Macroepidemics and unconventional monetary policy

Verónica Acurio Vásconez, Olivier Damette, David W Shanafelt

To cite this version:

Verónica Acurio Vásconez, Olivier Damette, David W Shanafelt. Macroepidemics and unconventional monetary policy. Economic Modelling, 2023, 126, pp.106431. 10.1016/j.econmod.2023.106431 . hal-04220462

HAL Id: hal-04220462
https://hal.inrae.fr/hal-04220462v1

Submitted on 27 Sep 2023

Economic Modelling 126 (2023) 106431

Contents lists available at ScienceDirect

Economic Modelling

journal homepage: www.journals.elsevier.com/economic-modelling

Macroepidemics and unconventional monetary policy[☆]

Verónica Acurio Vásconez [a,*], Olivier Damette [b], David W. Shanafelt [c]

[a] Université de Lorraine, Université de Strasbourg, Centre National de la Recherche Scientifique (CNRS), Bureau d'Economie Théorique et Appliquée (BETA), 13 Place Carnot, CO No. 026, 54035 Nancy Cedex, France
[b] Université de Lorraine, Université de Strasbourg, CNRS, BETA, 13 Place Carnot, CO No. 026, 54035 Nancy Cedex, France
[c] Université de Lorraine, Université de Strasbourg, AgroParisTech, CNRS, Institut National de Recherche pour l'Agriculture, l'Alimentation et l'Environnement (INRAE), BETA, 14 Rue Girardet - CS 14216, 54042 Nancy Cedex, France

ARTICLE INFO

Dataset link: https://osf.io/j7m65

JEL classification:
D58
E32
E52

Keywords:
New-Keynesian model
Financial DSGE
COVID-19
Epidemiology
Unconventional monetary policy

ABSTRACT

Although the current COVID-19 pandemic was neither the first nor the last disease to threaten a pandemic, only recently have studies incorporated epidemiology into macroeconomic theory. This paper uses a dynamic stochastic general equilibrium (DSGE) model with a financial sector to study the economic impacts of epidemics and the potential for unconventional monetary policy to remedy those effects. By coupling a macroeconomic model with a traditional epidemiological model, we can evaluate the pathways by which an epidemic affects a national economy. We find that no unconventional monetary policy can completely remove the negative effects of an epidemic crisis, save perhaps an exogenous increase in the shares of claims coming from the Central Bank ("*epi loans*"). To the best of our knowledge, our paper is one of the first to incorporate disease dynamics into a DSGE-SIR model with a financial sector and examine the use of an unconventional monetary policy.

1. Introduction

The economic effects of the COVID-19 pandemic are unprecedented and far-reaching, extending to virtually every member of the global market. Global growth was projected at minus 4.9 percent in 2020 (6 percent to 7.6 percent depending on the emergence of a second wave) (IMF, 2020) and reached a record value of minus 4.3 percent in that year. COVID-19 was not the first emerging zoonotic or epizootic disease to threaten a pandemic (Boissay and Rungcharoenkitkul, 2020; LePan, 2020), nor will it be the last (Daszak et al., 2001; Jones et al., 2008; Wu et al., 2017).

Nonetheless, before the COVID-19 pandemic, few studies incorporated epidemiology into macroeconomic theory, though this was not the case in microeconomics (see Horan and Wolf (2005), Horan and Fenichel (2007), Fenichel et al. (2011), Lenhart and Workman (2007), Morin et al. (2014) and Morin et al. (2015) for examples). Recent studies have examined the potential economic impacts of pandemics on a macroeconomic scale using susceptible–infected–recovered (SIR) epidemiological models in line with the macro models developed by Bodenstein et al. (2022) and Eichenbaum et al. (2021). However, previous papers have largely overlooked the effects of economic remedies in the form of monetary policies to reduce the economic burden of epidemics. The role of financial intermediaries in coupled macroeconomic-epidemic frameworks has not yet been studied.

Following Smets and Wouters (2007), this paper uses a dynamic stochastic general equilibrium (DSGE) model, but with a financial sector as in Gertler and Karadi (2011) (GK hereafter). We couple the financial DSGE model to an epidemiological SIR model to study the economic effects of an epidemic and the ability of monetary policy to remedy the crisis. Using a DSGE model enables us to study the effects of an epidemic at the level of the entire economy and, with the GK framework, assess the efficiency of unconventional monetary policy to combat the economic burdens of an epidemic.[1]

[☆] We would like to thank two anonymous reviewers for their comments and questions in improving our manuscript. All remaining errors are ours.

[*] Corresponding author.

E-mail addresses: veronica.acurio-vasconez@univ-lorraine.fr (V. Acurio Vásconez), olivier.damette@univ-lorraine.fr (O. Damette), david.shanafelt@inrae.fr (D.W. Shanafelt).

[1] The GK model was used to extensively examine the effects of unconventional monetary policy on macroeconomic outputs following the sub-prime crisis (Gertler and Karadi, 2011; Dedola et al., 2013; Gelain and Ilbas, 2017). Gertler and Karadi (2011) showed that when a financial crisis arises (understood as a negative shock in the quality of capital), the stronger the reaction of the Central Bank, and the smaller the total losses in GDP.

https://doi.org/10.1016/j.econmod.2023.106431
Received 17 February 2022; Received in revised form 28 June 2023; Accepted 1 July 2023
Available online 5 July 2023

We test the general effects of an epidemic on a model economy using a set of baseline parameters and then assess the ability of three sets of unconventional monetary policies to mitigate the effects of the recession. These policies include the steady-state leverage ratio (the total loans that a private bank can issue compared to its net worth), the sensitivity of the Central Banks' response to the spread in interest rates, and the use of what we call "*epi loans*". We model "*epi loans*" as a Central Bank liquidity injection into the real sector in the form of claims that do not pass through private banks, similar to those that followed the sub-prime crisis in Europe and the United States (US). This measure can be understood as a light form of "helicopter money" (Friedman, 1969), in the sense that the injected liquidity goes directly to the real sector without the direct involvement of fiscal authorities or private banks. However, unlike "helicopter money", our "*epi loans*" policy must be repaid, thus changing the Central Bank balance sheet by increasing its assets.[2]

