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's decision to cut back on consumption and work reduces the severity of the epidemic, as measured by total deaths. These decisions exacerbate the size of the recession caused by the epidemic. 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 optimal containment policy increases the severity of the recession but saves roughly half a million lives in the U.S.

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

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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.[1] While these models are very useful, they do have an important shortcoming: they do not allow for the interaction between economic decisions and rates of infection.

Policy makers certainly appreciate this interaction. For example, in their March 19, 2020 Financial Times op ed Ben Bernanke and Janet Yellen write that

> "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 classic SIR model proposed by Kermack and McKendrick (1927) to study the equilibrium interaction between economic decisions and epidemic dynamics.[2] Our model makes clear that people's decisions to cut back on consumption and work reduce the severity of the epidemic as measured by total deaths.[3] 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 people who are working to the virus. People react to that risk by reducing their labor supply. The demand effect arises because the epidemic exposes people who are purchasing consumption goods to the virus. People react to that risk by reducing their 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 person takes economy-wide infection rates as given.

[1] See, for example, Ferguson et al. (2020).

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

[3] Our paper is related to an emerging literature that combines economics and epidemiology. See Perrings et. al (2014) for a recent review. Our contribution is to focus on how employment and consumption decisions affect the equilibrium dynamics of an epidemic.

A natural question is: what policies should the government pursue to deal with the infection externality? We focus on containment policies that reduce consumption and 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 vaccines and treatments don't arrive before the epidemic is over and healthcare capacity is limited, optimal containment policy saves roughly half-a-million lives in the U.S.

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, epidemic-related policy issues. For example, we do not consider polices aimed at mitigating 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 which 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. We plan to address these important issues in future work. 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.

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 resulting framework as the SIR-macro model.

We choose parameters so that the Kermack-McKendrick SIR model is consistent with the scenario outlined by Angela Merkel in her March 11, 2020 speech.[4] According to this scenario, "60 to 70 percent of the population will be infected as long as this remains the situation." Using 60 percent as our benchmark value, the SIR model implies that the share of the initial population infected peaks at 6.8 percent. Applying this scenario to the U.S. implies that roughly 200 million Americans eventually become infected and one million

[4] "Merkel Gives Germans a Hard Truth About the Corona Virus," New York Times, March 11, 2020.

people die. When we embed the SIR model in a simple general equilibrium framework in which economic decisions do not affect the dynamics of the epidemic, we find that the epidemic causes a relatively mild recession. Average aggregate consumption falls by roughly 0.7 percent in the first year of the epidemic. In the long-run, population and real GDP decline permanently by 0.3 percent reflecting the death toll from the epidemic.

The impact of economic activity on 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 healthcare 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 versus 0.7 percent). This larger decline in economic activity reduces the infection peak (5.2 percent versus 6.8 percent) as well as the percentage of the population that becomes infected (54 versus 60 percent). Critically, the total number of U.S. deaths caused by the epidemic falls from one million to 880 thousand.

To understand the nature of optimal policy, it is important to understand how epidemics end. In both the SIR and SIR-macro models, epidemics end when a sufficiently high fraction of the population acquires immunity, 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 who never recover from an infection. In all versions of our model, it is optimal for policymakers to avoid recurrent epidemics. So a key questions 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. There are two problems with this approach. 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 optimal policy in this world is to build up the fraction of the population that is immune, curtailing 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 the critical immunity level.

An important concern in many countries is that the healthcare system will be overwhelmed by a large number of infected people. To analyze this scenario, we extend the simple SIR-macro model so that the mortality rate 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 mortality 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 of this model and the baseline SIR-macro model is quite small, both with respect to the competitive equilibrium and the optimal 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. These differences are not very important for the competitive equilibrium. But they are very important for the design of optimal policy. With vaccines 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 vaccination 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 mortality rate that rises with the number of infected people. The latter feature reflects capacity constraints in the healthcare 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. Optimal containment dramatically increases the magnitude of the recession. Absent containment measures, average

consumption falls by about 7 percent in the first year of the epidemic. With optimal containment, average containment falls by 22 percent. Notably, the size of the recession is smaller than in the medical preparedness model. The reason is that the prospect of vaccinations and treatments reduce 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 percent of the initial population. The optimal policy reduces the death toll as a percent of the initial population from 0.40 percent to 0.26 percent. For the U.S., 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.

Our paper is organized as follows. In section 2, we describe both the SIR and the SIR-macro model. In section 3, we describe the versions of the model that consider medical preparedness and the possibility of effective treatment and vaccines being discovered. In section 4, we discuss the properties of the competitive equilibrium in different variants of our model. In section 5, we solve the Ramsey policy problems and analyze their implications for the containment of the spread of the virus and for economic activity. In Section 6 we discuss our quantitative results for the benchmark model. Section 7 concludes.

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 pre-infection economy

The economy is populated by a continuum of identical agents with measure one. Prior to the start of the epidemic, all agents are identical and maximize 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 agent is:

$$(1 + \mu_{ct}) c_t = w_t n_t + \Gamma_t.$$

Here, w_t denotes the real wage rate, μ_{ct} is the tax rate on consumption, and Γ_t denotes lump-sum transfers from the government. As discussed below, we think of μ_{ct} as a proxy for containment measures aimed at reducing social interactions.[5] For this reason, we refer to μ_{ct} as the containment rate. The first-order condition for the representative-agent's problem is:

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

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:

$$C_t = A N_t.$$

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

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

The government's budget constraint is given by

$$\mu_{ct} c_t = \Gamma_t.$$

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

[5] See Adda (2016) for microeconomic evidence on the efficacy and cost-effectiveness of contaiment measures to slow the transmission of viral diseases.

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 classic 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.

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 $\pi_{s1}(S_t C_t^S)\left(I_t C_t^I\right)$. 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_{s1} 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 contact intensive than going to a grocery store. For simplicity we abstract from this type of heterogeneity.[6]

Second, susceptible and infected people can meet at work. The number of newly infected people that results from interactions at work is given by $\pi_{s2}(S_t N_t^S)\left(I_t N_t^I\right)$. 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_{s2} reflects the probability of becoming infected as a result of work interactions. We recognize that different jobs require different amounts of contact with people. 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 touching a contaminated surface. The number of random meetings between infected and susceptible people is $S_t I_t$. These meetings result

[6] See Faria-e-Castro (2020) for a model where the pandemic is modeled as a large negative shock to the utility of consumption of contact-intensive services.

in $\pi_{s3} S_t I_t$ newly infected people.

The total number of newly infected people is given by:

$$T_t = \pi_{s1} \left(S_t C_t^S\right) \left(I_t C_t^I\right) + \pi_{s2} \left(S_t N_t^S\right) \left(I_t N_t^I\right) + \pi_{s3} S_t I_t. \tag{1}$$

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 ($\pi_{s1} = 0$, $\pi_{s2} = 0$).

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 that 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 infected people that recovered ($\pi_r I_t$) and the number of infected people that 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 mortality rate, that is the probability that an infected person dies.

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:

$$Pop_{t+1} = Pop_t - \pi_d I_t,$$

with $Pop_0 = 1$.

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,

$$I_0 = \varepsilon,$$

$$S_0 = 1 - \varepsilon.$$

All agents are aware of the initial infection and understand the laws of motion governing population health dynamics.

