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Research paper

# 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.

## 1. Introduction

The spread of infectious diseases ("epidemics") has ravaged humanity, leaving indelible scars. The Great Influenza Pandemic of 1918–20 and the most recent COVID-19 are two examples of a long list of severe epidemics that led to substantial disruptions in economic 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 and growing literature tackles these questions in the context of models in which an epidemic and the policy response to it affect economic activity through changes in market hours worked, consumption, and physical capital investment. Bloom et al. (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, growing 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 policies that impose restrictions on the ability of people to engage in economic and social interactions like, for example, the lockdowns imposed in many places in response to COVID-19. How and to what extent this initial epidemic shock propagates through the economy depends on the specific characteristics of the economic environment and the mechanisms at play (see Section 2 for a review of the literature).

The objective of this paper is to build a tractable dynamic general equilibrium model to address important, yet overlooked, questions that relate the spread of an epidemic to market structure, innovation, and productivity growth. To achieve this goal, we integrate a standard Susceptible-Infected-Recovered-Deceased (SIRD) model of infectious diseases into a growth model featuring

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variety expansion and firm-specific cost-reducing innovation (Peretto and Connolly, 2007). The 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 steady-state growth rate remains unchanged because we work with a growth model that does not suffer from 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, this relationship can be characterized as a trajectory in the 2D space. If one can solve the equation describing this trajectory, one obtains the infection rate as a function of 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 to integrate 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 aggregate employment and so to the size of the market in which firms operate. The hump-shaped dynamics of 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.[1]

As a proof of concept of 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 lockdown. In our framework, such interventions are state-dependent and involve *policy rules* based on tracking the fraction of the susceptible or of the infected in the population. The policy rules are announced at the time of the outbreak and, crucially, anchor the 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 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 structure of the paper is 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.

## 2. Related literature

This paper adds to the literature that studies the economic effects of infectious diseases. This literature, recently reviewed by Bloom et al. (2022), predates COVID-19 and identifies several pathways through which diseases and health policy interventions affect the economy. The 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 et al. (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 coexists 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 et al. (2022) discuss several more examples of pre-COVID-19 contributions. A recent empirical paper that in spirit is related to ours is Madsen et al. (2022). It studies the extent 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.

[1] 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.

Focusing on COVID-19, the number of contributions studying different aspects of that epidemic has grown at a surprisingly fast rate.[2] Early contributions by Eichenbaum et al. (2020a,b,c) 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 an equilibrium with rational expectations 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 et al. (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.

## 3. Environment

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

The economy is populated by a representative household with $L(t) = e^{\lambda t}$ 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

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

where $C = \left[\int_0^N \left(X_i/L\right)^{\frac{\epsilon-1}{\epsilon}} di\right]^{\frac{\epsilon}{\epsilon-1}}$ is a constant-elasticity-of-substitution (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 the Dixit-Stiglitz price index of consumption.

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

$$r = r_A \equiv \rho + \frac{\dot{Y}}{Y} - \frac{\dot{L}}{L} = \rho + \frac{\dot{Y}}{Y} - \lambda,$$

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

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

Firm $i$ produces $X_i$ units of the differentiated good using $L_{X_i}$ units of labor and the technology $X_i = Z_i^{\theta}\left(L_{X_i} - \phi\right)$, 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 price $p_i$ and R&D labor $L_{Z_i}$ to maximize the value of the firm, $V_i(t) = \int_t^{\infty} e^{-\int_t^s [r(v)+\delta]v} \Pi_i(s) ds$, 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):

$$\dot{N} = \left(\frac{w}{\beta Y} N\right) L_N - \delta N. \tag{1}$$

[2] A partial list includes: Acemoglu et al. (2020), Alvarez et al. (2020), Atkeson (2020), Barro et al. (2020), Bethune and Korinek (2020), Bognanni et al. (2020), Brotherhood et al. (2020), Farboodi et al. (2020), Glover et al. (2020), Jones et al. (2020) and Krueger et al. (2020). See 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.

## 4. No-infection equilibrium dynamics

We now turn to the general equilibrium of the benchmark model without infection. As the equilibrium of the economy is symmetric, 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 numé raire, $w \equiv 1$. The value of the household's portfolio equals the value of securities issued by firms, $A = NV$.[3] Assets market equilibrium requires rates of return equalization, $r = r_A = r_Z = r_N$, where $r_Z$ and $r_N$ are the rates of return to cost reduction and to entry:

$$r = r_Z \equiv \alpha \left[ \frac{Y\sigma\theta(\epsilon-1)}{\epsilon N} - w\frac{L_Z}{N} \right] + \frac{\dot{w}}{w} - \delta; \tag{2}$$

$$r = r_N \equiv \frac{1}{\beta}\left[ \frac{1}{\epsilon} - \frac{N}{Y}\left( \phi + w\frac{L_Z}{N} \right) \right] + \frac{\dot{Y}}{Y} - \frac{\dot{N}}{N} - \delta. \tag{3}$$

These expressions describe the key role of market size, measured by the household's expenditure on differentiated consumption goods, $Y$, as the key driver of the model's impulse-response dynamics. When $Y$ falls as during the epidemic, both $r_z$ and $r_N$ fall, discouraging investment in the creation of new firms and in knowledge accumulation by existing firms.

*No-infection transition dynamics.* Let $y \equiv Y/L$ denote expenditure per capita and $x \equiv L/N$ denote the ratio of the population to the mass of firms. Note that since $L$ equals aggregate employment, $x$ is also "firm size" or employment per firm. When free entry holds, $y$ and $r$ are constant and equal to

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

When free entry does not hold,

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

The model exhibits a block-recursive property that facilitates the characterization and computation of the general equilibrium. Specifically, the model reduces to a differential equation for $x$. Then, equipped with the solution or time path for $x$, all other variables are easily calculated.

**Proposition 1.** *Consider the model without infection. The no-infection equilibrium dynamics of the population-to-firms ratio or firm size follows a piece-wise differential equation:*

$$\dot{x} = \begin{cases} (\delta+\lambda)x & \text{if} \quad \phi \le x \le x_N \\ \frac{1-\beta(\rho-\lambda)}{\beta}\phi - \left(\frac{1}{\beta\epsilon} - \rho - \delta\right)x & \text{if} \quad x_N < x \le x_Z \\ \frac{1-\beta(\rho-\lambda)}{\beta}\left(\phi - \frac{\rho+\delta}{\alpha}\right) - \left[\frac{1-\sigma\theta(\epsilon-1)}{\beta\epsilon} - \rho - \delta\right]x & \text{if} \quad x > x_Z \end{cases}, \tag{4}$$

*where the thresholds $x_N$ and $x_Z$ are*

$$x_N \equiv \frac{[1-\beta(\rho-\lambda)]\epsilon\phi}{1-\beta(\rho-\lambda)\epsilon} < x_Z \equiv \frac{[1-\beta(\rho-\lambda)]\epsilon(\rho+\delta)}{\sigma\alpha\theta(\epsilon-1)}. \tag{5}$$

*Using the equation for the rate of return to cost reduction (2), we obtain the growth rate of the stock of knowledge,*

$$z \equiv \frac{\dot{Z}}{Z} = \alpha\frac{L_Z}{N} = \begin{cases} 0 & \text{if} \quad x \le x_Z \\ x \cdot y\alpha\sigma\theta\left(\frac{\epsilon-1}{\epsilon}\right) - r - \delta & \text{if} \quad x > x_Z \end{cases}. \tag{6}$$

*Similarly, using the equation for the rate of return to entry (3), we obtain the growth rate of the mass of firms,*

$$n \equiv \frac{\dot{N}}{N} = \frac{1}{\beta}\left[\frac{1}{\epsilon} - \frac{1}{xy}\left(\phi + \frac{L_Z}{N}\right)\right] - r + \frac{\dot{y}}{y} - \delta. \tag{7}$$

As illustrated by Fig. 1, transition dynamics features three regions: (i) net firm exit with zero R&D ($\phi \le x \le x_N$); (ii) gross firm entry with zero R&D ($x_N < x \le x_Z$); (iii) gross firm entry with positive R&D ($x > x_Z$). To the left of the threshold $x_N$, firm size $x \le x_N$ is too small and there is no investment in gross entry. As a result, $x$ increases at the exit rate $\delta$, which increases profitability. As firm size $x$ crosses into the region $x_N < x \le x_Z$, new firms enter and the growth rate of firm size slows down. In this region, there is no accumulation of firm knowledge. Finally, to the right of the threshold $x_Z$, firm size is large enough that firm entry and firm knowledge accumulation coexist.

[3] Note that when free entry holds, $NV = \beta Y$, otherwise $NV < \beta Y$.

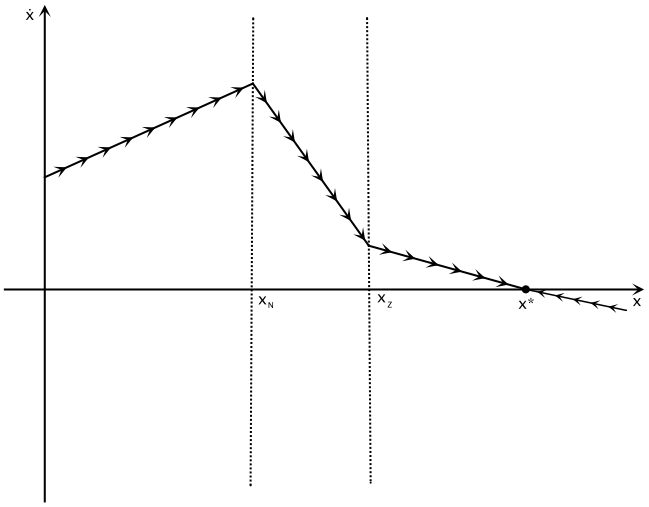



**Fig. 1.** Pre-infection equilibrium dynamics.
*Notes*: The figure depicts the equilibrium dynamics of the population-to-firms ratio as implied by the piece-wise differential equation (4), where the thresholds $x_N$ and $x_Z$ are as defined in (5), and the steady-state value $x^*$ is as in (10).

*No-infection steady state.* The model has a unique steady in which income per capita increases at a constant rate, independent of the size of the population.

**Proposition 2.** *Consider the model without infection. Assume parameters satisfy:*

$$\alpha\phi > \rho + \delta; \tag{8}$$

$$(\rho+\delta)\beta + \frac{\sigma\theta(\epsilon-1)}{\epsilon} < \frac{1}{\epsilon} < (\rho+\delta)\beta + \left(\frac{\alpha\phi}{\rho+\delta}\right)\frac{\sigma\theta(\epsilon-1)}{\epsilon}. \tag{9}$$

*The no-infection steady state values for firm size, $x$, firm knowledge growth, $z \equiv \dot{Z}/Z$, and the mass of firms, $N$, are, respectively:*

$$x^* = \frac{(1-\beta\rho)\epsilon\left(\phi - \frac{\rho+\delta}{\alpha}\right)}{1-\sigma\theta(\epsilon-1)-(\rho+\delta)\beta\epsilon}; \tag{10}$$

$$z^* = \frac{\phi\alpha-(\rho+\delta)}{1-\sigma\theta(\epsilon-1)-(\rho+\delta)\beta\epsilon}\theta(\epsilon-1)-(\rho+\delta); \tag{11}$$

$$N^* = \left[\frac{1-\sigma\theta(\epsilon-1)-(\rho+\delta)\beta\epsilon}{\epsilon\left(\phi-\frac{\rho+\delta}{\alpha}\right)(1-\beta\rho)}\right]L. \tag{12}$$

Note that $x^*$ and $z^*$ are *independent* of 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 $L$. Due to this property, the model does not suffer from the strong scale effect at work in first-generation models of endogenous growth à la Romer (1990). We stress that the proportionality of the mass of firms and population level is a steady-state phenomenon. The richer relation characterized by (4) holds when the economy is in transition dynamics.

Given the formula for the price index $p_C$, consumption per capita is

$$C = \frac{y^*}{p_C} = \frac{y^*}{c^*}\cdot Z^\theta N^{\frac{1}{\epsilon-1}},$$

where $c^* = \frac{\epsilon}{\epsilon-1}\cdot w$ captures the static drivers of the price index, i.e., those unrelated to endogenous technological change. (Recall the normalization $w = 1$.) Accordingly, the growth rate is $\left(\dot{C}/C\right)^* = \theta z^* + \frac{n^*}{\epsilon-1} = \theta z^* + \frac{\lambda}{\epsilon-1}$.