Since the beginning of the COVID-19 pandemic, an explosion of literature has investigated the macroeconomics of pandemics. Rather than offering a comprehensive literature review of the macro effects of pandemics, we limit our discussion to papers relevant to our research. We provide a detailed review in Appendix A. Differences in modeling frameworks largely depend on the focus of the study and the policy instruments chosen to combat the epidemic. For example, our paper is akin to Eichenbaum et al. (2021, 2020b,a), Angelini et al. (2020) or Krueger et al. (2020), who developed more-or-less simple macroeconomic neoclassical models, where agents consume goods and work, combined with standard disease models in the epidemiology literature. Their primary focus is the interplay between consumers, producers, and the labor market. They do not consider other sectors in the economy and, as a result of having a simpler model, can endogenize the feedback between the epidemic's effects on the labor market and economic behavior. In contrast, Costa Junior et al. (2021) and Kiley (2020) studied the ability of monetary policy – in the form of quantitative easing programs – to limit falls in consumption, inflation, and output and stimulate recovery following market shocks, including COVID-19. This approach required incorporating more sectors of the economy into the model and a more complex modeling framework; thus, they do not endogenize the epidemic, instead taking it as random, normally-distributed technical and preference shocks. Like Kiley (2020), we extend the GK framework to analyze the use of unconventional monetary policies but explicitly incorporate disease dynamics as a labor shock; we assume that the labor supply is given by the number of people in good health, which is exogenously driven by the SIR model. In this regard, we have a one-way epidemic effect on the economy as in Bodenstein et al. (2022). This approach is a subtle departure from traditional labor shocks. Traditionally, labor shocks are incorporated as stochastic, auto-regressive processes; however, epidemics follow particular profiles which, while exhibiting some stochasticity, cannot be captured by random probability distributions (Brauer and Castillo-Chavez, 2012).

In general, we find significant gross domestic product (GDP) losses due to an epidemic shock, with the effect on the labor market echoing throughout the economy. Regarding monetary policy, no unconventional monetary policy can altogether remove the adverse economic effects of the crisis, besides perhaps an exogenous increase in the share of claims from the Central Bank. We would like to stress that while our framework represents a general epidemic, it can be tailored to any combination of epidemiological or economic parameters, making it possible to calibrate the model to a specific disease or country. For example, we calibrate our model to the case of COVID-19 in the US. Using real data of weekly infections from March 2020 to March 2023, we analyze the effects of the pandemic on the US economy and evaluate the use of "*epi loans*". Our general results are robust in the COVID-19 application. While we believe that our model is relevant to the pandemic, we hope that its contribution can extend to general epidemics.

The paper is structured as follows. Section 2 presents the model, Section 3 describes the elements of the calibration and model simulation, and Section 4 analyzes the economy's response to the epidemic shock and investigates the effect of monetary policy and the case study of COVID-19. Finally, Section 5 concludes the paper.

2. The model

This paper constructs a financial DSGE model like the one developed in Gertler and Karadi (2011). However, in contrast to the usual financial DSGE models, we enlarge our model with a SIR block (see Atkeson (2020)). Traditionally, DSGE models are used for economic policy analysis, including monetary policy. They generally assume a single stable equilibrium for all model variables, initialize the model at equilibrium, perturb the system with a shock (such as a labor shock), and measure how the system returns to equilibrium. For more details, Galí (2015) explain DSGE models at length, and Christiano et al. (2018) provide an in-depth review of the literature. We also provide a more detailed introduction to DSGE models in Appendix B.

Our DSGE model is a neo-Keynesian micro-founded aggregate representation of a national economy (Fig. 1), in which there exists an infinite number of economic agents divided into households, financial intermediates, non-financial goods producers, capital producers, and retailers. Each agent individually chooses quantities of goods, production factors, bonds, and eventually prices to maximize their well-being (e.g., preferences for households and profits for bankers, capital producers, non-financial firms, and retailers). The model also includes a government and a Central Bank that conducts conventional and unconventional monetary policy.

We couple the DSGE model to a classic epidemiological model of an epidemic (Brauer and Castillo-Chavez, 2012, 1994; Hethcote, 2000) and suppose that labor supply ties directly to the proportion of healthy individuals (Fig. 2). To isolate the epidemic's effects on the model economy, we do not impose stochastic shocks on the economy. Instead, we take the trajectory of labor supply, which is affected by the disease, as a deterministic, exogenous shock to the economy.

This section describes the epidemiological model and how it relates to households and labor supply. We then describe how households behave and present the structure of financial and non-financial entities along with capital producers and retailers. Finally, we explain how the government intervenes in the economy and how the Central Bank conducts monetary policies. Variables, definitions, and parameters are summarized in Figs. 1 and 2 and Tables 1 to 3. Appendix B presents further details on the complete derivation of the model.

2.1. Epidemiological model

To model the spread of an epidemic, we use a (SIR) model, following Brauer and Castillo-Chavez (2012, 1994), Hethcote (2000), and Lenhart and Workman (2007). The SIR model is a type of compartmental epidemiological model in which the total population, N_t, is divided into three classes or types of individuals. These classes include susceptible individuals, S_t, who can incur the disease but are not yet infected; infected individuals, $\widetilde{I}_t$, who have the disease and can spread it to susceptible individuals; and recovered individuals, $\widetilde{R}_t$, who have contracted the disease but have recovered and are immune to future infections (Fig. 2). For simplicity, we assume a constant population size, abstracting from natural births and deaths, and normalize N_t to 1. Then S_t, $\widetilde{I}_t$ and $\widetilde{R}_t$ can be interpreted as shares or proportions of individuals of each class in the general population.

---

[2] While "helicopter money" may be highly inflationist, there is no proof that QE policies are, at least not in developed countries (Qianying et al., 2016; Albertazzi et al., 2018; Baumeister and Benati, 2013).