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 agent ($j = s, i, r$). The budget constraint of a type-j person is

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

where c_t^j and n_t^j denote the consumption and hours worker of agent j, 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 there is no way for agents to 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:

$$\tau_t = \pi_{s1} c_t^s \left(I_t C_t^I \right) + \pi_{s2} n_t^s \left(I_t N_t^I \right) + \pi_{s3} I_t. \tag{8}$$

Susceptible people take as given aggregate variables like $I_t C_t^I$ and $I_t N_t^I$. Critically, they understand that consuming and working less reduces the probability of becoming infected.

The first-order conditions for consumption and hours worked are:

$$u_1(c_t^s, n_t^s) - (1 + \mu_{ct})\lambda_{bt}^s + \lambda_{\tau t} \pi_{s1} \left(I_t C_t^I \right) = 0,$$

$$u_2(c_t^s, n_t^s) + w_t \lambda_{bt}^s + \lambda_{\tau t} \pi_{s2} \left(I_t N_t^I \right) = 0.$$

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 foregone utility of life (see Hall and Jones (2007) for a discussion).

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

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

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

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:

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

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

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

Government budget constraint The government budget constraint is

$$\mu_{ct} \left(S_t c_t^s + I_t c_t^i + R_t c_t^r \right) = \Gamma_t \left(S_t + I_t + R_t \right).$$

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

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

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

In the appendix, we describe our algorithm for computing the equilibrium.[7]

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

3 Medical preparedness, treatments and vaccines

In this section, we extend the SIR-macro model in three ways. First, we allow for the possibility that the mortality rate increases as the number of infections rises. Second, we 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 abstracted from the possibility that the efficacy of the healthcare system will deteriorate if a substantial fraction of the population becomes infected. A simple way to model this scenario is to assume that the mortality rate depends on the number of infected people, I_t:

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

This functional form implies that the mortality rate is a convex function of the fraction of the population that becomes infected. The basic SIR-macro 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 all subsequent periods 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 infected people become recovered. The number of deceased stabilizes once the treatment arrives:

$$D_t = D_{t^*} \text{ for } t \geq t^*.$$

Since anyone can be instantly cured, we normalize the number of susceptible and infected 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.[8] Suppose instead that a vaccine is discovered with probability δ_v per period. Once discovered, the vaccine is provided to all susceptible people in the period of discovery and in all subsequent periods.

The lifetime utility of a susceptible person is given by

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

This expression reflects the fact that with probability $1 - \delta_v$ a person who is susceptible at time t remains so 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 has no impact on people who were infected or have recovered. The lifetime utilities of infected and recovered people person 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 value of these variables are

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

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

[8] There is a sizable literature that analyses how private inventives to become vaccinated affect epidemic dynamics and optimal public health policy. See, for example, Philipson (2000) and Manski (2016).

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

Below, we report our choice of parameters. We are conscious that there is considerable uncertainty about the true values of these parameters. Below, we report robustness of our results to parameter configurations.

Each time period corresponds to a week. To choose the mortality 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 mortality rates based on data from other countries are probably biased upwards because the number of infected people is likely to be underestimated. We compute the weighted average of the mortality rates using weights equal to the percentage of the U.S. population for different age groups. If we exclude people aged 70 and over, because their labor-force participation rates is very low, we obtain an average mortality rate of 0.4 percent. If we exclude people aged 75 and over, we obtain an average mortality rate of 0.7 percent. Based on these estimates, we set the mortality rate equal to 0.5 percent and report robustness below. As in Atkeson (2020), we assume that it takes on average 18 days to either recover or die from the infection. Since our model is weekly, we set $\pi_r + \pi_d = 7/18$. A 0.5 percent mortality rate for infected people implies $\pi_d = 7 \times 0.005/18$.

[9] This estimate is roughly 8 times larger than the average flu death rate in the U.S.

We now discuss our calibration procedure to choose the values of π_{s1}, π_{s2}, and π_{s3}. It is common in epidemiology to assume that the relative importance of different modes of transmission is similar across airborne diseases. Ferguson et al. (2006) argue that, in the case of influenza, 30 percent of transmissions occur in the household, 33 percent in the general community, and 37 percent occurs in schools and workplaces.

To map these estimates into our transmission parameters, we proceed as follows. We use the Bureau of Labor Statistics 2018 Time Use Survey 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 time spent "eating and drinking outside the home," we multiply time spent "eating and drinking" by the fraction of total food expenditures on "food away from home" in 2018 (54 percent 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 percent. Since 33 percent of transmissions occur in the general community, we estimate that 16 percent of transmissions are related to consumption (0.33×0.48).

Turning to work, recall that 37 percent of transmissions 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 (2009). According to the Bureau of Labor Statistics, as of 2018 the number of students and workers in the population were 76.6 million and 162.1 million, respectively. These considerations imply that the fraction of transmissions occurring in the workplace is 46 percent ($162.1 \times 4 / (162.1 \times 4 + 76.6 \times 10)$). Since 37 percent of transmissions occur in schools and workplaces, 17 percent of transmissions are related to work (0.37×0.46).

We assume that virus transmission not related to consumption or work activities belong to the exogenous category ($\pi_{s3} S_t I_t$) emphasized in the SIR model. The values of π_{s1}, π_{s2} and π_{s3} are chosen to satisfy:

$$\frac{\pi_{s1} C^2}{\pi_{s1} C^2 + \pi_{s2} N^2 + \pi_{s3}} = 1/6,$$

[10] We classify the following entries in the time-use 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 and helping non-household members.

$$\frac{\pi_{s2}N^2}{\pi_{s1}C^2 + \pi_{s2}N^2 + \pi_{s3}} = 1/6.$$

Here, C and N are consumption and hours worked in the pre-infection steady state. In addition, we assume that in the limit of the simple SIR model 60 percent of the population either recovers from the infection or dies. This assumption corresponds to the Merkel scenario discussed in the introduction. The resulting values for π_{s1}, π_{s2}, and π_{s3}, are 7.8408×10^{-8}, 1.2442×10^{-4}, and 0.3901, respectively.

Our calibration procedure requires various judgement calls. For example, we had to chose 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 that 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 in 2019 from the U.S. Bureau of Economic Analysis and the average number of hours worked from the Bureau of Labor Statistics 2018 time-use survey. 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 decisions 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 the relative productivity of infected people is 0.8. This value is consistent with the notion that symptomatic agents don't work and the assumption that 80 percent of infected people are asymptomatic according to the China Center for Disease Control and Prevention. In the baseline SIR-macro model μ_{ct} is equal to zero.

In the medical preparedness model, we fix κ to 0.9, which implies a peak mortality rate of 1 percent, two times higher than that in the benchmark model. We obtain this higher mortality rate computing the weighted average of the mortality rates in Italy with weights equal to the percentage of different age groups in the U.S. population. As in our baseline scenario, we exclude people aged 70 and over.

[11] We chose to focus on the connection between market activities and the epidemic. This choice led us to abstract from the response of non-market 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).

In both the treatment and vaccination models, we set $\delta_c = \delta_v = 1/52$, which implies that it takes on average 52 weeks for these medical discoveries to become available.

