by the behavior of infected people. So, as the number of infected people rises, intensifying the containment measures is optimal. For example, at time zero, very few people are infected, so the externality is relatively unimportant. A high containment rate at time zero would have a high social cost relative to the benefit. As the infection rate rises, the externality becomes important, and the optimal containment rate rises.

The optimal containment policy greatly reduces the peak level of infections from 5.3% to 3.2%, thereby reducing the death toll from 0.27% to 0.21% of the initial population. For a country like the United States, this reduction represents roughly $200,000$ lives saved. This beneficial outcome is associated with a much more severe recession. The fall in average aggregate consumption in the first year of the epidemic more than triples, going from about 4.7% without containment measures to about 17% with containment measures. The mechanism underlying this result is straightforward: higher containment rates make consumption more costly, so people cut back on the amount they consume and work.

Why not choose initial containment rates that are sufficiently high to induce an immediate, persistent decline in the number of infected people? Absent vaccines, the only way to prevent a recurrence of the epidemic is for enough of the population to acquire immunity by becoming infected and recovering. The optimal way to reach this critical level of immunity is to gradually increase containment measures as infections rise and slowly relax them as new infections wane.

5.2 Simple command containment

One possible objection to our simple containment policy is that it is modeled as a Pigouvian consumption tax. An alternative formulation is to consider a planning problem in which the government chooses consumption and hours worked to maximize the objective function (14) subject to the population dynamics equations, the resource constraint, and the constraint that people have the same allocation regardless of health status:

$$C_t^s = C_t^i = C_t^r,$$

and

$$N_t^s = N_t^i = N_t^r.$$

The solution to this "simple command containment" problem is represented by the dash-dotted red line in Figure 3. This figure shows that the solution to this problem is very similar to the simple containment policy discussed above. A similar conclusion holds for optimal policy in the benchmark SIR model discussed below.[14]

5.3 Medical preparedness model

Comparing Figures 3 and 4, we see that the optimal containment policy is more aggressive in the medical preparedness model than in the basic SIR-macro model. The peak containment rate is higher in the medical preparedness model (110% vs. 72%) and occurs earlier (at week 33 vs. week 37). In addition, the containment rate comes down much more slowly in the medical preparedness model. These differences reflect that, other things equal, the social cost of the externality is much larger. People do not internalize the cost of consumption and work on infection rates nor do they internalize the aggregate increase in case fatality rates.

The optimal containment policy greatly reduces the peak level of infections from 4.7% without containment to 2.2% with containment. The death toll falls from 0.40% to 0.22% of the initial population. For a country like the United States, this reduction represents roughly $600,000$ lives saved.

5.4 The treatment and vaccination models

Comparing Figures 3 and 5, we see that the optimal containment policies in the treatment and basic SIR-macro models are quite similar. In the treatment model, along a path where no treatment is discovered, the optimal containment policy reduces the peak level of infections from 5.3% to 3.2%, reducing the death toll from 0.27% to 0.21% of the initial population. This reduction corresponds to roughly $200,000$ lives saved in the United States. The latter figure pertains to a worst-case scenario in which a treatment is never discovered.

[14] Both the peak in infections and the fall in aggregate consumption are slightly larger under simple command containment than under simple containment. This result reflects compositional effects. First, the consumption of recovered people drops by more under simple command containment because everybody must have the same consumption. Second, the consumption of infected people is substantially lower under simple containment than under simple command containment. This property reflects infected people's lower productivity and their response to the Pigouvian tax. Third, the consumption of susceptible people is similar under both policies. The net effect is that, even though aggregate consumption drops by more under simple command containment, infection rates are higher, reflecting the higher consumption of infected people.

The dashed black lines in Figure 6 show that optimal policy is very different in the basic SIR-macro model and the vaccination model. With vaccines as a possibility, it is optimal to immediately introduce severe containment measures to minimize the number of deaths. Those containment measures cause a very large, persistent recession: average consumption in the first year of the epidemic falls by about 17%. But this recession is worth incurring in the hope that the vaccines arrive before many people get infected.

It is optimal to reduce and delay the peak of the infections in anticipation of a vaccine being discovered. Figure 6 displays the behavior of the vaccination model under optimal containment policy on a path in which a vaccine does not arrive. Compared to the competitive equilibrium (dashed dotted red lines), the peak of the infection rate drops from 5.3% to 3.3% of the initial population. Moreover, the infection peak occurs in week 42 rather than in week 33. Absent a vaccine being discovered, the optimal containment policy reduces the death toll as a percentage of the initial population from 0.27% to 0.24%. For the United States, this reduction amounts to about $100,000$ lives. It is important to remember that this reduction pertains to a worst-case scenario in which vaccines do not arrive.

Above, we discuss why it is not optimal to introduce immediate containment measures in the basic SIR-macro and treatment models. But why is the optimal policy so different in the vaccination model? The basic reason is that, unlike curative treatments, a vaccine does not cure infected people. In addition, the expected arrival of a vaccine also reduces the importance of achieving herd immunity before the vaccine arrives.

6 Quantitative Predictions for the Benchmark Model

In the previous sections, we separately analyze the quantitative predictions of our model under different simplifying assumptions. Those exercises are useful for understanding the mechanisms at work. In our view, the most meaningful version of the model allows for both (a) the possibility of vaccines and medical treatment and (b) the impact of the number of infections on the efficacy of the health care system. We refer to this version of the model as the benchmark model. We describe the equations used to compute the competitive equilibrium of this model in the Appendix.

In the first subsection, we discuss the impact of optimal containment policy in the benchmark model. The second subsection considers the consequences of prematurely ending the containment policy. In the third subsection, we consider the implication of a delay in im-

plementing the optimal containment policy. In the fourth subsection, we study a first-best solution in which the planner chooses directly different levels of consumption and hours worked for susceptible, infected, and recovered people.

6.1 Optimal policy in the benchmark model

The solid blue and dashed black lines in Figure 7 represent the evolution of the economy in the competitive equilibrium and under the best simple containment policy, respectively. Consistent with previous figures, we display a path along which vaccines and treatments are not discovered.

From a qualitative point of view, the benchmark model inherits key features of its underlying components. Consistent with the vaccination model, it is optimal to immediately introduce severe containment ($\mu = 43\%$). Consistent with the treatment and medical preparedness models, it is optimal to ramp up containment as the number of infections rises. The maximal containment rate reaches 76% in week 32.

The optimal containment measures substantially increase the severity of the recession. Without containment, average consumption in the first year of the epidemic falls by about 7%. With containment, this fall is 22%. Notably, the size of the recession is smaller than in the medical preparedness model. The reason is that the prospect of vaccinations and treatments reduces the magnitude of the externality associated with the medical preparedness problem.

The benefit of the large recession associated with optimal containment in the combined model is a less severe epidemic. Compared to the competitive equilibrium, the peak infection rate drops from 4.7% to 2.5% of the initial population. The optimal policy reduces the death toll as a percentage of the initial population from 0.40% to 0.26%. For the United States, this reduction amounts to about half a million lives.

We emphasize that the latter reduction pertains to a worst-case scenario in which vaccines and treatments never arrive. If they do arrive, many more lives would be saved. Thankfully, they would be saved by medicine rather than by containment policies.

The dashed dotted red lines in Figure 7 represent the optimal containment policy implemented as a solution to a planner's problem in which the planner chooses consumption and hours worked subject to the constraint that people have the same allocation regardless of health status. We see that the solution under this "command containment" policy is very similar to the solution under the best simple containment policy.

