

# Market Size, Innovation, and the Economic Effects of an Epidemic\*

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## Abstract

We develop a framework for the analysis of the economic effects of an epidemic that incorporates firm-specific innovation and endogenous entry. Transition dynamics is characterized by two differential equations describing the evolution of the mass of susceptible in the population and the ratio of the population to the mass of firms. An epidemic propagates through the economy via changes in market size that disturb incentives to enter the market and to undertake innovative activity. We evaluate state-dependent interventions involving policy rules based on tracking susceptible or infected. Simple policy rules are announced at the time of the outbreak and anchors private sector's expectations about the time path of the intervention, including the end date. Welfare gains or losses relative to the do-nothing scenario are computed accounting for transition dynamics.

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# 1 Introduction

The spread of infectious diseases (“epidemics”) has ravaged humanity throughout its history, often leaving indelible scars. The Spanish Flu of 1918-20 and the most recent COVID-19 are only two examples of a long list of severe epidemics involving substantial disruptions in economy activity. Evaluating the economic consequences of an epidemic, including the trade-offs involved in public interventions, requires a structural model of the economy that allows for counterfactual analysis under alternative policy scenarios.

A large literature tackles these questions in the context of general equilibrium models in which an epidemic, and the policy response to it, affects economy activity through changes in market hours worked, consumption, and physical capital investment. [Bloom, Kuhn and Prettner \(2022\)](#) provide a timely and thorough review of this literature and show that under the stress of COVID-19 it has recently undergone a revival and grown massively. The typical paper augments an economic model with a canonical epidemiological model of an infectious disease and shows that the immediate effect of an epidemic is a reduction in the amount of labor services supplied to the market, akin to a negative “labor supply shock,” which can be amplified by, e.g., a lockdown policy. How and to what extent this initial epidemic shock propagates through the economy depends on the specific features of the economic environment and mechanisms at play (see [Section 2](#) for a literature review).

The objective of this paper is to build a tractable, dynamic general equilibrium model to address important, yet overlooked, questions relating the spread of an epidemic to market structure, innovation, and long-run productivity growth. To achieve this goal, we integrate a standard Susceptible-Infected-Recovered-Deceased (SIRD) model of infectious diseases into a growth model featuring variety expansion and firm-specific cost-reducing innovation ([Peretto and Connolly, 2007](#)). Endogenous market structure propagates the epidemic shock, leading to a very persistent response of the growth rate, featuring a sharp initial drop followed by a prolonged hump-shaped mean-reversion to the initial value that features overshooting. (The long-run growth rate remains unchanged because we work with a growth model without the strong scale effect.)

Methodologically, we propose the concept of “infection function” to reduce the system describing the equilibrium dynamics of the economy, in and out of the steady state, to two differential equations. Solving for the equilibrium of our growth model is easy despite the highly nonlinear structure of the dynamical system. Epidemiological models feature a precise relationship between the fraction of infected in the population and the fraction

of susceptible. In most cases, such relationship can be characterized as a trajectory in 2D space. If one can solve for such trajectory, one obtains a function representing the infection rate as determined by the fraction of susceptible. To our knowledge, epidemiologists do not refer to this object as the “infection function” but, on reflection, we noted that the concept is very useful in integrating epidemic dynamics in dynamic economic models because it allows one to characterize the law of motion of the epidemic as a shock with a precise and transparent dynamic structure.

In our model, the key to the propagation of the epidemic shock is the market size effect. The epidemic manifests itself as a shock to employment and thereby to the size of the market in which firms operate. The hump-shaped dynamics in the fraction of infected in the population due to the spread of the disease translates into U-shaped dynamics in the amount of labor services available for production. This leads to a drop in per capita consumption expenditure, which initiates transition dynamics fueled by changes in the incentives of entrepreneurs to enter the market with new goods and the incentives of incumbents firms to invest in innovation. More specifically, the model features highly non-linear transition dynamics: depending on the magnitude of the fall in expenditure, the economy may experience a prolonged period of net firm exit with shutdown of firm-specific innovation. In this extreme scenario, growth in total factor productivity (TFP) does not only slow down, but is negative for a while, implying a temporary fall in the *level* of TFP. In fact, if one allows for the possibility of death as in the standard SIRD case, and so a permanent reduction in population, the loss of product variety is permanent because the new steady-state associated with a smaller population size exhibits fewer firms and so less product variety.<sup>1</sup>

As a proof of concept for our framework, we use the model to study policy interventions that operate through a reduction in the transmission rate of the epidemic, as well as a direct reduction in the labor input, mimicking the effect of a mandated lockdown. Such interventions are state-dependent and involve *policy rules* based on tracking the fraction of susceptible or infected in the population, akin to the Taylor’s rule for monetary policy. Policy rules are announced at the time of the outbreak and anchor private sector’s expectations about the time path and the end date of the intervention. We restrict our attention to simple rules to retain analytical tractability, and compute welfare gains/losses relative

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<sup>1</sup>Because of the sterilization of the strong scale effect, the steady-state growth rate of the economy is invariant to population size. By contrast, the steady-state levels of per capita output, TFP, and the mass of firms do depend on population size. Notably, a smaller population size relative to the no-epidemic counterfactual, would permanently reduce the mass of firms and thereby product variety, with a permanent negative effect on per capita output and aggregate productivity.

to a do-nothing scenario, accounting for transition dynamics.

Numerical simulations illustrate a sharp policy trade-off. Slowing down contagion amounts to engineering a deeper fall in economic activity relative to the do-nothing scenario. The interventions that we study generally induce a persistent slowdown in aggregate productivity growth. Depending on the severity and duration of the intervention, transition dynamics may feature prolonged periods of net exit with negative TFP growth.

The paper is structured as follows. Section 2 discusses the related literature. In Section 3, we present the basic growth model, into which we embed the SIRD epidemiological model. In Sections 4 and 5, we analyze the equilibrium dynamics of the model pre- and post-infection. Section 6 discusses the effects of policy intervention. Section 7 presents numerical results. Section 8 concludes. Appendices A, B, and C contain details on model's derivations, parametrization, and additional results.

## 2 Related Literature

This paper adds to the large literature studying the economic effects of infectious disease. This literature, recently reviewed by Bloom, Kuhn and Prettnner (2022), predates COVID-19 and identifies several pathways through which diseases and the associated health policy interventions affect the economy. The unexpected break out of COVID-19 has spurred a massive revival of research along such lines and expanded our understanding of the mechanisms at play.

Goenka, Liu and Nguyen (2014) is an example of an early (pre-COVID-19) contribution that integrates epidemiological dynamics into a neoclassical growth model with investment in health which affects the recovery rate from the disease. It establishes that a disease-free steady state can coexist with a disease-endemic steady state in which health expenditure is either positive or zero depending on parameter values. Goenka and Liu (2020) extend this framework by allowing for investment in human capital. A disease-free balanced growth path with sustained economic growth coexists with multiple disease-endemic balanced growth paths in which the economy either grows at a slower rate or remains stuck in a poverty trap without human capital accumulation. Greenwood et al. (2019) quantitatively evaluate the economic impact of the HIV / AIDS epidemic in Malawi using a general equilibrium search model of sexual behavior. Bloom, Kuhn and Prettnner (2022) discuss several more examples of pre-COVID-19 contributions. A recent empirical paper that in spirit is related to ours is Madsen, Robertson and Ye (2022). It studies the ex-

tent to which the behavioral response of private agents to outbreaks of the bubonic plague over three centuries caused disruptions of trade and breakdowns of market integration. It thus focuses on a Smithian concept of “extent of the market” as the main transmission channel for the propagation of the effects of the disease throughout the economy.

Focusing on COVID-19, the number of contributions studying different aspects of that epidemic is growing at a surprisingly fast rate.<sup>2</sup> Early contributions by [Eichenbaum, Rebelo and Trabandt \(2020a\)](#), [Eichenbaum, Rebelo and Trabandt \(2020b\)](#), and [Eichenbaum, Rebelo and Trabandt \(2020c\)](#) study the economic consequences of COVID-19, testing and quarantining policies in the context of a neoclassical growth model (with/without monopolistic competition) and of a New Keynesian model with sticky prices. The main idea in these papers is that individual consumption and work decisions affect the transmission rate of the epidemic, creating an infection externality. The competitive equilibrium is thus not Pareto optimal. Testing and quarantining can improve upon the *laissez-faire* outcome.

Relative to the existing literature, we ask a different question and study a new mechanism in which market structure and transition dynamics take center stage. Notably, transition dynamics of economic variables is considerably slower than epidemiological dynamics. Through changes in the fraction of infected in the population, an epidemic leads to a fall in expenditure which shrinks the size of the market served by producers. This market-size effect reduces incentives to entry and incumbents’ cost-reducing activity, leading to the prolonged hump-shaped response of the growth rate described above. The source of this shape is the internal propagation mechanism at play in the economic block of the model, which yields that epidemiological and economic variables move at rather different speed. Further, we propose state-dependent intervention rules based on tracking the susceptible or the infected in the population. Such rules are an essential part of the definition of equilibrium and naturally allow for calculation of their effects on welfare. An aspect of our contribution worth stressing is that judging by the thorough review by [Bloom, Kuhn and Prettner \(2022\)](#) the literature has not explored the mechanism that we study (see especially Section 5, which lists several “key economic pathways” but never mentions market size and innovation and the associated long-run growth dynamics). Our contribution is thus to extend the study of the economic effects of infectious diseases along lines hitherto neglected.

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<sup>2</sup>A partial list includes: [Acemoglu et al. \(2020\)](#), [Alvarez, Argente and Lippi \(2020\)](#), [Atkeson \(2020\)](#), [Barro, Ursua and Weng \(2020\)](#), [Bethune and Korinek \(2020\)](#), [Bognanni et al. \(2020\)](#), [Brotherhood et al. \(2020\)](#), [Farboodi, Jarosch and Shimer \(2020\)](#), [Glover et al. \(2020\)](#), [Jones, Philippon and Venkateswaran \(2020\)](#), [Krueger, Uhlig and Xie \(2020\)](#). See [https://www.nber.org/wp\\_covid19.html](https://www.nber.org/wp_covid19.html) and <https://voxeu.org/pages/covid-19-page> for an extensive list of NBER/CEPR working papers on COVID-related research.

### 3 Environment

To streamline exposition, we keep the description of the environment for the benchmark model without infection to a minimum. We refer the reader to Appendix A for further details.

**Preferences and Budget Set.** The economy is populated by a representative household with  $L(t)$  infinitely-lived members, each endowed with one unit of time per period. Labor supply is inelastic, so that household's labor supply equal  $L(t)$  at all times. Preferences are described by

$$U(t) = \int_t^\infty e^{-\rho(s-t)} L(s) \log [C(s)] ds, \quad \rho > 0, \quad (1)$$

where  $C = \left[ \int_0^N (X_i/L)^{\frac{\epsilon-1}{\epsilon}} di \right]^{\frac{\epsilon}{\epsilon-1}}$  is a CES aggregator of differentiated consumer goods  $X_i$ , with elasticity of substitution  $\epsilon > 1$ , and  $N$  is the mass of consumer goods available for purchase. The budget constraint is  $\dot{A} = rA + wL - Y$ , where  $A$  is assets yielding a rate of return  $r$ ,  $w$  is the wage,  $Y = \int_0^N p_i X_i di = p_C C$  is consumption expenditures, and  $p_C = \left( \int_0^N p_i^{1-\epsilon} di \right)^{\frac{1}{1-\epsilon}}$  is a Dixit-Stiglitz price index.

The household's maximization problem yields a standard Euler equation,

$$r = r_A \equiv \rho + \frac{\dot{Y}}{Y} - \frac{\dot{L}}{L}, \quad (2)$$

which gives the household's reservation rate of return on savings,  $r_A$ , and a downward-sloping demand for differentiated goods,

$$X_i = Y \frac{p_i^{-\epsilon}}{\int_0^N p_j^{1-\epsilon} dj}. \quad (3)$$

**Production and Innovation.** Firm  $i$  produces  $X_i$  units of the differentiated good using  $L_{X_i}$  units of labor and the technology  $X_i = Z_i^\theta (L_{X_i} - \phi)$ , with  $0 < \theta < 1$ , and  $\phi > 0$ , where  $Z_i$  is the firm-specific stock of knowledge. Firm-specific knowledge evolves according to  $\dot{Z}_i = \alpha K L_{Z_i}^\sigma L_Z^{1-\sigma}$ , with  $\alpha > 0$ , and  $0 < \sigma \leq 1$ , where  $L_{Z_i}$  is R&D labor,  $K = (1/N) \int_0^N Z_j dj$ , and  $L_Z = (1/N) \int_0^N L_{Z_j} dj$ . An incumbent firm  $i$  chooses the paths of the unit price  $p_i$  and

R&D labor  $L_{Z_i}$  to maximize the value of the firm,

$$V_i(t) = \int_t^\infty e^{-\int_t^s [r(v) + \delta]v} \Pi_i(s) ds, \quad (4)$$

where  $\Pi_i \equiv (p_i - wZ_i^{-\theta})X_i - w\phi - wL_{Z_i}$  is profits and  $\delta > 0$  is a “death shock.”

An entrant pays a sunk cost  $\beta Y/N = wL_{N_i}$  and creates value  $V_i$ . Hence, free-entry implies  $V_i = \beta Y/N$ . One can think of the entry process in terms of an underlying entry technology (as typically done in literature):

$$\dot{N} = \left( \frac{w}{\beta Y} N \right) L_N - \delta N. \quad (5)$$

## 4 Pre-Infection Equilibrium Dynamics

We now turn to the general equilibrium of the benchmark model without infection. As the equilibrium of the economy is symmetric, henceforth, we drop the subscript  $i$  such that, for example,  $X = X_i$  indicates both firm-level and average production of consumer goods. Labor is the numéraire, i.e., we set  $w \equiv 1$ . The value of the household’s portfolio equals the value of securities issued by firms,  $A = NV$ . (Note that when free entry holds,  $NV = \beta Y$ , otherwise  $NV < \beta Y$ .) Assets market equilibrium requires rates of return equalization, i.e.,  $r = r_A = r_Z = r_N$ , where  $r_Z$  and  $r_N$  are the rates of return to cost reduction and to entry,

$$r = r_Z \equiv \alpha \left[ \frac{Y\sigma\theta(\epsilon - 1)}{\epsilon N} - w \frac{L_Z}{N} \right] + \frac{\dot{w}}{w} - \delta, \quad (6)$$

$$r = r_N \equiv \frac{1}{\beta} \left[ \frac{1}{\epsilon} - \frac{N}{Y} \left( \phi + w \frac{L_Z}{N} \right) \right] + \frac{\dot{Y}}{Y} - \frac{\dot{N}}{N} - \delta. \quad (7)$$

Let  $y \equiv Y/L$  denote per capita expenditure, and  $x \equiv L/N$  the ratio of population to the mass of firms. When free entry holds, expenditures per capita  $y$  and rate of return  $r$  are constant and equal to

$$y = y^* \equiv \frac{1}{1 - \beta\rho} \quad \text{and} \quad r = \rho.$$

When free entry does not hold, instead,

$$y = \frac{\epsilon}{\epsilon - 1} \left( 1 - \frac{\phi}{x} \right) \quad \text{and} \quad r = \rho + \frac{\dot{y}}{y}.$$

**Pre-Infection Transition Dynamics.** Transition dynamics of the pre-infection economy is characterized by one differential equation for the population-to-firms ratio,  $x$ :

$$\dot{x} = \begin{cases} \delta x & \text{if } \phi \leq x \leq x_N \\ \frac{1-\beta\rho}{\beta} \phi - \left( \frac{1}{\beta\epsilon} - \rho - \delta \right) x & \text{if } x_N < x \leq x_Z \\ \frac{1-\beta\rho}{\beta} \left( \phi - \frac{\rho+\delta}{\alpha} \right) - \left[ \frac{1-\sigma\theta(\epsilon-1)}{\beta\epsilon} - \rho - \delta \right] x & \text{if } x > x_Z \end{cases}, \quad (8)$$

where the thresholds  $x_N$  and  $x_Z$  are defined as

$$x_N \equiv \frac{(1-\beta\rho)\epsilon\phi}{1-\rho\beta\epsilon} \quad \text{and} \quad x_Z \equiv \frac{(1-\beta\rho)(\rho+\delta)}{\sigma\alpha\theta\frac{\epsilon-1}{\epsilon}}. \quad (9)$$

As illustrated by Figure 1, the transition dynamics features three regions: (i) net firms' exit with zero R&D labor ( $\phi \leq x \leq x_N$ ); (ii) gross firms' entry with zero R&D labor ( $x_N < x \leq x_Z$ ); (iii) gross firms' entry with positive R&D labor ( $x > x_Z$ ). On the left of the threshold  $x_N$ , firm size  $x \leq x_N$  is "too small," implying net firms' exit. As a result, the population-to-firms ratio grows at the rate  $\delta$  of firms' exit, which contributes to increase profitability. As firm size  $x$  enters the region  $x_N < x \leq x_Z$ , new firms enter, which slows down the growth rate of firm size. In this region, there is no accumulation of knowledge. Finally, on the right of the threshold  $x_Z$ , when average firm size becomes "large enough," firm entry and knowledge accumulation coexist.

Using the equation for the rate of return to cost reduction (6), we obtain the growth rate of the stock of knowledge:

$$z \equiv \frac{\dot{Z}}{Z} = \alpha \frac{L_Z}{N} = \begin{cases} 0 & \text{if } x \leq x_Z \\ x \cdot y \alpha \sigma \theta \left( \frac{\epsilon-1}{\epsilon} \right) - r - \delta & \text{if } x > x_Z \end{cases}. \quad (10)$$

Finally, using the Euler equation (2), and the rate of return to entry (7), we obtain the growth rate of the mass of firms,

$$n \equiv \frac{\dot{N}}{N} = \frac{1}{\beta} \left[ \frac{1}{\epsilon} - \frac{1}{xy} \left( \phi + \frac{L_Z}{N} \right) \right] - \rho + \frac{\dot{L}}{L} - \delta. \quad (11)$$

**Pre-Infection Balanced Growth Path.** Under the following conditions on parameters,

$$\alpha\phi > \rho + \delta, \quad (12)$$

$$(\rho + \delta)\beta + \frac{\sigma\theta(\epsilon - 1)}{\epsilon} < \frac{1}{\epsilon} < (\rho + \delta)\beta + \left(\frac{\alpha\phi}{\rho + \delta}\right) \frac{\sigma\theta(\epsilon - 1)}{\epsilon}, \quad (13)$$

the model exhibits a balance growth path (BGP) with endogenous growth, along which firm size,  $x$ , firm knowledge growth,  $z \equiv \dot{Z}/Z$ , and the mass of firms,  $N$ , take the following steady-state values:

$$x^* = \frac{(1 - \beta\rho)\epsilon \left(\phi - \frac{\rho + \delta}{\alpha}\right)}{1 - \sigma\theta(\epsilon - 1) - (\rho + \delta)\beta\epsilon'}, \quad (14)$$

$$z^* = \frac{\phi\alpha - (\rho + \delta)}{1 - \sigma\theta(\epsilon - 1) - (\rho + \delta)\beta\epsilon} \theta(\epsilon - 1) - (\rho + \delta), \quad (15)$$

$$N^* = \left[ \frac{1 - \sigma\theta(\epsilon - 1) - (\rho + \delta)\beta\epsilon}{\epsilon \left(\phi - \frac{\rho + \delta}{\alpha}\right) (1 - \beta\rho)} \right] L. \quad (16)$$

Note that along a BGP,  $x^*$  and  $z^*$  are *independent* of the population level  $L$ . This is an important feature of the model that comes from the property that the mass of firms  $N^*$  is proportional to  $L$ . Because of this property, the model does not suffer from the so-called “scale effect,” at work in the first-generation models of endogenous growth à la [Romer \(1990\)](#). We stress that the proportionality between mass of firms and the population level is a BGP phenomenon, which breaks when the economy is in transition dynamics.