4.1.1 The model's basic reproduction number

A widely used statistic 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 where everybody is susceptible ($S_t = 1$). 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, in our model is the ratio of the number of newly infected people to the total number of infected people. The value of γ is equal to T_0/I_0. The expected number of infections caused by a single infected person is

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

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

The value of $\mathcal{R}_0$ in the SIR and benchmark SIR-macro models is 1.50 and 1.45, respectively. These values are lower than current point estimates of $\mathcal{R}_0$ for COVID-19, but consistent with the evidence taking sampling uncertainty into account. For example, Riou and Althaus (2020) report a point estimate of 2.2 with a 90 percent confidence interval of 1.4 to 3.8.

4.2 The SIR model

The black dashed lines in Figure 1 display the equilibrium population dynamics implied by the SIR model. The share of the initial population that is infected peaks at 6.8 percent in week 31. Thereafter, this share falls because there are fewer susceptible people to infect. Eventually, 60 percent 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 mortality rate of 0.5 percent implies that the virus kills roughly one million people in the U.S.

Figure 1 shows that the epidemic induces a recession: aggregate consumption falls by roughly 1.5 percent 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 agents in the overall population. Second, the death toll caused by the epidemic permanently reduces the size of the work force.

Since production is constant returns to scale, per capita income is the same in the post- and pre-epidemic steady states. In the post-epidemic steady state, population and real GDP are both 0.3 percent 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 the SIR-macro model, susceptible households can lower the probability of being infected by reducing their consumption and hours worked. The solid blue lines in Figure 1 show how the epidemic unfolds in this model.

The share of the initial population that is infected peaks at 5.3 percent in week 33. The peak is substantially smaller and occurs somewhat later than the corresponding peak in the SIR model. Eventually, 54 percent of the population becomes infected. So, for the U.S., roughly 180 million people eventually become infected and 890 thousand 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. Consistent with Figure 2, there are no offsetting effects arising 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 percent, a fall seven times larger than in the SIR model.

For similar reasons, the dynamics and magnitude of the drop in hours work is very different in the two models. In the SIR model, hours worked decline smoothly falling by 0.30 percent 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 percent 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 agents. Interestingly, the long-run decline in hours worked is lower in the SIR-macro model (0.27 percent) than in the SIR model (0.30 percent). 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 red dashed-dotted lines in Figure 4 show that the competitive equilibrium with an endogenous mortality rate involves a much larger recession than in the basic SIR-macro model (blue solid lines). The reason is that people internalize the higher mortality rates associated with an healthcare system that is overburdened with infected people. Since the costs of becoming infected are much higher, people cut back 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 vaccines models

As discussed in the introduction, the possibility of treatment and vaccination have similar qualitative effects on the competitive equilibrium. Compared to the basic SIR-macro model people become more willing to engage in market activities. The reason is that the expected costs associated with being infected are smaller. Because of this change in behavior, the recession is less severe. In Figures 5 and 6 the blue-solid and red-dashed-dotted lines virtually coincide. So, in practice the quantitative effect of the possibility of treatments or vaccinations on the competitive equilibrium is quite small.

4.6 Robustness

Table 1 reports the results of a series of robustness exercises where we vary key parameters of the basic SIR-macro model. Recall that we choose our baseline parameters so that, in the SIR model, 60 percent of the population is eventually infected by the virus. We report results for two alternative long-run infection rates in the SIR model: 50 and 70 percent. Not surprisingly, the higher is the long-run infection rate, the larger is the drop in consumption, the peak infection rate, the cumulative death rate, and the total number of U.S. deaths.

Next we consider the parameter ϕ^i that controls the productivity of infected workers. The lower is ϕ^i, the larger is the average consumption drop, the peak infection rate, the cumulative death rate, and the total number of U.S. deaths.

Table 1 also reports the results for different parameters of the infection transmission function, (1). Recall that in the benchmark model we choose our baseline parameters so

that, in the beginning of the infection episode, economic decisions account for 1/3 of the infection rate. Table 1 summarizes results for the case where economic decisions account for 1/6 of the initial infection rate. In this scenario, the drop in consumption is smaller. The peak infection rate, the cumulative death rate, and the total number of U.S. deaths is larger. These results reflect the fact that people understand that economic activity has less of an impact on infection rates. Table 1 also reports the case in which economic decisions account for 2/3 of the initial infection rate. In this scenario, the drop in consumption is larger. But the peak infection rate and the cumulative death rate are smaller. These results reflect the fact that people cut back more on economic activities since these activities have a larger impact on infection rates.

Next, we increase the mortality rate from 0.5 percent to 1 percent. 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 death rate, and the number of U.S. deaths rise.

Finally, Table 1 summarizes 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 mortality rate in the medical preparedness model peaks at 1 percent. Table 1 shows that this higher value of κ is associated with a more severe recession as people curtail economic activity in response to higher mortality rates. While the peak level of infections fall, the cumulative death rate and the total number of U.S. deaths rise.

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

5 Economic policy

The competitive equilibrium of our model economy is not Pareto optimal. There is a classic externality associated with the behavior of infected agents. Because agents are atomistic, they don't take into account the impact of their actions on the infection and death rates of other agents. In this section, we consider a simple Ramsey problem designed to deal with this externality.

As with any Ramsey problem, we must take a stand on the policy instruments available. In reality, there are many ways in which governments can reduce social interactions. Exam-

ples of containment measures include shelter-in-place laws and shutting down of 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 all agents. We refer to this tax as the containment rate.

We compute the optimal sequence of 250 containment rates $\{\mu_{ct}\}_{t=0}^{249}$ that maximize social welfare, U_0, defined as a weighted average of the lifetime utility of the different agents:

$$U_0 = s_0 U_0^s + i_0 U_0^i + r_0 U_0^r.$$

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, it is optimal to escalate containment measures gradually over time. The optimal containment rate rises from 4.5 percent in period zero to a peak value of 72 percent in period 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 it is optimal to intensify containment measures. 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 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 percent, reducing the death toll from 0.27 to 0.21 percent of the initial population. For a country like the U.S., this reduction represents roughly two hundred thousand 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 percent without containment measures to about 17 percent 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? 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.

5.1 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 versus 72 percent) and occurs earlier (at week 33 versus 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. Not only do agents not internalize the cost of consumption and work on infection rates, they also don't internalize the aggregate increase in mortality rates.

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

5.2 The treatment and vaccines models

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

The black-dashed 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 percent. But this recession is worth incurring in the hope that the vaccination arrives 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 vaccines model under optimal containment policy on a path where a vaccine does not arrive. Compared to the competitive equilibrium (red-dashed-dotted lines), the peak of the infection rate drops from 5.3 to 3.3 percent of the initial population. Moreover, the infection peak occurs in period 42 rather

than in period 33. Absent a vaccine being discovered, the optimal containment policy reduces the death toll as a percent of the initial population from 0.27 percent to 0.24 percent. For the U.S., this reduction amounts to about a one hundred thousand lives. It is important to remember that this reduction pertains to a worst-case scenario in which vaccines never arrive.

Above we discussed why it is not optimal to introduce immediate containment measures in the basic SIR-macro and treatment models. But why is optimal policy so different in the vaccination model? The basic reason is that unlike treatment, a vaccine does not cure infected people. The expected arrival of a vaccine also reduces the importance of building up the fraction of the population that is immune to a level that prevents the recurrence of an epidemic.