6.2 The costs of ending containment too early

As a practical matter, policy makers could face intense pressure to prematurely end containment measures because of their negative impact on economic activity. In this subsection, we discuss the costs of doing so. The solid red lines in panels A and B of Figure 8 represent the response of the economy to an unanticipated end of the best simple containment policy after weeks 12 and 44, respectively. Week 44 is when infections peak under the best simple containment policy. The dashed black lines represent the behavior of the economy when the best simple containment policy is fully implemented.

From panel A, we see that abandoning containment initially generates a large recovery, with consumption surging by roughly 17%. Unfortunately, this surge results in a large rise in infection rates. The latter rise plunges the economy into a second, persistent recession.

So, prematurely abandoning containment produces a temporary rise in consumption but no long-lasting economic benefits. Tragically, abandonment leads to a substantial rise in the death toll of the epidemic.

Panel B shows that the longer policy makers pursue the optimal containment policy, the better. Both the temporary gains and the losses due to abandoning the optimal policy in panel B are smaller than those in panel A.

The implications of our model for the cost of ending containment too early are consistent with the evidence for the 1918 Spanish flu (Bootsma and Ferguson 2007). We conclude that it is important for policy makers to resist the temptation to pursue transient economic gains obtained by abandoning containment measures.

6.3 Costs of starting containment too late

Policy makers can also face pressure to delay implementing optimal containment measures. The dashed dotted red lines in Figure 9 represent the impact of beginning containment only in week 33, the week in which infections peak. We assume that the best simple containment policy is calculated and implemented from that point on. The dashed black lines represent the behavior of the economy when the best simple containment policy is implemented from week 0 on. The solid blue line represent the competitive equilibrium with no containment measures.

The best simple containment policy that begins in week 33 involves draconian containment measures that cause an enormous drop in economic activity. The reason is simple:

with infections raging, the externalities associated with economic activity are very large.

Despite the draconian measures, the total number of deaths caused by the epidemic is much larger than if the best simple containment policy is implemented without delay. Still, as far as the death toll of the epidemic is concerned, late containment (dashed dotted red lines) is better than no containment at all (solid blue lines).

The implications of our model for the cost of starting containment too late are consistent with the evidence for the 1918 Spanish flu (Hatchett, Mecher, and Lipsitch 2007; Bootsma and Ferguson 2007). We conclude that it is important for policy makers to resist the temptation to delay containment measures for the sake of initially higher short-run levels of economic activity.

6.4 Smart containment

Recall that in Section 4.1 we study simple containment policies corresponding to a Ramsey problem in which the government chooses the same consumption containment rate for everybody in the economy. In Section 4.2 we study the optimal simple command containment policy in which the government chooses the same consumption and hours worked for all health types.

In this section, we study smart containment, by which we mean the solution to a social planning problem in which the planner directly chooses *different levels* of consumption and hours worked for susceptible, infected, and recovered people.

The planner maximizes the social welfare, U_0, defined in equation (14). The planner chooses C_t^s, C_t^i, C_t^r, N_t^s, N_t^i, and N_t^r for all t to maximize U_0 subject to the expressions for the lifetime utility of the different people, the transmission function (1), and the laws of motion for the population, (2), (3), (4), and (5).

The lifetime utilities of susceptible, infected and recovered people are given by

$$U_t^s = u(C_t^s, N_t^s) + (1-\delta_v)(1-T_t)\beta U_{t+1}^s + \delta_v (1-T_t)\beta U_{t+1}^r + T_t \beta U_{t+1}^i,$$

$$U_t^i = u(C_t^i, N_t^i) + (1-\delta_c)\left[(1-\pi_r-\pi_{dt})\beta U_{t+1}^i + \pi_r \beta U_{t+1}^r\right] + \beta\delta_c U_{t+1}^r,$$

$$U_t^r = u(C_t^r, N_t^r) + \beta U_{t+1}^r.$$

The lifetime utility of susceptible people is computed using the aggregate transition probabilities because the planner internalizes the infection externality.

Figure 10 summarizes our results. Note that infected people do not work unless they recover. As a result, all susceptible people can work without fear of becoming infected. The planner sets the consumption of infected people to a minimum. In fact, there is no maximum to the social planning problem, only a supremum. Given the form of momentary utility, it is not optimal to set the consumption of infected people to zero because their utility would be equal to $-\infty$. But the closer the consumption of those infected is to zero, the higher is social welfare. Because infected people are completely isolated, the initial infection quickly dies out without causing a recession.

The previous analysis assumes that infected people have to be in contact with other people to obtain consumption goods. This assumption underlies the draconian policy implication that the consumption of infected people should be kept at a minimum. Suppose instead that the planner can directly deliver consumption goods to the infected so they do not need to go shopping. The solution to this modified problem continues to have the property that infected people don't work. But they consume the same as other people. Since there is such a small number of infected people at time zero, aggregate consumption and hours worked are essentially the same as in the pre-epidemic steady state.

One important limitation of smart containment is that herd immunity is never reached. Every infection case must be detected and dealt with until the virus is extinguished. Otherwise, the epidemic would begin anew as soon as smart containment ended.

Our simple analysis of smart containment assumes that policy makers know the health status of different individuals. In reality, this knowledge would require antigen and antibody tests for immunity and infection that are sufficiently accurate to act on. Our results suggest enormous social returns to having these tests and the policy instruments to implement smart containment.[15] This conclusion is consistent with the importance of early detection and early response emphasized by epidemiologists, such as Ginsberg et al. (2008). In a subsequent paper, Eichenbaum, Rebelo, and Trabandt (2020a) study the efficacy of smart containment policies in a setting in which people do not know their health status until they are tested.

[15] According to de Walque, Friedman, and Mattoo (2020), the average cost per test, including equipment, consumables, protective equipment, and labor, ranges from \$2 to \$5.

7 Model Performance in Retrospect

This paper was originally drafted in March 2020. Almost a year has passed, and a natural question now is: how do the predictions of the model compare to currently available data?

The first-order prediction of the model is that, in response to the epidemic, economic activity would undergo a sharp contraction followed by a robust recovery. This qualitative prediction is clearly supported by data for the U.S. and other developed economies. The model also does reasonably well at capturing the broad quantitative decline in economic activity. For example, the peak-to-trough decline in U.S. real GDP (between the last quarter of 2019 and the second quarter of 2020) is 10.7%. In our model, the analogue decline in output, absent containment, is 5.4%. The average decline in U.S. real GDP in 2020 relative to 2019 is 3.6%. In the model, the analogue decline in output, absent containment, is 4.7%. Going beyond these broad statements and providing a detailed comparison of model and data would require taking a stand on the timing and nature of the containment measures implemented in the United States.

Another important implication of our model is that economic activity should contract even *before* the government imposes containment measures. Substantial evidence supports this prediction. See, for example, results in Goolsbee and Syverson (2020) and Villas-Boas, Sears, Villas-Boas, and Villas-Boas (2020), based on mobility data, as well as the evidence in Chetty, Friedman, Hendren, and Stepner (2020) and Eichenbaum, Godinho de Matos, Lima, Rebelo, and Trabandt (2020), based on micro data on consumption.

The implications of our model are also consistent with evidence presented by Atkeson, Kopecky, and Zha (2020) for a large number of countries and U.S. states. These authors show that the growth rate of daily COVID-19 deaths fell much more rapidly than predicted by a canonical SIR model, suggesting strong behavioral changes that slowed down the transmission of the virus.