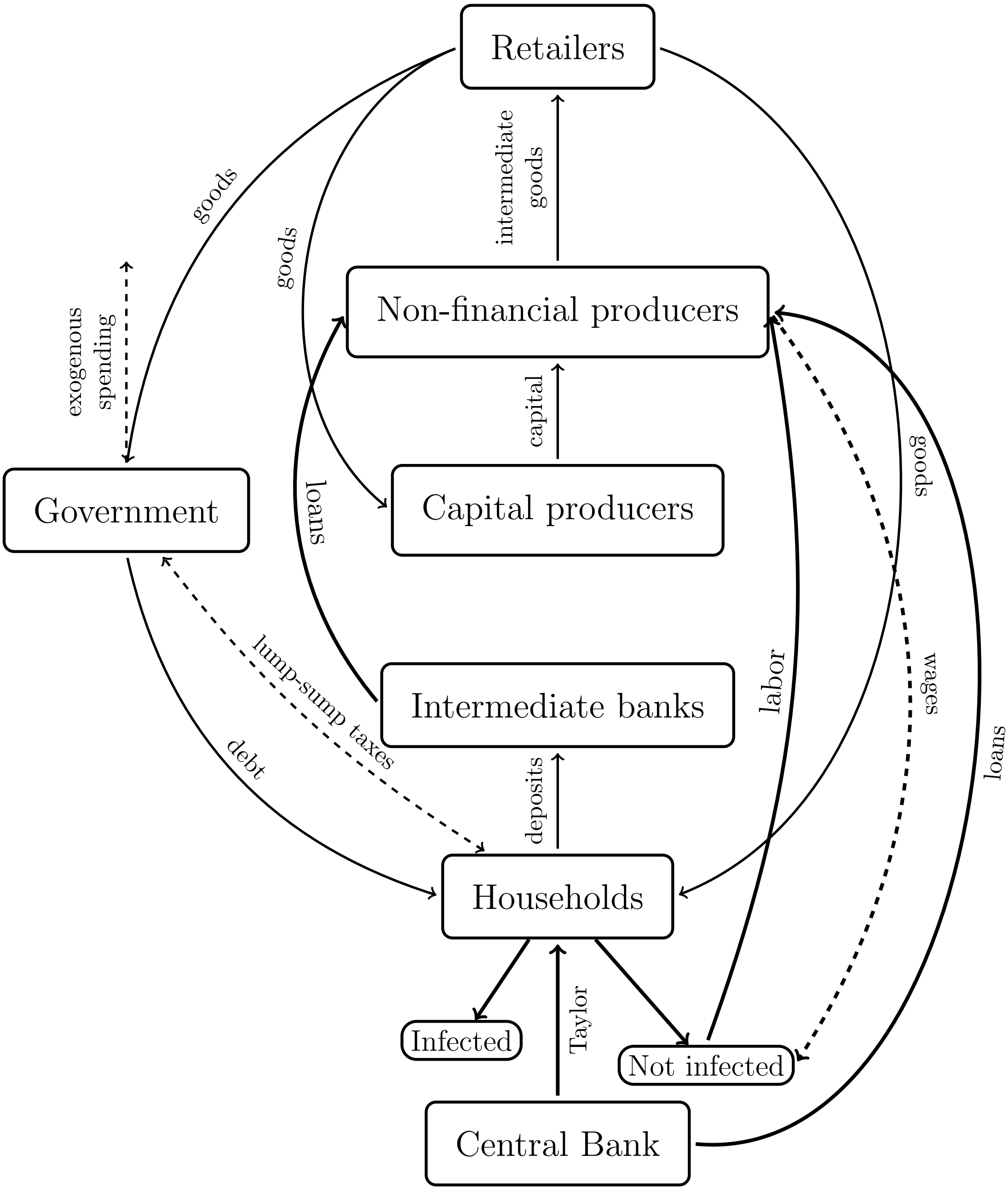

Fig. 1. Economic model Schema. In our model economy, households consume final goods and, when healthy, work for non-financial firms for which they receive a salary. They also pay/receive lump-sum taxes, lend funds to competitive financial intermediates, or buy government bonds. Private banks recover deposits from households and issue claims to non-financial producers. Claims can also be issued by the Central Bank, which also fixes nominal interest rates. Non-financial firms produce intermediate goods using workers and capital acquired from capital producers using claims. Capital producers create capital with final goods. Finally, the government collects lump-sum taxes and consumes final goods.

Fig. 2. SIR Schema.

Table 1
State and control variables.

Variable	Symbol	Type
Epidemic block		
Susceptible	S	State
Infected	$\tilde{I}$	State
Recovered	$\tilde{R}$	State
Households		
Labor	L	Control/State
Consumption	C	Control
Deposit = Government bonds	B	Control
Financial intermediates		
Quantity of financial claims issued by private banks	Z_p	Control
Non-financial intermediates and capital producers		
Intermediate non-financial goods	Y_m	Control
Capital	K	Control/State
Labor	L	Control/State
Capital investment	$I_{n,t}$	Control
Retailers and Capital producers		
Normal retailed good price	$P(h)$	Control

Table 2
Model definitions and outcomes.

Variable	Symbol
Households	
Total population	N
Real discount factor from date t to $t+1$	$\Lambda_{t,t+1}$
Good price = Aggregate retailer's price	P
Total real profits	D
Lump-sum taxes	T
Marginal lifetime discounted utility function	λ_c
Real wage	W
Financial intermediates	
Total quantity of financial claims	Z
Bankers' net worth	Ω
Expected discounted terminal wealth	V
Leverage ratio of private banks	ϕ
Auxiliary variable	Γ
Risk-less gross real rate of return	R
Claims gross real rate of return = Capital rate of return	R_k
Financial claims price	Q
Total leverage ratio (public and private)	Φ
Marginal value of banker's gain w.r.t claim income	ν
Marginal value of banker's gain w.r.t wealth	η
Existing banker's net worth	Ω_e
New banker's net worth	Ω_n
Private deposits	B_p
Private bank profit	$D_{b,t}$
Non-financial intermediates and capital producers	
Intermediate non-financial good price	P_m
Intermediate non-financial profit	$D_{m,t}$
Capital producer profit	$D_{k,t}$
Adjustment cost function of investment	$f(\cdot)$
Retailers and capital producers	
Aggregate super retailed good	Y
Normal retailed good	$Y(h)$
Normal retailed good price	$P(h)$
Optimal normal retailed good price	P^*
Normal retailer profit	$D_{r,t}$
Price dispersion	$\upsilon_{p,t}$
Central Bank and government	
Level of goods price inflation	Π
Fraction of total credits financed by the Central Bank	ψ
Quantity of financial claims issued by the government	Z_g
Government consumption	G
Nominal interest rate	i
GDP without disease	Y_{ss}
Inflation without disease	Π_{ss}
Government bonds	B_g
Exogenous fraction of publicly intermediate assets	$\tilde{\psi}$

We can write the dynamics of the epidemic over time as follows:

$$S_{t+1} - S_t = -\alpha_v S_t \widetilde{I}_t \tag{1}$$

$$\widetilde{I}_{t+1} - \widetilde{I}_t = \alpha_v S_t \widetilde{I}_t - \gamma_v \widetilde{I}_t \tag{2}$$

$$\widetilde{R}_{t+1} - \widetilde{R}_t = \gamma_v \widetilde{I}_t \tag{3}$$

where $1 = S_t + \widetilde{I}_t + \widetilde{R}_t$. The difference equations in (1)–(3) are equivalent to a system of ordinary differential equations solved via an Euler approximation. Susceptible and infected individuals make contact and transmit the disease with a constant probability, α_v, and infected individuals recover at a rate of γ_v. We assume that after recovery, individuals are immune from future infection.