## 5. Equilibrium dynamics with infection

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.

### 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:

$$\dot{S} = \xi_B L - \xi_M S - \frac{\xi_S I S}{L}; \tag{13}$$

$$\dot{I} = \frac{\xi_S I S}{L} - \xi_R I - \xi_M I - \xi_D I; \tag{14}$$

$$\dot{R} = \xi_R I - \xi_M R; \tag{15}$$

$$\dot{D} = \xi_D I; \tag{16}$$

$$\dot{L} = \xi_B L - \xi_M (S + I + R) - \xi_D I. \tag{17}$$

Here, $\xi_B$ is the birth rate (all newborn are $S$), $\xi_M$ is the non-disease death rate, $\xi_S$ is the transmission rate, $\xi_R$ is the recovery rate, $\xi_D$ is the fatality rate, and $S$, $I$, $R$ and $D$ are the mass of susceptible, infected, recovered, and deceased, respectively. Note that in the economic model presented in the previous section $\xi_B - \xi_M = \lambda$.

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

$$\frac{\dot{s}}{s} = \xi_B \left(\frac{1}{s} - 1\right) - \left(\xi_S - \xi_D\right) \imath; \tag{18}$$

$$\frac{\dot{i}}{\imath} = \xi_S s - \xi_R - \xi_B - \xi_D (1 - \imath); \tag{19}$$

$$\frac{\dot{L}}{L} = \xi_B - \xi_M - \xi_D \imath. \tag{20}$$

This system is simpler than it looks: (18)–(19) describe disease dynamics in a self-contained, autonomous way, while Eq. (20) describes the implied population dynamics.

It is important to note that epidemiologists have studied the dynamical system (13)–(17), and its lower-dimensional version (18)–(20), 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 $\xi_B = \xi_M = 0$. 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. Further inspecting the system (18)–(20), moreover, one can see that the first two equations are self-contained. This is the key to our concept of infection function.

*Infection function.* Figs. 2(a)–2(b) depict the phase diagrams for the SIRD and SIR model, respectively. In each case, given the initial state $(s_0, \imath_0)$, with $\imath_0 \approx 0$ and $s_0 \approx 1$, there exists a hump-shaped trajectory $\imath(s)$ in $(s, i)$ space that describes the progression of the epidemic. If one can compute the equation for such trajectory, one has an equation describing the infection rate, $i$, as a function of the susceptible rate, $s$. We interpret this mathematical object as the *"infection function"* mapping $s$ into $i$ at any point in time.

We leverage this concept of infection function to integrate epidemiology into an economic model. Specifically, we use the function $\imath(s)$ to compress the epidemic model to *one* equation:

$$\frac{\dot{s}}{s} = \xi_B \left(\frac{1}{s} - 1\right) - \left(\xi_S - \xi_D\right) \imath(s). \tag{21}$$

In macroeconomics jargon, this Eq. (21) represents the law of motion of the "epidemic shock" that we feed to our economy. However, we can do even better than this, because we can solve for the function $\imath(s)$ by taking the ratio of Eqs. (18) and (19) to obtain the partial differential equation (PDE),

$$\frac{d\imath}{ds} = \frac{\xi_S s - \xi_R - \xi_D - \xi_B + \xi_D \imath}{\xi_B \left(\frac{1}{s} - 1\right) - \left(\xi_S - \xi_D\right) \imath} \left(\frac{\imath}{s}\right). \tag{22}$$

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 for translating the epidemiology model into a tractable component of an integrated epidemic-economy model. As mentioned above, we look at two cases.

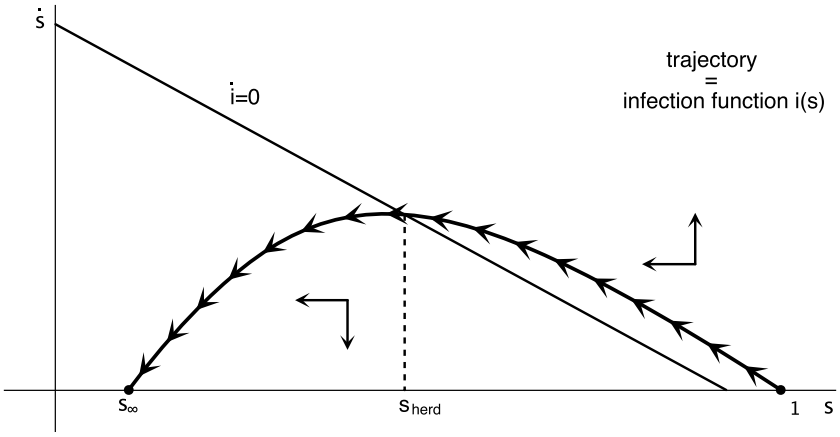
*Infection function: SIRD.* For $\xi_B = \xi_M = 0$, the PDE (22) reduces to the d'Alembert's equation

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

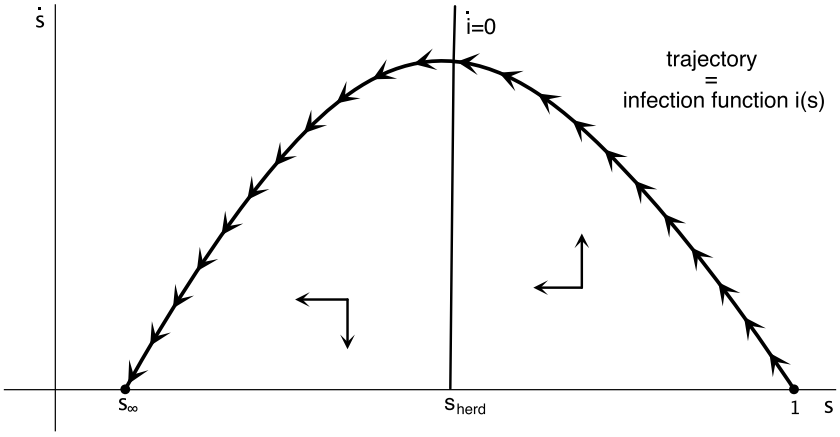
Solving the PDE with the boundary conditions $\imath(0) = \imath_0$ and $s(0) = 1 - \imath_0 \equiv s_0$ yields the infection function

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

which is a hump-shaped function with $s_\infty \equiv \arg\text{solve}\{\imath(s) = 0\} > 0$. Fig. 3(a) shows the infection function for the SIRD model. The epidemic shock that hits the economy then is simply a jump of $s$ from 1 to the initial value $s_0 < 1$ followed by convergence of $s$ to the long-run value $s_\infty$ according to the law of motion $\frac{\dot{s}}{s} = -\left(\xi_S - \xi_D\right) \imath(s)$.




(a) SIRD model




(b) SIR model

**Fig. 2.** Epidemiological models of infectious diseases.
*Notes*: The figures shows the phase diagram for the SIRD model (top panel) and for the SIR model (bottom panel).

*Infection function: SIR.* For $\xi_B = \xi_M = \xi_D = 0$ (i.e., constant population), the PDE (22) reduces to

$$\frac{d\imath}{ds} = \frac{1}{R_0 S} - 1, \quad R_0 \equiv \frac{\xi_S}{\xi_R}.$$

Solving the PDE with boundary conditions $\imath(0) = \imath_0$ and $s(0) = 1 - \imath_0 \equiv s_0$ yields the infection function

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

which is a hump-shaped function with $s_\infty \equiv \arg\text{solve}\{\imath(s) = 0\} > 0$. Fig. 3(b) shows the infection function for the SIR model. The epidemic shock that hits the economy is as in the SIRD case with the only difference that the law of motion of $s$ has the simpler form $\dot{s}/s = -\xi_S \imath(s)$.

### 5.2. Embedding the SIRD model into the growth model

We now embed the SIRD model without vital dynamics ($\xi_B = \xi_M = 0$, which also yields $\lambda = 0$) 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, $\dot{L}/L = -\xi_D \imath(s)$.

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

$$\underbrace{L_X + L_Z + L_N}_{\text{labor demand (allocation)}} = \underbrace{[1 - \imath(s)]\, L.}_{\text{labor supply (participation)}}$$

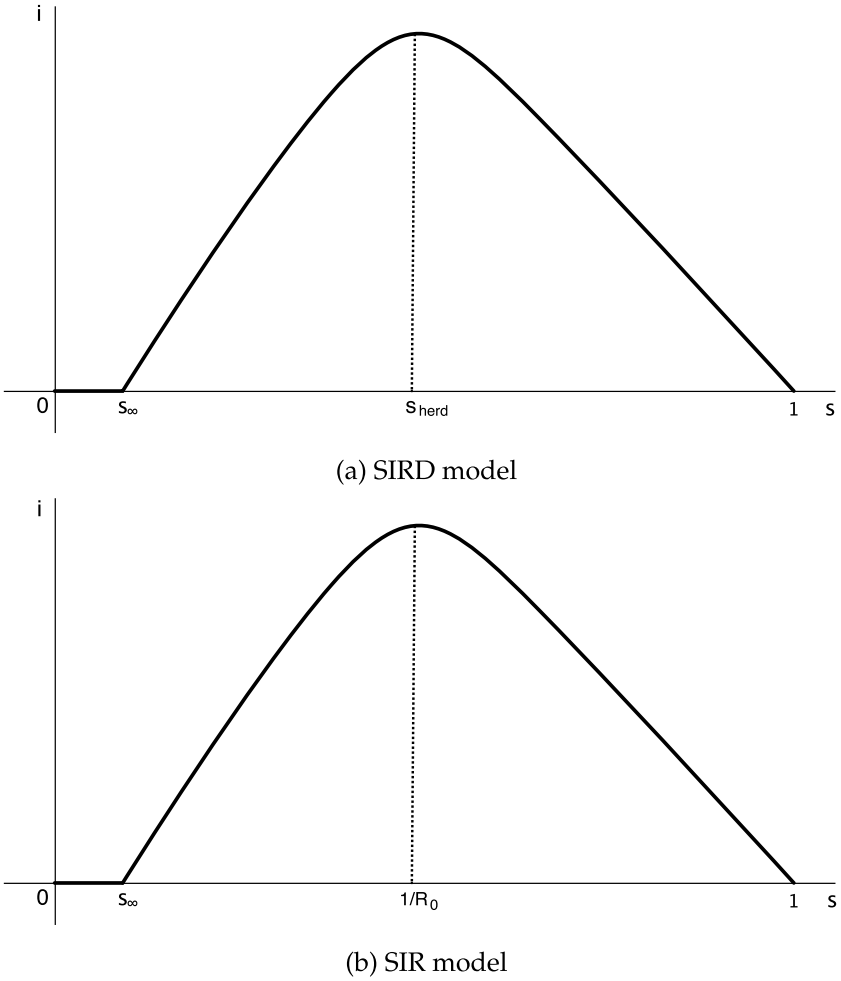



**Fig. 3.** Infection function. *Notes*: The figures depicts the infection function for the SIRD model (top panel) and for the SIR model (bottom panel).

This component is arguably the main cause of economic concern: the epidemic reduces the ability of people to work and interact. Interventions that further restrict people's participation in economic and social activities can exacerbate this recessionary effect.

Third, through the mortality rate $\xi_D$, the epidemic directly affects the *discount rate* of the utility flows, $L(t)\,u(t) = e^{-\int_0^t \xi_D \iota(v)dv} \log C$. As in 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, and therefore I discount the future for such risk.

### 5.2.1. Expenditures and interest rates

Consumption expenditure per capita is decreasing in the population share of infected, $\iota$. In general equilibrium, an epidemic also manifests itself as a reduction in market size. Through this channel, it alters incentives to enter the market and to perform in-house R&D, initiating transition dynamics.