Our model predicts that, absent a successful vaccination campaign, the death toll of the epidemic would be between $500,000$ and 1.5 million people, depending on the containment policy adopted. As of February 6, 2021, $460,000$ Americans have died of COVID-19. On February 4, 2021, the University of Washington's Institute for Health Metrics and Evaluation predicted that the total death toll will reach $630,000$ by June 2021. This forecast lies within the range of the model's predictions although somewhat at the lower end, reflecting in part the expected positive impact of the ongoing vaccination campaign.

An important failing of the model has to do with the timing of these deaths. In the data, a significant second wave of deaths took place between December 2020 and February 2021. Our model generates a second wave if we assume that containment ended prematurely (see Figure 8). But the magnitude of that wave is much smaller than that observed in the data. We do not think that this failing reflects the calibration that we used. Consistent with Atkeson (2021), we conjecture that a combination of seasonality in the transmission rate and changes in the way people react to infection risk are likely to be important factors in accounting for the second wave of COVID-19 deaths.

Finally, our model predicts that, absent instant mass vaccination, the epidemic episode would last between 18 months and two years. This prediction seems reasonable in light of the fact that the episode is not yet over but deaths are now declining. But, of course, now that vaccines are available, the end of the epidemic depends on the pace of the vaccination campaign and the efficacy of vaccines in the face of ongoing mutations of the COVID-19 virus.

8 Related Literature

Our work is related to a pre-Covid literature that combines economics and epidemiology (for a review, see Perrings et al. 2014). Examples include analyses of how private vaccination incentives affect epidemic dynamics and optimal public health policy (e.g., Philipson 2000, Manski 2010, and Adda 2016) and studies of the interaction between behavioral choice and the dynamics of the HIV/AIDS epidemic (e.g., Kremer 1996 and Greenwood, Kircher, Santos, and Tertilt 2019).

The COVID-19 crisis has stimulated a rapidly growing body of work on the economics of the epidemic. Below, we briefly summarize the first wave of this research program.

Atkeson (2020) provides an overview of SIR models and explores their implications for the COVID-19 epidemic. Alvarez, Argente, and Lippi (2020) study the optimal lockdown policy in a version of the canonical SIR model in which the case fatality rate increases with the number of infected people. Toxvaerd (2020) analyses the equilibrium amount of social distancing in a SIR model and argues that it is not socially optimal.

Jones, Philippon, and Venkateswaran (2020) study optimal mitigation policies in a model in which economic activity and epidemic dynamics interact. These authors emphasize learning-by-doing in working from home and assume that people have a fatalism bias about

the probability of being infected in the future. Other differences between our paper and theirs are as follows. First, we explicitly allow for the probabilistic arrival of vaccines and treatments. Second, we consider the social cost of starting containment too late or ending it too early. Third, we study "smart containment" policies that make allocations a function of whether people are infected, susceptible, or recovered.

Guerrieri, Lorenzoni, Straub, and Werning (2020) develop a theory of Keynesian supply shocks that trigger changes in aggregate demand that are larger than the shocks themselves. These authors argue that the economic shocks associated with the COVID-19 epidemic may have this feature. Guerrieri et al. (2020) analyze the efficacy of various fiscal and monetary policies at dealing with these shocks. In contrast with Guerrieri et al. (2020), we incorporate an extended version of SIR dynamics into our model.

Berger, Herkenhoff, Huang, and Mongey (2020) and Stock (2020) study the importance of randomized testing in estimating the health status of the population and designing optimal mitigation policies. In contrast with these authors, we explicitly model the two-way interaction between infection rates and economic activity.

A growing body of work studies the effects of the COVID-19 epidemic in models in which people differ in their health status as well as along other dimensions. For example, Glover, Heathcote, Krueger, and Rios-Rull (2020) study optimal mitigation policies in a model that takes into account the age distribution of the population. Kaplan, Moll, and Violante (2020) do so in a heterogeneous agent new-Keynesian model.

Faria-e-Castro (2021) studies the effect of an epidemic, modeled as a large negative shock to the utility of consumption of contact-intensive services, in a model with borrowers and savers. Buera, Fattal-Jaef, Neumeyer, and Shin (2020) study the impact of an unanticipated lockdown shock in a heterogeneous-agent model.

9 Conclusion

We extend the canonical epidemiology model to study the interaction between economic decisions and epidemics. In our model, the epidemic generates both supply and demand effects on economic activity. These effects work in tandem to generate a large, persistent recession.

We abstract from many important real-world complications to highlight the basic economic forces at work during an epidemic. The central message of our analysis should be

robust to allowing for those complications: there is an inevitable trade-off between the severity of the short-run recession caused by the epidemic and the health consequences of that epidemic. Dealing with this trade-off is a key challenge confronting policy makers.

Our model also abstracts from various forces that might affect the long-run performance of the economy. These forces include bankruptcy costs, unemployment hysteresis effects, and the destruction of supply chains. It is important to embody these forces in macroeconomic models of epidemics and study their positive and normative implications.

Appendix. Computing the Equilibrium

This appendix summarizes the equations used to compute the competitive equilibrium of the benchmark SIR-macro model. The basic SIR-macro corresponds to a particular case in which $\delta_c = \delta_v = \kappa = 0$.

For a given sequence of containment rates, $\{\mu_t\}_{t=0}^{H-1}$, for some large horizon, H, guess sequences for $\{n_t^s, n_t^i, n_t^r\}_{t=0}^{H-1}$. In practice, we solve the model for $H = 250$ weeks. Compute the sequence of the remaining unknown variables in each of the following equilibrium equations:

$$\theta n_t^r = A\lambda_{bt}^r,$$

$$(c_t^r)^{-1} = (1+\mu_t)\lambda_{bt}^r,$$

$$u_t^r = \ln c_t^r - \frac{\theta}{2}\left(n_t^r\right)^2.$$

Iterate backward from the post-epidemic steady-state values of U_t^r:

$$U_t^r = u(c_t^r, n_t^r) + \beta U_{t+1}^r.$$

Calculate the sequence for remaining unknowns in the following equations:

$$(1+\mu_t)c_t^r = An_t^r + \Gamma_t \qquad (\lambda_{bt}^r),$$

$$\theta n_t^i = \phi^i A\lambda_{bt}^i,$$

$$\left(c_t^i\right)^{-1} = \lambda_{bt}^i,$$

$$u_t^i = \ln c_t^i - \frac{\theta}{2}\left(n_t^i\right)^2,$$

$$(1+\mu_t)c_t^s = An_t^s + \Gamma_t \qquad (\lambda_{bt}^s),$$

$$u_t^s = \ln c_t^s - \frac{\theta}{2}\left(n_t^s\right)^2.$$

Given initial values for Pop_0, S_0, I_0, R_0, and D_0, iterate forward using the following seven equations for $t = 0, .., H-1$:

$$T_t = \pi_1(S_t c_t^s)\left(I_t c_t^i\right) + \pi_2(S_t n_t^s)\left(I_t n_t^i\right) + \pi_3 S_t I_t,$$

$$\text{Pop}_{t+1} = \text{Pop}_t - \pi_{dt} I_t,$$

$$S_{t+1} = S_t - T_t,$$

$$I_{t+1} = I_t + T_t - (\pi_r + \pi_{dt})\, I_t,$$

$$R_{t+1} = R_t + \pi_r I_t,$$

$$\pi_{dt} = \pi_d + \kappa I_t^2,$$

$$D_{t+1} = D_t + \pi_{dt} I_t.$$

Iterate backward from the post-epidemic steady-state values of U_t^s and U_t^i:

$$U_t^i = u(c_t^i, n_t^i) + (1-\delta_c)\left[(1-\pi_r-\pi_{dt})\,\beta U_{t+1}^i + \pi_r \beta U_{t+1}^r\right] + \beta\delta_c U_{t+1}^r.$$

$$\tau_t = \frac{T_t}{S_t},$$

$$U_t^s = u(c_t^s, n_t^s) + (1-\delta_v)\,(1-\tau_t)\,\beta U_{t+1}^s + \delta_v\,(1-\tau_t)\,\beta U_{t+1}^r + \tau_t \beta U_{t+1}^i.$$