The model assumes a closed population (no immigration or emigration) with a constant population size (no births or deaths) and a well-mixed population; that is, each individual in the population has an equal probability of interacting with every other individual. These assumptions capture the necessary dynamics of the epidemic while maintaining the tractability of the overall macro-epidemic model. Relaxing our assumptions complicates the analysis significantly. For example, including deaths gives rise to problems of non-constant population sizes, requiring the modeling numbers of individuals rather than proportions in Eqs. (1)–(3), births and immigration and emigration, and heterogeneous contact rates, rendering the macro-epidemic framework intractable. We leave this for future work.[3]

The epidemic affects the economy via the labor supply. Following Bodenstein et al. (2022), we assume that in the absence of disease, labor supply L_t is equal to the total working force, $L_t = N_t = 1$.; however, as the epidemic spreads in the general population, we assume that infected individuals stay home and do not work. Therefore, the labor force is reduced by the number of infected people $\widetilde{I}_t$. We find that this is a reasonable assumption. For most illnesses – such as polio, measles, rubella, chicken pox, certainly COVID-19, and even the cold and the flu – standard policy is to send employees home from work. In each period, labor supply is given as $L_t = 1 - \widetilde{I}_t$.[4]

In this way, the epidemic functions as a downward shock on the labor market, albeit with a specific profile. It is acknowledged in epidemiology that epidemic profiles cannot be captured by random probability distributions (Brauer and Castillo-Chavez, 2012); therefore, it cannot be treated as a usual $AR(1)$ labor supply shock. Appendix D shows that a SIR epidemic profile behaves very differently than a stochastic $AR(1)$ process, with significant deviations in how the

---

[3] Extensions of the basic SIR model relax these assumptions to consider multiple populations of individuals (Bichara et al., 2015), endemic disease (Hethcote, 2000), heterogeneous mixing (Morin et al., 2014, 2015; Toxvaerd, 2020), age structure (Hethcote, 2000), other classes of individuals, such as exposed or asymptomatic, vaccinated or hospitalized (Chowell et al., 2003; Hethcote, 2000; Lenhart and Workman, 2007), and management strategies, such as treatment and vaccination (Hethcote, 2000; Lenhart and Workman, 2007; Toxvaerd and Rowthorn, 2020). The validity of the SIR extensions varies according to the spatial and temporal scale of the analysis and the necessary dynamics of the disease in question. Consider the baseline SIR model (no births or deaths) with a single, localized epidemic where the disease could reasonably circulate throughout the entire population. For diseases like the cold, flu, or measles, an epidemic may last weeks or months, and accounting for births and deaths would not be appropriate; for diseases lasting years or a lifetime (AIDS/HIV, hepatitis C, or tuberculosis), including births and deaths is more reasonable (Hethcote, 2000). Similar arguments exist for assumptions of heterogeneous mixing and contact rates, treatments, or vaccination.

[4] However, we test an alternative specification on how the epidemic affects labor supply in Appendix C. Instead of having infected workers not work, we assume that the epidemic negatively affects labor productivity. While the overall economic effects of the epidemic are less severe, we find no qualitative difference in our results.

Table 3
Parameter calibration.

Parameter	Symbol	Calibrated Value/Baseline
Epidemic block		
Initial condition of susceptible	S_0	0.009
Initial condition of infected	$\widetilde{I}_0$	0.001
Initial condition of recovered	$\widetilde{R}_0$	0
Transmission rate	α_v	0.08
Recovery rate	γ_v	0.04
Households		
Discount factor	β	0.99
Internal habit formation	h	0.71
Financial intermediates		
Bankers' survival rate	θ	0.972
Fraction of claims income that can be diverted	λ	Function of risk premium at steady-state, leverage ratio at steady-state and θ
Proportional transfer to the new bankers	ϵ	Function of risk premium at steady-state, leverage ratio at steady-state, θ and $\bar{\psi}$
Risk premium at steady-state	$R_k - R$	0.01/4
Leverage ratio at steady-state	ϕ	4
Non-financial intermediates and capital producers		
Capital depreciation	δ	0.025
Price indexation to inflation	χ	0.24
Calvo price parameter	θ_p	0.66
Capital share	α	0.33
Retailers and capital producers		
Adjustment cost constant	κ	5.74
Elasticity of substitution between normal retailers	ϵ_p	4.167
Price markup	$\mathcal{M}$	Function of θ_p
Central Bank and government		
Efficiency cost	τ	0.001
Feedback parameter	ω	10
Taylor rule response to inflation	ϕ_π	2.04
Taylor rule response to output gap	ϕ_y	0.08
Taylor rule inertia	ϕ_i	0.81
Steady-state share of GDP that government expends	ω_g	0.18

economy responds to each shock. A stochastic $AR(1)$ shock specifies that the shocked variable depends linearly on its previous values (auto-regressive) and a stochastic term (stochastic). In contrast, a SIR trajectory is nonlinear and deterministic; thus, while they may appear similar at the beginning of a simulation, the complete profiles of how each affects labor supply differ. For example, in a SIR model, the labor supply decreases until the epidemic peaks and then increases to its steady-state value; for an $AR(1)$ shock, labor decreases after the shock but immediately begins to return to its steady-state. Furthermore, differences in the deterministic versus stochastic nature of the shocks affect agent behavior. If the trajectory of the shock (as opposed to the distribution of the shock) is known, agents can anticipate future variables and behave accordingly.

2.2. Households

We assume a continuum of perfectly competitive households in the economy indexed by $j \in [0,1]$. Susceptible, infected, and recovered individuals are assumed to be evenly distributed among households, and each household consumes domestic goods and, if healthy, supplies identical labor services to the non-financial production sector. Households pay/receive lump-sum taxes, collect profits from all firms, and can lend funds to competitive financial intermediates or buy government bonds. Households are Ricardian, meaning that they are forward-looking and, in response to increases in government spending, will choose to save today, expecting to pay higher taxes later.

At each time period t, a typical household j chooses consumption C_t to maximize the following lifetime expected utility function:

$$\mathbb{E}_t \left[\sum_{k=0}^{\infty} \beta^k U\left(C_{t+k}(j)\right) \right] \tag{4}$$

where $U(C_t(j))$ is the net utility of household consumption of non-financial goods, and $\beta \in (0,1)$ is the discount factor.

Following Christiano et al. (2005), we allow for internal habit formation in consumption. Thus, the instantaneous utility at time t is given by:

$$U(C_t(j)) = log(C_t(j) - hC_{t-1}(j)) \tag{5}$$

where $h \in [0,1)$ represents the internal habit formation parameter. The latter governs how household preferences for past consumption affect utility over time. A high value of h means that past consumption is important; a household must consume at least the same quantity as the last period to maintain the current level of utility. A low value of h implies that households only care about present consumption. Note that we do not introduce a trade-off between consumption and labor since the epidemic determines labor supply. With this formulation, we implicitly assume that all those who can work are willing to work.

Each household may have a portion of infected people that do not work. The remaining individuals – susceptible, recovered, or both – may be divided into workers and bankers. Workers are employed by non-financial intermediate firms and receive a real salary W_t in exchange for the total amount of labor provided L_t. Bankers manage financial intermediaries and gain earnings without supplying labor. We assume that each household member gives their respective revenues to the household, and perfect consumption insurance is provided; that is, consumption is equally distributed within households regardless if members can work.