When free entry does not hold, per capita expenditure is

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

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

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

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

Log-differentiating the expression for $y$ yields

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

Next, using the Euler equation and the expression for $i$ gives us

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

The interest rate is below $\rho$ and decreases in $\iota$ when the term in square brackets on the right-hand side $[\cdot]\equiv\left[\xi_S s-\xi_R-\xi_D+\xi_D\iota(s)\right]$ is greater than zero; and it is above $\rho$ and increases 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-shaped and then U-shaped in the fraction of susceptible, $s$. Since initially $[\cdot]>0$, the outbreak results in a fall in $r$ at $t=0$. As $s$ falls throughout, the interest rate continues to fall, it turns around at the peak of the epidemic, overshoots and finally returns to $\rho$ from above.[4]

### 5.2.2. Market structure, productivity, and welfare

Using the expressions for $y(s)$ and $r(s)$, the general-equilibrium dynamics reduces to a system of two differential equations: (i) the first equation describes the evolution of the model's economic state variable, 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, $s$. As stated, in the jargon of macroeconomics the second equation describes the epidemiological shock or driving force that we feed to the economy. The first equation then describes how economic forces propagate such epidemiological shock.

*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:

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

This drop in expenditure starts the transition dynamics, during which the economic state variable follows $\dot{x}/x=-\xi_D\iota-n$, with initial condition $x_0=L_0/N_0\geq 0$ and $n\equiv\dot{N}/N$ denoting the growth rate of the mass of firms.

**Proposition 3.** *Consider the model with infection. The equilibrium 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.*** *The reduction in expenditures is large enough that the economy hits the two corner solutions with no firm innovation and no firm entry, $L_Z=0$ and $L_N=0$. The boundary of this region in the $x$-dimension is*

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

*The dynamical system is:*

$$\frac{\dot{s}}{s} = -\left(\xi_S+\xi_D\right)\iota(s); \tag{23}$$

$$\frac{\dot{x}}{x} = \delta-\xi_D\iota(s). \tag{24}$$

*The underlying net exit process is $n=-\delta\leq 0$.*

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

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

*The dynamical system is:*

$$\frac{\dot{s}}{s} = -\left(\xi_S+\xi_D\right)\iota(s); \tag{25}$$

$$\frac{\dot{x}}{x} = \frac{\phi}{\beta y(s)}\cdot\frac{1}{x}-\frac{1}{\beta\epsilon}+\rho+\delta. \tag{26}$$

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

[4] To visualize some of the qualitative features of the transition dynamics, Appendix Figs. A.1 and A.2 show per capita expenditures and interest rate dynamics as a function of the fraction of susceptible and time, respectively.

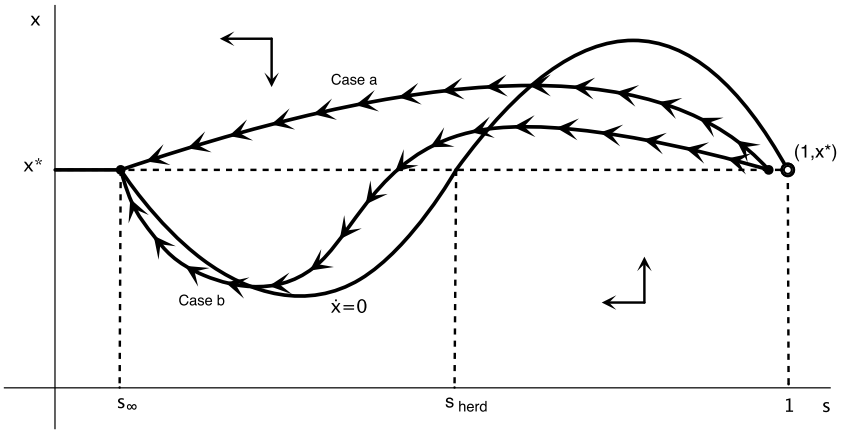



**Fig. 4.** Post-infection dynamics.
*Notes*: The figure depicts the phase diagram for the post-infection economy. On the $x$-axis, $s$ denotes the fraction of susceptible in the population; on the $y$-axis, $x$ denotes the ratio of the population to the mass of firms.

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

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

*The dynamical system is:*

$$\frac{\dot{s}}{s} = -\left(\xi_S+\xi_D\right)\iota(s); \tag{27}$$

$$\frac{\dot{x}}{x} = \frac{1}{\beta y(s)}\left(\phi - \frac{r(s)+\delta}{\alpha}\right)\frac{1}{x} - \frac{1-\sigma\theta(\epsilon-1)}{\beta\epsilon} + \rho + \delta. \tag{28}$$

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

*R&D labor allocation and TFP.* To provide some insight into the transition dynamics produced by the model, Fig. 4 illustrates two potential trajectories in $(s,x)$ space. The population-to-firms ratio, $x$, grows initially because of net exit due to falling profitability caused by falling expenditure. Both expenditure, $y$, and the interest rate, $r$, 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 steady-state level. Possibly, undergoing a phase with zero firm growth.[5]

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

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

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

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

Importantly, in calculating the welfare integral, effective discounting accounts for the mortality due to the spread of the epidemics through the factor $\exp\left\{-\rho t - \int_0^t \xi_D \iota(s(v))\,dv\right\}$.

## 6. Policy interventions

We now study policy interventions that operate through a reduction in the transmission rate of the epidemic engineered via restrictions on the people's ability to supply labor. In the model, this replicates the main effect of a lockdown. We follow the

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

textbook representation of this class of interventions. Specifically, we let $h \geq 0$ denote the policy variable of interest that captures in reduced-form the intervention's intensity. We then model the immediate cost and benefit of the intervention as follows:

$$\text{benefit:} \quad \frac{\xi_S}{1+h}\iota(s)S; \tag{29}$$

$$\text{cost:} \quad \frac{L}{1+h}\left[1-\iota(s)\right]. \tag{30}$$

The intervention's intensity parameter, $h$, reduces the transmission rate of the disease.[6] That is, the new transmission rate, $\xi_S/(1+h)$, equals the old transmission rate, $\xi_S$, in (13)–(14) divided by $1+h$. In addition, it introduces a wedge between the endowment of labor services, $L$, and the effective supply of labor, $L/(1+h)$.[7]

In terms of utility $u(t) = \log C(t)$, interventions of the type described by (29)–(30) have a (negative) direct effect through the term $-\log(1+h)$, which captures the interventions' the direct adverse effect on total labor supply and the indirect effects that work through general equilibrium forces that we characterize below. At any point in time, utility is

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

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

### 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. The first thing to notice is that we must recompute the infection functions to take into account that the intervention changes the transmission rate of the disease to $\xi_S/(1+h)$:

$$\text{SIRD:} \quad \frac{d\iota}{ds} = \frac{1}{s}; \tag{31}$$

$$\text{SIR:} \quad \frac{d\iota}{ds} = \frac{1+h}{R_0 s} - 1. \tag{32}$$

Solving (31)–(32) with boundary conditions $\iota(0) = \iota_0$ and $s(0) = 1-\iota_0 \equiv s_0$ yields the new infection functions:

$$\text{SIRD:} \quad \iota(s) = \frac{\xi_R}{\xi_D}\left[1-\left(\frac{s}{s_0}\right)^{-\frac{\xi_D}{\xi_S/(1+h)-\xi_D}}\right] + 1 - s; \tag{33}$$

$$\text{SIR:} \quad \iota(s) = \frac{1+h}{R_0}\log\left(\frac{s}{s_0}\right) + 1 - s. \tag{34}$$

Again, there are two cases.

When free entry does not hold, per capita expenditure is

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

When free entry holds, per capita expenditure is

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

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

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

[6] Allowing for endogenous labor supply, i.e., the representative household choosing the fraction of non-infected household members working, is possible, but at the cost of losing tractability without adding any substantive insight into the propagation mechanism of the epidemics. Moreover, insofar as the labor supply constraint implied by the lockdown binds, arguably the empirically relevant case, the constrained employment dynamics would naturally follow those of the policy variable, $h$.

[7] 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.

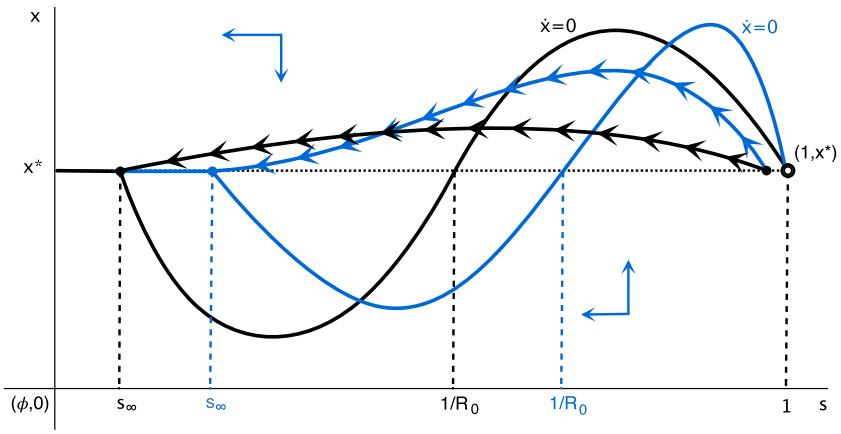



**Fig. 5.** Post-infection SIR dynamics with policy intervention.
*Notes*: The figure depicts the equilibrium dynamics of the population-to-firms ratio for the post-infection SIR economy with (blue lines) and without (black lines) policy intervention. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

The first term on the right-hand side captures the negative direct effect that the policy exercises on 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:

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

Next we look at the interest rate. Log-differentiating $y(s)$ and using the Euler equation, yields

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

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.

Fig. 5 shows the phase diagram of the dynamical system for the SIR case, with (blue lines) and without (black lines) policy intervention. Under the 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 the 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 the epidemiological literature 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.

### 6.2. A state-dependent wedge approach

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

#### 6.2.1. Tracking the susceptible

*Intervention policy #2.* We start with the policy based on tracking the susceptible, i.e., $h(s)$. To simplify the analysis, we set $\xi_D = 0$ (SIR model). To keep things tractable, we consider a simple policy rule of the form

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

This rule has the property that the intervention relaxes as $s$ falls and vanishes at the target $\bar{s}$. A few remarks are in order. First, let $\bar{s} = 0$. Then: (i) we rule out $\mu = 1$ because it implies the total shutdown of the economy at $s_0 \approx 1$; (ii) we interpret $\mu$ as the upper bound on the restrictions, e.g., $\mu = 0.2$ implies that initially $1/(1+0.2) = 83\%$ of the healthy work; (iii) the parameter $\eta$ regulates the sensitivity of the restrictions to the state variable $s$. Second, let $\bar{s} > 0$. Then there is the endogenous termination of the intervention at the policy target $\bar{s}$. The target can be consistent with the achievement of natural herd immunity, $s_{herd} = 1/R_0$, with the natural terminal state, $s = s_\infty$, or with the development of a vaccine at some expected date. The rationale for the last example is that the solution of the epidemiological component of the model yields the invertible function $s(t)$ and therefore allows us to compute $\bar{s} = s^{-1}(t_v)$, where $t_v$ is the time at which the vaccine becomes available for mass administration to the population and/or it halts the progression of the disease.

As stated, we need to recompute the infection function. We show in Appendix A that solving the PDE problem with initial condition $(s_0, \iota_0)$, and verifying continuity (value matching) and smooth-pasting at $s = \bar{s}$, yields the new infection function, which takes into account the rule-based policy response to the epidemiological dynamics of the susceptible:

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

This expression 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

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

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 $s_\infty^{int} \lesseqgtr s_\infty$ for $\bar{s}^\eta \log\bar{s} + \frac{1}{\eta}(1-\bar{s}^\eta) \lesseqgtr 0$. To get $s_\infty^{int} = s_\infty$, set $\bar{s}$ such that $\bar{s}^\eta \log\bar{s} + \frac{1}{\eta}(1-\bar{s}^\eta) = 0$.