6 Quantitative predictions for the benchmark model

In the previous sections, we separately analyzed 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 the possibility of vaccines and medical treatment, as well as the impact of a large number of infections on the efficacy of the healthcare system.

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

6.1 Optimal policy in the complete model

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

From a qualitative point of view, the complete model inherits key features of its underlying components. Consistent with the vaccination model, it is optimal to immediately introduce severe containment (43 percent). Consistent with the treatment and medical preparedness models, it is optimal to ramp containment up as the number of infections rise.

The maximal containment rate reaches 76 percent in period 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 percent. With containment, this fall is 22 percent. Notably, the size of the recession is smaller than in the medical preparedness model. The reason is that the prospect of vaccinations and treatments reduce 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 percent of the initial population. The optimal policy reduces the death toll as a percent of the initial population from 0.40 percent to 0.26 percent. For the U.S., 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 arrived, many more lives would saved. Thankfully, they would be saved by medicine rather than by containment policies.

6.2 The costs of ending containment too early

As a practical matter, policymakers could face intense pressure to prematurely end containment measures because of their 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 display the response of the economy to an unanticipated end of optimal containment policy after weeks 12 and 44, respectively. Week 44 is when infections peak under optimal containment. The black dashed lines pertain to the behavior of the economy when optimal policy is fully implemented.

From Panel A, we see that abandoning containment initially generates a large recovery with consumption surging by roughly 17 percent. 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 brings about a temporary rise in consumption but no long-lasting economic benefits. Tragically, abandonment leads to a substantial rise in the total number of deaths caused by the epidemic.

Panel B shows that the longer policy makers pursue optimal containment policy, the better. Both the temporary gains and the losses of abandoning optimal policy in Panel B are smaller than those in Panel A. We conclude that it is important for policymakers to resist the temptation to pursue transient economic gains associated with abandoning containment

measures.

6.3 The costs of starting containment too late

Policymakers can also face pressures to delay implementing optimal containment measures. The red dashed-dotted lines in Figure 9 display the impact of only beginning containment in week 33, the period in which infections peak. We assume that optimal policy is calculated and implemented from that point on. The black dashed lines pertain to the behavior of the economy when the optimal containment policy is implemented from week zero on. The solid blue line corresponds to the competitive equilibrium with no containment measures.

The optimal policy that begins in week 33 involves draconian containment measures that lead to an enormous drop in economic activity. The reason is simple: with infections raging, the economic externalities associated with economic activity are very large.

Despite the draconian measures, the total number of deaths associated with the epidemic is much larger than if the optimal containment policy is implemented without delay. Still, as far as the death toll of the epidemic is concerned, late containment (red dashed-dotted lines) is better than no containment at all (blue solid lines). We conclude that it is important for policymakers to resist the temptation to delay optimal containment measures for the sake of initially higher short-run levels of economic activity.

7 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 policymakers.

Finally, we note that our model 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-side chains. It is important to embody

these forces in macroeconomic models of epidemics and study their positive and normative implications.

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Appendix A Computing the Equilibrium

For a given sequence of containment rates, $\{\mu_{ct}\}_{t=0}^{H-1}$, 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:

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

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

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

Iterate backwards 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:

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

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

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

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

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

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

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

$$T_t = \pi_{s1}(S_t c_t^s)\left(I_t c_t^i\right) + \pi_{s2}(S_t n_t^s)\left(I_t n_t^i\right) + \pi_{s3} S_t I_t,$$

$$Pop_{t+1} = Pop_t - \pi_d I_t,$$

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

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

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

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

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

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

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

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

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

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

$$(c_t^s)^{-1} - \lambda_{bt}^s(1+\mu_{ct}) + \lambda_{\tau t}\pi_{s1}\left(I_t C_t^I\right) = 0.$$

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:

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

$$\mu_{ct}\left(S_t c_t^s + I_t c_t^i + R_t c_t^r\right) = \Gamma_t\left(S_t + I_t + R_t\right),$$

$$-\theta n_t^s + A\lambda_{bt}^s + \lambda_{\tau t}\pi_{s2}\left(I_t n_t^I\right) = 0.$$

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

	Consumption %[b]	**Infection Rate** %[c]	**Death Rate** %[d]	**U.S. Deaths** Millions[e]
Percent of population eventually infected in canonical SIR model				
50	−3.42	3.20	0.21	0.72
60 (baseline)	−4.66	5.23	0.26	0.88
70	−5.21	8.15	0.31	1.05
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 healthcare 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

[a] See section 4.6 for a discussion of the results provided in this table.
[b] Average drop of consumption in first year relative to pre-infection steady state.
[c] Peak infection rate relative to pre-infection population.
[d] Death rate at the end of the epidemic relative to pre-infection population.
[e] Total number of deaths in the U.S. at the end of the epidemic.