Given the formula for the price index  $p_C = \left[ \int_0^N p_i^{1-\epsilon} di \right]^{\frac{1}{1-\epsilon}}$ , along a BGP, real per capita consumption expenditure is

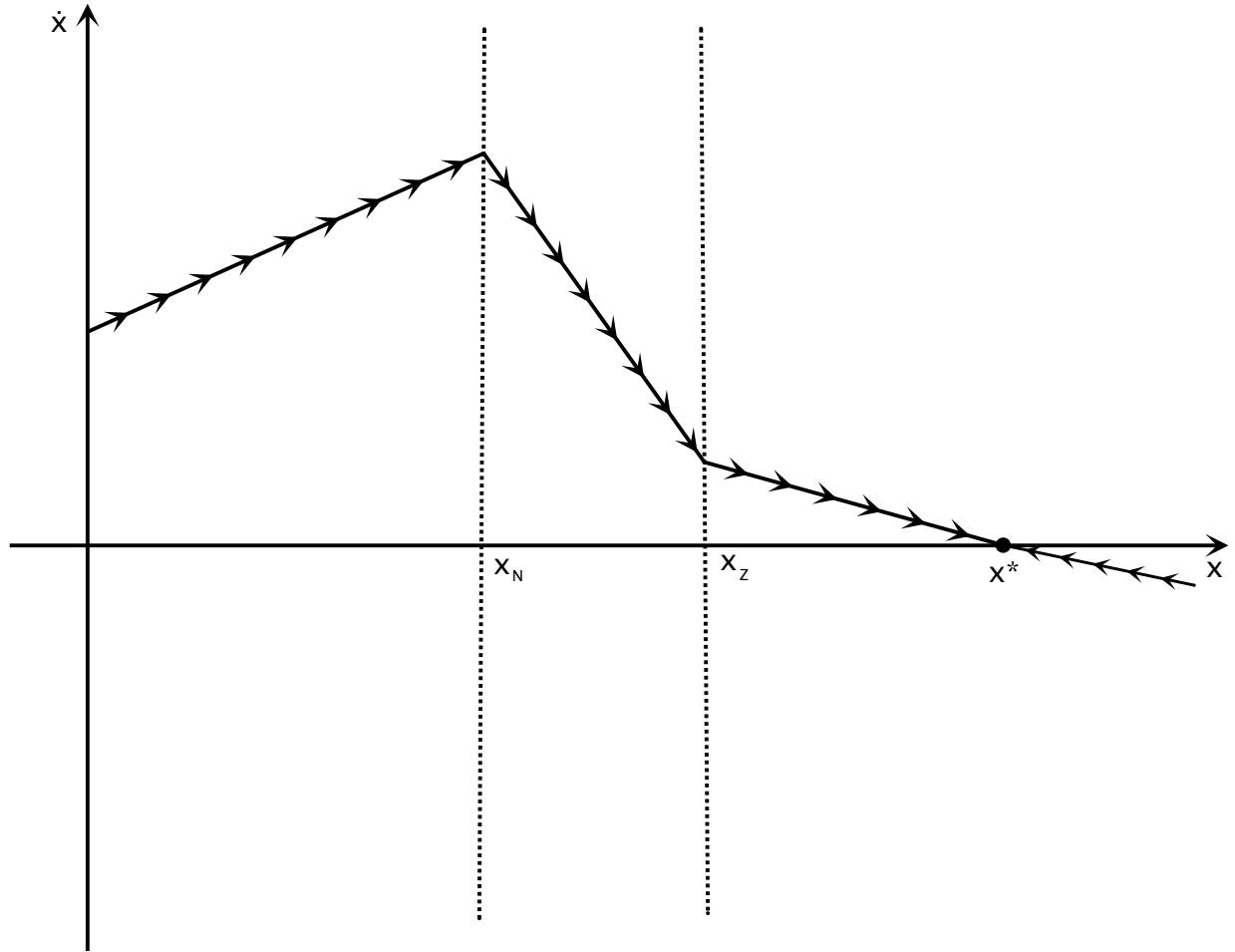
$$\frac{y^*}{p_C} = \frac{y^*}{c^*} \cdot Z^\theta N^{\frac{1}{\epsilon-1}}, \quad (17)$$

where  $c^* = \frac{\epsilon}{\epsilon-1} \cdot w$  captures “static” drivers of the price index, i.e., those unrelated to endogenous technological change. (Recall the normalization  $w = 1$ .)

## 5 Post-Infection Equilibrium Dynamics

In this section, we provide a cursory description of the Susceptible-Infected-Recovered-Deceased (SIRD) epidemiological model and integrate it into the basic growth model we

Figure 1: Pre-Infection Equilibrium Dynamics



*Notes:* The figure depicts the equilibrium dynamics of the population-to-firms ratio as implied by the piece-wise differential equation (8), where the thresholds  $x_N$  and  $x_Z$  are as defined in (9), and the steady-state value  $x^*$  is as in (14).

presented above.

## 5.1 SIRD Model with Vital Dynamics

In its general formulation, the SIRD model with births and non-disease deaths amounts to a high-dimensional dynamical system:

$$\dot{S} = \zeta_B L - \zeta_M S - \frac{\zeta_S I S}{L}, \quad (18)$$

$$\dot{I} = \frac{\zeta_S I S}{L} - \zeta_R I - \zeta_M I - \zeta_D I, \quad (19)$$

$$\dot{R} = \zeta_R I - \zeta_M R, \quad (20)$$

$$\dot{D} = \zeta_D I, \quad (21)$$

$$\dot{L} = \zeta_B L - \zeta_M (S + I + R) - \zeta_D I, \quad (22)$$

where  $\zeta_B$  is the birth rate (all newborn are  $S$ ),  $\zeta_M$  is the non-disease death rate,  $\zeta_S$  is the transmission rate,  $\zeta_R$  is the recovery rate,  $\zeta_D$  is the fatality rate, and  $S$ ,  $I$ ,  $R$  and  $D$  are the mass of susceptible, infected, recovered, and deceased, respectively.

One can conveniently rewrite the dynamical system above in terms of the rates  $s \equiv S/L$  and  $\iota \equiv I/L$ , where  $L = S + I + R$ , as

$$\frac{\dot{s}}{s} = \zeta_B \left( \frac{1}{s} - 1 \right) - (\zeta_S - \zeta_D) \iota, \quad (23)$$

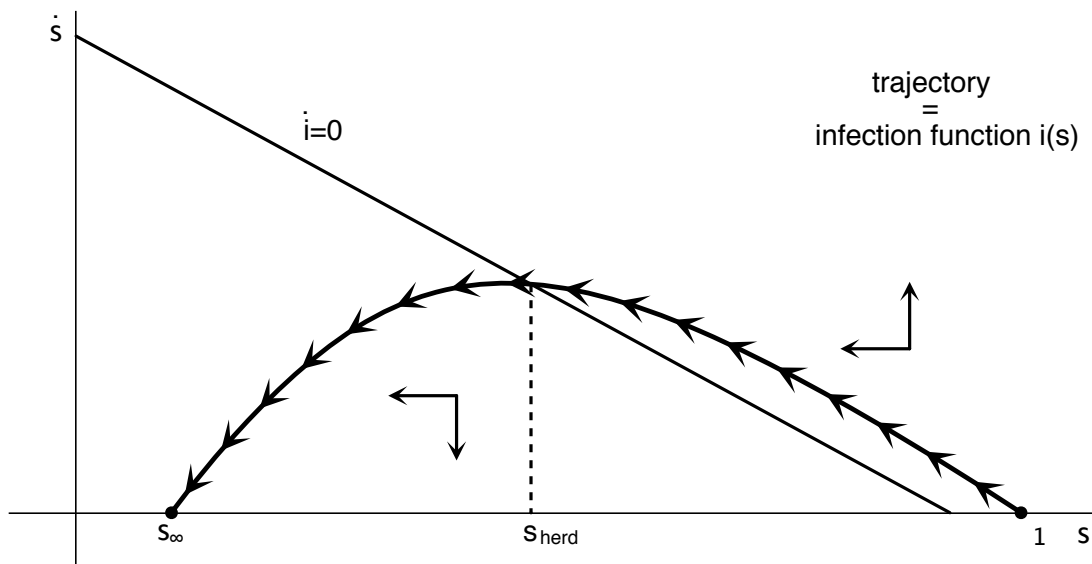
$$\frac{\dot{\iota}}{\iota} = \zeta_S s - \zeta_R - \zeta_B - \zeta_D (1 - \iota), \quad (24)$$

$$\frac{\dot{L}}{L} = \zeta_B - \zeta_M - \zeta_D \iota, \quad (25)$$

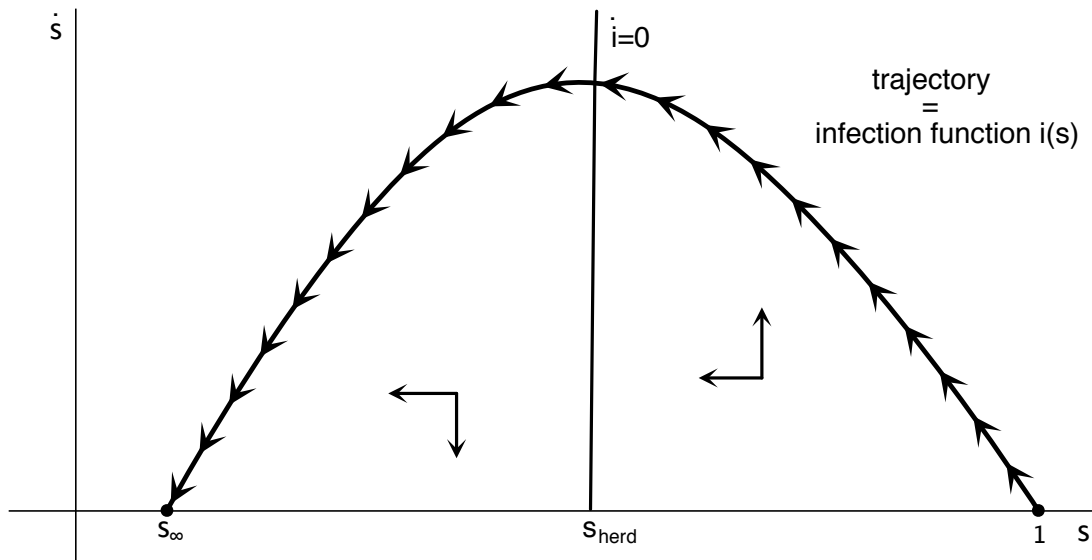
where equations (23)-(24) describe disease dynamics, and equation (25) describes the implied population dynamics.

It is important to note that epidemiologists have studied the dynamical system (18)-(22), and its lower-dimensional version (23)-(25), thoroughly and rigorously (see, e.g., Hethcote, 2000; Brauer, 2008). Widely used simplifying assumptions yield a dynamical system that is easy to incorporate in economic models. For instance, recent applications of the epidemiological framework to economics typically set  $\zeta_B = \zeta_M = 0$ . This is the case known as “no vital dynamics” that is usually rationalized with the relatively fast dynamics of epidemics, which typically play out over a time-horizon of several weeks.

**Infection Function.** Figures 2a-2b depict the phase diagrams for the SIRD and SIR model, respectively. In each case, for the initial state  $(s_0, \iota_0)$ , with  $\iota_0 \approx 0$  and  $s_0 \approx 1$ , there exists a



(a) SIRD model



(b) SIR model

Notes: The figures shows the phase diagram for the SIRD model (top panel) and for the SIR model (bottom panel).

Figure 2: Epidemiological Models of Infectious Diseases

hump-shaped trajectory  $i(s)$  that we interpret as the “infection function.” Next, we leverage this concept of infection function to integrate epidemiology into an economic model.

We use  $\iota(s)$  to compress the epidemic model to *one* equation:

$$\frac{\dot{s}}{s} = \xi_B \left( \frac{1}{s} - 1 \right) - (\xi_S - \xi_D) \iota(s). \quad (26)$$

In macroeconomics jargon, equation (26) represents the law of motion of the “epidemic shock.”

Next, to solve for  $\iota(s)$ , we take the ratio of equations (23) and (24), which gives the partial differential equation (PDE),

$$\frac{d\iota}{ds} = \frac{\xi_S s - \xi_R - \xi_D - \xi_B + \xi_D \iota}{\xi_B \left( \frac{1}{s} - 1 \right) - (\xi_S - \xi_D) \iota} \left( \frac{\iota}{s} \right). \quad (27)$$

The existence, meaning and, in some cases, solution of this PDE is well-known in the epidemiological literature (see, e.g., Brauer, 2008). It turns out to be an extremely useful device to translate the epidemiology model into a tractable component of an integrated epidemic-economy model.

Infection function: SIRD. For  $\xi_B = \xi_M = 0$ , the PDE (27) reduces to the d’Alembert’s equation

$$\frac{d\iota}{ds} = \frac{1}{s} \frac{\xi_R + \xi_D(1 - \iota)}{\xi_S - \xi_D} - \frac{\xi_S}{\xi_S - \xi_D}. \quad (28)$$

Solving the PDE (28) with the boundary conditions  $\iota(0) = \iota_0$  and  $s(0) = 1 - \iota_0 \equiv s_0$  yields the infection function

$$\iota(s) = \frac{\xi_R}{\xi_D} \left[ 1 - \left( \frac{s}{s_0} \right)^{-\frac{\xi_D}{\xi_S - \xi_D}} \right] + 1 - s, \quad (29)$$

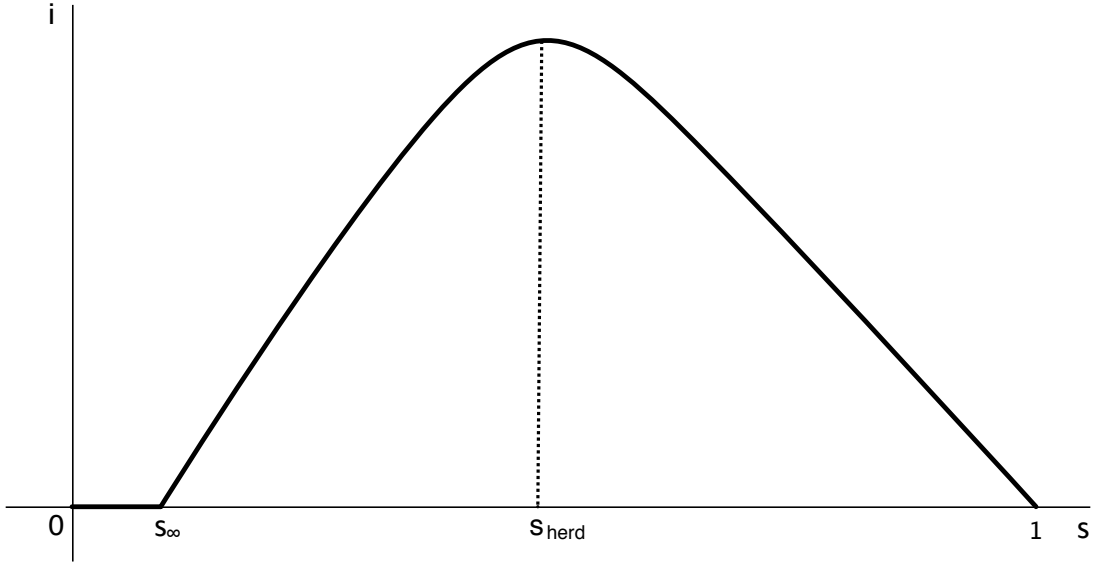
which is a hump-shaped function with  $s_\infty \equiv \arg \text{solve} \{ \iota(s) = 0 \} > 0$ . Figure 3a depicts the infection function (29) for the SIRD model.<sup>3</sup>

Infection function: SIR. For  $\xi_B = \xi_M = \xi_D = 0$  (i.e., constant population), the PDE (27) reduces to

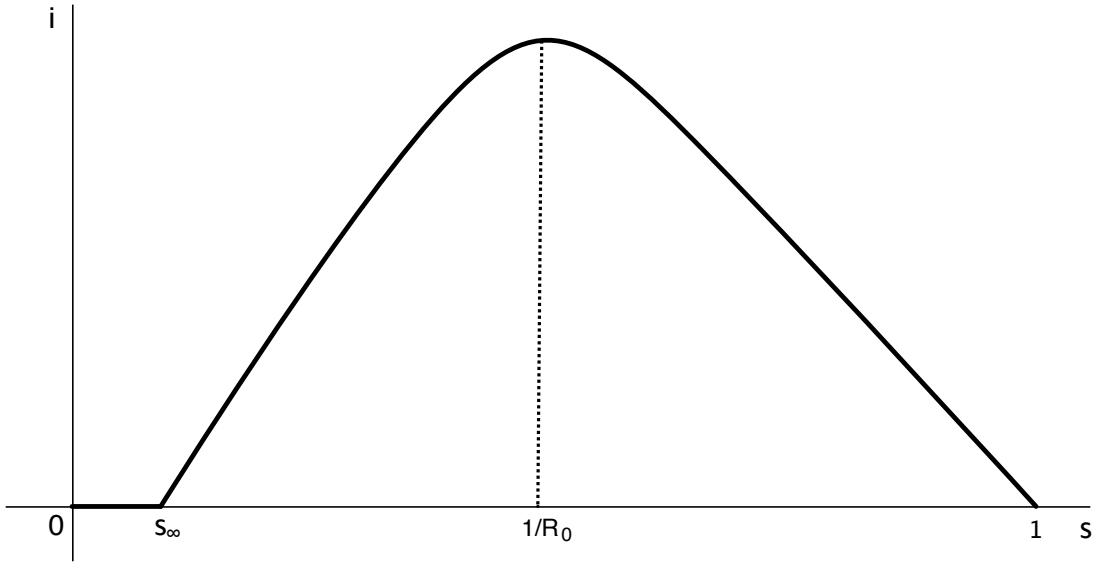
$$\frac{d\iota}{ds} = \frac{1}{R_0 s} - 1, \quad R_0 \equiv \frac{\xi_S}{\xi_R}. \quad (30)$$

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<sup>3</sup>In the SIRD case, the epidemic shock that hits the economy is  $\frac{\dot{s}}{s} = -(\xi_S - \xi_D) \iota(s)$ .