*Per capita expenditure and the interest rate.* When free entry does not hold, per capita expenditure is

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

When free entry holds, it is

$$y(s) = \frac{1}{1-\rho\beta}\frac{1-\iota(s)}{1+h(s)}.$$

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

$$r(s) = \rho - \frac{1 - (\rho + \xi_D)\beta}{\left[1 - \rho + \xi_D \iota(s)\right]\beta}\frac{\iota(s)}{1-\iota(s)}\xi_R\left(R_0 s - 1\right) - \frac{h'(s)\dot{s}}{(1+h(s))^2}.$$

Setting $\xi_D = 0$ and using the expression $\dot{s}/s = -\xi_S \iota(s)$, we then obtain

$$r(s) = \rho - \frac{\iota(s)}{1-\iota(s)}\xi_R\left(R_0 s - 1\right) + \frac{h'(s)s}{(1+h(s))^2}\xi_S\left[1 - s + \frac{1-\mu\bar{s}^\eta}{R_0}\log\left(\frac{s}{s_0}\right) + \frac{\mu}{\eta R_0}\left(s^\eta - s_0^\eta\right)\right]. \tag{35}$$

These expressions make the model's short-run propagation channel analytically transparent because they describe expenditure per capita and the interest rate as simple functions of the epidemiological state variable $s$ (see Fig. 6).

### 6.2.2. Tracking the infected

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

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

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

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

Note the infection-tracking case, $h(\iota)$, is much simpler than the susceptible-tracking case, $h(s)$, because there is no need to construct the new infection function piece by piece. The epidemic fed to the economy takes the same form $\dot{s}/s = -\xi_S \iota(s)$. Qualitatively, the

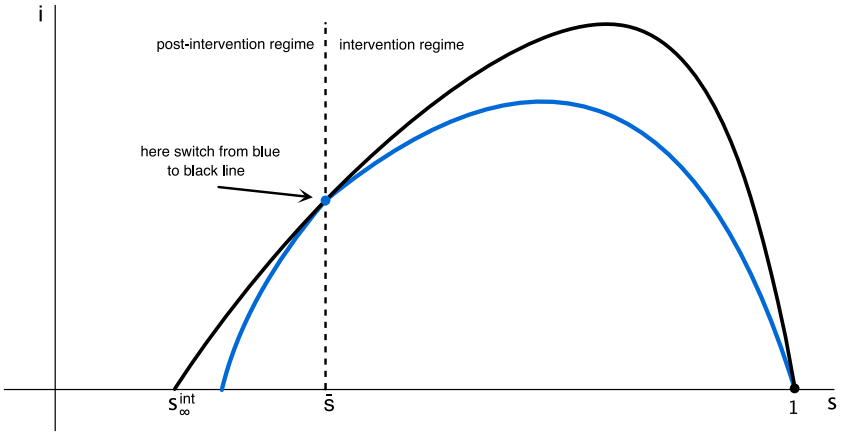



**Fig. 6.** Post-infection dynamics with state-dependent policy intervention.
*Notes*: The figure depicts the phase diagram for the post-infection economy with state-dependent policy intervention. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

intervention policy that tracks the infected delivers a similar phase diagram; the difference is that there is only one regime since by construction the intervention lasts as long as $\iota > 0$.[8]

## 7. A numerical illustration

To further illustrate how an epidemic affects the economy, we parameterize the model and simulate equilibrium paths of four 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 of the disease is left to running its own course; (iii) the state-dependent policy that tracks the susceptible (Intervention Policy #2 or IP2); (iv) the state-dependent policy that tracks the infected (Intervention Policy #3 or IP3). Intervention policies go inactive at about week 75.[9]

The parameter values for the SIR model are from the by now established literature on COVID-19.[10] We stress that we use COVID-19 as just an example, prominent because of the events of the last few years, useful to build our proof of concept exercise. The values for the transmission and recovery rates imply $R_0 \equiv \xi_S/\xi_R = 0.301/0.155 \approx 1.94$. This value is broadly consistent with the evidence. For example, Riou and Althaus (2020) reports a point estimate of 2.2 with a 90 percent confidence interval of 1.4 to 3.8. See Table 1 for our baseline parameter values.

### 7.1. Takeaways

Two main lessons emerge from the numerical analysis. First, market size is a powerful propagation mechanism of the epidemic. The endogenous decrease in the mass of firms in response to the epidemic shock considerably amplifies the epidemic's recessionary effect on economic activity. Specifically, the epidemic, as well as the associated intervention policies that reduce the effective labor supply by restricting the ability of people to engage in economic activity and social interactions, are akin to a negative labor supply shock that causes a sharp fall in consumption expenditure and a sharp increase in the interest rate relative to the no-disease scenario. In response to these events, the incentives to set up new firms weaken drastically and there is a dramatic drop in net firm entry. Likewise, the incentives to invest in-house to improve the technology of the firm weaken drastically and there is a dramatic drop in firm knowledge growth. These dynamics propagate over time the recessionary effects of the epidemics, and they do so very slowly. Indeed we find that the macroeconomic dynamics are much, much slower than the epidemiological dynamics due to the sluggishness of the mass of firms. The latter plays out over about 100–120 weeks, as documented by all parametrizations in the literature of the pure SIR/SIRD model that does not have a slow-moving economic state variable, while the former plays out over several decades. In other words, we find that the time horizon of the adjustment of the economic block of the model is almost ten times longer than the time horizon of the adjustment of the epidemiological block of the model.

Second, for plausible parameterizations of the model, we find that the susceptible-tracking intervention policy (IP2) unambiguously worsens welfare. Welfare with intervention is lower than the do-nothing scenario during the transition to the new steady state.

[8] Note that the analysis can be extended to intervention stopping at target $\bar{\iota} > 0$.

[9] We refer the reader to Appendix B for details on the parameterization of the model. 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.

[10] A useful ready-to-use repository of estimates is https://www.mathworks.com/matlabcentral/fileexchange/74658-fitviruscovid19.

**Table 1**
Parameterization.

| Parameter | Description | Value |
|---|---|---|
| A. Preferences & technology | | |
| $\rho$ | 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 |
| B. SIR model | | |
| $\xi_S$ | Transmission rate | 0.301 |
| $\xi_R$ | Recovery rate | 0.155 |
| $S_0$ | Susceptible at outbreak | 0.999 |
| C. Intervention policy | | |
| $\bar{s}$ | 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.

This negative outcome is not hard-wired into the model. To see this, note that the infected-tracking intervention policy (IP3) does worse than the do-nothing scenario in the first 400 weeks but raises welfare relative to the do-nothing over the longer run. Thus, whether an intervention policy raises or reduces welfare depends critically on the values of the coefficients of the policy rule in place that determine the stringency and duration of the intervention, and whether the costs of the policy are more or less 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 at the onset of the epidemic and decreases throughout its course. Interventions tied to it, therefore, concentrate their effects at the beginning of the epidemic, exacerbating the initial 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.

### *7.2. Epidemiological dynamics*

We now turn to our detailed results. We begin by discussing the numerical results related to the epidemiological evolution of the disease. Fig. 7(a) and Appendix Fig. C.1(a) show the infection functions resulting from the parametrized model for the IP2 and IP3 cases, respectively. A key insight stands out: the policy interventions yield an across-the-board downward shift in the infection function so that there are fewer infected per susceptible in the population throughout the course of the epidemic.

As evident in Figs. 7(b)–7(c) and Appendix Figs. C.1(b)–C.1(c), such a shift slows down 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. As highlighted in the literature, however, interventions of this type prolong the duration of the epidemic precisely because they "flatten the curve", namely, they push down in $(s, i)$ space the trajectory of the epidemic, which is exactly what our concept of infection function captures analytically.

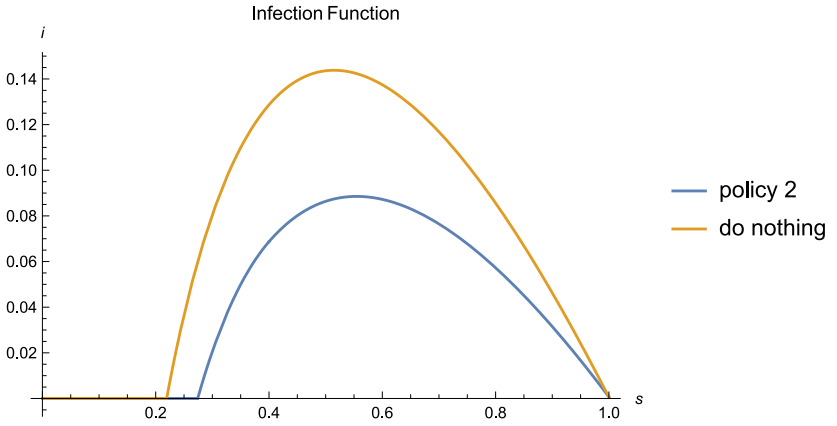
To summarize: (i) the time path of the infection rate remains hump-shaped under the policy intervention; (ii) the peak of the infection rate is smaller in magnitude and occurs later; (iii) the end of the epidemic is delayed.

### *7.3. Macroeconomic dynamics*

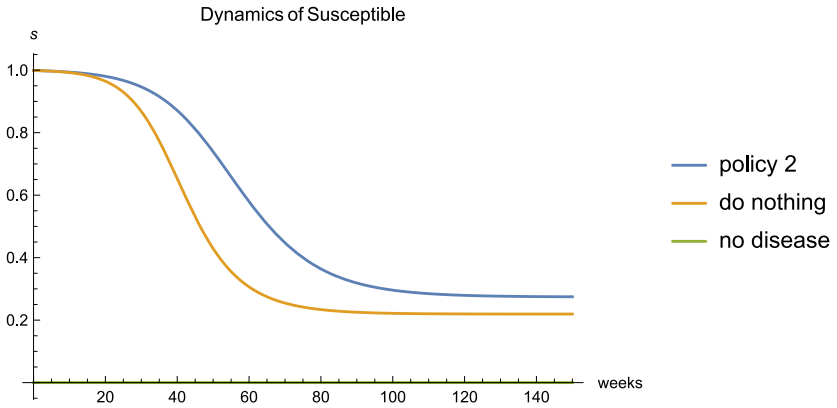
As stated above, in the model the transition dynamics of economic variables lasts considerably longer than the epidemiological dynamics. For our baseline parameterization, while the epidemic runs its natural course by approximately week 120, the dynamics of the economic state variable, i.e., the population-to-firm ratio, $x \equiv L/N$, 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, therefore, epidemiological and economic variables move at very different speeds.

Figs. 8(a)–8(c) and Appendix Figs. C.2(a)–C.2(c) show simulated time paths for the population-to-firms ratio, per capita expenditure, and the interest rate under IP2 and IP3, respectively. The last two variables are fast-adjusting (i.e., jumping variables) whose dynamics are almost entirely dictated by the evolution of the epidemiological state variable, $s$. The population-to-firms ratio, in contrast, is a slow-adjusting variable (i.e., a pre-determined variable) whose dynamics are mostly governed by its own internal response to the anticipated evolution of market size and the interest rate. This response features a pronounced hump-shaped time profile that is very sensitive to the intervention policy in place.

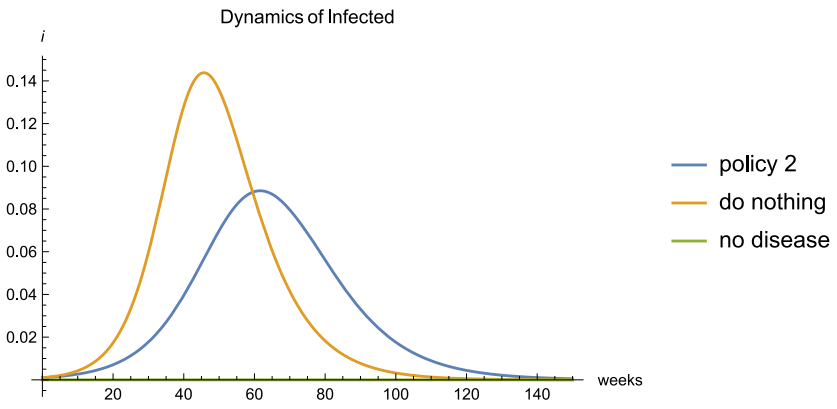
Starting with the case of susceptible-tracking policy intervention (IP2), the economy experiences an abrupt drop in per capita expenditure and a sharp rise in the interest rate, with slow reversion to their steady-state values (Figs. 8(b)–8(c)). The associated




(a) Fraction of infected as a function of fraction of susceptible: $\imath(s)$




(b) Fraction of susceptible in population: $s(t)$




(c) Fraction of infected in population: $\imath(t)$

**Fig. 7.** Epidemiological dynamics after the Outbreak—Susceptible-Tracking policy.
*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.

rise in the population-to-firms ratio in Fig. 8(a) is considerably larger relative to the do-nothing scenario, meaning that the rise in the firm net *exit* rate is quite large. The peak in the population-to-firms ratio occurs when the epidemic and the policy intervention run out (about week 120). This pattern subsumes an important lesson: after these two joint events, the dynamics of the effective labor supply and so market size and the interest rate are back to normal, restoring the incentives driving our Schumpeterian growth model. Hence, the post-epidemic dynamics is solely due to the endogenous market structure dynamics of the economic block of our model, a process that is notoriously very slow.