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

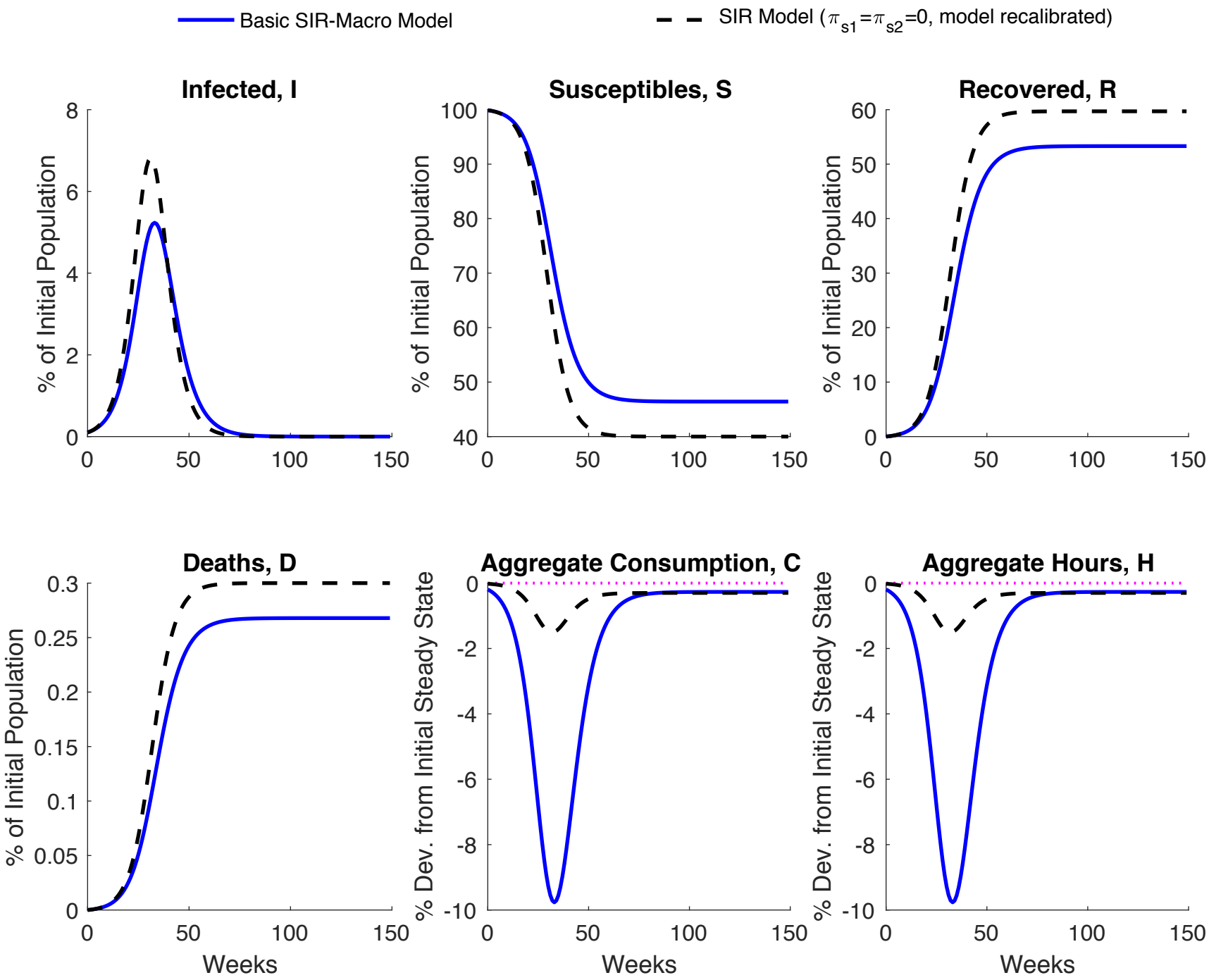


Figure 2: Consumption and Hours by Type in Basic SIR-Macro Model

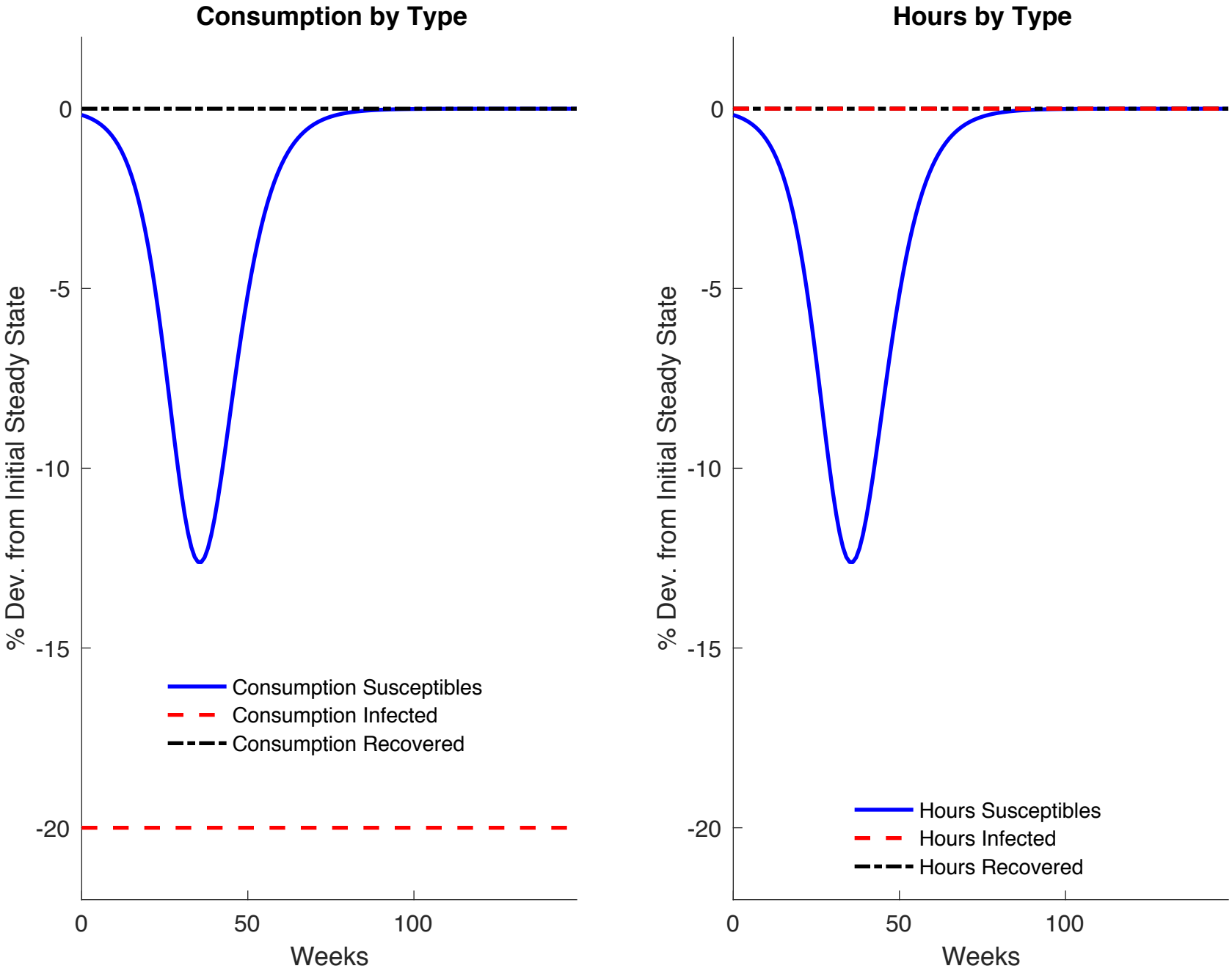