(a) SIRD model



(b) SIR model

*Notes:* The figures depicts the infection function for the SIRD model (top panel) and for the SIR model (bottom panel).

Figure 3: Infection Function

Solving the PDE (30) with boundary conditions  $i(0) = i_0$  and  $s(0) = 1 - i_0 \equiv s_0$  yields the infection function

$$i(s) = \frac{1}{R_0} \log \left( \frac{s}{s_0} \right) + 1 - s, \quad (31)$$

which is a hump-shaped function with  $s_\infty \equiv \arg \text{solve } \{\iota(s) = 0\} > 0$ . Figure 3b depicts the infection function (31) for the SIR model.<sup>4</sup>

## 5.2 Embedding the SIRD Model into the Growth Model

We now embed the SIRD model without vital dynamics (i.e.,  $\xi_B = \xi_M = 0$ ) into the basic growth model. First, in the SIRD case with  $\xi_D > 0$ , the epidemic *permanently* removes people from the economy leading to negative population growth:

$$\frac{\dot{L}}{L} = -\xi_D \iota(s). \quad (32)$$

Second, the disease *temporarily* removes people from the labor force, akin to a negative “labor supply shock,”

$$\underbrace{L_X + L_Z + L_N}_{\substack{\text{labor demand} \\ \text{(allocation)}}} = \underbrace{[1 - \iota(s)] L}_{\substack{\text{labor supply} \\ \text{(participation)}}} \quad (33)$$

Third, through the mortality rate  $\xi_D$ , the epidemic directly affects the *discount rate* of utility flows:

$$L(t) u(t) = e^{-\int_0^t \xi_D \iota(v) dv} \log C. \quad (34)$$

Akin to the Yaari-Blanchard perpetual youth model, one can interpret discounting through the term  $e^{-\int_0^t \xi_D \iota(v) dv}$  as an individual life expectancy effect: the disease can kill *me*, not just my siblings.

### 5.2.1 Expenditures and Interest Rates

**Expenditures.** Overall, per capita consumption expenditure is decreasing in the fraction of infected in the population,  $\iota$ . In general equilibrium, an epidemic manifests itself on the demand side of the economy, too, as a reduction in market size. Through this channel, it alters firms’ incentives to enter the market and incumbents’ allocation of R&D labor, initiating transition dynamics.

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<sup>4</sup>In the SIR case, the epidemic shock that hits the economy is  $\dot{s}/s = -\xi_S \iota(s)$ .

When free entry does not hold, per capita expenditure is

$$y(s, x) = \frac{\epsilon}{\epsilon - 1} \left(1 - \frac{\phi}{x}\right) [1 - \iota(s)], \quad (35)$$

which is U-shaped in  $s$  since the infection function  $\iota(s)$  is hump-shaped in the fraction of susceptible in population,  $s$ . On the other hand, when free entry holds, per capita expenditure is

$$y(s) = \frac{1 - \iota(s)}{1 - [\rho + \xi_D \iota(s)] \beta}. \quad (36)$$

Assuming  $1 > \beta(\rho + \xi_D)$  to guarantee existence for  $\iota \in [0, 1]$ , it implies  $dy/d\iota < 0$ . Again,  $y$  is U-shaped in  $s$  since  $\iota(s)$  is hump-shaped in  $s$ . Importantly, as  $\iota$  goes from 0 to  $\iota_0 > 0$ , an outbreak yields a fall in  $y$  at  $t = 0$ . As  $s$  falls throughout,  $y$  initially keeps falling, turns around at the peak of epidemic and returns to  $y^*$  from below.

**Interest Rates.** Log-differentiating the expression for  $y$  yields

$$\frac{\dot{y}}{y} = -\frac{1 - (\rho + \xi_D) \beta}{1 - [\rho + \xi_D \iota(s)] \beta} \cdot \frac{\dot{\iota}(s)}{1 - \iota(s)}. \quad (37)$$

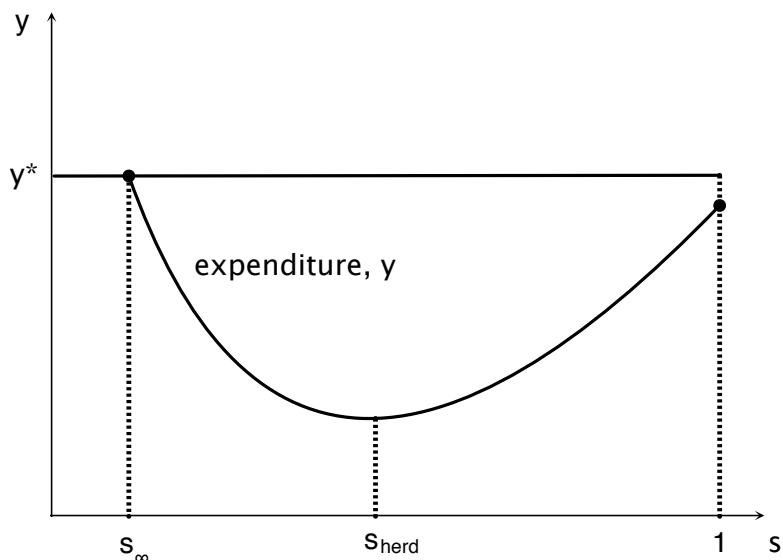
Next, using the Euler equation, and the expression for  $i$  gives an expression for the interest rate:

$$r(s) = \rho - \underbrace{\frac{1 - (\rho + \xi_D) \beta}{1 - [\rho + \xi_D \iota(s)] \beta}}_{\text{increasing in } \iota} \cdot \underbrace{\frac{\iota(s)}{1 - \iota(s)}}_{\text{increasing in } \iota} \cdot \underbrace{[\xi_S s - \xi_R - \xi_D + \xi_D \iota(s)]}_{\text{increasing in } \iota}. \quad (38)$$

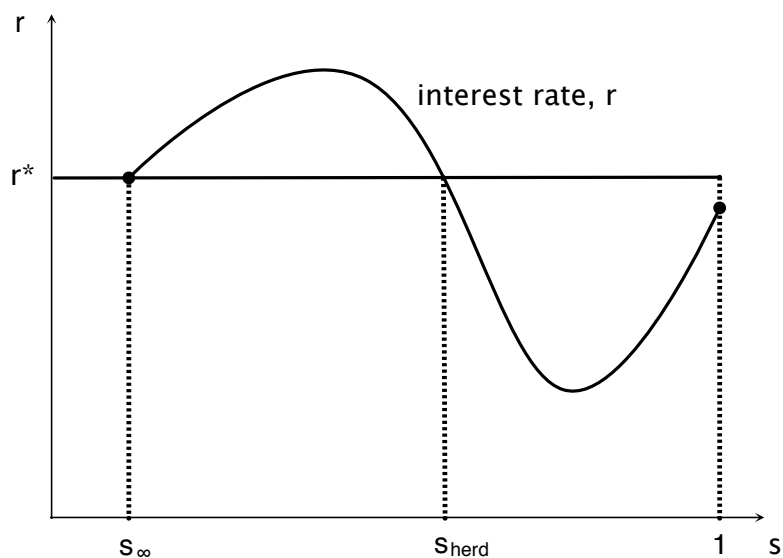
A few remarks are in order here. (i) The interest rate is below  $\rho$  and decreasing in  $\iota$  when the term in square brackets on the right-hand side  $[\cdot] \equiv [\xi_S s - \xi_R - \xi_D + \xi_D \iota(s)]$  is larger than zero; and (ii) it is above  $\rho$  and increasing in  $\iota$  when  $[\cdot] < 0$ . The fraction of infected  $\iota(s)$  is decreasing in  $s$  for  $[\cdot] > 0$  and increasing in  $s$  when  $[\cdot] < 0$ . As a result, the interest rate  $r(s)$  is first hump- and then U-shaped in the fraction of susceptible,  $s$ . Since initially  $[\cdot] > 0$ , the outbreak yields a fall in  $r$  at  $t = 0$ . As  $s$  falls throughout, the interest rate continues falling, it turns around at the peak of the epidemic, overshoots and finally returns to  $\rho$  from above.

To visualize some of the qualitative features of the transition dynamics, Figures 4 and 5 show per capita expenditures and interest rate dynamics as a function of the fraction of

susceptible and time, respectively.



(a) Per capita expenditure as function of susceptible:  $y(s)$

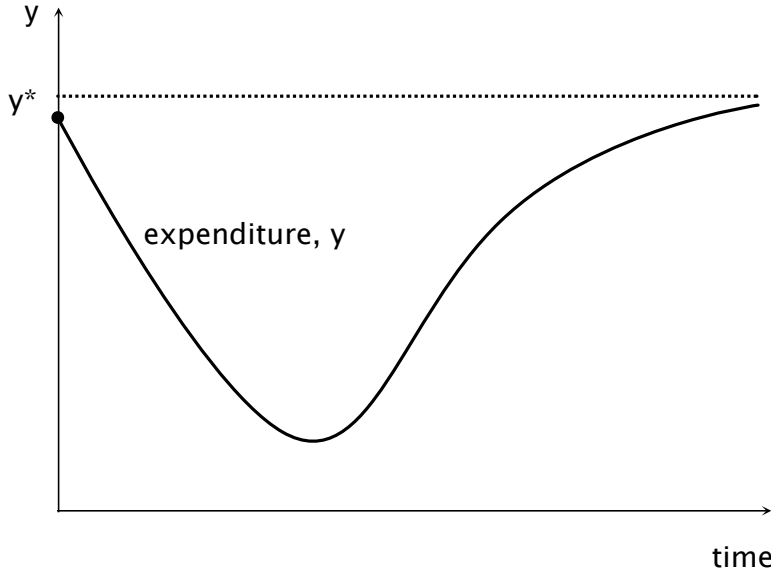


(b) Interest rate as function of susceptible:  $r(s)$

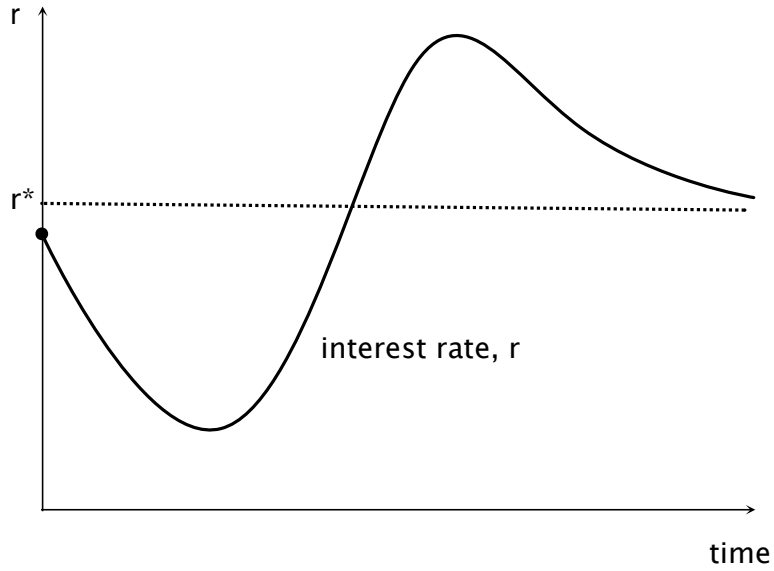
Figure 4: Expenditure and Interest Rate as Function of Susceptible: A Visualization

## 5.2.2 Market Structure, Productivity, and Welfare

Using the expressions for  $y(s)$  and  $r(s)$  in equations (36) and (38), respectively, the general-equilibrium dynamics reduces to a tractable system of two differential equations: (i) the first equation describes the evolution over time of the economic state variable of the



(a) Per capita expenditure over time:  $y(t)$



(b) Interest rate over time:  $r(t)$

Figure 5: Expenditure and Interest Rate as Function of Time: A Visualization

model, i.e. the ratio of the population to the mass of firms,  $x \equiv L/N$ ; (ii) the second equation describes the evolution of the epidemiological state variable, i.e. the fraction of susceptible in the population,  $s$ .

**Outbreak.** At  $t = 0$ , per capita expenditure  $y$  falls from its steady-state value  $y^*$  to the lower value associated with  $\iota(s_0)$  or  $\iota_0$  for short:

$$y(s_0) = \frac{1 - \iota_0}{1 - (\rho + \xi_D \iota_0) \beta} < y^*. \quad (39)$$

Such a drop in expenditure initiates transition dynamics, during which the economic state variable follows

$$\frac{\dot{x}}{x} = -\xi_D \iota - n, \quad (40)$$

with initial condition  $x_0 = L_0/N_0 \geq 0$  and  $n \equiv \dot{N}/N$  indicating the growth rate of the mass of firms. The dynamical system features three regions that differ qualitatively in terms of market structure and R&D labor allocations.

**Region 1: Net Exit with R&D Shutdown.** In the net exit region, expenditure falls so much that the economy hits two corners with no firm-level innovation and no firm entry, i.e.,  $L_Z = 0$  and  $L_N = 0$ , which yields  $n = -\delta \leq 0$ . The boundary of this region in the  $x$ -dimension is determined by the following condition:

$$\frac{1 - [\rho + \xi_D \iota(s)] \beta}{1 - \iota(s)} \epsilon \phi \leq x \leq \frac{1 - [\rho + \xi_D \iota(s)] \beta}{\frac{1}{\epsilon} - [\rho + \xi_D \iota(s)] \beta} \cdot \frac{\phi}{1 - \iota(s)}.$$

In this region, the dynamical system is

$$\frac{\dot{s}}{s} = -(\xi_S + \xi_D) \iota(s), \quad (41)$$

$$\frac{\dot{x}}{x} = \delta - \xi_D \iota(s). \quad (42)$$

**Region 2: Gross Entry with R&D Shutdown.** The boundary of this region in the  $x$ -dimension is determined by the following condition:

$$\frac{1 - [\rho + \xi_D \iota(s)] \beta}{\frac{1}{\epsilon} - [\rho + \xi_D \iota(s)] \beta} \cdot \frac{\phi}{1 - \iota(s)} < x \leq \frac{r(s) + \delta}{\sigma \alpha \theta \frac{\epsilon - 1}{\epsilon}} \cdot \frac{1 - [\rho + \xi_D \iota(s)] \beta}{1 - \iota(s)}. \quad (43)$$

In this region, the dynamical system is

$$\frac{\dot{s}}{s} = -(\xi_S + \xi_D) \iota(s), \quad (44)$$

$$\frac{\dot{x}}{x} = \frac{\phi}{\beta y(s)} \cdot \frac{1}{x} - \frac{1}{\beta \epsilon} + \rho + \delta. \quad (45)$$

The underlying net entry process follows  $n = -\dot{x}/x - \xi_D \iota(s)$ .

**Region 3: Gross Entry with Active R&D.** Finally, the boundary of the region featuring gross entry with active R&D is determined by the following inequality:

$$x > \frac{r(s) + \delta}{\sigma \alpha \theta \frac{\epsilon-1}{\epsilon}} \cdot \frac{1 - [\rho + \xi_D \iota(s)] \beta}{1 - \iota(s)}. \quad (46)$$

In this region, the dynamical system is

$$\frac{\dot{s}}{s} = -(\xi_S + \xi_D) \iota(s), \quad (47)$$

$$\frac{\dot{x}}{x} = \frac{1}{\beta y(s)} \left( \phi - \frac{r(s) + \delta}{\alpha} \right) \frac{1}{x} - \frac{1 - \sigma \theta (\epsilon - 1)}{\beta \epsilon} + \rho + \delta. \quad (48)$$

The underlying net entry process follows  $n = -\dot{x}/x - \xi_D \iota(s)$  and knowledge growth is positive and equal to  $z = x \alpha \sigma \theta \frac{\epsilon-1}{\epsilon} y(s) - r(s) - \delta > 0$ .

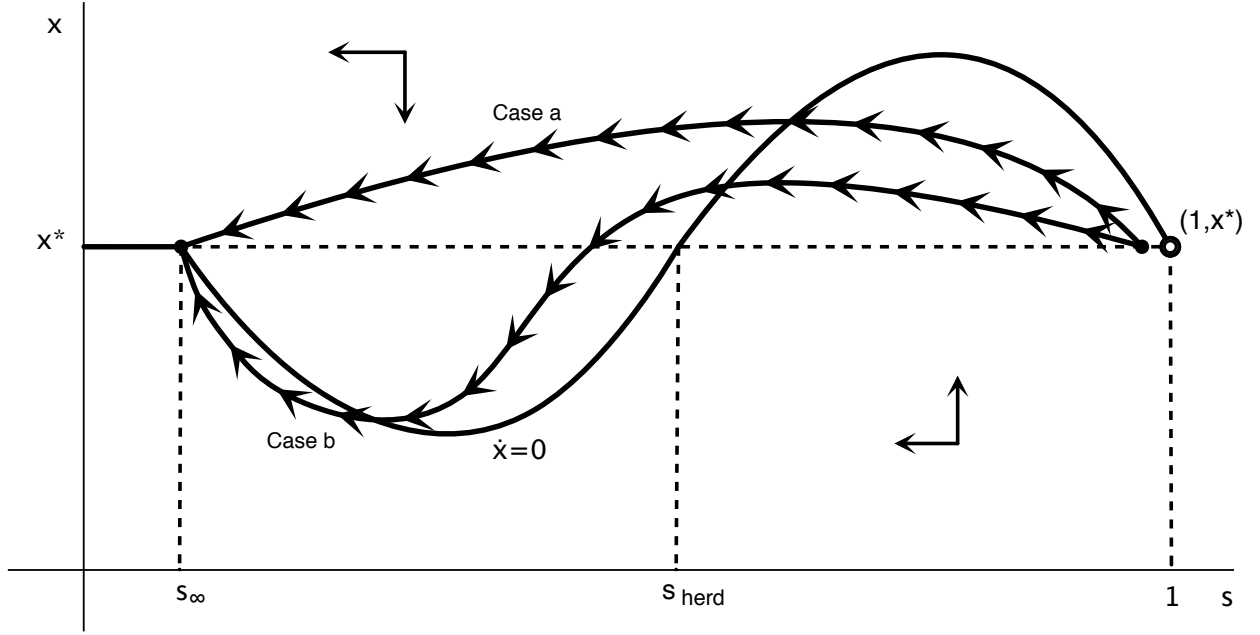
**R&D Labor Allocation and TFP.** To provide some insight into the transition dynamics implied by the model, Figure 6 illustrates two potential trajectories in the  $(s, x)$  space for the case of next exit. The population-to-firms ratio,  $x$ , grows initially because of net exit due to falling profitability caused by falling expenditure. Both  $y$  and  $r$  jump down and initially follow U-shaped paths. The net effect can be a fall in firm knowledge growth,  $z$ . Overall, the transition dynamics features an initial deceleration of firm productivity growth with reversion to the long-run level. Possibly, a phase with zero firm growth.<sup>5</sup>

Such a temporary deceleration delivers a *permanent* TFP loss relative to the baseline no-disease path. Note also that period of net exit means period of falling TFP. This is a *real* loss, not just relative to the no-disease baseline (i.e., a “lost opportunity” loss). In the SIRD case, the transition features overall net exit because the new steady state exhibit

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<sup>5</sup>Formally, the constant population-to-firms ratio  $\dot{x} \geq 0$  locus is (i)  $x \leq \frac{\epsilon \phi}{1 - (\rho + \delta) \beta \epsilon} \cdot \frac{1 - [\rho + \xi_D \iota(s)] \beta}{1 - \iota(s)}$  for Region 2 and (ii)  $x \leq \frac{\epsilon \left( \phi - \frac{r(s) + \delta}{\alpha} \right)}{1 - \sigma \theta (\epsilon - 1) - (\rho + \delta) \beta \epsilon} \cdot \frac{1 - [\rho + \xi_D \iota(s)] \beta}{1 - \iota(s)}$  for Region 3.