Calculate the sequence of the remaining unknowns in the following equations:

$$\beta\left(U_{t+1}^i - U_{t+1}^s\right) - \lambda_{\tau t} = 0,$$

$$(c_t^s)^{-1} - \lambda_{bt}^s(1+\mu_t) + \lambda_{\tau t}\pi_1\left(I_t C_t^i\right) = 0.$$

Finally, use a gradient-based method to adjust the guesses $\{n_t^s, n_t^i, n_t^r\}_{t=0}^{H-1}$ so that the following three equations hold with arbitrary precision:

$$(1+\mu_t)c_t^i = \phi^i A n_t^i + \Gamma_t \qquad (\lambda_{bt}^i),$$

$$\mu_t\left(S_t c_t^s + I_t c_t^i + R_t c_t^r\right) = \Gamma_t\left(S_t + I_t + R_t\right),$$

$$-\theta n_t^s + A\lambda_{bt}^s + \lambda_{\tau t}\pi_2\left(I_t n_t^i\right) = 0.$$

To solve the Ramsey problem, we compute the sequence of optimal containment rates, μ_t, by maximizing the social welfare function, (14), using the Matlab routine fmincon.m. We explored the robustness of the numerical results by using Matlab's global optimization toolbox, including fminunc.m, patternsearch.m, ga.m, particleswarm.m, surrogateopt.m, and globalsearch.m. None of these routines finds an optimum that is superior to the one based on fmincon.m.

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Figure 1: Basic SIR-Macro Model vs. SIR Model