Figure 3: Basic SIR-Macro Model With and Without Containment

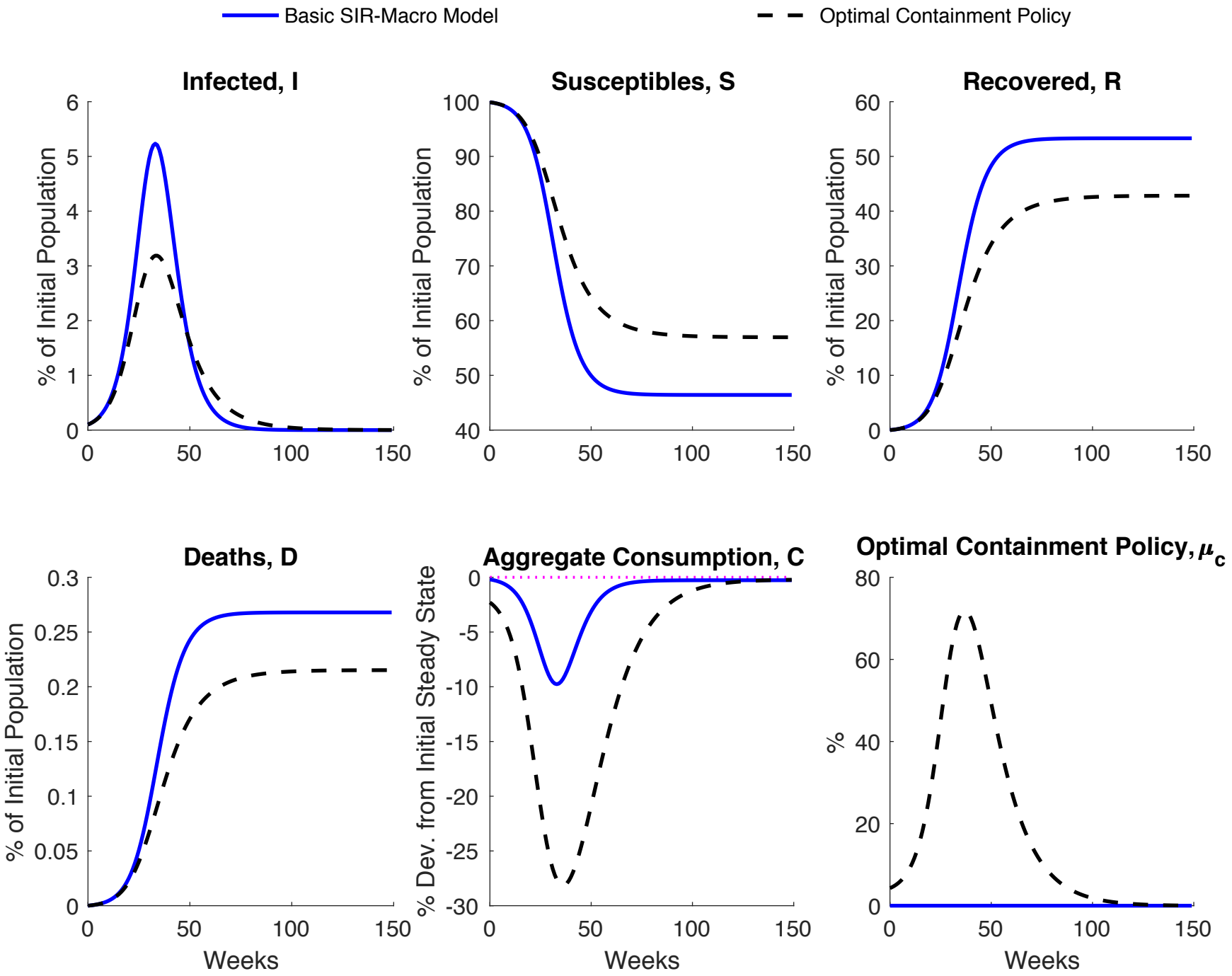


Figure 4: Medical Preparedness

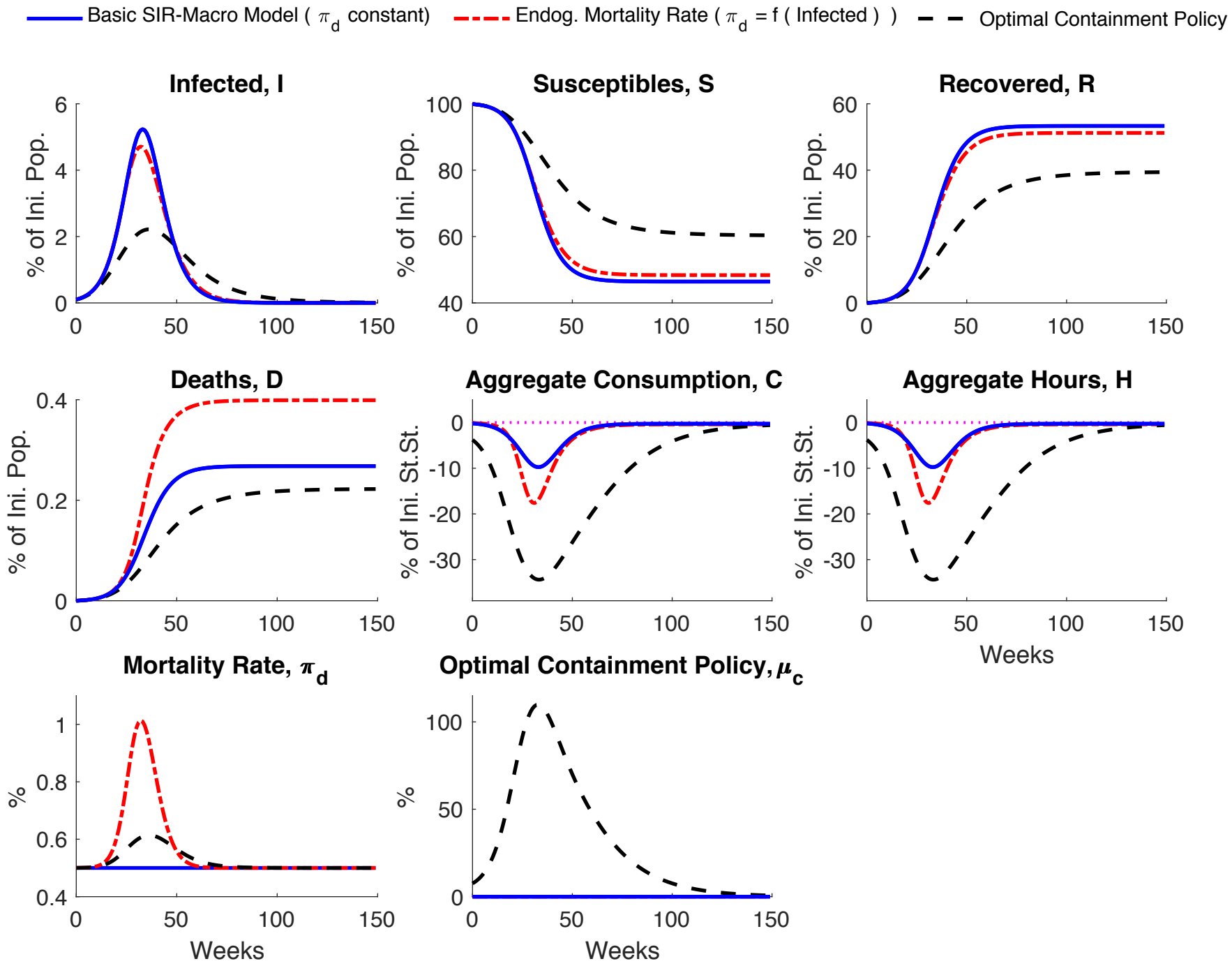


Figure 5: SIR-Macro Model With Treatments