Each household consumes final goods produced by retailers at price P_t and invests/deposits B_t in government bonds and intermediary deposits. We assume that investing in government bonds and depositing into intermediate banks are equivalent and perfectly substitutable, as both are risk-less and pay the same rate. Each is a one-period real bond, which pays a gross real rate of return R_t, such that $R_{t+1} := \frac{1+i_t}{\Pi_{t+1}}$; i_t is the nominal interest rate fixed by the Central Bank, and $\Pi_{t+1} := \frac{P_{t+1}}{P_t}$ represents gross price inflation.

We assume that each household owns an equal share of all firms and receives an aliquot share $D_t(j)$ of aggregate profits D_t, i.e., the sum of dividends of all retailers $D_{r,t}$, intermediate private banks $D_{f,t}$, intermediate non-financial firms $D_{m,t}$, and capital producers $D_{k,t}$. Thus, $\int_0^1 D_t(j) = D_t := \int_0^1 (D_{r,t}(i) + D_{b,t}(i) + D_{m,t}(i) + D_{k,t}(i))di$, where i indexes an individual firm in each sector. Households pay/receive T_t lump-sum transfers.

For tractability, all households are identical and choose consumption and investment similarly. Dropping the j subscript, we can write the real budget constraint for each household as:

$$C_t + B_{t+1} \leq W_t L_t + R_t B_t + T_t + D_t \tag{6}$$

Each household solves (4) under the budget constraint (6). The solution to this maximization problem gives us the following Euler equation, which describes the evolution of consumption along an optimal path:

$$1 = \beta \mathbb{E}_t \left[\frac{\lambda_{c,t+1}}{\lambda_{c,t}} R_{t+1} \right] \tag{7}$$

where $\lambda_{c,t}$ represents the marginal lifetime discounted utility function at t. Eq. (7) indicates that, at the optimum, each consumer is indifferent to consuming one more unit today and saving that unit (by buying bonds) to consume in the future.

Assuming internal habit formation yields:

$$\lambda_{c,t} = \frac{1}{C_t - hC_{t-1}} - \beta h \mathbb{E}_t \left[\frac{1}{C_{t+1} - hC_t} \right] \tag{8}$$

Thus, we define the stochastic real discount factor for the entire economy from period t to $t+i$ as:

$$\Lambda_{t,t+i} := \beta^i \frac{\lambda_{c,t+i}}{\lambda_{c,t}} \tag{9}$$

2.3. Financial intermediates

This section presents the financial intermediate's problem assuming that the Central Bank does not apply unconventional monetary policy, i.e., it does not directly lend to non-financial firms. We relax this hypothesis in the next section.

We assume an infinite continuum of financial intermediates indexed by j. Each intermediate recovers a quantity $B_{t+1}(j)$ of deposits from households, which pays a gross interest rate R_{t+1}, and issues a quantity $Z_t(j)$ of financial claims to non-financial producers at a real price of Q_t per claim. In reality, the Central Bank also issues claims, and we could differentiate private claims $Z_{p,t}$ and government claims $Z_{g,t}$; however, for the sake of presentation, we abstract from this distinction in this section.

Denote $\Omega_t(j)$ as the net worth of banker j in period t such that:

$$\Omega_t(j) = Q_t Z_t(j) - B_{t+1}(j) \tag{10}$$

Assets acquired by bankers earn a rate of return $R_{k,t+1}$ on claims; thus, using Eq. (10), bankers' wealth at period $t+1$ is:

$$\Omega_{t+1}(j) = (R_{k,t+1} - R_{t+1}) Q_t Z_t(j) + R_{t+1}\Omega_t(j) \tag{11}$$

Note the difference in subscripts between the banker rate of return ($R_{k,t+1}$) and the gross interest rate (R_{t+1}).

We assume that bankers cannot default on their loans; therefore, a banker j operates if and only if the following condition holds:

$$\mathbb{E}_t \Lambda_{t,t+1+i} \left(R_{k,t+1+i} - R_{t+1+i} \right) \geq 0, \quad i \geq 0 \tag{12}$$

where $\Lambda_{t,t+1+i}$ is defined as in (9). In other words, if a banker must borrow more than its income, then it will not remain a banker.

In each period t, a fraction f of household members are bankers; the remaining proportion are workers. We assume that a fraction θ of bankers in the current period remains bankers in the next period. That is, $(1-\theta)f$ bankers become workers, and a similar number of workers become bankers. As explained in Gertler and Karadi (2011), this assertion implies that the average "survival time" for a banker at any period is $\frac{1}{1-\theta}$, which ensures that bankers cannot fund all investments from their capital and that the relative proportion of each type of household remains constant over time. Note that bankers do not need labor for their activities; therefore, the epidemic does not directly affect them.

Accordingly, each banker has the following expected discounted terminal wealth:

$$V_t(j) = \sum_{i=0}^{\infty} (1-\theta)\theta^i \Lambda_{t,t+1+i} \left((R_{k,t+1+i} - R_{t+1+i}) Q_{t+i} Z_{t+i}(j) + R_{t+1+i}\Omega_{t+i}(j) \right) \tag{13}$$

Under condition (12), bankers may want to increase their assets indefinitely by borrowing more and more funds from households. Furthermore, a banker can divert funds, i.e., transfer a fraction or even the totality of assets to its own household for personal gain. Creditors are aware of this possibility as they know that there may be a fraction λ of funds that will never be recovered; however, they can impose a borrowing constraint to ensure that bankers do not divert all funds. Therefore, households are willing to supply funds to a bank only if the banker's expected discounted terminal wealth $V_t(j)$ is at least as large as the banker's gain form diverting funds $\lambda Q_t Z_t(j)$:

$$V_t(j) \geq \lambda Q_t Z_t(j) \tag{14}$$

where in each period t, banker j chooses $Z_t(j)$ to maximize (13) subject to constraint (14). Effectively, Eq. (14) prevents bankers from diverting all their claims income to their households. See Gertler and Karadi (2011) for an extensive explanation of this condition.

We denote the leverage ratio of banker j as $\phi_t(j)$ and we define it as:

$$\phi_t(j) := \frac{Q_t Z_t(j)}{\Omega_t(j)} \tag{15}$$

The leverage ratio is the value of the total loans of a banker to non-financial producers divided by that banker's net worth. It is a measure of the proportion of worth that a banker lends. Note that the leverage ratio can be greater than one (e.g., bankers can lend more than they have), depending on interest rates.