Figure 6: Post-infection Dynamics



*Notes:* The figure depicts the phase diagram for the post-infection economy. On the  $x$ -axis,  $s$  denotes the fraction of susceptible in the population; on the  $y$ -axis,  $x$  denotes the ratio of the population to the mass of firms.

fewer firms, i.e.,  $N < N^*$  due to a smaller population. This causes TFP to fall permanently. In the SIR case instead, such an effect is only temporary.

**Welfare.** Real expenditure per capita is equal to

$$C = \frac{y}{p_C} = \left( \frac{\epsilon - 1}{\epsilon} \right) y \cdot \underbrace{Z^\theta N^{\frac{1}{\epsilon-1}}}_{\text{TFP}} \quad (49)$$

which implies a utility flow of  $\log C(t) = \log \left( \frac{\epsilon-1}{\epsilon} \right) + \log y(t) + \theta \log Z(t) + \frac{1}{\epsilon-1} \log N(t)$ .

Further, normalizing  $\log \left( \frac{\epsilon-1}{\epsilon} Z_0^\theta N_0^{\frac{1}{\epsilon-1}} \right) = 0$ , we rewrite the utility flow as

$$\log C(t) = \log y(t) + \theta \int_0^t z(s) ds + \frac{1}{\epsilon-1} \int_0^t n(s) ds. \quad (50)$$

Importantly, in calculating the welfare integral in (1), effective discounting accounts

for fatality due to the spread of the epidemics:

$$\exp \left\{ -\rho t - \int_0^t \xi_{D^I}(s(v)) dv \right\}. \quad (51)$$

## 6 Policy Intervention

In this section, we study policy interventions that operate through a reduction in the transmission rate of the epidemic, which entails a reduction in the supply of labor, akin to a lockdown. Let  $h \geq 0$  denote the policy variable of interest, that captures in a reduced-form lockdown intensity. Following the literature, we model the immediate cost and benefit of intervention as follows:

$$\text{benefit: } \frac{\xi_S}{1+h} \iota(s) S; \quad (52)$$

$$\text{cost: } \frac{L}{1+h} [1 - \iota(s)]. \quad (53)$$

The lockdown intensity parameter,  $h$ , reduces the transmission rate of the disease. That is, the new transmission rate,  $\xi_S/(1+h)$ , equals the old transmission rate,  $\xi_S$ , in (18)-(19) divided by  $1+h$ . In addition, it introduces a wedge between the endowment of labor services,  $L$ , and the effective supply of labor,  $L/(1+h)$ .<sup>6</sup>

In terms of utility  $u(t) = \log C(t)$ , interventions of the type described by (52)-(53) have a (negative) direct effect through the term  $-\log(1+h)$ , which captures policies' adverse impact on total labor supply, and indirect effects that work through general equilibrium forces, in a way that we explain further below:

$$\log C(t) = \underbrace{-\log(1+h)}_{\text{direct effect}} + \log y(t) + \theta \int_0^t z(v) dv + \frac{1}{\epsilon-1} \int_0^t n(v) dv, \quad (54)$$

where  $y$  is per capita expenditure, and  $z$  and  $n$  are the growth rates of knowledge and of the mass of firms, respectively.

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<sup>6</sup>In the current formulation, the percentage change in the flow of new infected with respect to the policy variable  $h$  on the benefit side equals the percentage change in available labor services on the cost side. This assumption can be readily relaxed by introducing an additional parameter  $\chi \geq 0$ , such that the labor cost of policy intervention is  $L/(1+\chi h)$ . In this alternative formulation, the ratio of elasticities of the labor cost to the reduction of new infected becomes  $\varsigma \equiv \chi(1+h)/(1+\chi h)$ , akin to a "sacrifice ratio." A one percent reduction in new infected comes at the cost of a  $\varsigma$  percent loss in labor services. Note that when  $\chi = 1$ ,  $\varsigma = 1$ , which nests our baseline formulation.

## 6.1 A Constant Wedge Approach

**Intervention Policy #1.** To build intuition on the key trade-offs at play, we consider first the case where  $h$  is constant. Note that the infection functions need to be re-computed to take in account the fact that the new transmission rate,  $\xi_S / (1 + h)$ , is reduced by the factor  $1 + h$ :

$$\text{SIRD: } \frac{d\iota}{ds} = \frac{1}{s} \left[ \frac{\xi_R + \xi_D (1 - \iota)}{\xi_S / (1 + h)} \right] - \xi_D - \frac{\xi_S / (1 + h)}{\xi_S / (1 + h) - \xi_D}; \quad (55)$$

$$\text{SIR: } \frac{d\iota}{ds} = \frac{1 + h}{R_0 s} - 1. \quad (56)$$

Solving (55)-(56) with boundary conditions  $\iota(0) = \iota_0$  and  $s(0) = 1 - \iota_0 \equiv s_0$  yields the new infection functions:

$$\text{SIRD: } \iota(s) = \frac{\xi_R}{\xi_D} \left[ 1 - \left( \frac{s}{s_0} \right)^{-\frac{\xi_D}{\xi_S / (1 + h) - \xi_D}} \right] + 1 - s; \quad (57)$$

$$\text{SIR: } \iota(s) = \frac{1 + h}{R_0} \log \left( \frac{s}{s_0} \right) + 1 - s. \quad (58)$$

Again, there are two cases. (i) When free entry does not hold, per capita expenditure is equal to

$$y(s, x) = \frac{\epsilon}{\epsilon - 1} \left( 1 - \frac{\phi}{x} \right) \left[ \frac{1 - \iota(s)}{1 + h} \right]. \quad (59)$$

(ii) When instead free entry holds, per capita expenditure is equal to

$$y(s) = \frac{1}{1 - [\rho + \xi_D \iota(s)] \beta} \left[ \frac{1 - \iota(s)}{1 + h} \right]. \quad (60)$$

Note that, in general, one can think of a family of  $y(s; h)$  functions parametrized by the policy variable  $h$ . The policy intervention with a constant wedge has two opposing effects:

$$\frac{dy(s; h)}{dh} = \underbrace{\frac{\partial y(s; h)}{\partial h}}_{-} + \overbrace{\frac{\partial y(s; h)}{\partial \iota(s)} \cdot \frac{\partial \iota(s)}{\partial h}}^{+}. \quad (61)$$

The first term on the right-hand side of (61) captures the negative direct effect that the

policy exercises on the total labor supply. The second term captures the positive indirect effect that the policy has through the reduction in the transmission rate. In the SIR case, the policy intervention has an unambiguous negative net effect:

$$\frac{dy(s;h)}{dh} = \frac{1}{R_0} \log\left(\frac{s}{s_0}\right) < 0, \text{ since } s < s_0. \quad (62)$$

Log-differentiating  $y(s)$  and using the Euler equation, yields

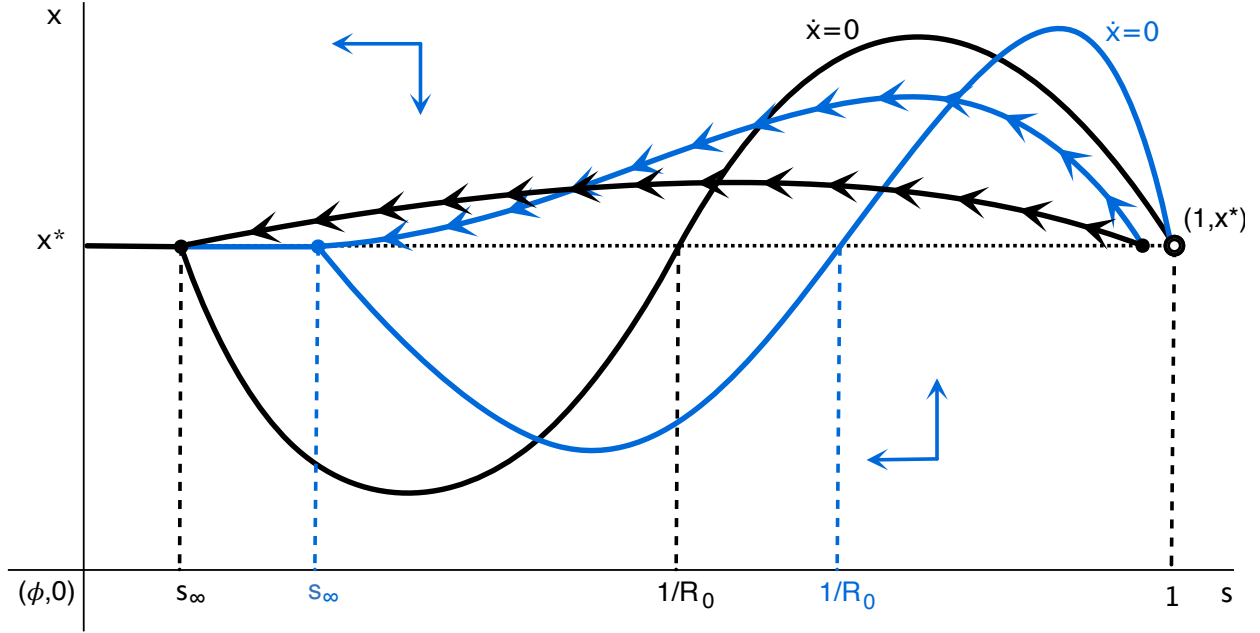
$$r(s) = \rho - \frac{1 - (\rho + \xi_D) \beta}{1 - [\rho + \xi_D \iota(s)] \beta} \cdot \frac{\iota(s)}{1 - \iota(s)} \cdot \underbrace{[\xi_{SS} - \xi_R - \xi_D + \xi_D \iota(s)]}_{\xi_R(R_0 s - 1) \text{ for } \xi_D=0}.$$

Note that a policy intervention involving a constant wedge  $h$  has no direct affect on the interest rate; it only operates through the dynamic effects of the fraction of infected,  $\iota(s)$ , on per capita expenditures.

Figure 7 shows the phase diagram of the dynamical system for the SIR case, with (blue lines) and without (black lines) policy intervention. Under policy intervention, herd immunity occurs at a smaller fraction of susceptible; this flatten-the-curve effect is the immediate result of a lower value of  $R_0$ . Qualitatively, under intervention, the population-to-firms ratio rises at a faster rate after the outbreak, and it converges at a slower rate to the steady state.

The result above that the policy does not affect the interest rate points to the importance of expectations and the role of the details of the policy in anchoring them. The implicit assumption driving the result is that the policy is permanent. If the policy were explicitly time-dependent, i.e., it featured an announced expiration date, then it would have an effect on the interest rate due to the forward-looking behavior of agents. In this sense, the constant and permanent wedge  $h$  is unrealistic and we view it as a useful device to illustrate how the canonical representation of policies in epidemiological model applies seamlessly to our integrated epidemic-economy model. Rather than walking the reader through the specifics of time-dependent policies, however, we think it more insightful to move directly to the state-dependent policies studied in the next subsection since, to a large extent, they subsume the core properties of time-dependent policies.

Figure 7: Post-Infection SIR Dynamics with Policy Intervention



Notes: The figure depicts the equilibrium dynamics of the population-to-firms ratio for the post-infection SIR economy with (blue lines) and without (black lines) policy intervention.

## 6.2 A State-Dependent Wedge Approach

Here we turn to study *state-dependent* policy interventions. More specifically, we consider two classes of policies: (i) the first is based on tracking susceptible, such that the policy variable  $h$  is a function of the fraction of susceptible in the population, i.e.,  $h(s)$ ; (ii) the second policy is based on tracking infected, so that the policy variable is a function of the fraction of infected in the population, i.e.,  $h(i)$ . Modelling state-dependent policies has two key advantages over the simple policy in the previous subsection. First, it allows for an *endogenous* termination date of intervention based on a pre-specified target for either  $s$  or  $i$ . Second, it allows us to think in terms of a policy *rule* which anchors private sectors' expectations: at the time of the announcement, agents are aware of the intervention policy rule, so they anticipate what will happen and make self-fulfilling plans.

### 6.2.1 Tracking Susceptible

**Intervention Policy #2.** We start with the policy based on tracking susceptible, i.e.,  $h(s)$ . To simplify the analysis, we set  $\xi_D = 0$  (SIR model). To proceed, we need to recompute

the infection function.<sup>7</sup> To keep things tractable, let us consider a simple policy rule of the following form:

$$h = \begin{cases} \mu s^\eta - \mu \bar{s}^\eta & \bar{s} < s \leq 1 \\ 0 & 0 \leq s \leq \bar{s} \end{cases}, \quad 0 \leq \mu < 1, \eta > 0. \quad (63)$$

This policy rule has the property that intervention relaxes as  $s$  falls and vanishes at the target  $\bar{s}$ . A few remarks are in order. First, for  $\bar{s} = 0$ , (i) we can rule out  $\mu = 1$  because it implies the total shutdown of the economy at  $s_0 \approx 1$ ; (ii) we interpret  $\mu$  as upper bound on restrictions, e.g.,  $\mu = 0.2$  implies that initially  $1 / (1 + 0.2) = 83\%$  of healthy work; (iii) the parameter  $\eta$  regulates sensitivity of restrictions. Second, for  $\bar{s} > 0$ , there is endogenous termination at the policy target  $\bar{s}$ . For example, policy targets might be consistent with the achievement of natural herd immunity,  $s_{herd} = 1/R_0$ , or with the natural terminal state,  $s = s_\infty$ .

Recall that the infection function is a trajectory in the  $(s, \iota)$  space. Solving the PDE problem with initial condition  $(s_0, \iota_0)$ , and verifying continuity (value matching) and smooth-pasting at  $s = \bar{s}$ , it yields the new infection function, that takes into account the systematic policy response to the epidemiological dynamics of susceptible:

$$\iota(s) = \begin{cases} 1 - s + \frac{1}{R_0} \log\left(\frac{s}{s_0}\right) - \frac{\mu \bar{s}^\eta}{R_0} \log\left(\frac{\bar{s}}{s_0}\right) + \frac{\mu}{\eta R_0} (\bar{s}^\eta - s_0^\eta) & s_\infty^{int} \leq s \leq \bar{s} \\ 1 - s + \frac{1}{R_0} \log\left(\frac{s}{s_0}\right) - \frac{\mu \bar{s}^\eta}{R_0} \log\left(\frac{s}{s_0}\right) + \frac{\mu}{\eta R_0} (s^\eta - s_0^\eta) & \bar{s} < s \leq s_0 \end{cases}. \quad (64)$$

This adds two phase-specific intervention terms to the do-nothing solution. Allowing for  $s_0 \approx 1$ , we have the terminal state under intervention  $(s_\infty^{int}, 0)$  where

$$s_\infty^{int} = \arg \text{solve} \left\{ R_0 (1 - s) + \log s = \mu \bar{s}^\eta \log \bar{s} + \frac{\mu}{\eta} (1 - \bar{s}^\eta) \right\}. \quad (65)$$

At  $s = \bar{s}$  the economy reverts smoothly to the do-nothing regime, converging toward the terminal state  $(s, \iota) = (s_\infty^{int}, 0)$ . Recall that in the do-nothing regime, the terminal state is  $s_\infty = \arg \text{solve} \{ R_0 (1 - s) + \log s = 0 \}$ , so that  $s_\infty^{int} \leq s_\infty$  for  $\bar{s}^\eta \log \bar{s} + \frac{1}{\eta} (1 - \bar{s}^\eta) \leq 0$ . To get  $s_\infty^{int} = s_\infty$ , set  $\bar{s}$  such that  $\bar{s}^\eta \log \bar{s} + \frac{1}{\eta} (1 - \bar{s}^\eta) = 0$ .

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<sup>7</sup>Recall that the fraction of infected and susceptible evolve over time according to  $\dot{i} = \left( \frac{R_0 s}{1+h(s)} - 1 \right) \zeta_R \iota$  and  $\dot{s} = -\frac{\zeta_s}{1+h(s)} \iota s$ . Taking ratio of the two equations yields the PDE  $\frac{dt}{ds} = \frac{1+h(s)}{R_0 s} - 1$ .