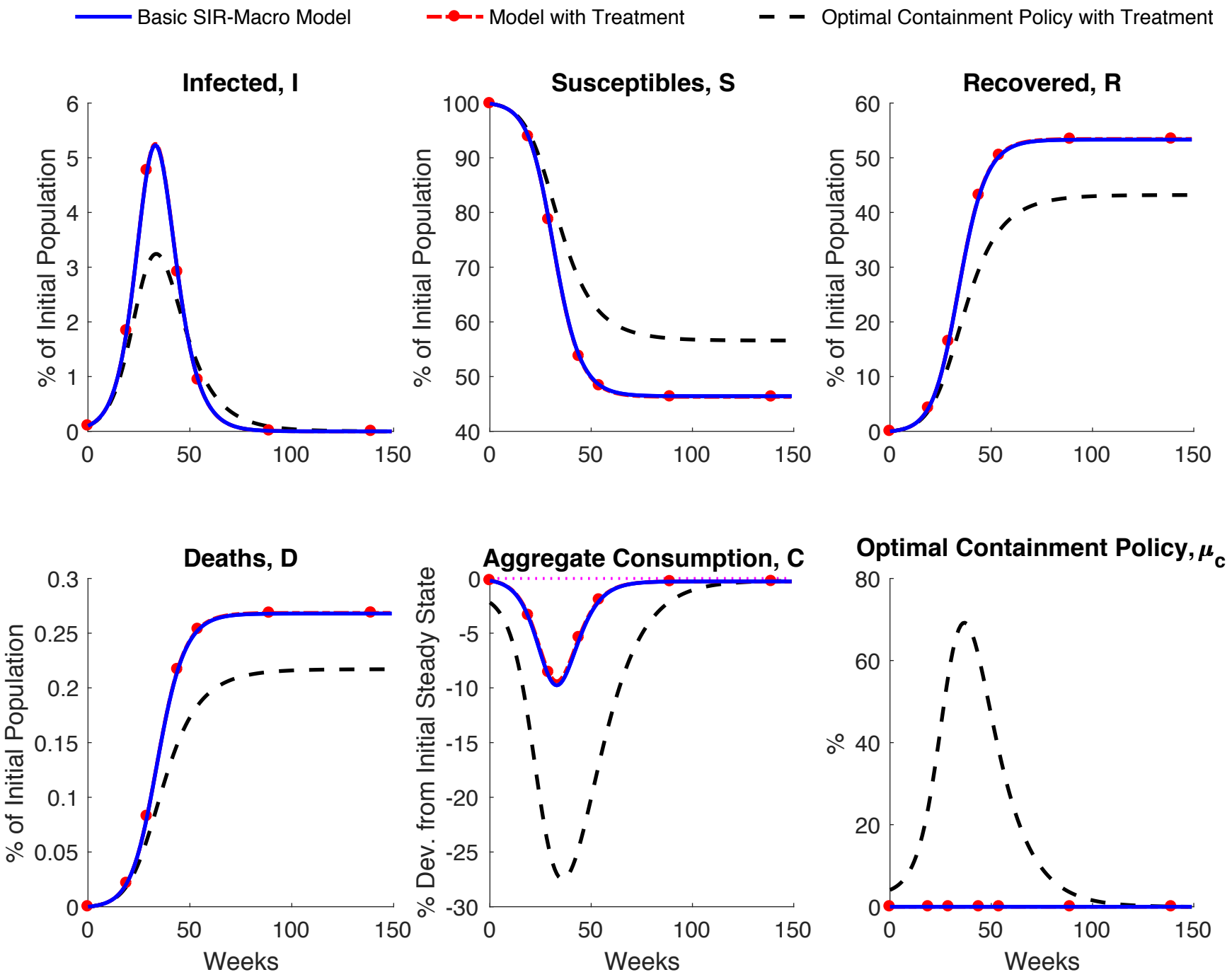


Figure 6: SIR-Macro Model With Vaccines

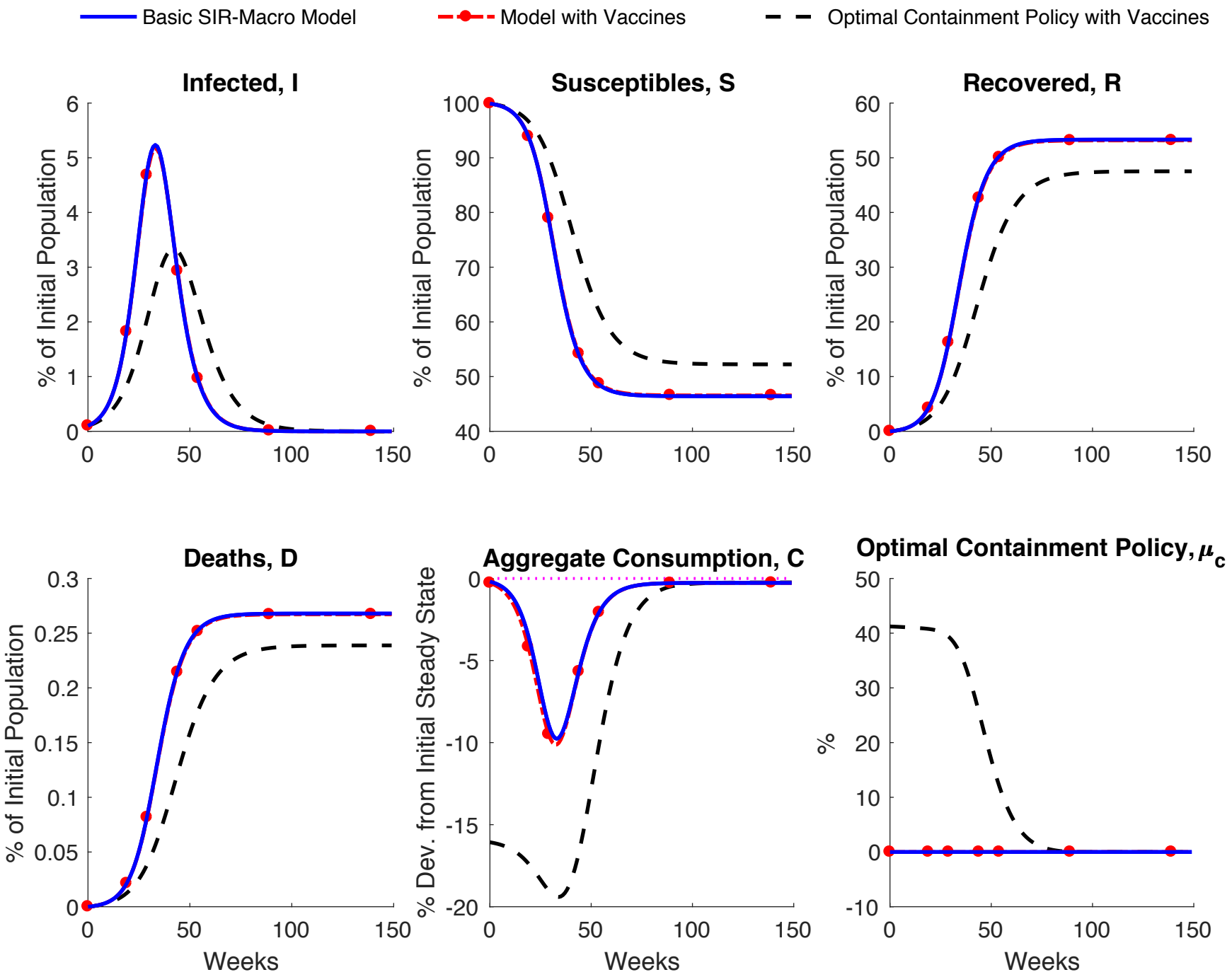


Figure 7: Benchmark SIR-Macro Model (Vaccines, Treatment, Med. Preparedness)