Following Gertler and Karadi (2011), if constraint (14) is binding, the interior solution is:

$$\nu_t = \mathbb{E}_t \Lambda_{t,t+1} \Gamma_{t+1} \left(R_{k,t+1} - R_{t+1} \right), \quad \eta_t = \mathbb{E}_t \Lambda_{t,t+1} \Gamma_{t+1} R_{t+1} \tag{16}$$

$$\Gamma_{t+1} = 1 - \theta + \theta \left(\nu_{t+1}\phi_{t+1}(j) + \eta_{t+1} \right), \quad \phi_t(j) = \frac{\eta_t}{\lambda - \nu_t} \tag{17}$$

where ν represents the expected discounted marginal value that the banker gains by expanding claims. η represents the expected marginal value of an extra unit of wealth; see Appendix B for details of its derivation.

Provided $0 < \nu_t < \lambda$, the incentive constraint holds, and the banker will increase its assets. In contrast, when $\nu_t > \lambda$, the incentive constraint is not binding, and the expected discounted value of the banker consistently exceeds gains from diverting funds.

Aggregating the wealth of all existing bankers, we have the following:

$$\Omega_{t+1} = \left(\left(R_{k,t+1} - R_{t+1} \right) \phi_t + R_{t+1} \right) \Omega_t \tag{18}$$

Since all bankers are created equal and choose the same quantity of claims, their choice of $Z_t(j)$ will not depend upon j, nor deposits $B_t(j)$. Then ϕ_t is independent of j.

Given that the probability of a banker at time t remaining a banker at time $t+1$ is θ, then at each date t, not all bankers remain bankers to the following period, and a portion of households become new bankers. We assume that bankers who exit give their earnings to their household, and the household gives the new banker startup funds equal to a fraction $\frac{\epsilon}{1-\theta}$ of the value of assets that existing bankers earned in their last operating period.

Accordingly, the total net worth of all bankers is the sum of the existing bankers and new bankers, such that:

$$\Omega_t = \theta \left(\left(R_{k,t} - R_t \right) \phi_{t-1} + R_t \right) \Omega_{t-1} + \epsilon Q_t Z_{t-1} \tag{19}$$

2.4. Central Bank and public loans

Thus far, we have assumed that only private banks receive deposits from households (B_t) and lend funds to intermediate producers (Z_t). This subsection relaxes this assumption to consider a Central Bank which conducts unconventional monetary policy, managing the epidemic by issuing bonds and lending money to non-financial firms.

As Gertler and Kiyotaki (2010) explained, the Central Bank may behave in many ways. Since we aim to study how the public authority may fight an epidemic crisis using public loans, we assume that the Central Bank issues government bonds $B_{g,t}$ to consumers at gross interest rate R_t and – using that income subject to its budget constraint – issues financial claims $Z_{g,t}$ to intermediate non-financial producers at price Q_t, for which the government earns a rate of return $R_{k,t+1}$.

Let $Q_t Z_{p,t}$ be the value of assets coming from private banks, $Q_t Z_{g,t}$ the value of assets coming from the Central Bank, and $Q_t Z_t$ the total value of intermediate assets (i.e., the sum of assets from private and Central banks). In our model, borrowers and lenders view private deposits/claims and government bonds/claims as equivalent because they have the same price and interest rates.

The Central Bank has both an advantage and a disadvantage concerning private lenders. We assume that government assets come with an efficiency cost of τ per claim. As Gertler and Karadi (2011) and Gertler and Kiyotaki (2010) explained, the government faces additional costs of evaluating and monitoring borrowers that private banks do not have because private banks possess specific knowledge of the market not readily available to the Central Bank. Simultaneously, we assume that the government can always honor its debts, with no limitations in the number of bonds it can supply.[5] Therefore, it is not subject to an incentive constraint. Consequently, the Central Bank may also issue government debt to financial intermediates without constraint. Private banks fund government bonds by issuing household deposits at the same rate as they lend them from the Central Bank; thus, only private assets financed with private banks face the incentive constraint.

Suppose the Central Bank lends a fraction ψ_t of total credit in each period. Then, using Eq. (15), we write the total value of intermediate assets as:

$$Q_t Z_t = \phi_t \Omega_t + \psi_t Q_t Z_t = \Phi_t \Omega_t \tag{20}$$

where $\Phi_t := \frac{\phi_t}{1-\psi_t}$ is the leverage ratio for total intermediate funds (public and private). The choice of ψ_t will be explained in Section 2.8.

2.5. Intermediate non-financial firms

Suppose a continuum exists of perfectly competitive, homogeneous intermediate goods producers that produce a differentiated non-financial good sold at the real price $P_{m,t}$. Following Gertler and Karadi (2011), we do not introduce price stickiness through intermediate goods producers; instead, we do so by assuming that retailers are monopolistic. Each intermediate goods producer uses two inputs: labor L and capital K.

Following Gertler and Kiyotaki (2010), we assume that at the end of period t, each intermediate producer acquires a quantity K_{t+1} of capital from the capital producers to be used in production in time $t+1$. After production in period $t+1$, the firm may sell non-depreciated capital back to the capital producer. Thus, intermediate-goods firms face a static problem, solving their profit maximization problem one period at a time rather than maximizing expected profit over the firm's lifetime.

Producers of intermediate goods finance physical capital by borrowing from financial intermediates, with private and public financial intermediaries being perfect substitutes in their eyes. Note that borrowers are not constrained by the number of claims Z_t they want to issue. However, intermediate private banks are constrained by the funds they obtain from households, and thus, the interest rate $R_{k,t}$ indirectly affects the intermediate-goods producer's dynamics.

Each goods producer then issues a quantity Z_t of capital claims, where each claim equals one unit of capital $Z_t = K_{t+1}$, and the price per unit capital is Q_t. It follows that $Q_t K_{t+1} = Q_t Z_t$.

Recall that goods producers are homogeneous, and all behave in the same fashion. We can then write the quantity of intermediate non-financial goods $Y_{m,t}$ produced by the representative physical goods producer at time t as a Cobb–Douglas production function involving capital and labor, such that:

$$Y_{m,t} := K_t^{\alpha} L_t^{1-\alpha} \tag{21}$$

where the subscript m differentiates intermediate-goods ($Y_{m,t}$) from final goods (Y_t), and α is the elasticity of production with respect to capital. We assume that retailers are monopolistic; thus, one unit of intermediate-good $Y_{m,t}$ does not necessarily equal one unit of final good Y_t. These quantities are related by the equation $Y_{m,t} = v_{p,t} Y_t$ at equilibrium, where $v_{p,t}$ is the price dispersion of the aggregated final good (see Appendix B for details). Furthermore, we assume no stochastic shocks and abstract here from quality capital shocks as in Merton (1973) and a total factor productivity shock as in classic DSGE models (Smets and Wouters, 2007).