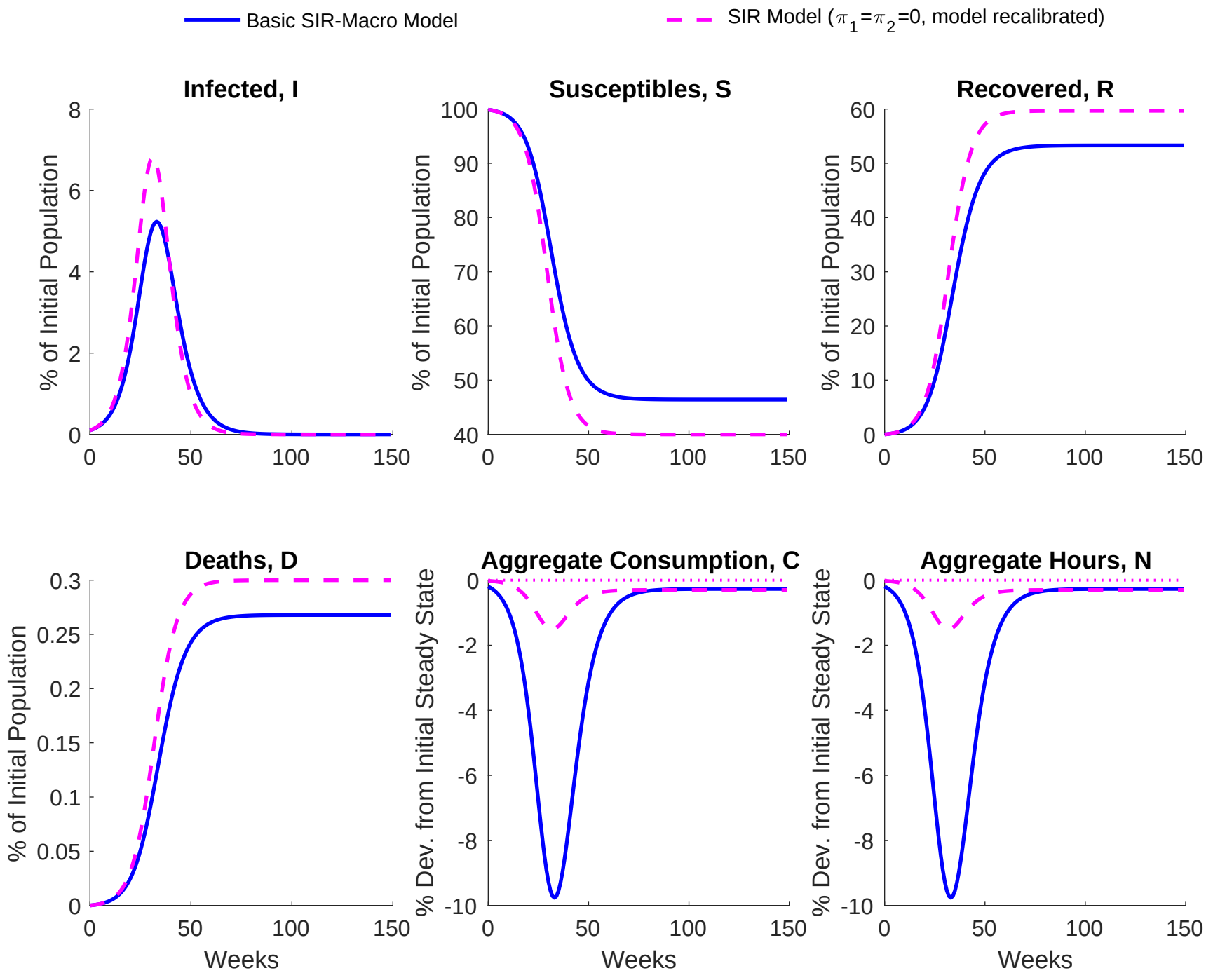


Figure 2: Consumption and Hours by Type in Basic SIR-Macro Model

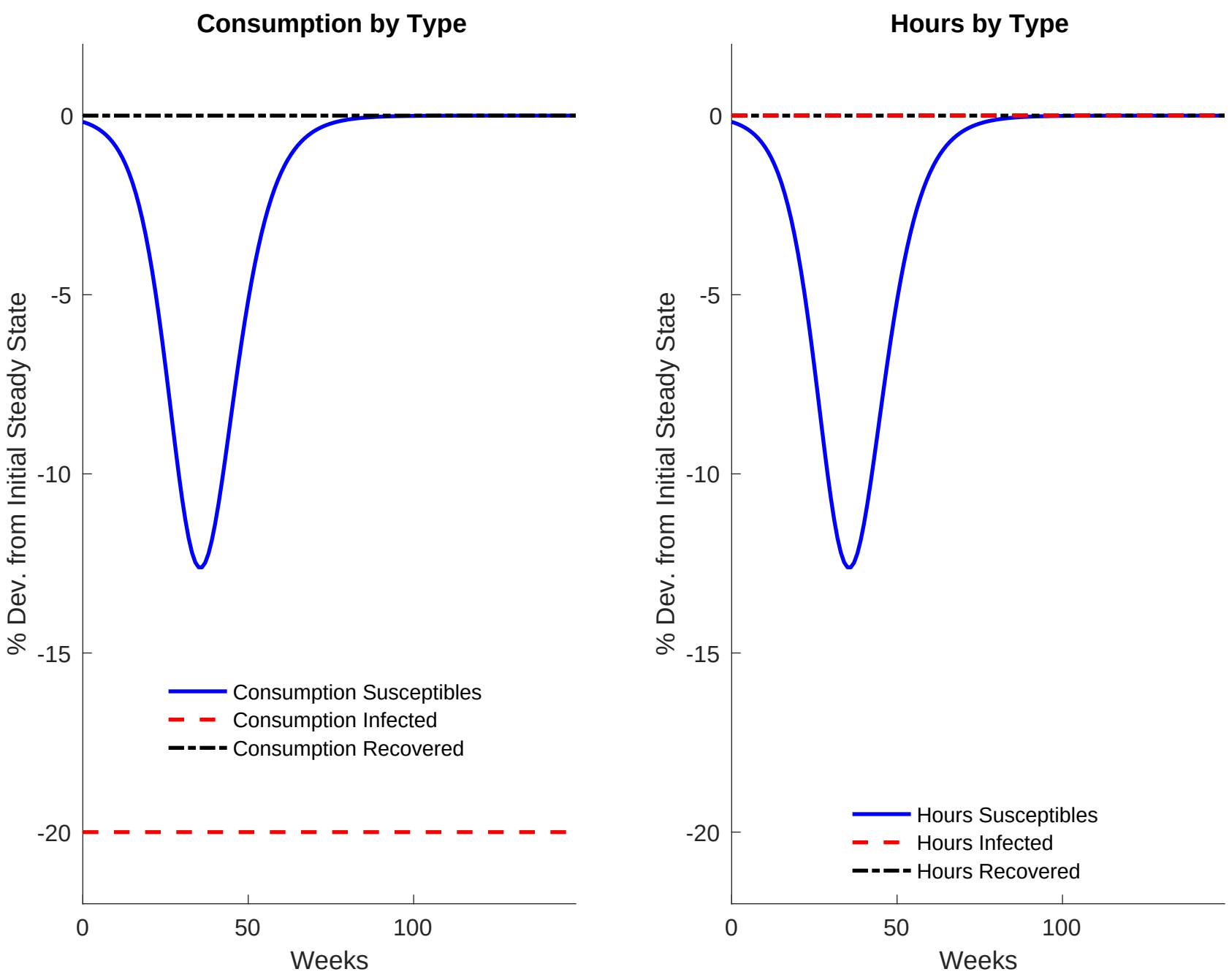


Figure 3: Basic SIR-Macro Model with and without Containment