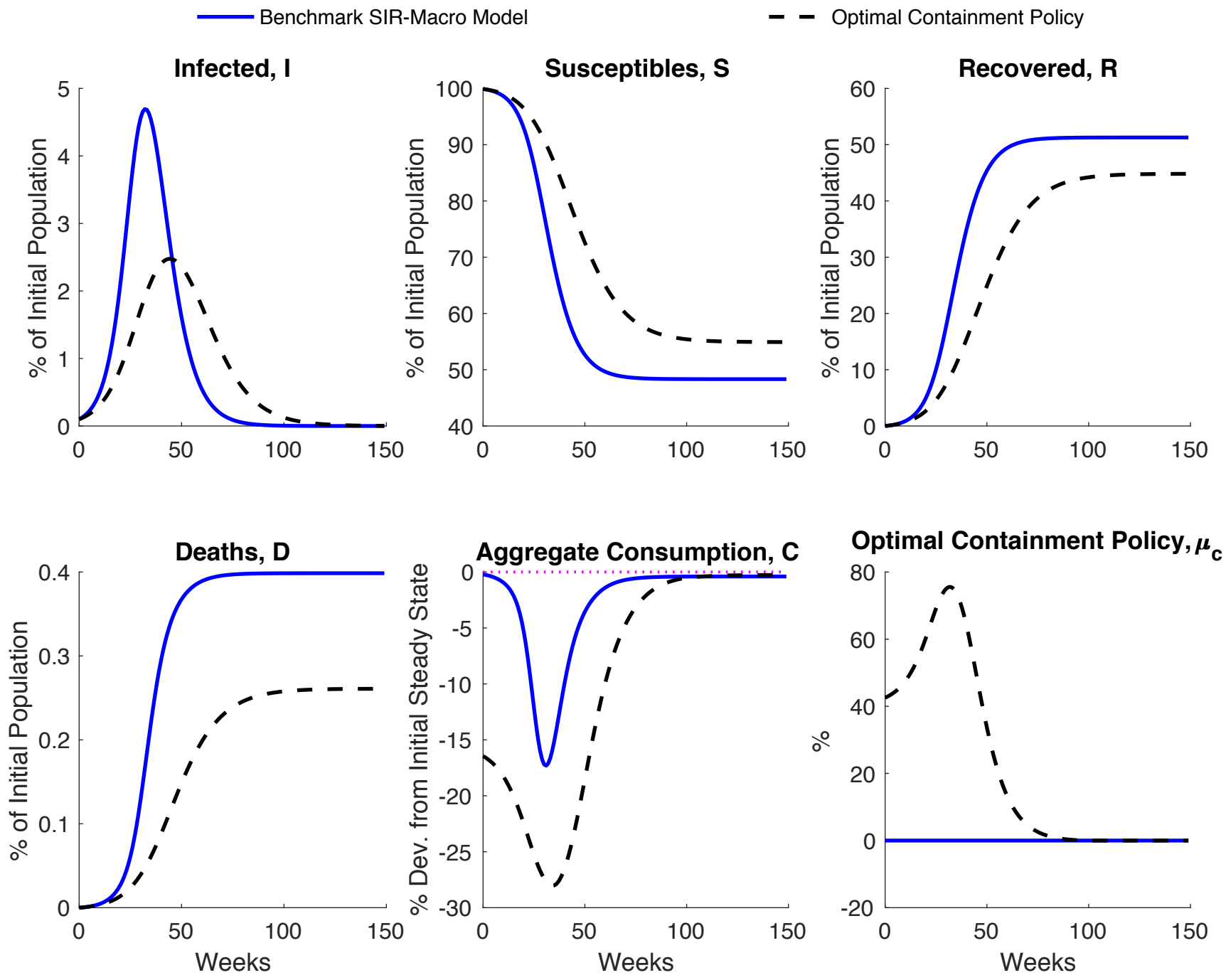
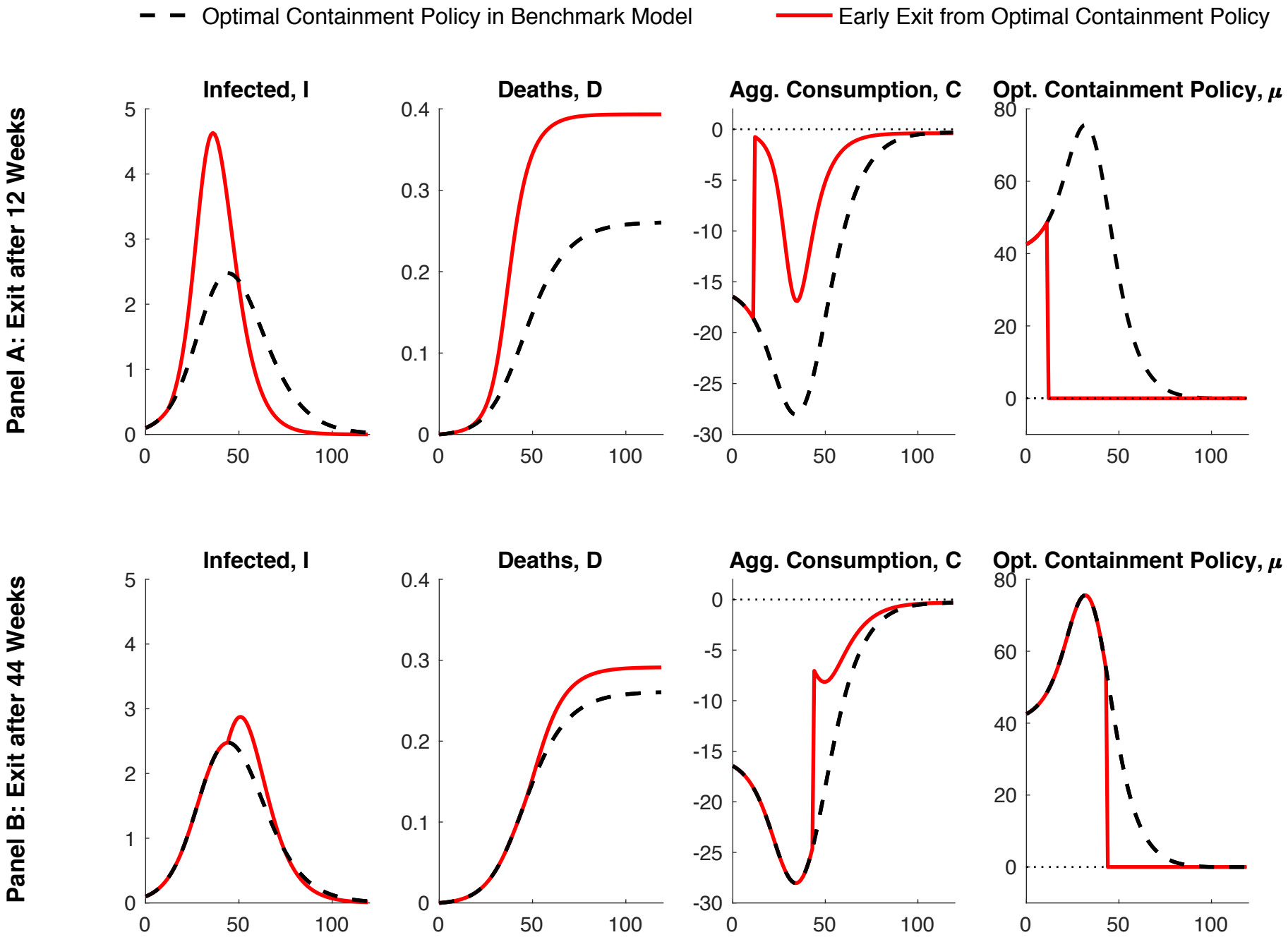


Figure 8: Benchmark SIR-Macro Model (Vaccines, Treatment, Med. Preparedness)



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

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