The main differences between susceptible- versus infected-tracking interventions, i.e., IP2 versus IP3, are in the paths of per capita expenditures and so the interest rate. In Fig. 8(c), the interest rate dynamics under IP2 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 IP2 scenario instead, the interest rate remains above its steady-state level along the entire transition. The interest rate jumps down when the intervention ends because of the associated kink in the time profile of expenditure, which is due to the forward-looking behavior with perfect foresight of the agents populating our macroeconomic model. By contrast, the interest rate dynamics under IP3 in Appendix Fig. C.2(c) is qualitatively similar to the do-nothing scenario. Such a result derives from the expenditures dynamics under IP3 being qualitatively similar to those of the do-nothing scenario (see Fig. 8(b) versus Appendix Fig. C.2(b)).

Figs. 9(a)–9(b) show transition dynamics for firm size, defined as $f \equiv [1-\iota(s)]\,L/N$, and net firm entry, denoted $n \equiv \dot{N}/N$. Firm size, $f$, and the population-to-firms ratio, $x \equiv L/N$, differ during the course of the epidemic because of the "epidemic wedge" $\iota(s) > 0$ that reduces the effective labor supply. 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 larger due to the larger drop in expenditures relative to the do-nothing scenario. Appendix Figs. C.3(a) and C.3(b) show simulated paths for firm size and net entry under infected-tracking policy intervention.

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 the rate of firm knowledge growth and $n$ is again the growth rate of the mass of firms. Fig. 10(a) shows that under IP2 the economy hits the second corner solution as well, zero R&D labor, which causes a halt in firm knowledge accumulation.[11] As a result, the economy experiences a period of negative TFP growth (see Fig. 10(b)). Net entry and TFP growth resume when the epidemic runs out (about week 140 in our simulations). Similar dynamics arise under IP3 in Appendix Figs. C.4(a) and C.4(b).

### 7.4. Welfare effect of policy intervention

An important feature of the simple policy rules of the kind we propose, is that they are amenable to welfare evaluation, accounting for transition dynamics. Fig. 11 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. As evident from the figure, for our baseline parameterization, 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 Appendix Fig. C.5). The rules that we consider are simple but rather crude and this is likely the reason 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. Thus, it front-loads the economic damage caused by the loss of employment. The second policy, in contrast, tracks the infection rate and thus its intensity follows a hump-shaped profile that concentrates the loss of employment around the peak of the epidemic.

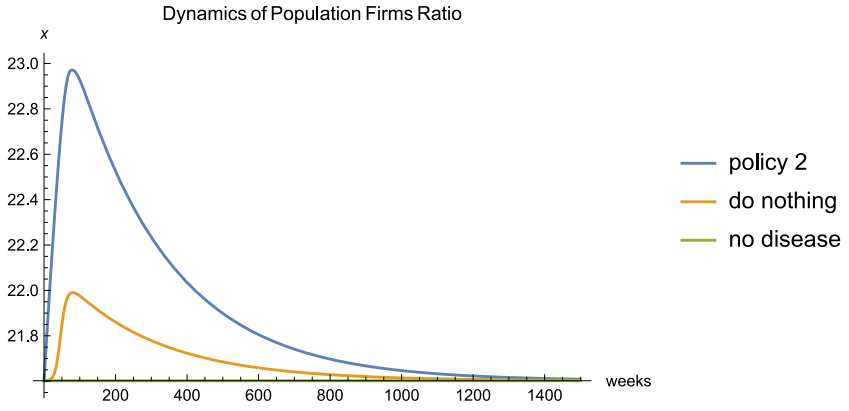
Overall, the message that we extract from these exercises is that crude policies can be very damaging and that minimizing the damage requires careful examination of the channels through which policies operate.

## 8. Conclusion

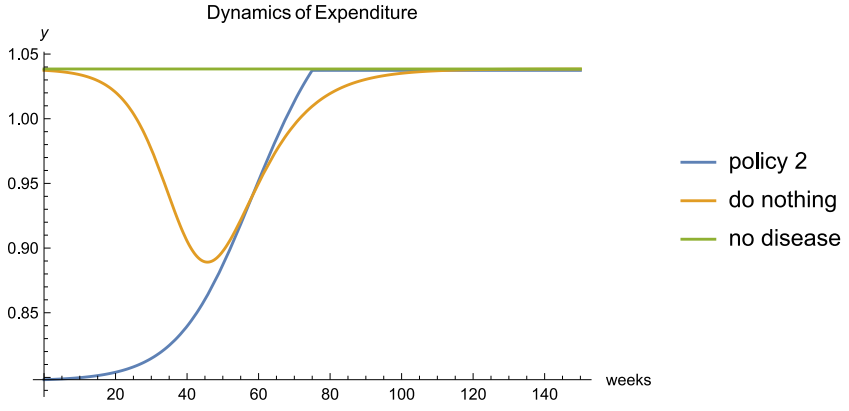
We develop a dynamic general equilibrium model for the analysis of the economic effects of an epidemic. The model combines the standard epidemiological SIRD model with key features of Schumpeterian endogenous growth theory, such as endogenous firm entry, which expands product variety, and cost-reducing innovation. Transition dynamics is analytically tractable and governed 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 large 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,

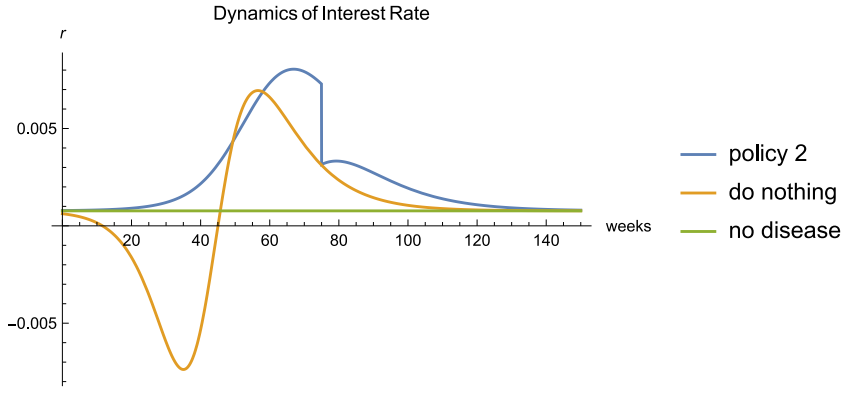
[11] If one assumed a positive depreciation rate of firm knowledge, transition dynamics would generate negative firm knowledge growth.




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

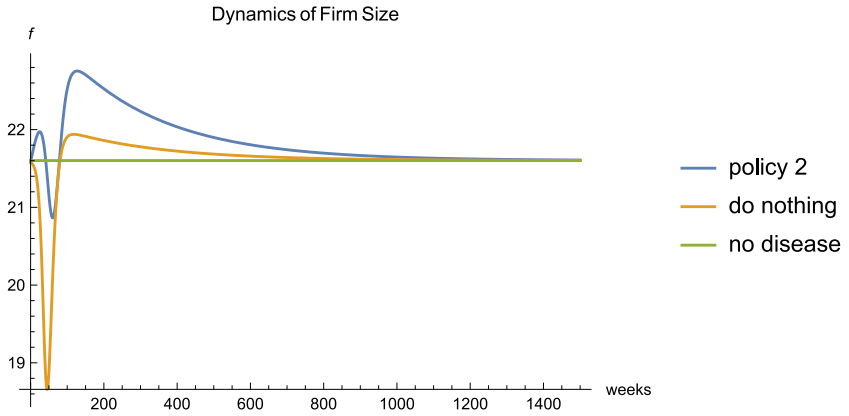



(b) Per capita expenditure: $y \equiv Y/L$

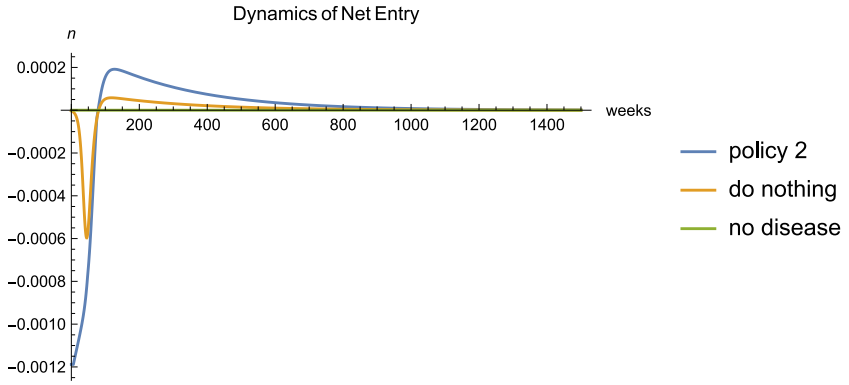



(c) Interest rate: $r$

**Fig. 8.** Market size and interest rates—Susceptible-Tracking policy.
*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.




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




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

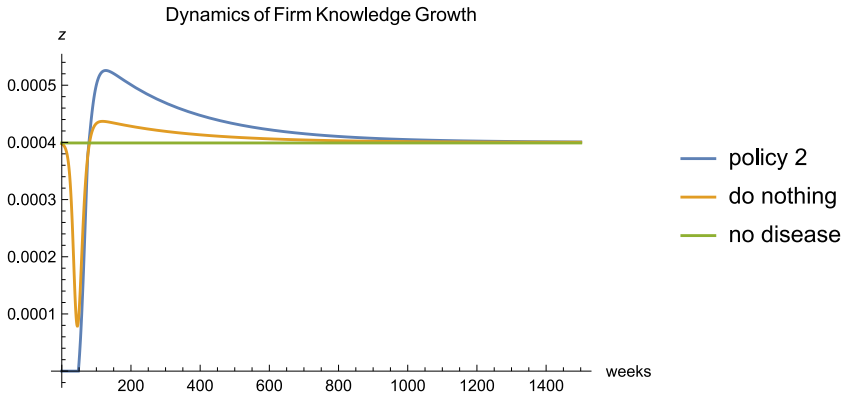
**Fig. 9.** Firm size and net entry—Susceptible-Tracking policy.
*Notes*: The figure shows simulations from a parametrized version of the model. The susceptible-tracking intervention (policy 2) becomes inactive at about week 75. See Appendix B for details on the parameterization of the model.

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 the susceptible or of the infected in the population. Our simple rules are an essential part of the definition of the model's equilibrium in that they serve to anchor agents' (rational) expectations about the time path and the end date of the intervention.

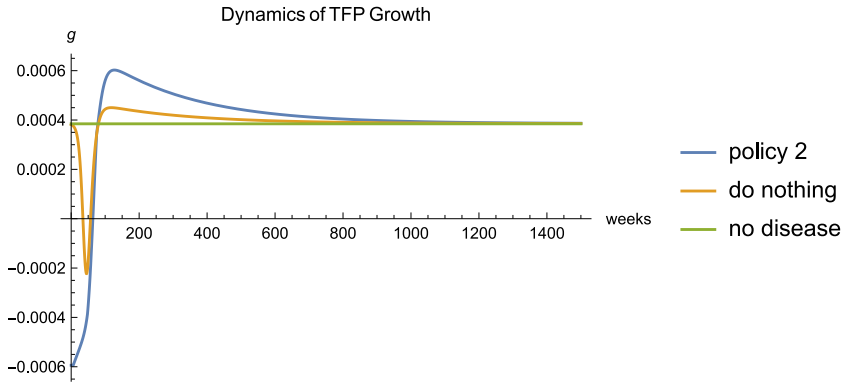
There are several promising avenues for future research. First, while studying optimal intervention policies was beyond the scope of this paper, our result reinforce the notion that the systematic analysis of the welfare effects of simple policy rules is of first-order importance. In particular, one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




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




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

**Fig. 10.** Knowledge and TFP growth—Susceptible-Tracking policy.
*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.