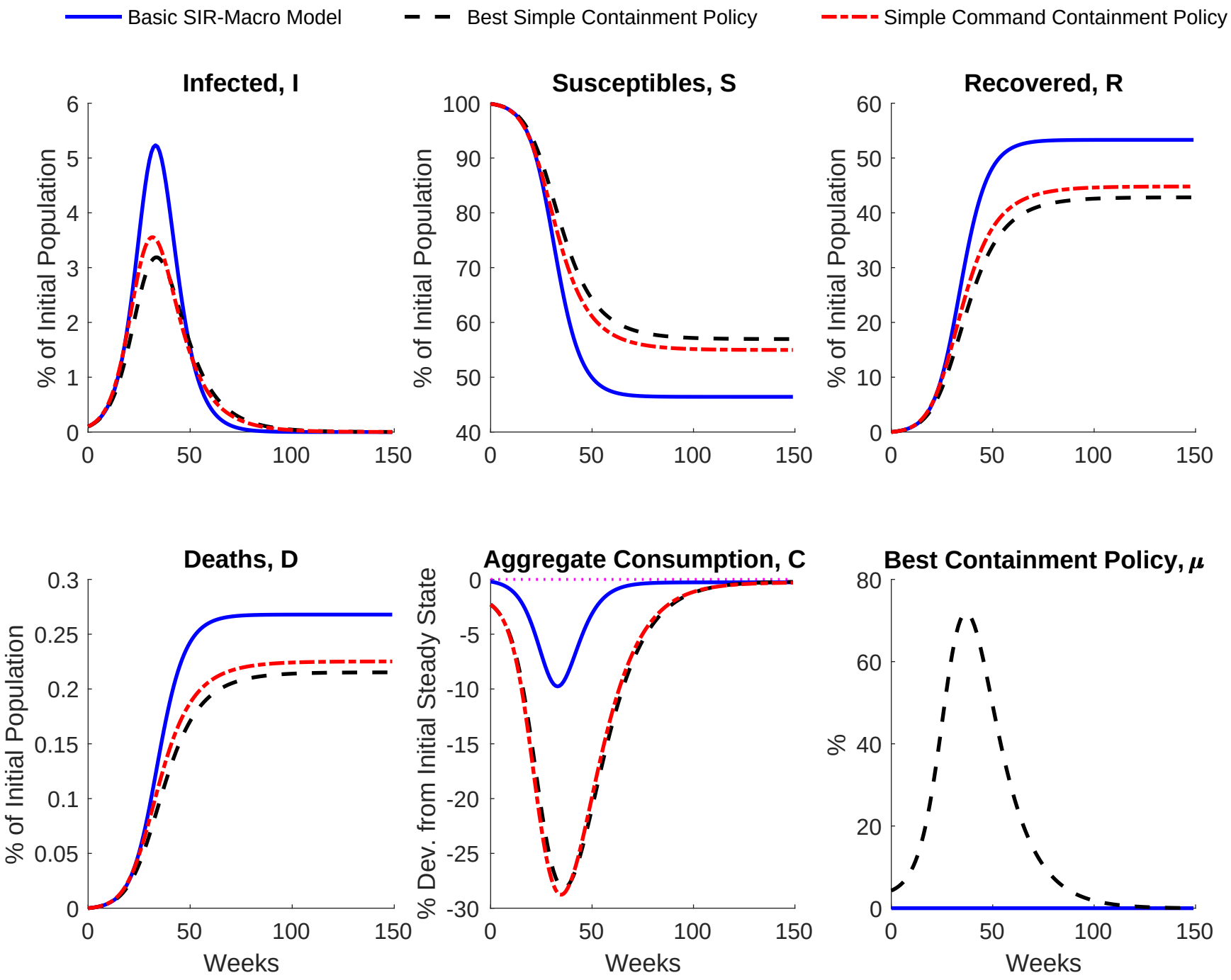


Figure 4: Medical Preparedness

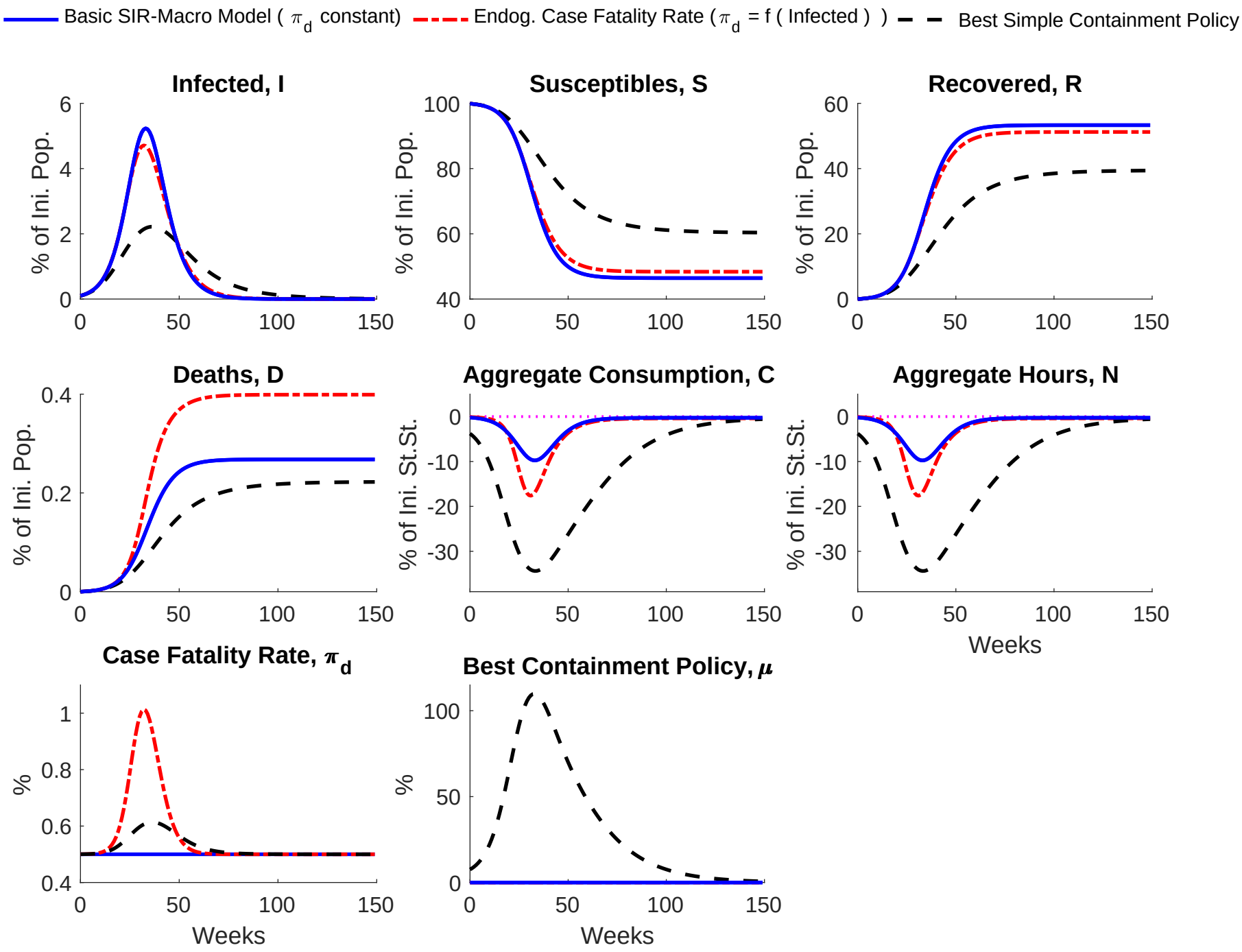


Figure 5: SIR-Macro Model with Treatments

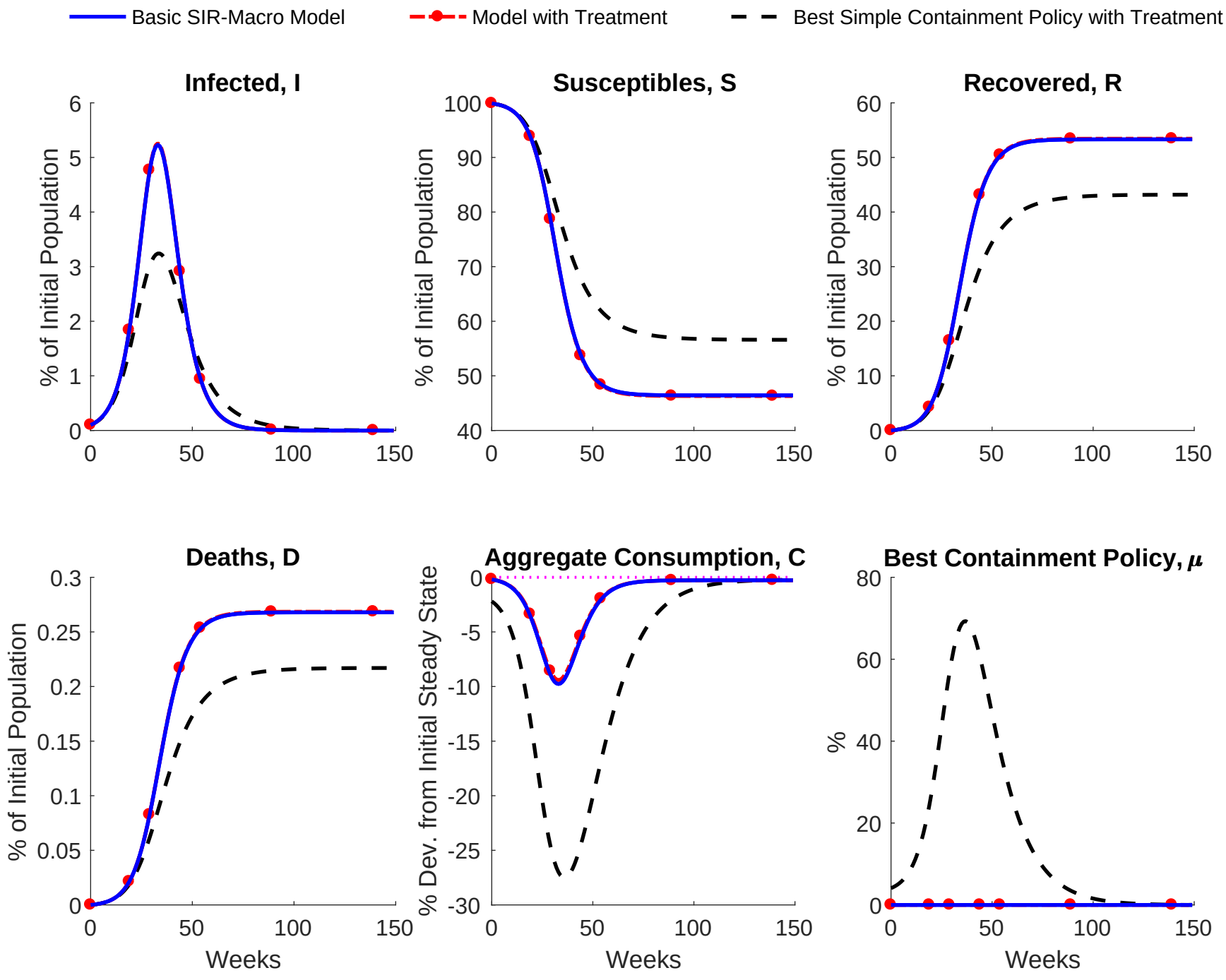


Figure 6: SIR-Macro Model with Vaccines