**Per Capita Expenditure and the Interest Rate.** When free entry does not hold, per capita expenditure is

$$y(s, x) = \frac{\epsilon}{\epsilon - 1} \left( 1 - \frac{\phi}{x} \right) \left[ \frac{1 - \iota(s)}{1 + h(s)} \right]. \quad (66)$$

When free entry holds, it is

$$y(s) = \frac{1}{1 - \rho\beta} \left[ \frac{1 - \iota(s)}{1 + h(s)} \right]. \quad (67)$$

Importantly, in contrast with the constant-wedge policy in the previous subsection, the state-dependent policy rule  $h(s)$  in (63) introduces a direct effect of intervention on the interest rate. To see this, log-differentiating  $y(s)$  and using the Euler equation, yields

$$r(s) = \rho - \frac{1 - (\rho + \zeta_D)\beta}{[1 - \rho + \zeta_D \iota(s)]\beta} \frac{\iota(s)}{1 - \iota(s)} \zeta_R (R_0 s - 1) - \frac{h'(s) \dot{s}}{(1 + h(s))^2}. \quad (68)$$

And setting  $\zeta_D = 0$  and using the expression  $\dot{s}/s = -\zeta_S \iota(s)$ ,

$$\begin{aligned} r(s) = & \rho - \frac{\iota(s)}{1 - \iota(s)} \zeta_R (R_0 s - 1) \\ & + \frac{h'(s) s}{(1 + h(s))^2} \zeta_S \left[ 1 - s + \frac{1 - \mu \bar{s}^\eta}{R_0} \log \left( \frac{s}{s_0} \right) + \frac{\mu}{\eta R_0} (s^\eta - s_0^\eta) \right]. \end{aligned} \quad (69)$$

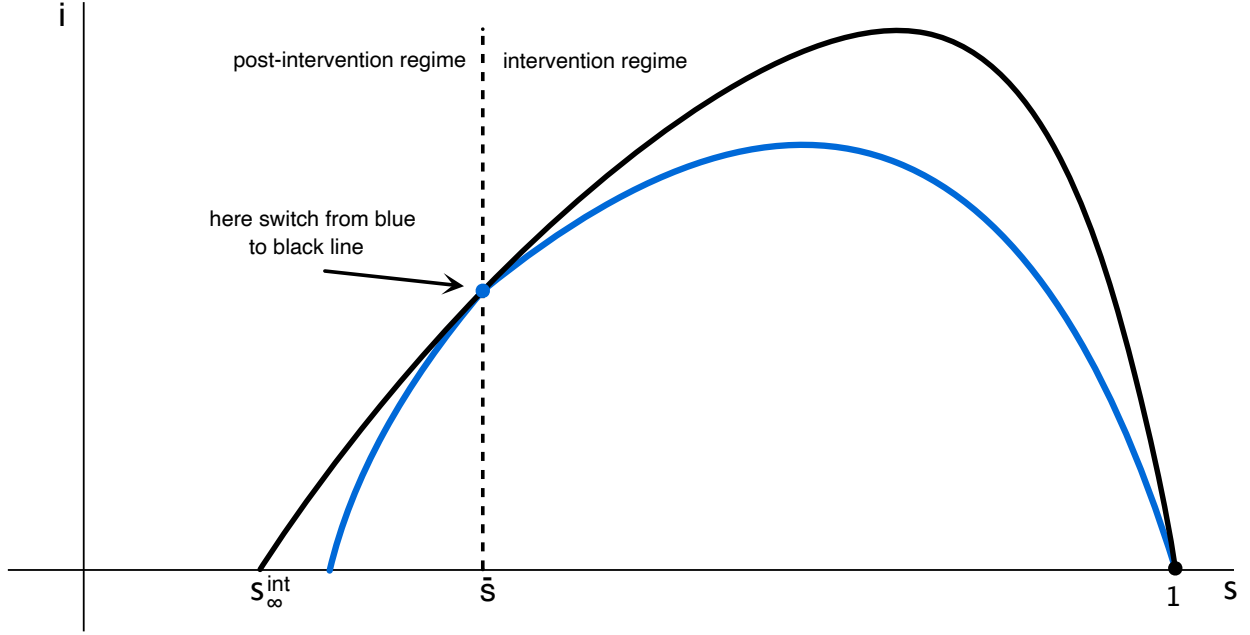
### 6.2.2 Tracking Infected

**Intervention Policy #3.** We now turn to a policy based on tracking infected:  $h(\iota)$ . Again, to simplify the analysis, we set  $\zeta_D = 0$  (SIR model), and recompute the infection function. To keep things tractable, we consider  $h = \mu \iota$ , with  $\mu > 0$ , and solving the PDE with initial condition  $(s_0, \iota_0)$ , we obtain

$$\iota(s) = \frac{R_0 (1 + \mu s) - \mu}{(\mu - R_0) \mu} - \left( \frac{s}{s_0} \right)^{\frac{\mu}{R_0}} \frac{R_0 (1 + \mu s_0) - \mu}{(\mu - R_0) \mu}. \quad (70)$$

As for the previous infection functions, for  $\mu > R_0$ , the function (70) is hump-shaped with two zeros, one at  $(1, 0)$  and the other at  $(s_\infty^{int}, 0)$ , where for  $(s_0, \iota_0) \approx (1, 0)$ , we get

Figure 8: Post-Infection Dynamics with State-Dependent Policy Intervention



Notes: The figure depicts the phase diagram for the post-infection economy with state-dependent policy intervention.

the terminal state under intervention as

$$s_{\infty}^{int} = \arg \text{solve} \left\{ \frac{\mu - R_0 (1 + \mu s)}{\mu - R_0 (1 + \mu)} = s^{\frac{\mu}{R_0}} \right\}. \quad (71)$$

Note that the infection-tracking case  $h(i)$  is much simpler than the susceptible-tracking case  $h(s)$  in that there is no need to construct the new infection function piece by piece. The epidemic fed to the economy takes the same form  $\dot{s}/s = -\xi_S i(s)$ . Qualitatively, the intervention policy the tracks infected delivers a similar phase diagram; the difference is that there is only one regime since by construction intervention lasts as long as  $i > 0$ .<sup>8</sup>

## 7 A Numerical Illustration

To further illustrate how an epidemic affects allocations and prices, and the implications of policy intervention, we parameterize the model and simulate equilibrium paths of three counterfactual economies: (i) the no-disease benchmark, in which the economy is moving along a disease-free BGP; (ii) the do-nothing policy scenario, in which the spread

<sup>8</sup>Note that the analysis can be extended to intervention stopping at target  $\bar{i} > 0$ .

of the disease is left to its own course; and (iii) the state-dependent policy scenario based on tracking susceptible (Intervention Policy #2). In our simulations, we focus on the SIR case, and the intervention policy goes inactive at about week 75. We refer the reader to Appendix B for details on the parameterization of the model, and to Appendix C for additional results based on a policy that tracks infected (Intervention Policy #3). We have run all simulations for the SIRD version of the model as well and noted that the numerical difference between the two cases is vary small. This is due to the fact that, thankfully, the mortality rate turned out to be rather small. We thus decided to report only results for the simpler SIR case.

**Model Parameters.** The parameter values for the SIR model are from <https://www.mathworks.com/matlabcentral/fileexchange/74658-fitviruscovid19>. The values for the transmission and recovery rate imply an  $R_0 \equiv \xi_S/\xi_R = 0.301/0.155 \approx 1.94$ . This value is broadly consistent with the evidence. 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. See Table 1 for baseline parameter values.

**Takeaways.** Two main lessons emerge from the numerical analysis. First, market size acts as a powerful propagation mechanism of the epidemic. The endogenous reduction in the mass of firms in response to the epidemic shock amplifies the recessionary effect on economic activity. The epidemic as well as the associated intervention policies that restrict the effective labor supply are akin to a negative “labor supply shock,” which leads to a sharp fall in consumption expenditure relative to the no-disease scenario, and thereby to a drop in net firm entry, which slowly propagates over time. Indeed, macroeconomic dynamics is much, much slower than epidemiological dynamics due to the sluggishness of the mass of firms.

Second, for a plausible parameterization of the model, we find that the susceptible-tracking intervention policy unambiguously worsens welfare. Welfare under intervention is lower than the do-nothing scenario over all the transition to the new steady state. We stress that this negative outcome is not hard-wired into the model. As an example, the infected-tracking intervention policy continues to do worse than the do-nothing scenario in the first 400 weeks, but it raises welfare relative to the do-nothing over the longer run. Overall, whether intervention policies raise or reduce welfare critically depends on the values of the coefficients of the policy rule in place that determine the stringency, and the duration of the intervention, and whether the costs of the policies are more or less

Table 1: Parameterization

Parameter	Description	Value
<i>A. Preferences &amp; technology</i>		
$\rho$	Discount rate	0.04/52
$\epsilon$	Prod. fcn	3
$\theta$	Prod. fcn	0.9639
$\beta$	Entry cost	$0.926 \times 52$
$\alpha$	Knowledge prod. fcn	0.0961/52
$\sigma$	Knowledge prod. fcn	0.0885
$\phi$	Fixed operating cost	5.1465
$\delta$	Firms' death rate	0.0618/52
<i>B. SIR model</i>		
$\xi_S$	Transmission rate	0.301
$\xi_R$	Recovery rate	0.155
$S_0$	Susceptible at outbreak	0.999
<i>C. Intervention policy</i>		
$\bar{s}$	Target for $s \equiv S/L$	0.4
$\mu$	Policy rule	0.5
$\eta$	Policy rule	1

Notes: Calibration at weekly frequency: 52 weeks per year.

front-loaded.

The last observation is very important and deserves emphasis: the core difference between tracking the susceptible and tracking the infected is that the fraction of susceptible is high and the onset and decreases throughout the epidemic. Interventions tied to it, therefore, concentrate their effects at the beginning of the epidemic, exacerbating the fall in economic activity. In contrast, the fraction of infected is initially very small and rises gradually before starting to decrease and eventually vanish. Interventions tied to it gradually tighten and then relax as they track the hump-shaped dynamics of the disease.

## 7.1 Epidemiological Dynamics

We begin by discussing the numerical results related to the epidemiological evolution of the disease. Figure 9a shows the infection function resulting from the parametrized model. A key insight stands out: policy intervention implies an across-the-board downward shift in the infection function, such that there are fewer infected per susceptible in the population. As evident in Figures 9b-9c, such a shift implies a slowdown in the spread of the epidemic: the fraction of susceptible falls less steeply under intervention relative to the do-nothing case, so that the peak in the fraction of infected occurs later in time. The time path of infected remains hump-shaped under the policy intervention, its peak is smaller in magnitude, but delayed.

## 7.2 Macroeconomic Dynamics

In the model, transition dynamics of economic variables lasts considerably longer than epidemiological dynamics. For our baseline parameterization, while the epidemic runs its natural course by approximately week 120, the dynamics of the economic state variable (i.e., population-to-firm ratio,  $x \equiv L/N$ ) and that of firm-specific knowledge growth is substantially slower. By week 600, the dynamical system remains far from the steady state. Due to the internal propagation mechanism at play in the model, epidemiological and economic variables move at rather different speed.

Figures 10a-10c show simulation results for the population-to-firms ratio, per capita expenditure, and the interest rate, respectively. After the outbreak, the population-to-firms ratio features hump-shaped dynamics: under the susceptible-tracking policy (policy 2), the rise in  $x$  is considerably larger relative to the do-nothing scenario. The peak in the population-to-firms ratio occurs when the epidemic runs out; the post-epidemic dynamics is due to endogenous market structure (Schumpeterian) dynamics. Under the susceptible-tracking policy intervention, the economy experiences an abrupt drop in per capita expenditure, which slowly reverts back to the steady state (see Figure 10b).

In Figure 10c, the interest rate dynamics reveals a striking qualitative difference between the policy intervention and the do-nothing scenario. In the do-nothing scenario, the interest rate falls first, then it rises, overshooting its long-run steady state level. In the policy intervention scenario instead, the interest rate remains above its long-run level along all the transition dynamics. The interest rate jumps down at the time the intervention ends because of the associated kink in the time profile of expenditure, due to the

forward-looking behavior with perfect foresight.

Figures 11a-11b show transition dynamics for “firm size,”  $f \equiv [1 - \iota(s)] L/N$ , and net firm entry,  $n \equiv \dot{N}/N$ , respectively. (Firm size  $f$  and the population-to-firms ratio,  $x$ , differ during the course of the epidemic insofar as  $\iota(s) > 0$ .) After the outbreak, the economy experiences a period of net exit in which the mass of firms shrinks at the firms’ exit rate  $\delta$ , i.e.,  $n = -\delta < 0$ . Under the policy intervention, the fall in net entry is of a larger magnitude due to the bigger drop in expenditures relative to the do-nothing scenario (see Figure 10b).

Finally, note that in the model TFP growth is equal to  $g = \theta z + \frac{n}{\epsilon-1}$ , where  $z \equiv \dot{Z}/Z$  denotes firm knowledge growth, and  $n$  is again growth in the mass of firms. Figures 12a shows that under policy intervention the economy hits a second corner, that is that of zero R&D labor, which implies a halt in firm’s knowledge accumulation.<sup>9</sup> As a result, the economy experiences a period of negative TFP growth (see Figure 12b). Net entry with a rebound in TFP growth re-starts when the epidemic runs out (about week 140 in our simulations).

### 7.3 Welfare Analysis of Policy Intervention

An important feature of the simple policy rules of the kind we propose here, is that they are amenable to welfare evaluation, accounting for transition dynamics. Figure 13 shows discounted utility relative to the baseline of the no-disease economy for the do-nothing and the *susceptible-tracking* intervention scenario with policy rule (63). As evident from the figure, for our baseline parametrization, severity, and duration of the intervention, the welfare in the counterfactual economy under intervention is lower than the do-nothing scenario over all the transition dynamics. (Note that as we consider the SIR case, the epidemic has no effect on the discount rate of utility flows.) Hence, this front-loaded policy intervention unambiguously worsens welfare. This is *not* a general result, rather, it critically depends on the intervention policy rule in place, whose coefficients contribute to determine the duration of the intervention. As we discussed, the infected-tracking policy intervention operates very differently: it does worse than the do-nothing scenario in the short-run (0-400 weeks period) but delivers welfare improvements in the medium- and long-run (see Figure C.5, in Appendix C).

The rules that we consider are simple but rather crude and this is likely the reason

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<sup>9</sup>If one assumed a positive depreciation rate of firm knowledge, transition dynamics would generate negative firm knowledge growth.

why they cause such deep and prolonged responses. Note also that the key difference between the two rules is that, because it tracks the susceptible rate, the first starts out at maximum intensity and then decays monotonically. It thus front-loads the economic damage caused by the loss of employment. The second policy, in contrast, tracks the infections rate and thus its intensity follows a hump-shaped profile that concentrates the loss of employment around the peak of the epidemic. Overall, the message that we extract from these exercises is that crude policies can be very damaging and that minimizing the damage requires careful examination of the channels through which policies operate.

## 8 Conclusion

We develop a dynamic general equilibrium model for the analysis of the economic effects of an epidemic. The model combines the standard epidemiological SIRD model with key features of second-generation endogenous growth theory, such as endogenous firm entry, which expands product variety, and cost-reducing innovation. Transition dynamics is analytically tractable and characterized by two differential equations describing the mass of susceptible in the population and the ratio of the population to the mass of firms. Overall, our results point to market size as an important mechanism through which an epidemic exerts its recessionary effect on economic activity.

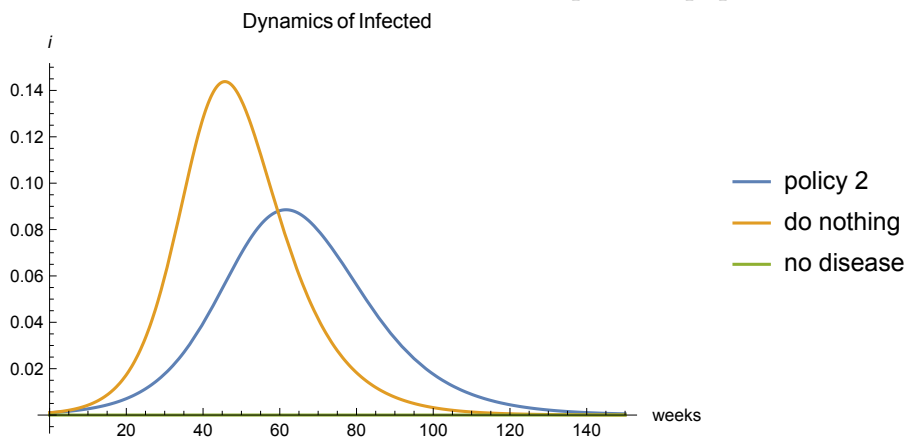
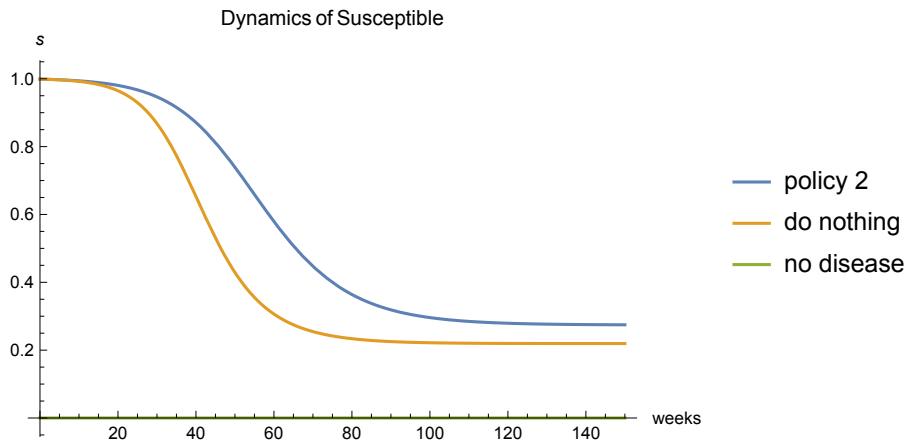
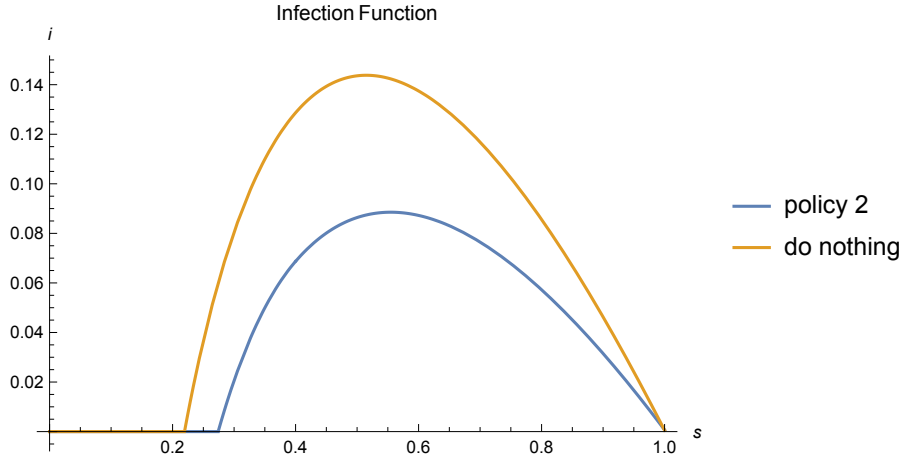
In the model, an outbreak propagates through the economy via changes in market size that alter the incentives of new firms to enter the market and of incumbents firms to invest in cost-reducing innovation. A typical epidemic is associated with a persistent fall in per capita expenditures and an aggregate productivity growth slowdown. If the initial fall in expenditure is big enough, a prolonged period of net firm exit with negative TFP growth ensues. Further, if the epidemics leads to death, as in the SIRD case, the new steady state exhibits fewer firms, less product variety, and a permanently lower level of real output per capita. Through the lens of the model, we evaluate state-dependent intervention policies based on tracking the fraction of susceptible or infected in the population. Akin to the well-known Taylor’s rule for monetary policy, our simple rules are an essential part of the definition of the equilibrium in that they serve to anchor private sector’s expectations about the time path and end date of the intervention.

There are several promising avenues for future research. First, while studying optimal intervention policies was beyond the scope of this paper, the systematic welfare analysis of simple policy rules is of first-order importance. In particular, one of the points that we

made in this paper is that, in the spirit of the Lucas' critique, the quantitative assessment of the economic effects of an epidemic cannot be conducted independently of the specific policy rule in place because such a rule anchors agents' expectations about the future. We plan to explore further this idea in future work.