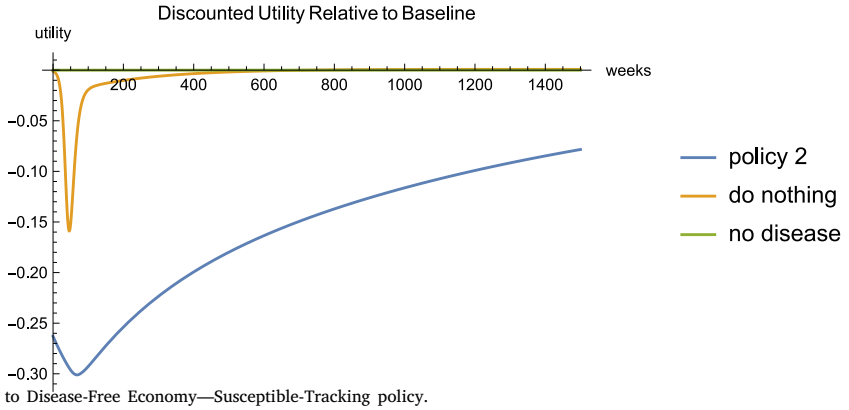



**Fig. 11.** Welfare relative to Disease-Free Economy—Susceptible-Tracking policy.
*Notes*: The figure shows simulations from a parametrized version of the model. The susceptible-tracking intervention (policy 2) becomes inactive at about week 75. See Appendix B for details on the parameterization of the model.

determines the effectiveness of lockdown policies, is in our view a margin to be taken into account in the quantitative evaluation of intervention policies.

## Declaration of competing interest

The author declare that they have no relevant or material financial interests that relate to the research described in this paper.

## Appendix A. Model

See Figs. A.1 and A.2.

### A.1. The basic growth model

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

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

where $X_i$ is output, $L_{X_i}$ is labor employment and $\phi$ is a fixed operating cost. The knowledge accumulation technology is

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

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 firm maximizes the present discounted value of profit,

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

where $\Pi_i \equiv P_i X_i - L_{X_i} - wL_{Z_i}$.

The firm's Hamiltonian is

$$\mathrm{CVH}_i = P_i X_i - L_{X_i} - wL_{Z_i} + q_{Z_i}\alpha K L_{Z_i}^{\sigma} L_Z^{1-\sigma}.$$

The FOCs are:

$$P_i = \frac{\epsilon}{\epsilon - 1} Z_i^{-\theta};$$

$$w = q_{Z_i}\alpha\sigma K L_{Z_i}^{\sigma-1} L_Z^{1-\sigma} \Rightarrow 1 = q_Z \alpha\sigma K,$$

$$r + \delta = \frac{\partial \Pi_i}{\partial Z_i} \cdot \frac{1}{q_{Z_i}} + \frac{\dot{q}_{Z_i}}{q_{Z_i}}.$$

The pricing rule yields that the firm's instantaneous profit is

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

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

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

This expression characterizes the return to knowledge accumulation for firm $i$. Then we have that under symmetry

$$r + \delta = \frac{Y}{N}\alpha\sigma\theta\frac{\epsilon - 1}{\epsilon} - \alpha\frac{L_Z}{N}.$$

The return to horizontal innovation (entry) is

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

It is useful to check that we have well-behaved no entry region with $z = 0$. From the household's budget constraint:

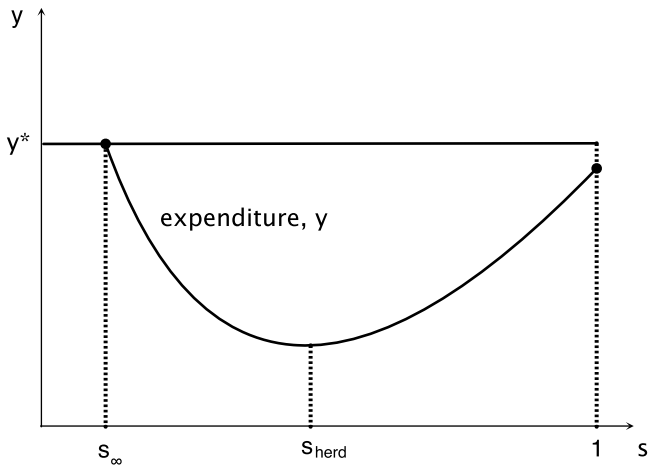
$$\dot{N}V + N\dot{V} = \left(\frac{\frac{Y}{\epsilon N} - \phi}{V} + \frac{\dot{V}}{V} - \delta\right)NV + wL - Y,$$

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

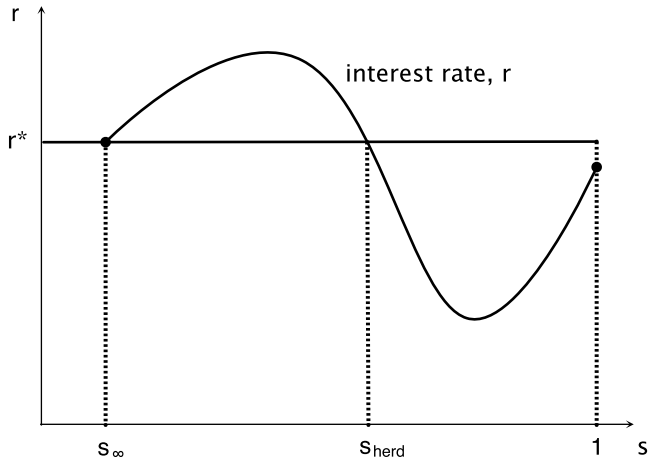
$$0 = \left(\frac{Y}{\epsilon N} - \phi\right)N + L - Y, \quad \text{since } w = 1.$$

Hence, we obtain

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

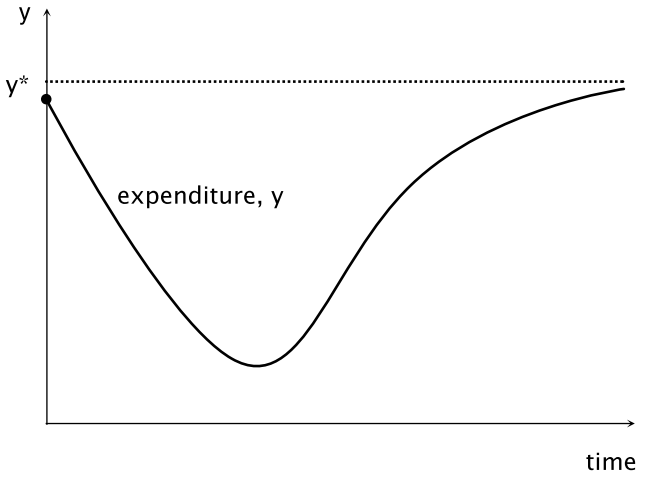



(a) Per capita expenditure as function of susceptible: $y(s)$

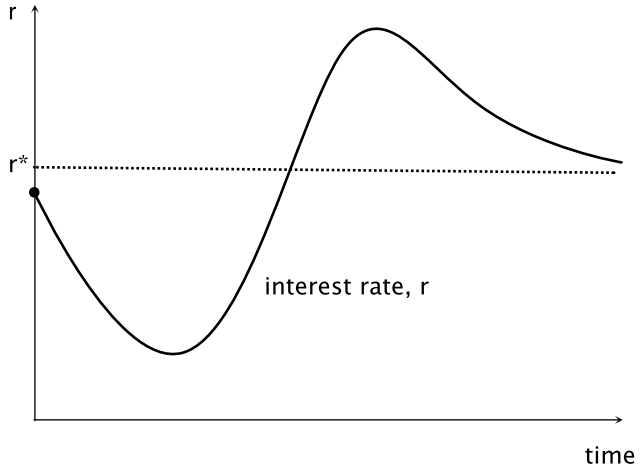



(b) Interest rate as function of susceptible: $r(s)$

**Fig. A.1.** Expenditure and interest rate as function of susceptible.




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




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

**Fig. A.2.** Expenditure and interest rate as function of time.

### A.2. Three examples of epidemiological trajectories

Epidemiological SIRD equations:

$$\frac{\dot{s}}{s} = \xi_B\left(\frac{1}{s} - 1\right) - \left(\xi_S - \xi_D\right)\iota; \tag{A.1}$$

$$\frac{\dot{i}}{\iota} = \xi_S s - \xi_R - \xi_B - \xi_D\left(1 - \iota\right). \tag{A.2}$$

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

$$\frac{\dot{s}}{s} = -\left(\xi_S - \xi_D\right)\iota < 0; \tag{A.3}$$

$$i \geq 0: \quad \iota \geq 1 + \frac{\xi_R}{\xi_D} - \frac{\xi_S}{\xi_D}s. \tag{A.4}$$

*Example 2: SIR w/ no vital dynamics.* (i.e., $\xi_B = \xi_M = \xi_D = 0$):

$$\frac{\dot{s}}{s} = -\xi_S\iota < 0; \tag{A.5}$$

$$i \geq 0: \quad s \geq \frac{\xi_S}{\xi_R} = R_0. \tag{A.6}$$

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

$$\dot{s} \geq 0: \quad \iota \leq \frac{\xi_B}{\xi_S}\left(\frac{1}{s} - 1\right); \tag{A.7}$$

$$i \geq 0: \quad s \geq \frac{\xi_R + \xi_B}{\xi_S}. \tag{A.8}$$

### A.3. Three types of interventions

#### A.3.1. Example 1: Constant wedge policy

The economy reverts to the same steady-state growth rate because the permanent change in expenditure is offset by a permanent rise in firm size. Of course, one can think that a 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 $\iota = i_\infty > 0$. 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

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

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

$$\frac{d\iota}{ds} = \frac{1 + \mu s^\eta - \mu\bar{s}^\eta}{R_0 s} - 1$$

and has solution

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

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

$$\iota_0 = c - 1 + \iota_0 + \frac{1 - \mu\bar{s}^\eta}{R_0}\log s_0 + \frac{\mu}{\eta R_0}s_0^\eta.$$

We thus get

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

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

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

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

$$\iota\left(\bar{s}^{+}\right) = 1 - \bar{s} + \frac{1 - \mu\bar{s}^{\eta}}{R_0}\log\left(\frac{\bar{s}}{s_0}\right) + \frac{\mu}{\eta R_0}\left(\bar{s}^{\eta} - s_0^{\eta}\right);$$
$$\iota\left(\bar{s}^{-}\right) = c - \bar{s} + \frac{1}{R_0}\log\bar{s}.$$

Continuity holds when

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

which yields

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

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

$$\begin{aligned}\iota(s) &= 1 - s + \frac{1}{R_0}\log\left(\frac{\bar{s}}{s_0}\right) + \frac{1}{R_0}\log\left(\frac{s}{\bar{s}}\right) - \frac{\mu\bar{s}^{\eta}}{R_0}\log\left(\frac{\bar{s}}{s_0}\right) + \frac{\mu}{\eta R_0}\left(\bar{s}^{\eta} - s_0^{\eta}\right),\\ &= 1 - s + \frac{1}{R_0}\log\left(\frac{s}{s_0}\right) + \frac{\mu}{\eta R_0}\left(\bar{s}^{\eta} - s_0^{\eta}\right) - \frac{\mu\bar{s}^{\eta}}{R_0}\log\left(\frac{\bar{s}}{s_0}\right).\end{aligned}$$

At $s = \bar{s}$ the two pieces connect as follows:

$$-\frac{\mu\bar{s}^{\eta}}{R_0}\log\left(\frac{\bar{s}}{s_0}\right) + \frac{\mu}{\eta R_0}\left(\bar{s}^{\eta} - s_0^{\eta}\right) = -\frac{\mu\bar{s}^{\eta}}{R_0}\log\left(\frac{\bar{s}}{s_0}\right) + \frac{\mu}{\eta R_0}\left(\bar{s}^{\eta} - s_0^{\eta}\right) \Rightarrow 0 = 0.$$

Continuity thus holds. Next, we check the derivatives at $s = \bar{s}$:

$$-1 + \frac{1}{R_0}\cdot\frac{1}{\bar{s}} = -1 + \frac{1}{R_0}\cdot\frac{1}{\bar{s}} + \frac{\mu\bar{s}^{\eta-1}}{R_0} - \frac{\mu\bar{s}^{\eta-1}}{R_0} \Rightarrow 0 = 0.$$