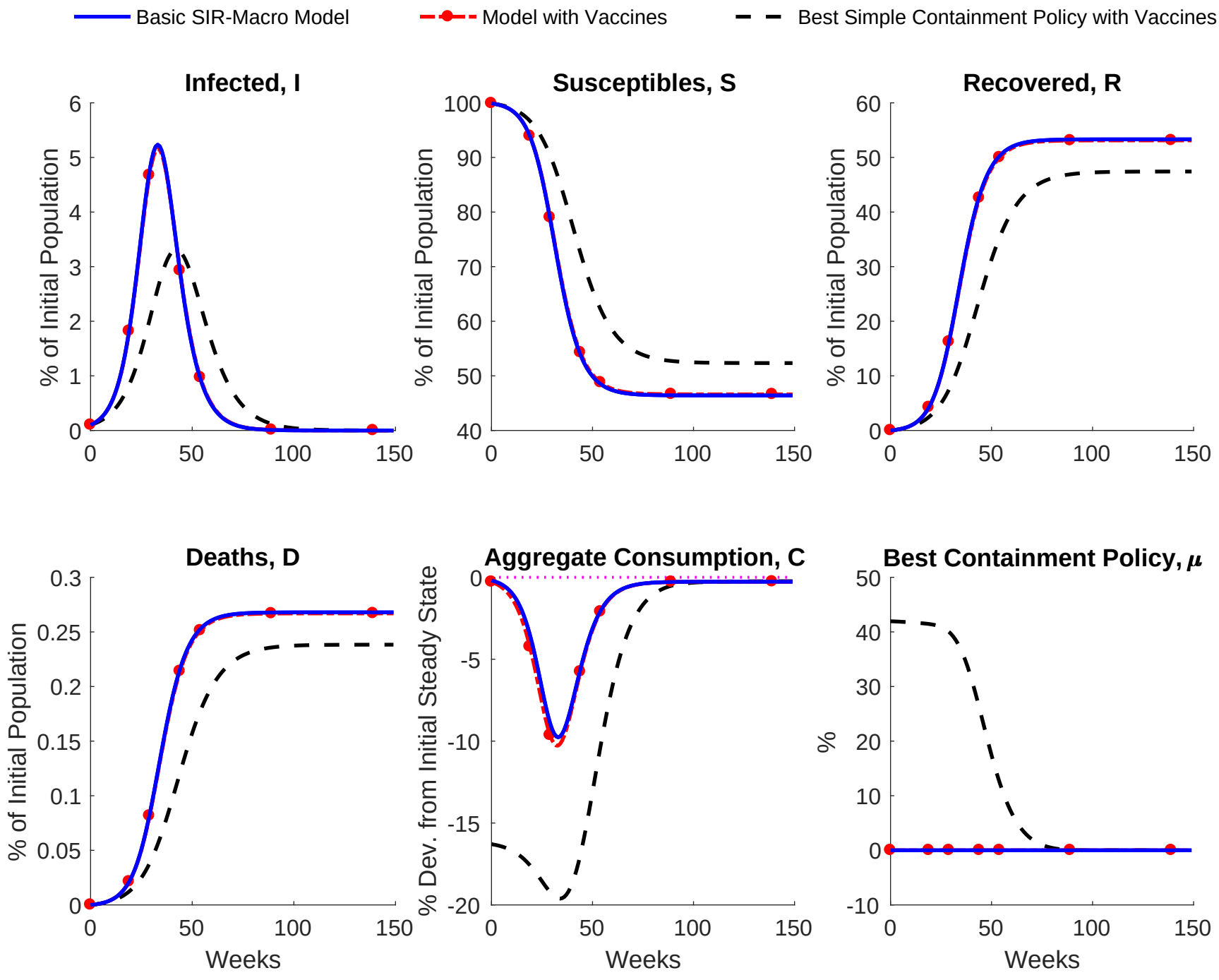


Figure 7: Benchmark SIR-Macro Model (Vaccines, Treatment, Med. Preparedness)

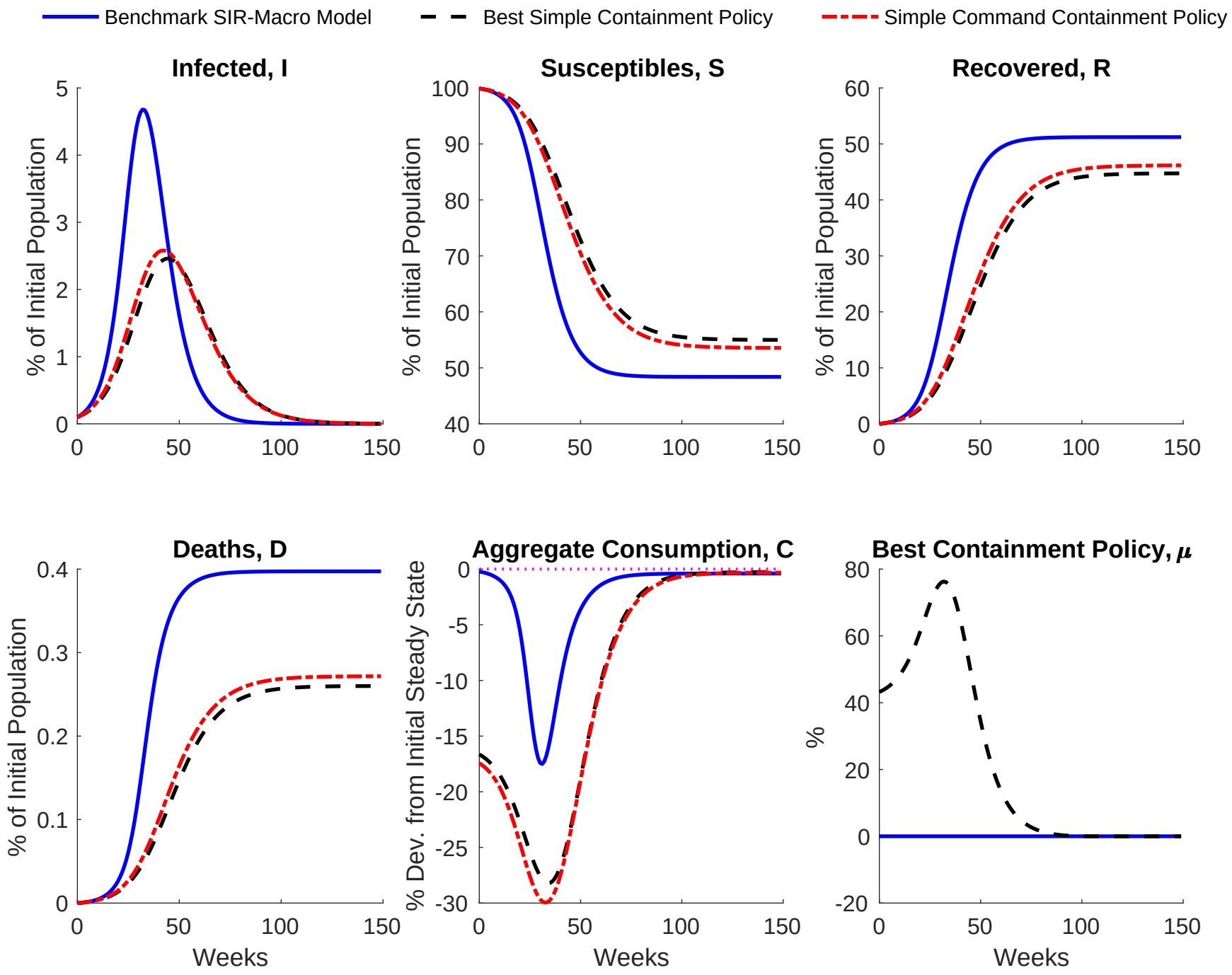
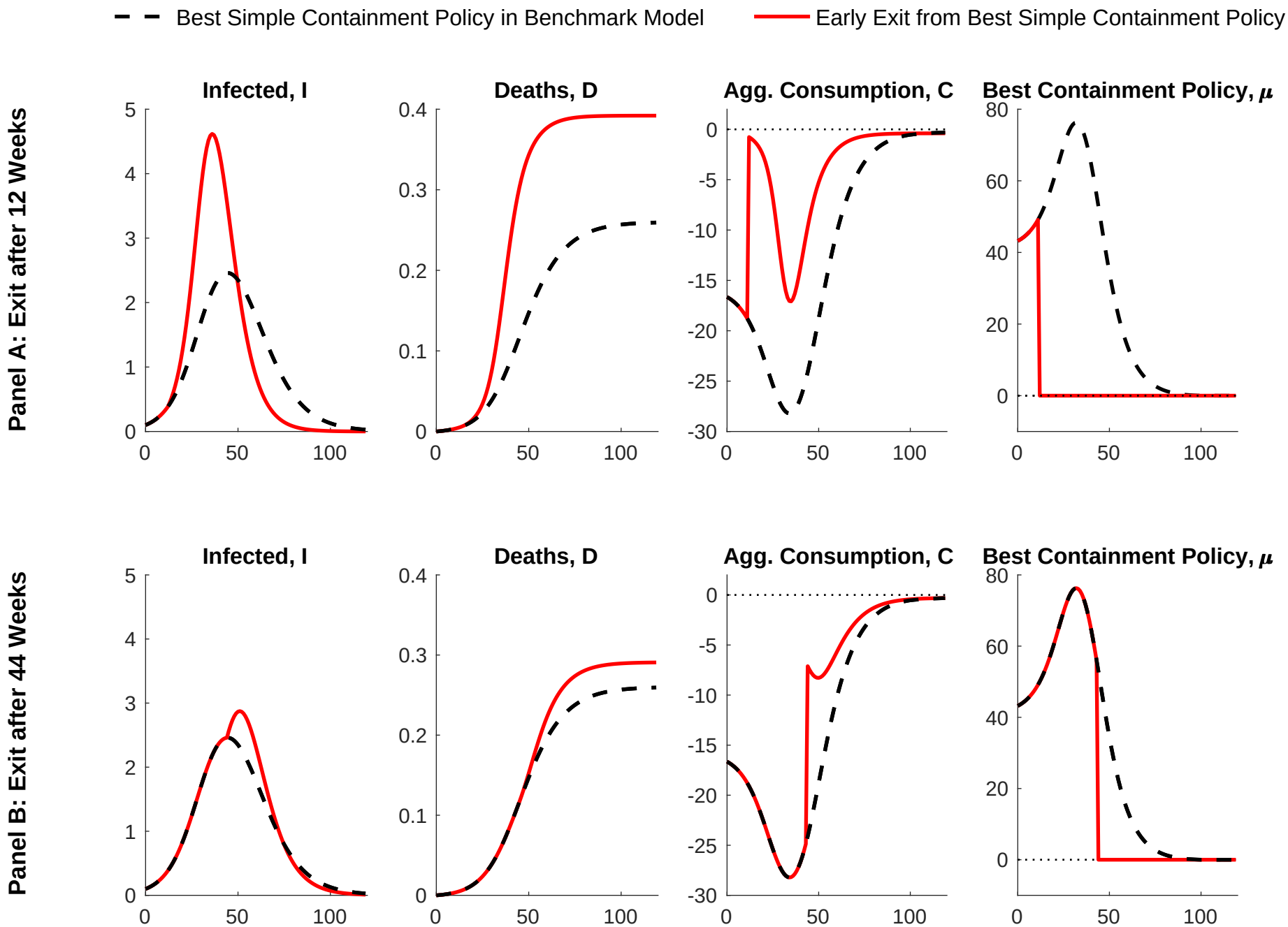