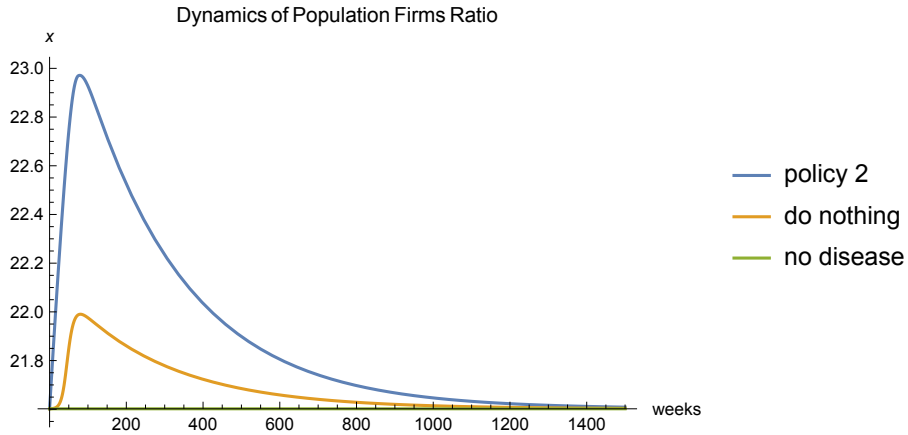
Second, in our basic setup we abstracted from several features that are important in an epidemic crisis. For example, the need for caregiving services to the infected calls for a reallocation of household expenditure and aggregate employment to the health-care sector. Similarly, the development of a vaccine and its mass production and provision requires the reallocation of resources from entry and cost-reducing innovation. Such reallocation is likely very important for the quantification of the economic effects of an epidemic. These questions can be addressed in the context of a multi-sector version of the model that we used here.

Third, and finally, in response to the COVID-19 epidemic, several OECD countries implemented fiscal packages aimed at offsetting the recessionary effects of mandated lockdowns. These fiscal measures will likely lead to a sizable increase in government debt. Understanding how the private sector's expectations of a future fiscal stabilization affect current behavior, and how this in turn determines the effectiveness of lockdown policies, is in our view a margin to be taken into account in the quantitative evaluation of intervention policies.

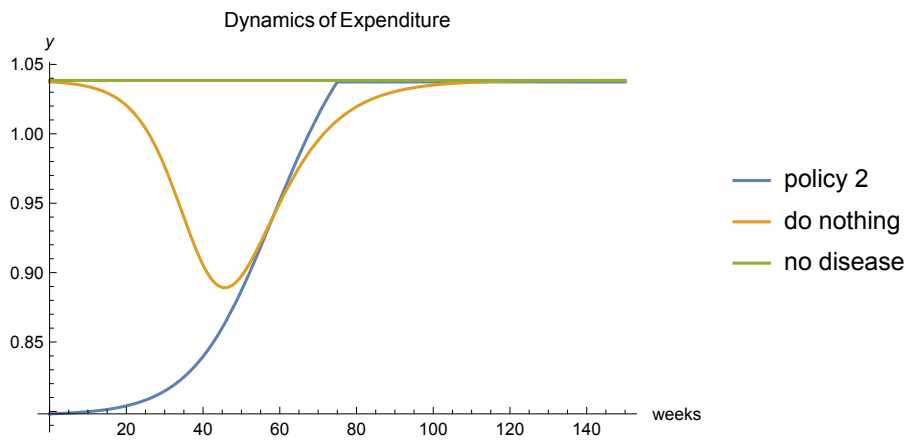


*Notes:* The figure shows the infection function and the dynamics of the fraction of susceptible and infected in the population after the outbreak for a parametrized version of the model. The susceptible-tracking intervention (policy 2) becomes inactive at about week 75. See Appendix B for details on the parameterization of the model.

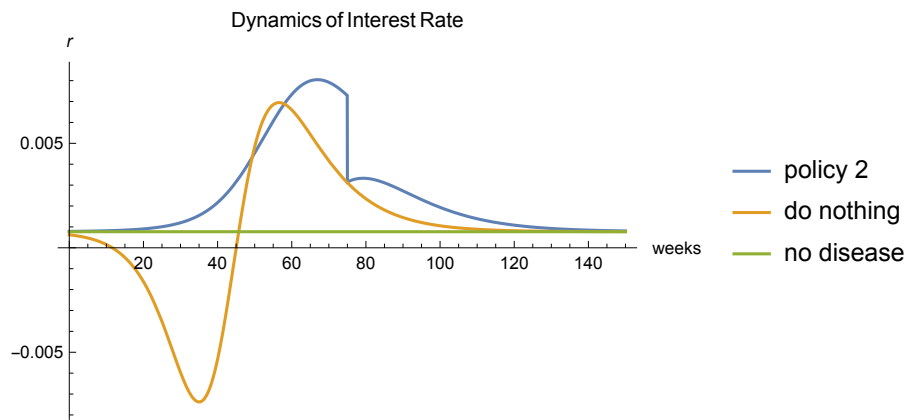
Figure 9: Epidemiological Dynamics after the Outbreak



(a) State variable:  $x \equiv L/N$



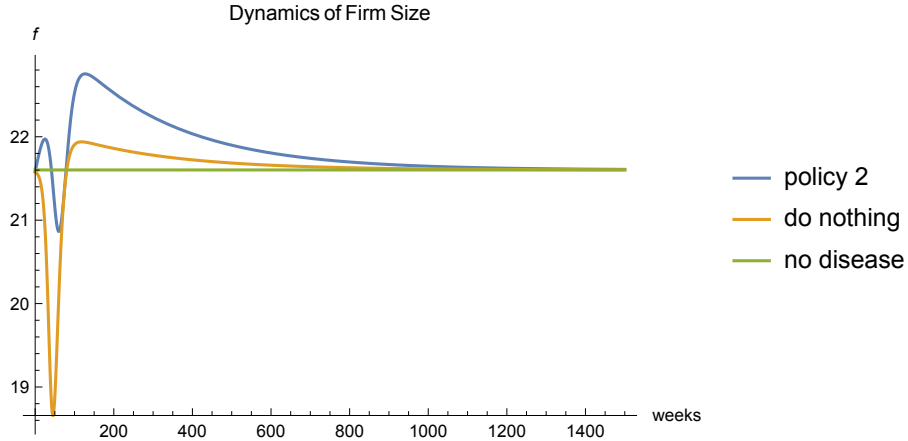
(b) Per capita expenditure:  $y \equiv Y/L$



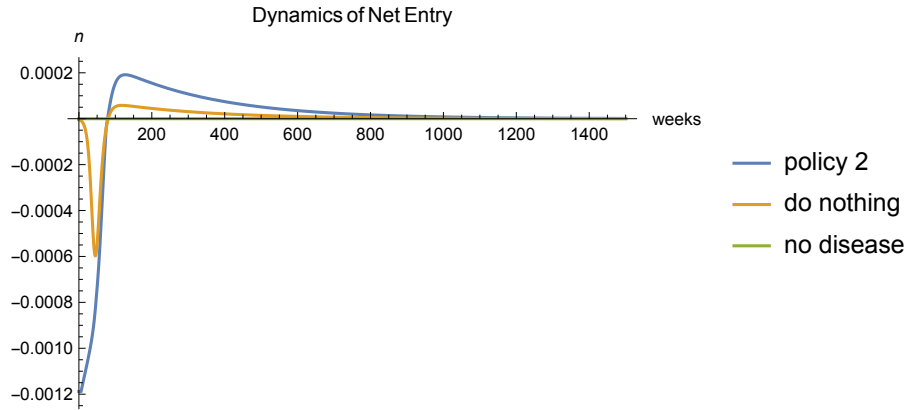
(c) Interest rate:  $r$

*Notes:* The figure shows simulations from a parametrized version of the model. The susceptible-tracking intervention (policy 2) becomes inactive at about week 75. See Appendix B for details on the parameterization of the model.

Figure 10: Market Size and Interest Rates



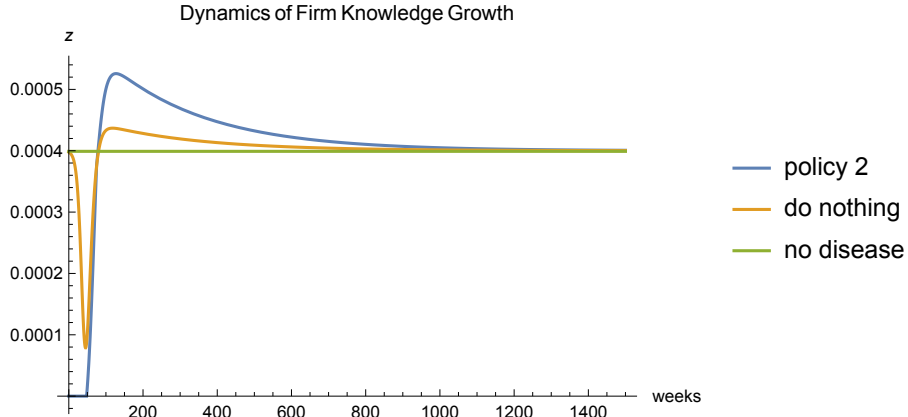
(a) Firm size:  $f \equiv [1 - \iota(s)] L/N$



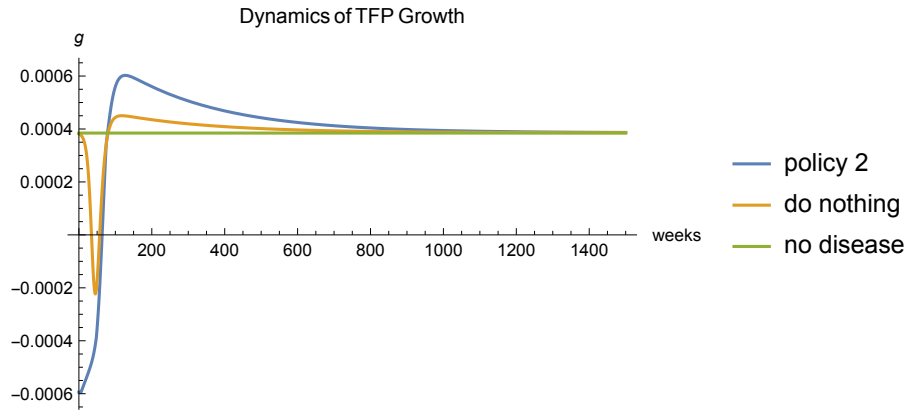
(b) Net entry:  $n \equiv \dot{N}/N$

*Notes:* The figure shows simulations from a parametrized version of the model. The susceptible-tracking intervention (policy 2) becomes inactive at about week 75. See Appendix B for details on the parameterization of the model.

Figure 11: Firm Size and Net Entry



(a) Firm knowledge growth:  $z = \dot{Z}/Z$

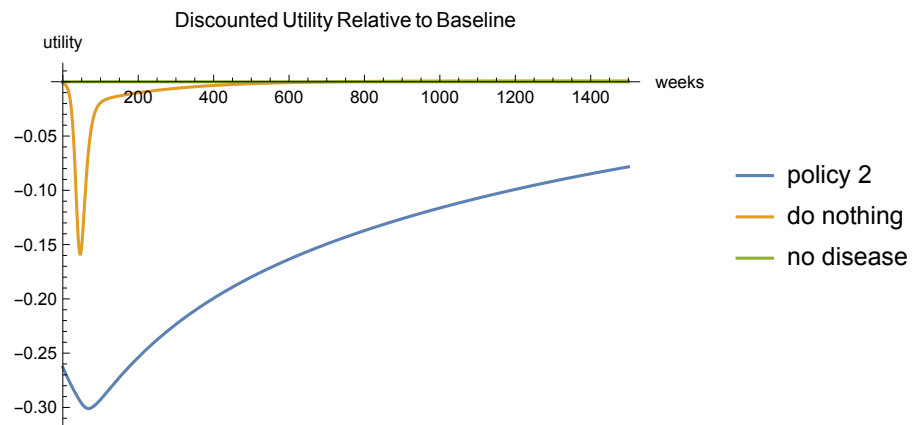


(b) TFP:  $Z^\theta N^{\frac{1}{\epsilon-1}}$

*Notes:* The figure shows simulations from a parametrized version of the model. The susceptible-tracking intervention (policy 2) becomes inactive at about week 75. See Appendix B for details on the parameterization of the model.

Figure 12: Knowledge and TFP Growth

Figure 13: Welfare Relative to Disease-Free Economy



*Notes:* The figure shows simulations from a parametrized version of the model. The susceptible-tracking intervention (policy 2) becomes inactive at about week 75. See Appendix B for details on the parameterization of the model.

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# Appendix

## A Model Derivations

In this appendix, we provide details on mathematical derivations.

### A.1 The Basic Growth Model

Henceforth, to simplify notation, we suppress time from endogenous variables whenever confusion does not arise.

**Production and innovation** Each consumption good is supplied by one firm. Thus,  $N$  also denotes the mass of firms. Each firm produces with the technology

$$X_i = Z_i^\theta (L_{X_i} - \phi), \quad 0 < \theta < 1, \quad \phi > 0, \quad (A.1)$$

where  $X_i$  is output,  $L_{X_i}$  is labor employment and  $\phi$  is a fixed operating cost. The firm maximizes the present discounted value of profit,

$$V_i = \int_0^\infty e^{-\int_0^t r(s)ds} \Pi_i dt, \quad (A.2)$$

where  $\Pi_i \equiv P_i X_i - L_{X_i} - w L_{Z_i}$ . The firm's FOC is

$$r = \frac{\partial \Pi_i}{\partial Z_i} \cdot \frac{1}{q_{Z_i}} + \frac{\dot{q}_{Z_i}}{q_{Z_i}}. \quad (A.3)$$

The associated pricing strategy is a constant mark-up rule:

$$P_i = \frac{\epsilon}{\epsilon - 1} Z_i^{-\theta}. \quad (A.4)$$

The firm's instantaneous profit can be written as

$$\Pi_i = \frac{\Upsilon}{\epsilon} \cdot \frac{Z_i^{\theta(\epsilon-1)}}{\int_0^N Z_j^{\theta(\epsilon-1)} dj} - \phi - L_{Z_i}. \quad (A.5)$$

Differentiating under the assumption that the firm takes the denominator as given, substituting the resulting expression into the asset-pricing equation derived above, and rear-

ranging terms yields

$$r = \frac{Y}{\epsilon} \theta (\epsilon - 1) \frac{Z_i^{\theta(\epsilon-1)-1}}{\int_0^N Z_j^{\theta(\epsilon-1)} dj} \cdot \frac{1}{q_{Z_i}} + \frac{\dot{q}_{Z_i}}{q_{Z_i}}. \quad (A.6)$$

This expression characterizes the return to knowledge accumulation for firm  $i$ . The cost of knowledge accumulation is determined by the technology

$$\dot{Z}_i = \alpha K L_{Z_i}^\sigma L_Z^{1-\sigma}, \quad \alpha > 0, \quad 0 < \sigma < 1, \quad (A.7)$$

where  $\dot{Z}_i$  is the flow of new knowledge generated by employing  $L_{Z_i}$  units of labor for an interval of time  $dt$ . The FOCs are:

$$CVH_i = P_i X_i - L_{X_i} - w L_{Z_i} + q_{Z_i} \alpha K L_{Z_i}^\sigma L_Z^{1-\sigma}, \quad (A.8)$$

$$w = q_{Z_i} \alpha \sigma K L_{Z_i}^{\sigma-1} L_Z^{1-\sigma} \Rightarrow 1 = q_{Z_i} \alpha \sigma K, \quad (A.9)$$

$$r + \delta = \frac{\partial \Pi_i}{\partial Z_i} \cdot \frac{1}{q_{Z_i}} + \frac{\dot{q}_{Z_i}}{q_{Z_i}} \Rightarrow \alpha \frac{L_Z}{N} = \frac{Y}{\epsilon N} \sigma \alpha \theta (\epsilon - 1) - r - \delta. \quad (A.10)$$

Then we have under symmetry,

$$r + \delta = \frac{Y}{N} \alpha \sigma \theta \frac{\epsilon - 1}{\epsilon} - \alpha \frac{L_Z}{N}. \quad (A.11)$$

The return to horizontal innovation (entry) is

$$r = \left[ \frac{Y}{\epsilon N} - \phi - L_Z \right] \frac{\epsilon N}{Y \beta (\epsilon - 1)} - \frac{\dot{N}}{N} + \frac{\dot{Y}}{Y}. \quad (A.12)$$

Check that we have well-behaved no entry region with  $z > 0$  since calibration yields  $x_Z \ll x_N$ . From the household's budget constraint:

$$\dot{N}V + N\dot{V} = \left( \frac{\frac{Y}{\epsilon N} - \phi - \frac{L_Z}{N}}{V} + \frac{\dot{V}}{V} - \delta \right) NV + wL - Y, \quad (A.13)$$

$$-\delta NV = \left( \frac{Y}{\epsilon N} - \phi - \frac{L_Z}{N} \right) N - \delta NV + wL - Y, \quad (A.14)$$

$$0 = \left( \frac{Y}{\epsilon N} - \phi - \frac{L_Z}{N} \right) N + L - Y, \quad \text{since } w = 1. \quad (A.15)$$

Also,

$$\frac{L_Z}{N} = \frac{Y}{\epsilon N} \theta (\epsilon - 1) - \frac{r + \delta}{\alpha}. \quad (A.16)$$

Hence,

$$\alpha \phi - x \alpha \left( 1 - y \left[ 1 - \frac{1 - \theta (\epsilon - 1)}{\epsilon} \right] \right) = r + \delta. \quad (A.17)$$

Then we have the dynamical system:

$$\frac{\dot{y}}{y} = \alpha \phi - x \alpha + \alpha x y \left[ 1 - \frac{1 - \theta (\epsilon - 1)}{\epsilon} \right] - \rho - \delta, \quad (A.18)$$

$$\frac{\dot{x}}{x} = \delta. \quad (A.19)$$

Check that we have well-behaved no entry region with  $z = 0$ . From the household's budget constraint:

$$\dot{N}V + N\dot{V} = \left( \frac{Y}{\epsilon N} - \phi + \frac{\dot{V}}{V} - \delta \right) NV + wL - Y, \quad (A.20)$$

$$-\delta NV = \left( \frac{Y}{\epsilon N} - \phi \right) N - \delta NV + wL - Y, \quad (A.21)$$

$$0 = \left( \frac{Y}{\epsilon N} - \phi \right) N + L - Y, \quad \text{since } w = 1. \quad (A.22)$$

Hence,

$$y = \frac{\epsilon}{\epsilon - 1} \left( 1 - \frac{\phi}{x} \right). \quad (A.23)$$

## A.2 Three Examples of Epidemiological Trajectories

Epidemiological SIRD equations:

$$\frac{\dot{s}}{s} = \zeta_B \left( \frac{1}{s} - 1 \right) - (\zeta_S - \zeta_D) \iota, \quad (A.24)$$

$$\frac{\dot{i}}{i} = \zeta_S s - \zeta_R - \zeta_B - \zeta_D (1 - \iota). \quad (A.25)$$

**Example 1: SIRD w/ no vital dynamics** (i.e.,  $\zeta_B = \zeta_M = 0$ ):

$$\frac{\dot{s}}{s} = -(\zeta_S - \zeta_D) \iota < 0, \quad (A.26)$$

$$i \geq 0 : \quad \iota \geq 1 + \frac{\zeta_R}{\zeta_D} - \frac{\zeta_S}{\zeta_D} s. \quad (A.27)$$

**Example 2: SIR w/ no vital dynamics** (i.e.,  $\zeta_B = \zeta_M = \zeta_D = 0$ ):

$$\frac{\dot{s}}{s} = -\zeta_S \iota < 0, \quad (A.28)$$

$$i \geq 0 : \quad s \geq \frac{\zeta_S}{\zeta_R} = R_0. \quad (A.29)$$

**Example 3: SIR w/ vital dynamics** (i.e.,  $\zeta_D = 0$ ):

$$\dot{s} \geq 0 : \quad \iota \leq \frac{\zeta_B}{\zeta_S} \left( \frac{1}{s} - 1 \right), \quad (A.30)$$

$$i \geq 0 : \quad s \geq \frac{\zeta_R + \zeta_B}{\zeta_S}. \quad (A.31)$$

### A.3 Three Types of Interventions

#### A.3.1 Example 1: Constant Wedge Policy

The economy reverts to the same long-run growth rate because the permanent change in expenditure is offset by a permanent rise in firm size. Of course, permanent intervention is not sensible since when the epidemic runs its course and the economy is at  $(s_\infty, 0)$ , one would think that intervention should be lifted. However, note that if one allows for the possibility of newborn, we have a continuous inflow of susceptible and thus the system does *not* converge to a disease-free state with  $\iota = 0$ , but to an endemic state with  $\iota = \bar{\iota} > 0$ . One might then think that there exists an equilibrium with permanent intervention absent a vaccine and/or a cure.