Each goods producer chooses quantities of labor and capital to maximize its profit. The solution to this problem yields the following first-order conditions:

$$W_t = (1-\alpha) P_{m,t} \frac{Y_{m,t}}{L_t} \tag{22}$$

$$R_{k,t} = \frac{\alpha P_{m,t} \frac{Y_{m,t}}{K_t} + (1-\delta) Q_t}{Q_{t-1}} \tag{23}$$

where δ is the capital depreciation rate. As we are in a perfect competitive framework, Eqs. (22) and (23) establish that intermediate-good producers choose the quantity of labor to equate real wages and the marginal product of labor. They also choose the quantity of capital such that the capital real price equals the net return after depreciation. At equilibrium, the quantity of labor is given by the share of non-infected workers, while wages are determined by Eq. (22).

2.6. Capital producers

A continuum of perfectly competitive, homogeneous capital producers firms exists. At the end of each period t, capital producers produce capital by buying final goods from retailers (i.e., investing I_t). They purchase non-depreciated capital from intermediate-good producers at Q_t, and sell capital to intermediate goods producers at Q_t. Total aggregate capital accumulates in the following fashion:

$$K_{t+1} := (1-\delta) K_t + I_t \tag{24}$$

where δ is the capital depreciation rate.

Furthermore, we assume that producing capital faces an adjustment cost associated with changing the level of investment; thus, capital

---

[5] By abstracting from solvency problems, we assume that the government can always print money to pay its debts. Solvency problems can emerge and be aggravated by sovereign debt and credit-rating agencies. We leave this for future work.

producer profit can be written as[6]:

$$D_{k,t} = \left((Q_t - 1)I_t - f\left(\frac{I_t}{I_{t-1}}\right) I_t \right) \tag{25}$$

A representative capital producer chooses the quantity of capital investment, I_t, to maximize its discounted profits:

$$\mathbb{E}_t \sum_{i=0}^{\infty} \Lambda_{t,t+i} \left((Q_{t+i} - 1)I_{t+i} - f\left(\frac{I_{t+i}}{I_{t-1+i}}\right) I_{t+i} \right) \tag{26}$$

where the adjustment cost function ($f(\cdot)$) depends on net capital investment at times t and $t-1$. Specifically, it is defined as:

$$f\left(\frac{I_t}{I_{t-1}}\right) = \frac{\kappa}{2}\left(\frac{I_t}{I_{t-1}} - 1\right)^2, \kappa > 0 \tag{27}$$

The adjustment cost is zero at the steady-state, and this cost increases with temporal changes in investment.

The first-order condition for profit maximization yields:

$$Q_t = 1 + f\left(\frac{I_t}{I_{t-1}}\right) + f'\left(\frac{I_t}{I_{t-1}}\right)\frac{I_t}{I_{t-1}} - \mathbb{E}_t \Lambda_{t,t+1} f'\left(\frac{I_{t+1}}{I_t}\right)\left(\frac{I_{t+1}}{I_t}\right)^2 \tag{28}$$

This equation is the marginal Tobin's "Q", which, given asset prices, defines the optimal investment demand function. With no adjustment costs, $Q_t = 1$.

2.7. Retailers

Let there be a continuum of monopolistic *normal retailers* indexed by $h \in [0,1]$ and a continuum of perfectly competitive *super retailers* that purchase and assemble final goods produced by *normal retailers* to produce an aggregate final good that will be sold at P_t. We assume that *super retailers* are homogeneous and all behave in the same fashion (*normal retailers* are not treated as homogeneous).

The *super retailer* is characterized by the following CES production function:

$$Y_t := \left(\int_0^1 Y_t(h)^{\frac{\epsilon_p - 1}{\epsilon_p}} dh \right)^{\frac{\epsilon_p}{\epsilon_p - 1}} \tag{29}$$

where $Y_t(h)$ is final good produced by *normal retailer* h, and ϵ_p is the elasticity of substitution for choosing between *normal retailer* goods.

Given the prices of normal retailer goods $P_t(h)_{h\in[0,1]}$ and the final aggregated good price P_t, the *super retailer* chooses the quantities of *normal retailers* goods $(Y_t(h))_{h\in[0,1]}$ to maximize its profit. The solution yields the following demand function for good h:

$$Y_t(h) = \left(\frac{P_t(h)}{P_t}\right)^{-\epsilon_p} Y_t \quad \forall h \tag{30}$$

Notice that the production function of the *super retailer* includes constant returns to scale and that firms are perfectly competitive, meaning that firms experience zero profits at equilibrium; therefore, we obtain the following equation for the price of the final aggregate good:

$$P_t = \left(\int_0^1 P_t(h)^{1-\epsilon_p} dh \right)^{\frac{1}{1-\epsilon_p}}. \tag{31}$$

Each *normal retailer* h uses intermediate goods produced by the intermediate goods firms to "pack" the intermediate goods and sell them to the *super retailers* at $P_t(h)$. We assume that one unit of intermediate-good is required to produce one unit of normal final output; thus, the marginal cost for each *normal retailer* is the intermediate price $P_{m,t}$, which is the same for all *normal retailers*.

We introduce nominal price rigidity following Calvo (1983). A fraction $(1-\theta_p)$ of *normal retailers* can re-optimize their nominal price $(P_t(h) = P_t^*(h))$ in each period t, while the remaining fraction can only partially adjust their prices according to past inflation. If firm h cannot change its price for i periods, then its normalized price after i periods is:

$$\prod_{s=1}^{i} \Pi_{t+s-1}^{\chi} \frac{P_t(h)}{P_{t+i}} \tag{32}$$

where $\chi \in (0,1)$ reflects the price response to inflation and $\Pi_t := \frac{P_t}{P_{t-1}}$ represents the level of inflation from period $t-1$ to t.

Profits for *normal retailer* h at date t is then given by:

$$\left(\prod_{s=1}^{i} \Pi_{t+s-1}^{\chi} \frac{P_t(h)}{P_t} - P_{m,t} \right) Y_t(h) \tag{33}$$

Given the option, each *normal retailer* firm will choose to readjust its price. The choice of $P_t^*(h)$ does not depend on the specific household h because all firms that can choose their prices will do so in the same fashion. Furthermore, firms only consider future states in which re-optimization is not possible; thus, each firm h chooses $P_t(h)$ to maximize expected discounted profits:

$$\mathbb{E}_t \sum_{i=0}^{\infty} \theta_p^i \Lambda_{t,t+i} \left(\prod_{s=1}^{i} \Pi_{t+s-1}^{\chi} \frac{P_t(h)}{P_{t+i}} - P_{m,t+i} \right) Y_{t+i}(h) \tag{34}$$

subject to Eq. (30).