Figure 8: Benchmark SIR-Macro Model (Vaccines, Treatment, Med. Preparedness)



Notes: x-axis in weeks; infected and deaths in % of ini. population; consumption in % dev. from ini. steady state; best containment policy in %.

Figure 9: Benchmark SIR-Macro Model (Vaccines, Treatment, Med. Preparedness)

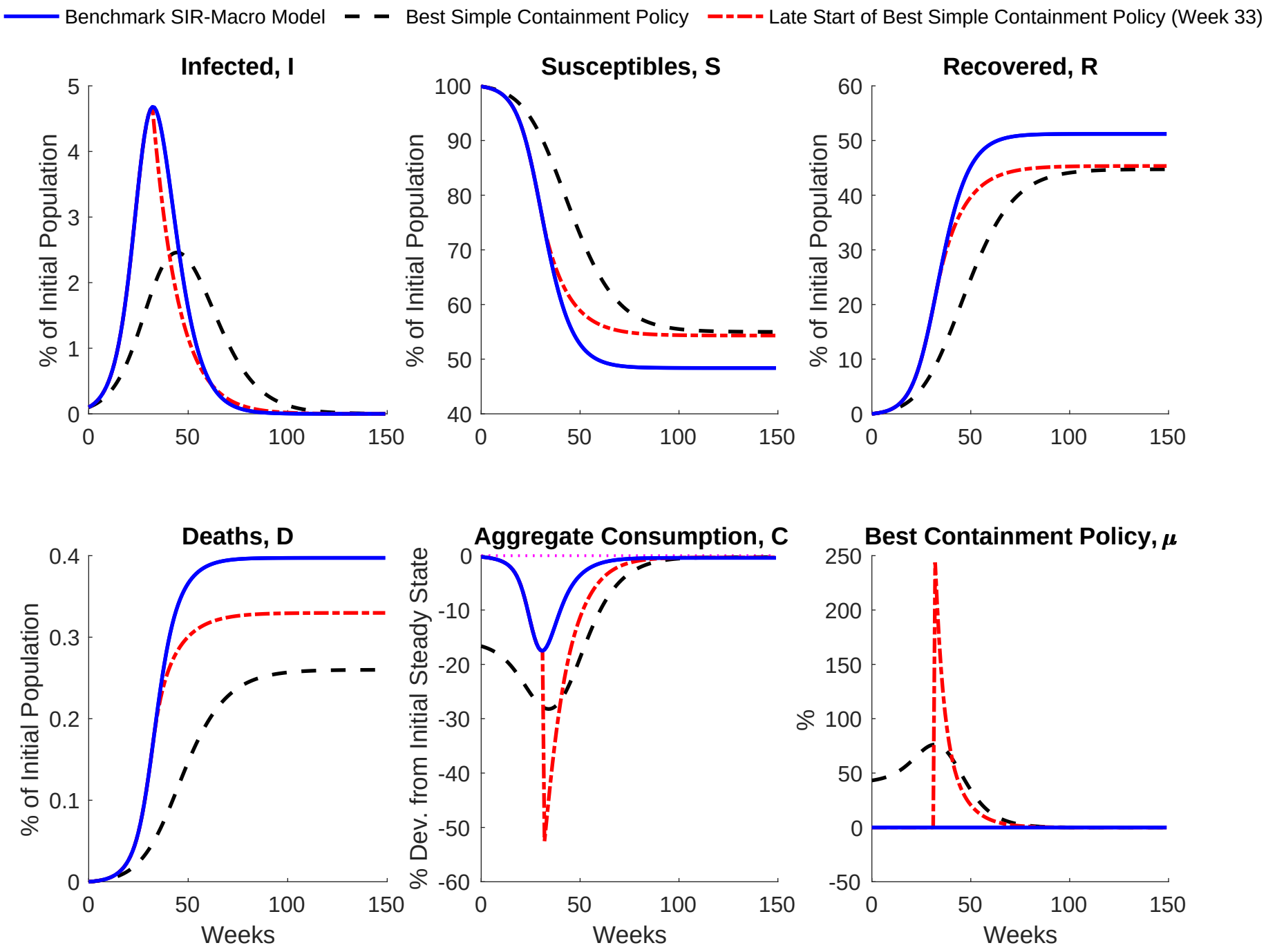


Figure 10: Smart Containment in the Benchmark SIR-Macro Model