#### A.3.2 Example 2: Susceptible-tracking Policy

Define the policy

$$h(s) = \begin{cases} \mu s^\eta - \mu \bar{s}^\eta & \bar{s} < s \leq 1 \\ 0 & 0 \leq s \leq \bar{s} \end{cases}. \quad (A.32)$$

This policy rule has the property that intervention relaxes as  $s$  falls and vanishes at the target  $\bar{s}$ . The PDE problem is

$$\frac{d\iota}{ds} = \frac{1 + \mu s^\eta - \mu \bar{s}^\eta}{R_0 s} - 1, \quad (\text{A.33})$$

and has solution

$$\iota(s) = c - s + \frac{1 - \mu \bar{s}^\eta}{R_0} \log s + \frac{\mu}{\eta R_0} s^\eta. \quad (\text{A.34})$$

Using the initial condition  $(s_0, \iota_0)$  yields

$$\iota_0 = c - 1 + \iota_0 + \frac{1 - \mu \bar{s}^\eta}{R_0} \log s_0 + \frac{\mu}{\eta R_0} s_0^\eta. \quad (\text{A.35})$$

We thus get

$$\iota(s) = 1 - s + \frac{1}{R_0} \log \left( \frac{s}{s_0} \right) + \underbrace{\frac{\mu}{\eta R_0} (s^\eta - s_0^\eta) - \frac{\mu \bar{s}^\eta}{R_0} \log \left( \frac{s}{s_0} \right)}_{\text{intervention term}}. \quad (\text{A.36})$$

Let us check that the infection function switches continuously from this one to the one that holds with no intervention, which has solution

$$\iota(s) = c - s + \frac{1}{R_0} \log s. \quad (\text{A.37})$$

We choose  $c$  so that continuity holds at  $s = \bar{s}$ . The two values are:

$$\iota(\bar{s}^+) = 1 - \bar{s} + \frac{1 - \mu \bar{s}^\eta}{R_0} \log \left( \frac{\bar{s}}{s_0} \right) + \frac{\mu}{\eta R_0} (\bar{s}^\eta - s_0^\eta), \quad (\text{A.38})$$

$$\iota(\bar{s}^-) = c - \bar{s} + \frac{1}{R_0} \log \bar{s}. \quad (\text{A.39})$$

Continuity holds when

$$c - \bar{s} + \frac{1}{R_0} \log \bar{s} = 1 - \bar{s} + \frac{1 - \mu \bar{s}^\eta}{R_0} \log \left( \frac{\bar{s}}{s_0} \right) + \frac{\mu}{\eta R_0} (\bar{s}^\eta - s_0^\eta), \quad (\text{A.40})$$

$$c = 1 + \frac{1 - \mu \bar{s}^\eta}{R_0} \log \left( \frac{\bar{s}}{s_0} \right) + \frac{\mu}{\eta R_0} (\bar{s}^\eta - s_0^\eta) - \frac{1}{R_0} \log \bar{s}. \quad (\text{A.41})$$

Thus, for  $s \leq \bar{s}$  we have

$$\iota(s) = 1 - s + \frac{1}{R_0} \log\left(\frac{\bar{s}}{s_0}\right) + \frac{1}{R_0} \log\left(\frac{s}{\bar{s}}\right) - \frac{\mu \bar{s}^\eta}{R_0} \log\left(\frac{\bar{s}}{s_0}\right) + \frac{\mu}{\eta R_0} (\bar{s}^\eta - s_0^\eta), \quad (A.42)$$

$$= 1 - s + \frac{1}{R_0} \log\left(\frac{s}{s_0}\right) + \frac{\mu}{\eta R_0} (\bar{s}^\eta - s_0^\eta) - \frac{\mu \bar{s}^\eta}{R_0} \log\left(\frac{\bar{s}}{s_0}\right). \quad (A.43)$$

Let us check continuity. At  $s = \bar{s}$  the two pieces connect as follows:

$$-\frac{\mu \bar{s}^\eta}{R_0} \log\left(\frac{\bar{s}}{s_0}\right) + \frac{\mu}{\eta R_0} (\bar{s}^\eta - s_0^\eta) = -\frac{\mu \bar{s}^\eta}{R_0} \log\left(\frac{\bar{s}}{s_0}\right) + \frac{\mu}{\eta R_0} (\bar{s}^\eta - s_0^\eta), \quad (A.44)$$

$$0 = 0. \quad (A.45)$$

Continuity holds. Next, let us check derivatives at  $s = \bar{s}$ :

$$-1 + \frac{1}{R_0} \cdot \frac{1}{\bar{s}} = -1 + \frac{1}{R_0} \cdot \frac{1}{\bar{s}} + \frac{\mu \bar{s}^{\eta-1}}{R_0} - \frac{\mu \bar{s}^{\eta-1}}{R_0}, \quad (A.46)$$

$$0 = 0. \quad (A.47)$$

Smooth pasting also holds. Hence, to sum:

$$\iota(s) = \begin{cases} 1 - s + \frac{1}{R_0} \log\left(\frac{s}{s_0}\right) - \frac{\mu \bar{s}^\eta}{R_0} \log\left(\frac{\bar{s}}{s_0}\right) + \frac{\mu}{\eta R_0} (\bar{s}^\eta - s_0^\eta) & s_\infty^{int} \leq s \leq \bar{s}, \\ 1 - s + \frac{1}{R_0} \log\left(\frac{s}{s_0}\right) - \frac{\mu \bar{s}^\eta}{R_0} \log\left(\frac{s}{s_0}\right) + \frac{\mu}{\eta R_0} (s^\eta - s_0^\eta) & \bar{s} < s \leq s_0 \end{cases}. \quad (A.48)$$

### A.3.3 Example 3: Infected-tracking Policy

Define the policy

$$h(\iota) = \mu \iota. \quad (A.49)$$

This policy rule has the property that intervention intensifies as  $\iota$  rises and relaxes as  $\iota$  falls, vanishing exactly when  $\iota = 0$ . The PDE problem is

$$\frac{d\iota}{ds} = \frac{1 + \mu \iota}{R_0 s} - 1, \quad (A.50)$$

and has solution

$$i(s) = \frac{\mu - R_0(1 + \mu s)}{(R_0 - \mu)\mu} + s^{\frac{\mu}{R_0}} c. \quad (A.51)$$

Using initial condition  $(s_0, i_0)$  yields

$$c = \left[ i_0 - \frac{\mu - R_0(1 + \mu s_0)}{(R_0 - \mu)\mu} \right] s_0^{-\frac{\mu}{R_0}}. \quad (A.52)$$

Therefore, we have

$$i(s) = \frac{R_0(1 + \mu s) - \mu}{(\mu - R_0)\mu} - \left( \frac{s}{s_0} \right)^{\frac{\mu}{R_0}} \frac{R_0(1 + \mu s_0) - \mu}{(\mu - R_0)\mu}. \quad (A.53)$$

For  $\mu > R_0$  this is hump-shaped with two zeros, one at  $(1, 0)$  and the other at  $(s_\infty^{int}, 0)$ , where for  $(s_0, i_0) \approx (1, 0)$ ,

$$s_\infty^{int} = \arg \text{solve} \left\{ \frac{\mu - R_0(1 + \mu s)}{\mu - R_0(1 + \mu)} = s^{\frac{\mu}{R_0}} \right\}. \quad (A.54)$$

## B Parametrization

In this appendix, we discuss the parametrization of the model. Our strategy consists of two steps. First, in Subsection B.1, we set data targets and back out parameter values at the annual frequency. Second, in Subsection B.2, we calculate the weekly counterparts of the annual parameter values, so that the weekly model remains consistent with the annual data targets.

### B.1 Annual Calibration

**Standard targets** At the annual frequency,

- Interest rate:  $r = \rho = 0.04$ .
- Per capita GDP growth rate:  $g = 0.02$ .
- Consumption expenditure to GDP ratio:  $Y/G = 0.675$ .
- Labor share of GDP:  $wL/G = 0.65$ .

- Population to firms ratio: using data from the Business Dynamics Statistics (BDS), we calculate an average population-to-firms ratio of  $x \equiv L/N = 21.6$ .
- Firms' death rate: using data from the BDS, we calculate an average exit rate of  $\delta = 0.0618$ .

**Elasticity of substitution b/w intermediate goods ( $\epsilon$ )** To calibrate  $\epsilon$  we use data on the Net Operating Surplus (NOS) to GDP ratio from the BEA. In U.S. data, the average NOS/GDP ratio is 23%. In the model,  $NOS = Y/\epsilon N$ . Using the relationship  $Y/G = \epsilon NOS/G$ , we obtain  $\epsilon = (Y/G) / NOS/G = 0.675/0.23 = 2.9348 \simeq 3$ .

**Sunk entry cost ( $\beta$ )** Using the household budget, it gives

$$y = \frac{1}{1 - \beta\rho} \Rightarrow \beta = \left(1 - \frac{wL}{G} \frac{G}{Y}\right) \frac{1}{\rho} = \left(1 - \frac{0.65}{0.675}\right) \frac{1}{0.04} = 0.926. \quad (B.1)$$

This procedure is immediate and only uses the labor share,  $wL/G$ , and the consumption ratio,  $Y/G$ .

**Fixed operating cost ( $\phi$ )** We note that the parameter  $\phi$  is identified independently of the R&D technology since

$$\phi = x \left[ \frac{\frac{1}{\epsilon} - (r + \delta) \beta}{1 - r\beta} - \frac{L_Z}{L} \right]. \quad (B.2)$$

Needed: data on  $L_Z/L$ . From the InfoBrief, October 2016, NSF 17-302: “Companies active in research and development (those that paid for or performed R&D) employed 1.5 million R&D workers in the United States in 2013 (table 1), according to the Business R&D and Innovation Survey (BRDIS).[2] R&D employees are defined in BRDIS as all employees who work on R&D or who provide direct support to R&D, such as researchers, R&D managers, technicians, clerical staff, and others assigned to R&D groups. Although these R&D workers account for just over 1% of total business employment in the United States, they play a vital role in creating the new ideas and technologies that keep companies competitive, create new markets, and spur economic growth.[3] This InfoBrief presents data from BRDIS on the characteristics of these R&D workers, highlighting similarities and differences between different types of R&D-active companies.” (See <https://www.nsf.gov/statistics/2017/nsf17302/>).

So, using  $L_Z/L = 0.01$ , for  $\delta = 0.0618$ , it gives

$$\phi = 21.6 \left[ \frac{\frac{1}{3} - (0.1018) 0.926}{1 - 0.04 \times 0.926} - 0.01 \right] = 5.1465. \quad (B.3)$$

**The “innovation” triplet  $(\alpha, \theta, \sigma)$**  Let us write the TFP growth rate along a BGP as

$$g = \alpha\theta \times \frac{L}{N} \times \frac{L_Z}{L} \Rightarrow \alpha\theta = \frac{g}{\frac{L}{N} \times \frac{L_Z}{L}} = \frac{0.02}{\frac{L}{N} \times \frac{L_Z}{L}} = \frac{0.02}{21.6 \times 0.01} = 0.0926. \quad (B.4)$$

Thus, the data moments ( $g = 2\%$ ,  $L_Z/L = 1\%$ ,  $L/N = 21.6$ ) pin down the value of  $\alpha\theta$ . To proceed, consider the Current Value Hamiltonian (CVH) for the firm’s problem:

$$CVH_i = P_i X_i - L_{X_i} - w L_{Z_i} + q_{Z_i} \cdot \alpha K L_{Z_i}^\sigma L_Z^{1-\sigma}. \quad (B.5)$$

The FOCs are:

$$w = q_{Z_i} \alpha \sigma K L_{Z_i}^{\sigma-1} L_Z^{1-\sigma} \Rightarrow 1 = q_Z \alpha \sigma K, \quad (B.6)$$

$$r + \delta = \frac{\partial \Pi_i}{\partial Z_i} \cdot \frac{1}{q_{Z_i}} + \frac{\dot{q}_{Z_i}}{q_{Z_i}} \Rightarrow \frac{L_Z}{N} = \frac{Y}{N} \sigma \theta \frac{\epsilon - 1}{\epsilon} - \frac{r + \delta}{\alpha}. \quad (B.7)$$

After some manipulations, we get

$$r + \delta + z = \sigma \alpha \theta \left( \frac{\epsilon - 1}{\epsilon} \right) xy \Rightarrow \left( r + \delta + \frac{g}{\theta} \right) \frac{1}{y} \frac{\epsilon}{\epsilon - 1} = \sigma \alpha \theta. \quad (B.8)$$

The associated entry process is unchanged, i.e.,

$$\frac{\dot{N}}{N} = \frac{1}{\beta} \left[ \frac{1}{\epsilon} - \frac{N}{Y} \left( \phi + \frac{L_Z}{N} \right) \right] - \rho - \delta \quad (B.9)$$

$$= \frac{1}{\beta \epsilon} - \frac{1}{\beta y} \left( \phi + \frac{z}{\alpha} \right) \frac{1}{x} - \rho - \delta. \quad (B.10)$$

The expression for the steady-state population-to-firms ratio is

$$x = \frac{\phi - \frac{\rho + \delta}{\alpha}}{1 - \sigma \theta (\epsilon - 1) - (\rho + \delta) \beta \epsilon} \times \frac{\epsilon}{y}. \quad (B.11)$$

Note that the parameter  $\sigma$  only enters the return to in-house innovation and it scales the activation threshold  $x_Z$  leaving the rest the same. We now have two moment conditions

and three parameters with the needed degree of freedom to obtain the “right” threshold ordering, i.e.  $x_N < x_Z$ . Specifically, we have:

$$r + \delta + \frac{g}{\theta} = \sigma \alpha \theta \left( \frac{\epsilon - 1}{\epsilon} \right) xy. \quad (B.12)$$

Given the value of  $\alpha \theta$ , this equation identifies  $\theta$  and  $\sigma$  as follows. First, let us isolate  $\theta$  such that

$$\theta = \frac{0.02}{\sigma \alpha \theta y^{\frac{\epsilon-1}{\epsilon}} x - r - \delta} = \frac{0.02}{\sigma \frac{0.0926}{1-0.04 \times 0.926} \frac{2}{3} 21.6 - 0.1018}. \quad (B.13)$$

Then, we use the ratio of thresholds,

$$\frac{x_Z}{x_N} = \frac{1}{\sigma} \frac{\rho + \delta}{\alpha \theta (\epsilon - 1)} \frac{1 - \rho \beta \epsilon}{\phi} = \frac{1}{\sigma} \frac{0.1018}{0.0926 \times 2} \frac{1 - 0.04 \times 0.926 \times 3}{5.0177}, \quad (B.14)$$

where the thresholds are:

$$x_Z = (1 - \rho \beta) \frac{\epsilon (\rho + \delta)}{\sigma \alpha \theta (\epsilon - 1)} = \frac{1}{\sigma} (1 - 0.04 \times 0.926) \frac{0.1018 \times 3}{0.0926 \times 2} = \frac{1}{\sigma} 1.5879, \quad (B.15)$$

$$x_N = \frac{1 - \beta \rho}{\frac{1}{\epsilon} - \rho \beta} \phi = \frac{1 - 0.04 \times 0.926}{\frac{1}{3} - 0.04 \times 0.926} 5.0177 = 16.308. \quad (B.16)$$

We thus are free to set  $\sigma$  to obtain the desired threshold ratio. For example, let us say we aim to obtain the following targets:

$$\begin{aligned} \frac{x_Z}{x_N} = \frac{5}{4} : \quad \sigma &= \frac{1.5879}{5/4 \times 16.308} = 0.0779, \\ \frac{x_Z}{x_N} = \frac{11}{10} : \quad \sigma &= \frac{1.5879}{11/10 \times 16.308} = 0.0885, \\ \frac{x_Z}{x_N} = \frac{101}{100} : \quad \sigma &= \frac{1.5879}{101/100 \times 16.308} = 0.0964. \end{aligned}$$

And in terms of the threshold for cost-reducing innovation:

$$x_Z = \frac{5}{4} 16.308 = 20.385, \quad (B.17)$$

$$x_Z = \frac{11}{10} 16.308 = 17.939, \quad (B.18)$$

$$x_Z = \frac{101}{100} 16.308 = 16.471. \quad (B.19)$$

The first is very close to steady state, while the last is very close to  $x_N$ . Then we get:

$$\theta = \frac{0.02}{0.0779 \times \frac{0.0926}{1-0.04 \times 0.926} \frac{2}{3} 21.6 - 0.1018} = 3.2946, \quad (B.20)$$

$$\theta = \frac{0.02}{0.0885 \times \frac{0.0926}{1-0.04 \times 0.926} \frac{2}{3} 21.6 - 0.1018} = 0.9639, \quad (B.21)$$

$$\theta = \frac{0.02}{0.0964 \times \frac{0.0926}{1-0.04 \times 0.926} \frac{2}{3} 21.6 - 0.1018} = 0.6312. \quad (B.22)$$

Notice how quickly the value of  $\theta$  drops as we make the distance between the thresholds smaller. Next, we get:

$$\alpha = \frac{0.0926}{3.2946} = 0.0281, \quad (B.23)$$

$$\alpha = \frac{0.0926}{0.9639} = 0.0961, \quad (B.24)$$

$$\alpha = \frac{0.0926}{0.6312} = 0.1467. \quad (B.25)$$

Need to check that in all cases  $1 - \sigma\theta(\epsilon - 1) = 1 - 0.0779 \times 3.2946 \times 2 = 0.4867 > 0$ .