Smooth pasting also holds. Putting all of this together, we obtain the infection function

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

*A.3.3. Example 3: Infected-tracking policy*

Define the policy

$$h(\iota) = \mu\iota.$$

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

$$\frac{d\iota}{ds} = \frac{1 + \mu\iota}{R_0 s} - 1$$

and has solution

$$\iota(s) = \frac{\mu - R_0(1 + \mu s)}{(R_0 - \mu)\mu} + s^{\frac{\mu}{R_0}}c.$$

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

$$c = \left[\iota_0 - \frac{\mu - R_0(1 + \mu s_0)}{(R_0 - \mu)\mu}\right] s_0^{-\frac{\mu}{R_0}}.$$

Therefore, we have

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

For $\mu > R_0$ this function is hump-shaped with two zeros, one at $(1, 0)$ and the other at $(s_{\infty}^{int}, 0)$, where for $(s_0, \iota_0) \approx (1, 0)$,

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

## Appendix B. Parametrization

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

### B.1. Annual calibration

*Standard targets.*

- 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

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

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

*Fixed operating cost* ($\phi$). Since

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

the parameter $\phi$ is identified independently of the R&D technology if we have data on $L_Z/L$. We take the value $L_Z/L = 0.01$ from the InfoBrief, October 2016, NSF 17–302.[12] This gives us

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

*The "innovation" triplet* ($\alpha, \theta, \sigma$). We note that the TFP growth rate along a BGP is

$$g = \alpha\theta \times \frac{L}{N} \times \frac{L_Z}{L} \Rightarrow \alpha\theta = \frac{g}{\frac{L}{N} \times \frac{L_Z}{L}} = \frac{0.02}{\frac{L}{N} \times \frac{L_Z}{L}} = \frac{0.02}{21.6 \times 0.01} = 0.0926.$$

Thus, the data moments $g = 2\%$, $L_Z/L = 1\%$, $L/N = 21.6$ pin down the value of $\alpha\theta$. We then use the relation

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

and the expression for the steady-state population-to-firms ratio

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

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 desired threshold ordering, i.e., $x_N < x_Z$. Specifically, we have:

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

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

$$\theta = \frac{0.02}{\sigma\alpha\theta y\frac{\epsilon-1}{\epsilon}x - r - \delta} = \frac{0.02}{\sigma\frac{0.0926}{1-0.04\times0.926}\frac{2}{3}21.6 - 0.1018}.$$

Then, we write the ratio of thresholds,

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

[12] Specifically, the InfoBrief states: *"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).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/).

where the thresholds are:

$$x_Z = (1-\rho\beta)\frac{\epsilon(\rho+\delta)}{\sigma\alpha\theta(\epsilon-1)} = \frac{1}{\sigma}(1-0.04\times 0.926)\frac{0.1018}{0.0926}\frac{3}{2} = \frac{1}{\sigma}1.5879;$$
$$x_N = \frac{1-\beta\rho}{\frac{1}{\epsilon}-\rho\beta}\phi = \frac{1-0.04\times 0.926}{\frac{1}{3}-0.04\times 0.926}5.0177 = 16.308.$$

We thus are free to set $\sigma$ to obtain the target threshold ratio. After experimenting with this structure, we settled on

$$\frac{x_Z}{x_N} = \frac{11}{10} \Rightarrow \sigma = \frac{1.5879}{11/10\times 16.308} = 0.0885.$$

The threshold for cost-reducing innovation is $x_Z = \frac{11}{10}16.308 = 17.939$. Then we obtain:

$$\theta = \frac{0.02}{0.0885\times\frac{0.0926}{1-0.04\times 0.926}\frac{2}{3}21.6-0.1018} = 0.9639$$

and

$$\alpha = \frac{0.0926}{0.9639} = 0.0961.$$

We need to check that $1-\sigma\theta(\epsilon-1) = 1-0.0779\times 3.2946\times 2 = 0.4867 > 0$.

### 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:

$$\frac{wL}{Y} = 1-\beta\rho \Rightarrow \beta\rho = 1-\frac{wL}{G}\times\frac{G}{Y} = \frac{0.65}{0.675} = 0.96296.$$

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:

$$\frac{1-\sigma\theta(\epsilon-1)}{\beta\epsilon}-(\rho+\delta) = \left[\frac{1-\sigma\theta(\epsilon-1)}{\beta\epsilon}-\frac{\rho+\delta}{52}\right]52$$
$$\frac{1-\sigma\theta(\epsilon-1)}{\beta_a\epsilon}-(\rho+\delta) = \frac{1-\sigma\theta(\epsilon-1)}{\beta_w\epsilon}\frac{1}{52}-(\rho+\delta)$$
$$\frac{1-\sigma\theta(\epsilon-1)}{\beta_a\epsilon} = \frac{1-\sigma\theta(\epsilon-1)}{\beta_w\epsilon}52.$$

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

Also, since

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

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

$$\frac{0.02/54}{\theta} = \alpha\frac{L_Z}{L}\times\frac{L}{N} \Rightarrow \alpha\theta = \frac{0.02/52}{\frac{L}{N}\times\frac{L_Z}{L}},$$

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

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

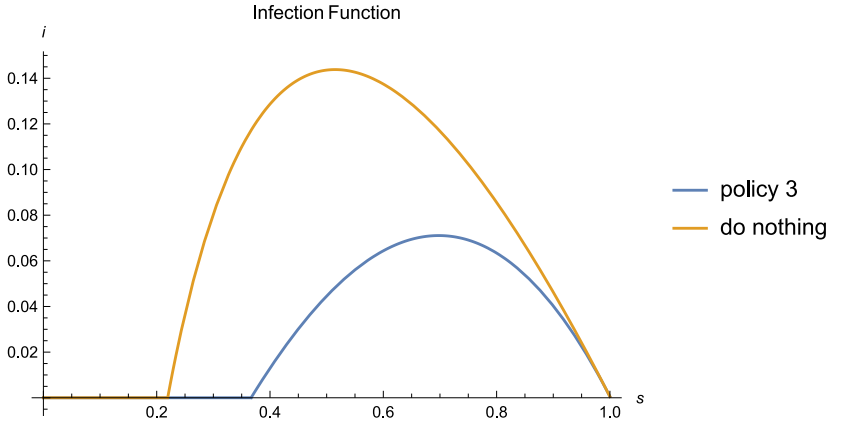
Hence, the parameters $\theta$ and $\sigma$ are invariant from the annual to the weekly frequency since the conversion factor 52 drops out of these expressions. Then, going to the weekly calibration, implies that $\alpha$ changes according to

$$\alpha_w = \frac{1}{\theta}\frac{0.02}{\frac{L}{N}\times\frac{L_Z}{L}}\frac{1}{52} = \frac{\alpha_a}{52}.$$

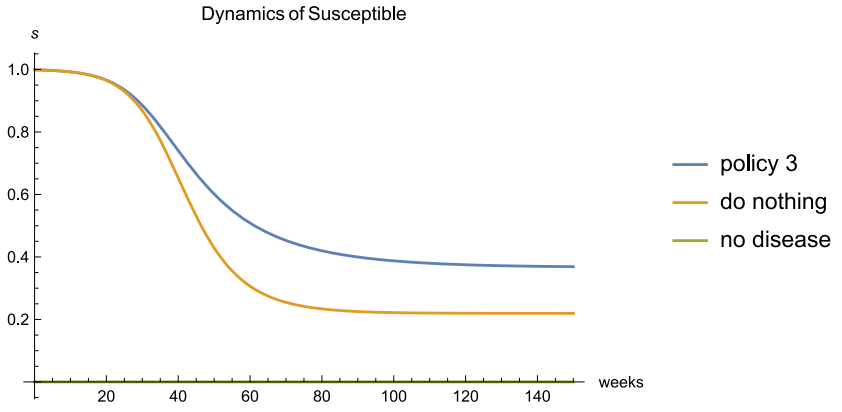
To summarize, going from an annual to a weekly calibration, only the rates $(r,g,\delta)$ change and their changes are absorbed one-for-one by $\alpha$ and $\beta$.

## Appendix C. Results

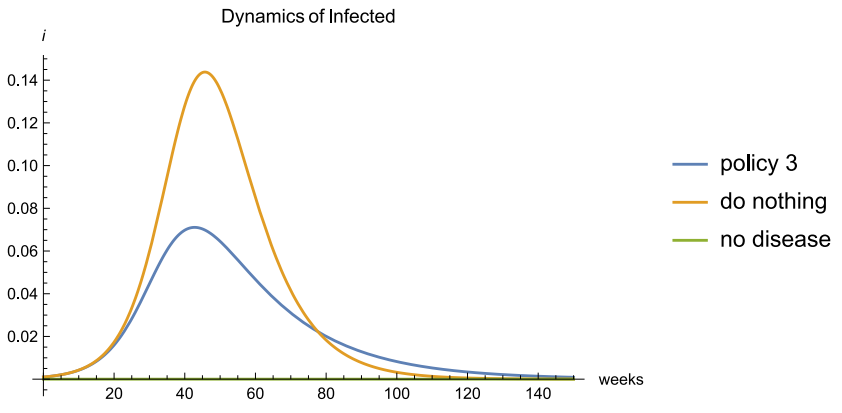
See Figs. C.1–C.5.




(a) Fraction of infected as a function of fraction of susceptible: $\imath(s)$

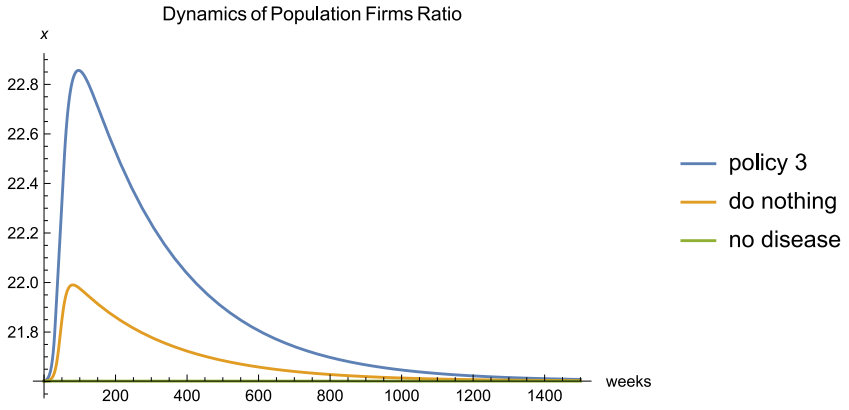



(b) Fraction of susceptible in population: $s(t)$

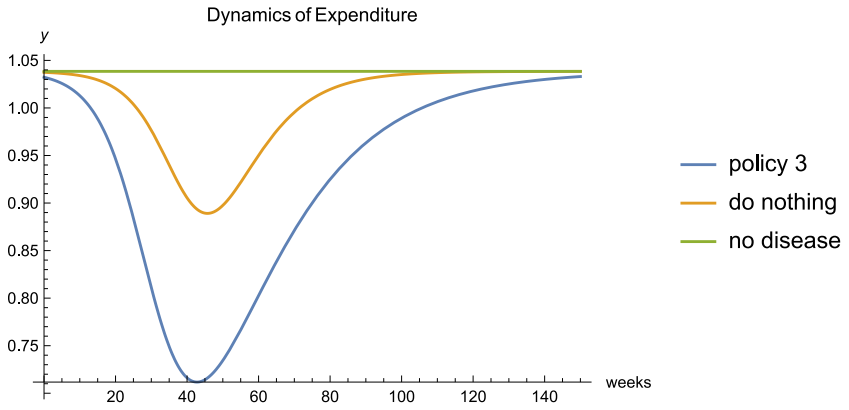



(c) Fraction of infected in population: $\imath(t)$

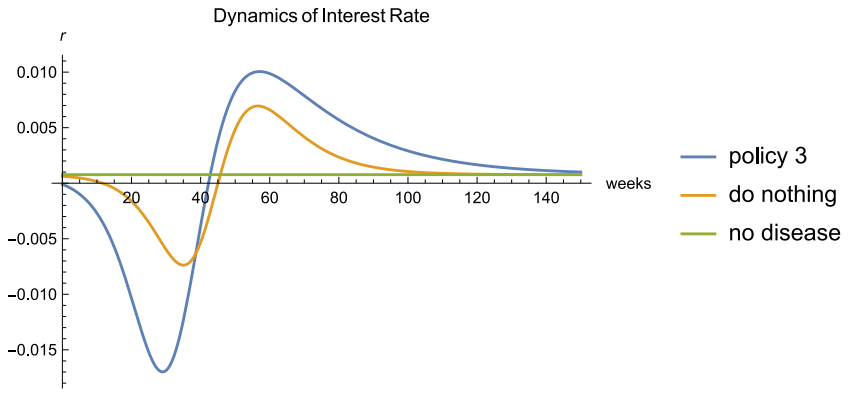
**Fig. C.1.** Epidemiological dynamics after the Outbreak—Infected-Tracking policy.
*Notes*: The figure shows the infection function and the dynamics of the fraction of susceptible and infected in the population after the outbreak for a parametrized version of the model. The infected-tracking intervention (policy 3) becomes inactive at about week 140. See Appendix B for details on the parameterization of the model.




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

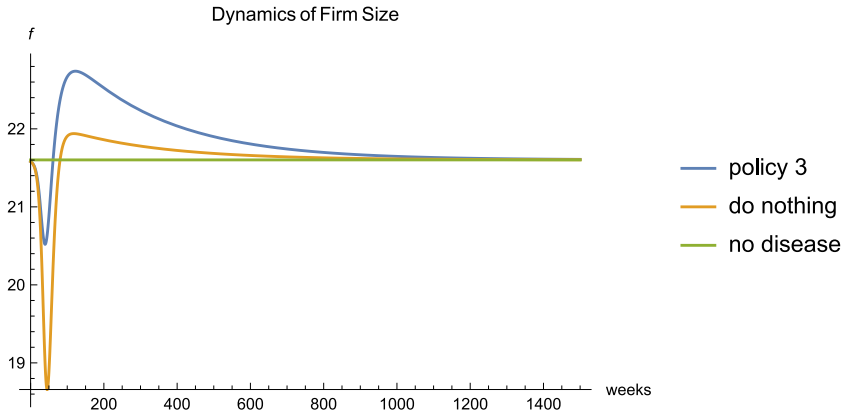



(b) Per capita expenditure: $y \equiv Y/L$

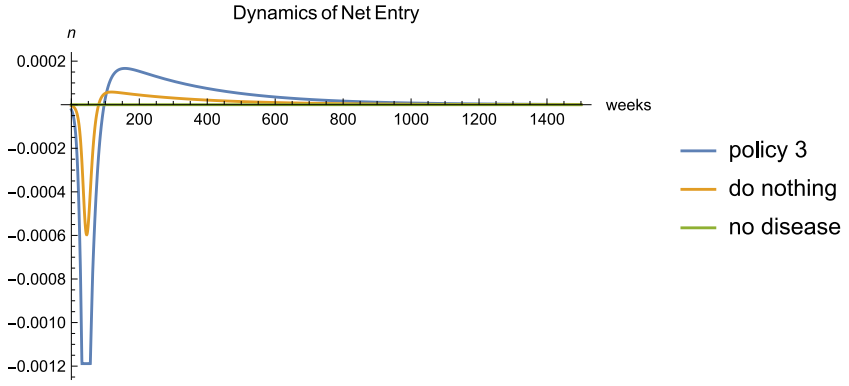



(c) Interest rate: $r$

**Fig. C.2.** Market size and interest rates—Infected-Tracking policy.
*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.

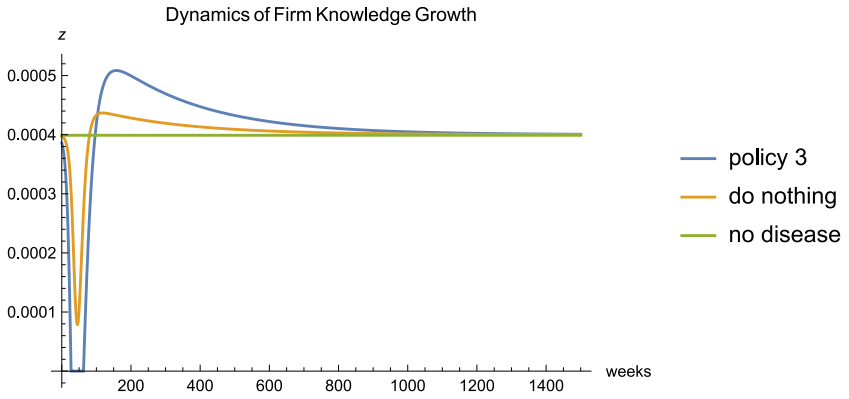



(a) Firm size: $[1-\iota(s)]\,L/N$

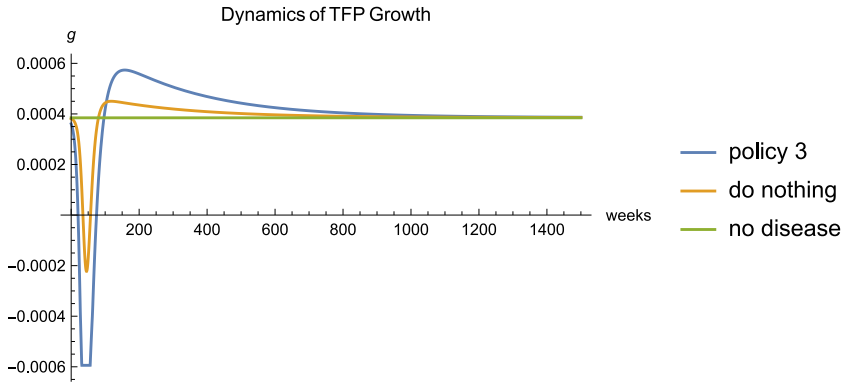



(b) Net entry: $n \equiv \dot{N}/N$

**Fig. C.3.** Firm size and net entry—Infected-Tracking policy.
*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.




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




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

**Fig. C.4.** Knowledge and TFP growth—Infected-Tracking policy.
*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.

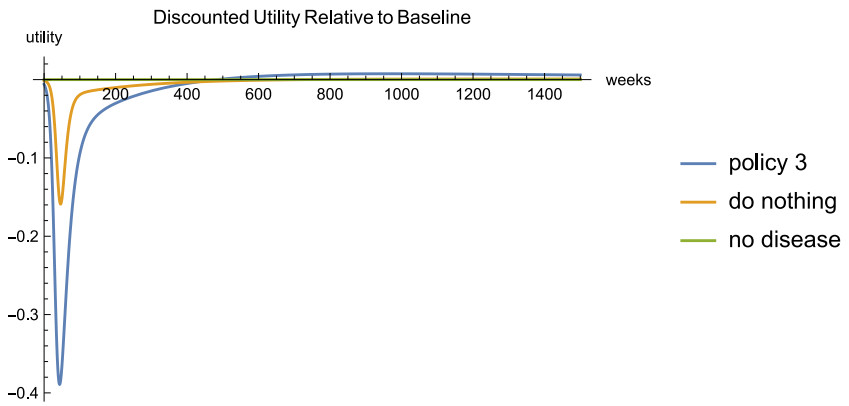



**Fig. C.5.** Welfare relative to Disease-Free Economy—Infected-Tracking policy.
*Notes*: The figure shows simulations from a parametrized version of the model. The infected-tracking intervention (policy 3) becomes inactive at about week 140. See Appendix B for details on the parameterization of the model.

## Data availability

Data will be made available on request.

## References


Acemoglu, Daron, Chernozhukov, Victor, Werning, Iván, Whinston, Michael D., 2020. A Multi-Risk SIR Model with Optimally Targeted Lockdown. NBER Working Paper, No. 27102, National Bureau of Economic Research.

Alvarez, Fernando E., Argente, David, Lippi, Francesco, 2020. A Simple Planning Problem for COVID19 Lockdown. NBER Working Paper, No. 26981, National Bureau of Economic Research.

Atkeson, Andrew, 2020. What Will be the Economic Impact of COVID-19 in the US? Rough Estimates of Disease Scenarios. NBER Working Paper, No. 26867, National Bureau of Economic Research.

Barro, Robert J., Ursua, Jose F., Weng, Joanna, 2020. The Coronavirus and the Great Influenza Epidemic: Lessons from the "Spanish Flu" for the Coronavirus' Potential Effects on Mortality and Economic Activity. NBER Working Paper, 26866, JSTOR.

Bethune, Zachary A., Korinek, Anton, 2020. Covid-19 Infection Externalities: Trading Off Lives vs. Livelihoods. National Bureau of Economic Research.

Bloom, David E., Kuhn, Michael, Prettner, Klaus, 2022. Modern infectious diseases: Macroeconomic impacts and policy responses. J. Econ. Lit. 60 (1), 85–131.

Bognanni, Mark, Hanley, Doug, Kolliner, Daniel, Mitman, Kurt, 2020. Economic activity and COVID-19 transmission: Evidence from an estimated economic-epidemiological model. Mimeo.

Brauer, Fred, 2008. Compartmental models in epidemiology. In: Mathematical Epidemiology. Springer, pp. 19–79.

Brotherhood, Luiz, Kircher, Philipp, Santos, Cezar, Tertilt, Michèle, 2020. An economic model of the COVID-19 epidemic: The importance of testing and age-specific policies. Mimeo.

Eichenbaum, Martin S., Rebelo, Sergio, Trabandt, Mathias, 2020a. The Macroeconomics of Epidemics. NBER Working Paper, No. 26882, National Bureau of Economic Research.

Eichenbaum, Martin S., Rebelo, Sergio, Trabandt, Mathias, 2020b. The Macroeconomics of Testing and Quarantining. NBER Working Paper, No. 27104, National Bureau of Economic Research.

Eichenbaum, Martin S., Rebelo, Sergio, Trabandt, Mathias, 2020c. Epidemics in the Neoclassical and New Keynesian Models. NBER Working Paper, No. 27430, National Bureau of Economic Research.

Farboodi, Maryam, Jarosch, Gregor, Shimer, Robert, 2020. Internal and External Effects of Social Distancing in a Pandemic. NBER Working Paper, No. 27059, National Bureau of Economic Research.

Glover, Andrew, Heathcote, Jonathan, Krueger, Dirk, Ríos-Rull, José-Víctor, 2020. Health versus Wealth: On the Distributional Effects of Controlling a Pandemic. NBER Working Paper, No. 27046, National Bureau of Economic Research.

Goenka, Aditya, Liu, Lin, 2020. Infectious diseases, human capital and economic growth. Econom. Theory 70, 1–47.

Goenka, Aditya, Liu, Lin, Nguyen, Manh-Hung, 2014. Infectious diseases and economic growth. J. Math. Econom. 50, 34–53.

Greenwood, Jeremy, Kircher, Philipp, Santos, Cezar, Tertilt, Michele, 2019. An equilibrium model of the african HIV/AIDS epidemic. Econometrica 87 (4), 1081–1113.

Hethcote, Herbert W., 2000. The mathematics of infectious diseases. SIAM Rev. 42 (4), 599–653.

Jones, Callum J., Philippon, Thomas, Venkateswaran, Venky, 2020. Optimal Mitigation Policies in a Pandemic: Social Distancing and Working from Home. NBER Working Paper, No. 26984, National Bureau of Economic Research.

Krueger, Dirk, Uhlig, Harald, Xie, Taojun, 2020. Macroeconomic Dynamics and Reallocation in an Epidemic. National Bureau of Economic Research.

Madsen, Jakob, Robertson, Peter E., Ye, Longfeng, 2022. Lives versus livelihoods in the middle ages: The impact of the plague on markets over 400 years. Unpublished Manuscript.

Peretto, Pietro F., Connolly, Michelle, 2007. The manhattan metaphor. J. Econ. Growth 12 (4), 329–350.

Riou, Julien, Althaus, Christian L., 2020. Pattern of early human-to-human transmission of Wuhan 2019-nCoV. BioRxiv.

Romer, Paul M., 1990. Endogenous technological change. J. Political Econ. 98 (5, Part 2), S71–S102.