## B.2 Weekly Calibration

For the labor share to hit the same target at the annual and weekly frequency, the model implies sharp parameter restrictions:

$$\frac{wL}{Y} = 1 - \beta\rho \Rightarrow \beta\rho = 1 - \frac{wL}{G} \times \frac{G}{Y} = \frac{0.65}{0.675} = 0.96296. \quad (B.26)$$

The key is then to keep  $\beta\rho$  constant, so that scaling  $\rho$  by the factor 52,  $\rho/52$ , mandates to inflate the entry cost by the same factor,  $\beta \times 52$ . This is consistent with the argument that the weekly eigenvalue should be equal to the annual eigenvalue divided by 52:

$$\frac{1 - \sigma\theta(\epsilon - 1)}{\beta\epsilon} - (\rho + \delta) = \left[ \frac{1 - \sigma\theta(\epsilon - 1)}{\beta\epsilon} - \frac{\rho + \delta}{52} \right] 52, \quad (B.27)$$

$$\frac{1 - \sigma\theta(\epsilon - 1)}{\beta_a\epsilon} - (\rho + \delta) = \frac{1 - \sigma\theta(\epsilon - 1)}{\beta_w\epsilon} \frac{1}{52} - (\rho + \delta), \quad (B.28)$$

$$\frac{1 - \sigma\theta(\epsilon - 1)}{\beta_a\epsilon} = \frac{1 - \sigma\theta(\epsilon - 1)}{\beta_w\epsilon} 52. \quad (B.29)$$

Insofar as  $\sigma$  and  $\theta$  keep the same value, we have exactly that  $\beta_w = \beta_a \times 52$ .

Also, since

$$\phi = x \left[ \frac{\frac{1}{\epsilon} - (\rho + \delta) \beta}{1 - \rho \beta} - \frac{L_Z}{L} \right], \quad (B.30)$$

we note that  $\phi$  retains the same value since both  $(\rho + \delta) \beta$  and  $\rho \beta$  remain constant across calibrations. (For the firms' exit rate, we simply re-scale the annual death rate by 52 weeks,  $\delta = 0.0618/52$ .)

Next, note that

$$\frac{0.02/54}{\theta} = \alpha \frac{L_Z}{L} \times \frac{L}{N} \Rightarrow \alpha \theta = \frac{0.02/52}{\frac{L}{N} \times \frac{L_Z}{L}}, \quad (B.31)$$

implying that the conversion to the weekly frequency requires dividing the annual value of the product  $\alpha \theta$  by 52:

$$r + \delta = \alpha \left[ \frac{Y \sigma \theta (\epsilon - 1)}{\epsilon N} - \frac{L_Z}{N} \right] \Rightarrow z = \sigma \alpha \theta \left( \frac{\epsilon - 1}{\epsilon} \right) xy - r - \delta, \quad (B.32)$$

$$\frac{0.02/52}{\theta} = \sigma \frac{\alpha \theta}{52} \left( \frac{\epsilon - 1}{\epsilon} \right) xy - (r + \delta) / 52. \quad (B.33)$$

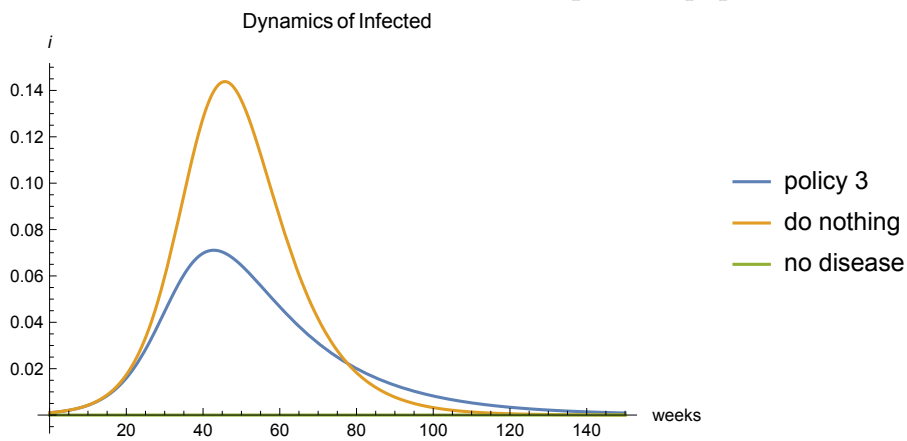
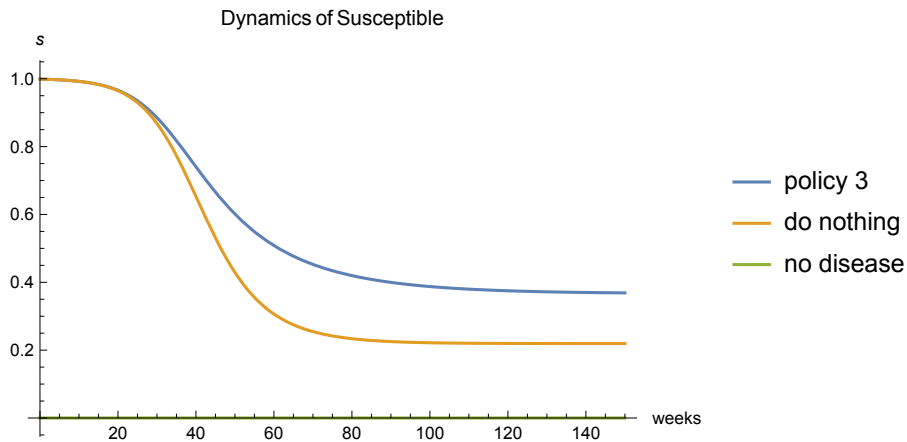
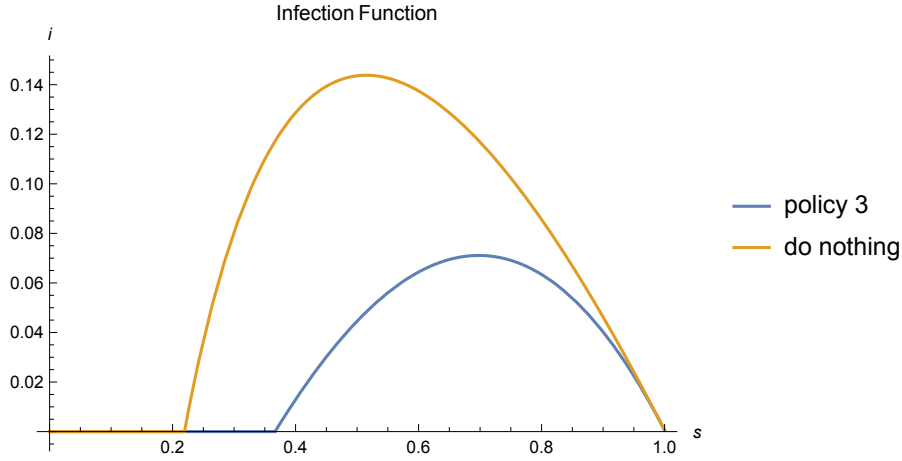
Hence, the parameters  $\theta$  and  $\sigma$  are invariant from the annual to the weekly frequency since the conversion factor 52 drops out of these expressions. Then, going to the weekly calibration, implies that  $\alpha$  changes according to

$$\alpha_w = \frac{1}{\theta} \frac{0.02}{\frac{L}{N} \times \frac{L_Z}{L}} \frac{1}{52} = \frac{\alpha_a}{52}. \quad (B.34)$$

To sum, going from an annual to a weekly calibration, only the rates  $(r, g, \delta)$  change and their changes are absorbed one-for-one by  $\alpha$  and  $\beta$ .

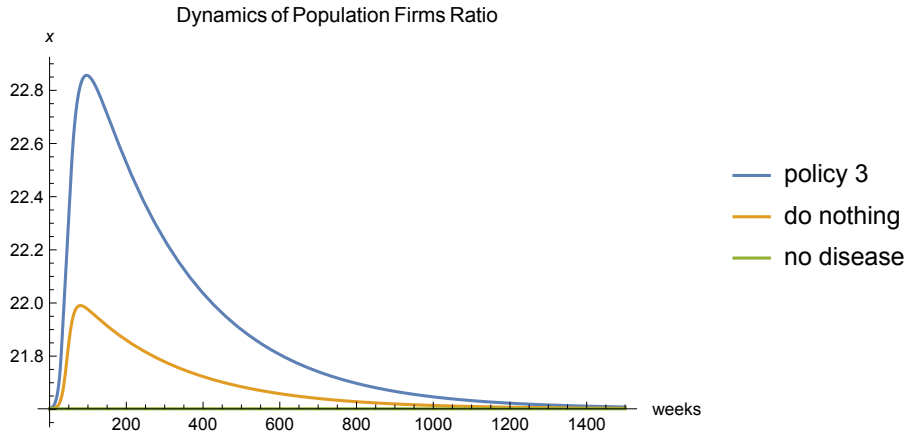
## C Additional Results

In this appendix, we provide additional results from numerical experiments based on an intervention policy that tracks the fraction of infected in the population (policy 3).

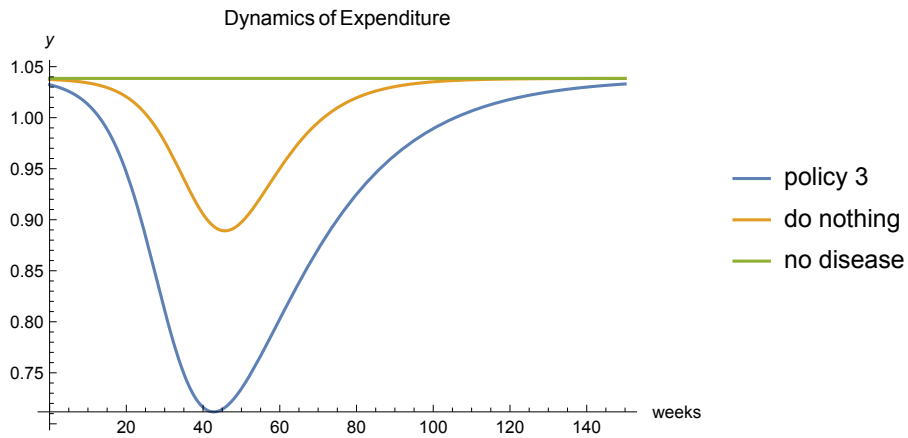


*Notes:* The figure shows the infection function and the dynamics of the fraction of susceptible and infected in the population after the outbreak for a parametrized version of the model. The infected-tracking intervention (policy 3) becomes inactive at about week 140. See Appendix B for details on the parameterization of the model.

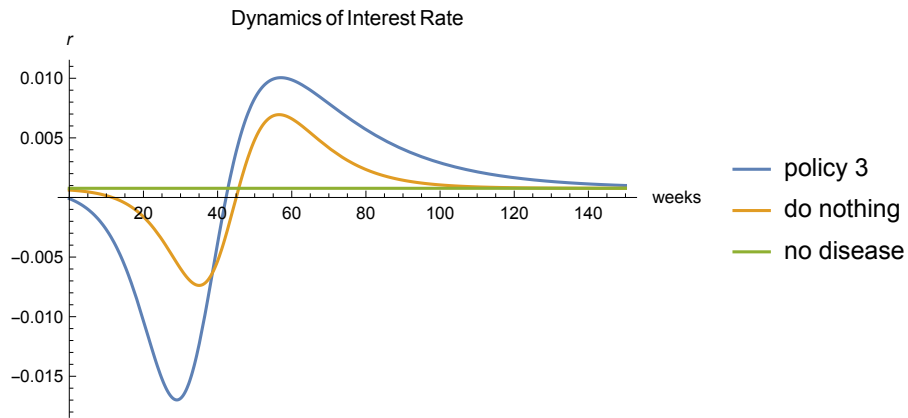
Figure C.1: Epidemiological Dynamics after the Outbreak



(a) State variable:  $x \equiv L/N$



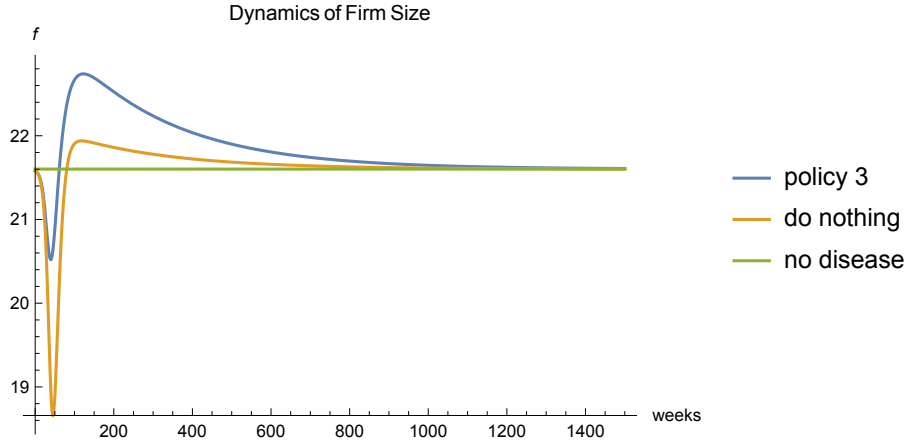
(b) Per capita expenditure:  $y \equiv Y/L$



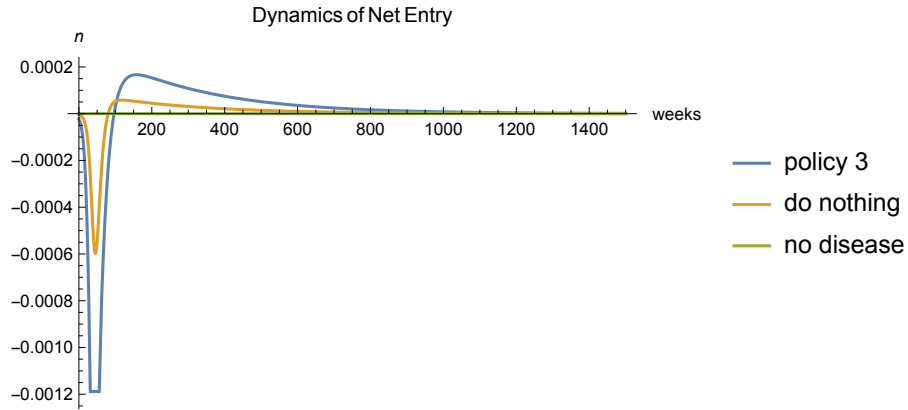
(c) Interest rate:  $r$

*Notes:* The figure shows simulations from a parametrized version of the model. The infected-tracking intervention (policy 3) becomes inactive at about week 140. See Appendix B for details on the parameterization of the model.

Figure C.2: Market Size and Interest Rates



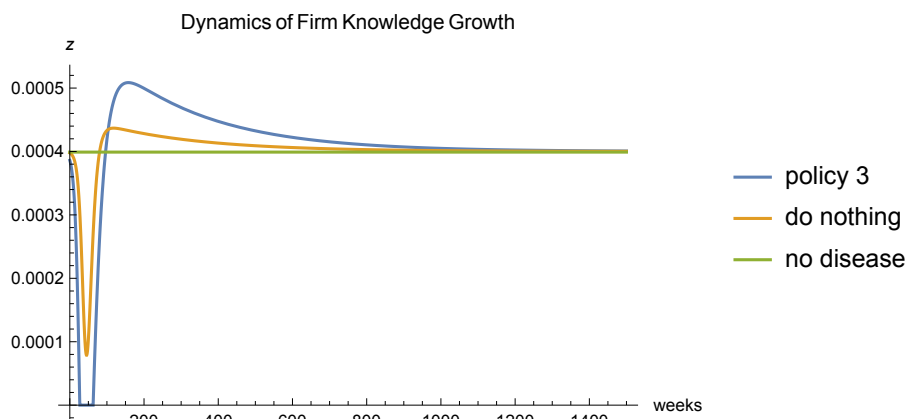
(a) Firm size:  $[1 - \iota(s)] L/N$



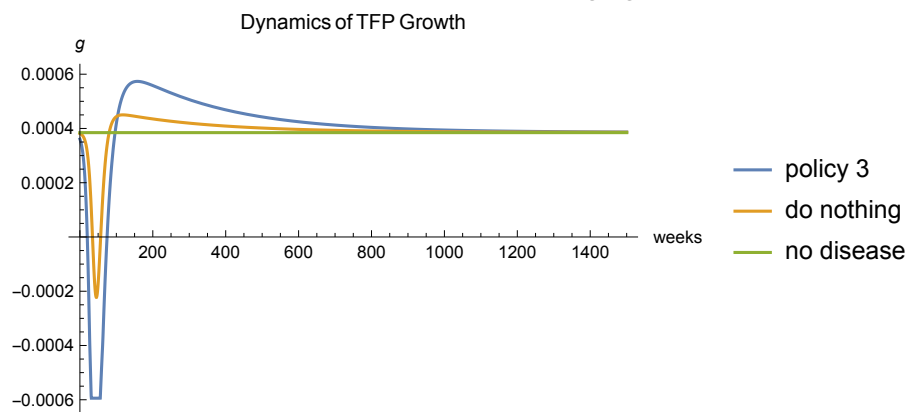
(b) Net entry:  $n \equiv \dot{N}/N$

*Notes:* The figure shows simulations from a parametrized version of the model. The infected-tracking intervention (policy 3) becomes inactive at about week 140. See Appendix B for details on the parameterization of the model.

Figure C.3: Firm Size and Net Entry



(a) Firm knowledge growth:  $z = \dot{Z}/Z$

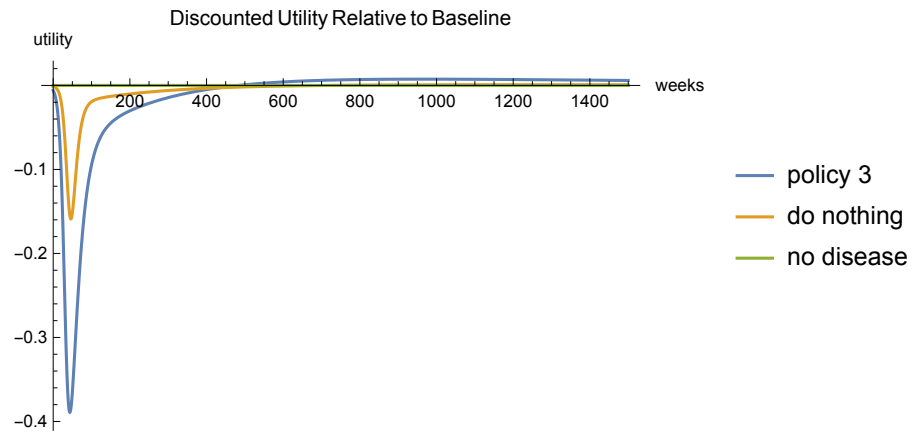


(b) TFP:  $Z^\theta N^{\frac{1}{\epsilon-1}}$

*Notes:* The figure shows simulations from a parametrized version of the model. The infected-tracking intervention (policy 3) becomes inactive at about week 140. See Appendix B for details on the parameterization of the model.

Figure C.4: Knowledge and TFP Growth

Figure C.5: Welfare Relative to Disease-Free Economy



*Notes:* The figure shows simulations from a parametrized version of the model. The infected-tracking intervention (policy 3) becomes inactive at about week 140. See Appendix B for details on the parameterization of the model.