The first-order condition of this problem yields:

$$\mathbb{E}_t \sum_{i=0}^{+\infty} \theta_p^i \Lambda_{t,t+i} Y_{t+i}(h) \left(\frac{P_t^*}{P_{t+1}} \prod_{s=1}^{i} \Pi_{t+s-1}^{\chi} - \mathcal{M} P_{m,t+i} \right) = 0 \tag{35}$$

where $\mathcal{M} := \frac{\epsilon_p}{\epsilon_p - 1}$ is the desired price markup, absent from inflation. This equation gives the optimal price-setting condition.

Finally, a fraction $(1-\theta_p)$ of *normal retailers* can optimize prices while the rest index prices to past inflation; thus, Eq. (31) can be written as:

$$P_t^{1-\epsilon} = \theta_p \left(\Pi_{t-1}^{\chi} P_{t-1} \right)^{1-\epsilon} + (1-\theta_p)\left(P_t^*\right)^{1-\epsilon} \tag{36}$$

2.8. Government, monetary policy and the market clearing condition

The government issues public debt $B_{g,t}$ to households for which it pays a gross interest rate R_t, purchases claims $Z_{g,t}$ from non-financial firms at Q_t and gross interest rate of return of $R_{k,t}$, recovers/pays lump-sum taxes, and applies its expenditures G_t.

As explained in Section 2.4, through the Central Bank, the government lends a fraction ψ_t of total credit to non-financial intermediates in each period. However, government assets come with an inefficiency cost of $\tau \in [0,1]$ per claim (recall that private banks are more efficient as they have better access to market information). Then government expenditure on financial intermediation is given by $(1+\tau)\psi_t Q_t K_{t+1}$.

We also assume that the share of government consumption out of final goods ω_g is constant, such that $G_t := \omega_g Y_t$. Assuming that transfers automatically adjust at each date, the government faces the following budget constraint:

$$G_t + \tau\psi_t Q_t K_{t+1} = T_t + \left(R_{k,t} - R_t\right) B_{g,t-1} \tag{37}$$

Eq. (37) equates all expenditures (final good consumption and expenditures in inefficiency costs) to revenue (lump-sum taxes and the difference between what the government gains from claims and its debt). We assume that the totality of government claims are financed with debt, where $B_{g,t} = \psi_{t-1} Q_{t-1} Z_{t-1}$. This rule does not apply to the inefficiency cost of implementing claims financed with lump-sum taxes and profits from financial intermediation.

Unconventional monetary policy ψ_t is set in the following manner:

$$\psi_t = \bar{\psi}_t + \omega \mathbb{E}_t \left[\left(log R_{k,t+1} - log R_{t+1} \right) - \left(log R_k - log R \right) \right] \tag{38}$$

where $\bar{\psi}_t$ is defined as our "*epi loans*", $\omega > 0$ is the Central Bank credit feedback parameter, and $log R_k - log R$ is the steady-state risk

---

[6] See Appendix B for a detailed derivation.

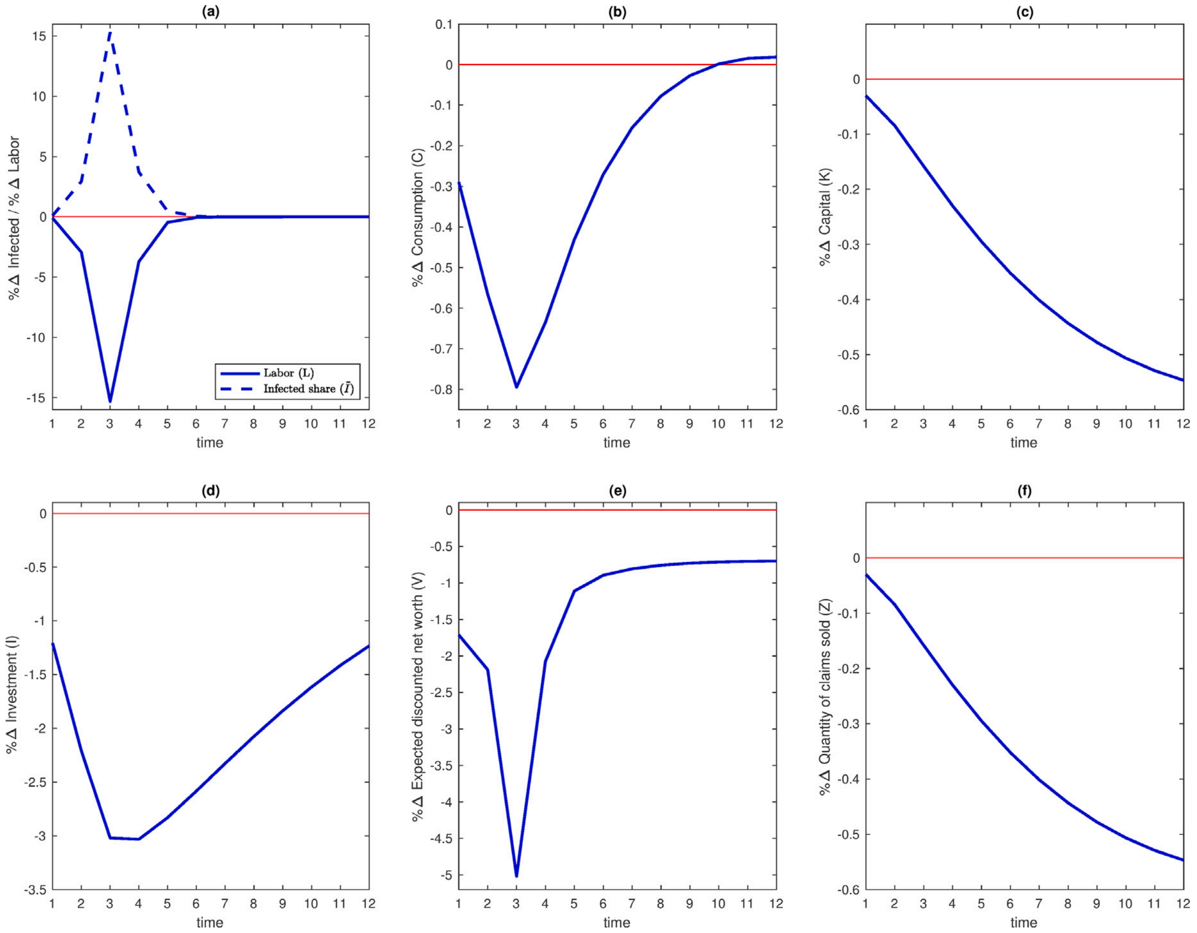

Fig. 3. Baseline results for labor (a), consumption (b), capital (c), investment (d), expected discounted net worth (e), and the number of claims sold (f). Reported values are the percent deviation from the no-disease case (i.e., the long-term steady-state value without disease). The red line corresponds to a zero percent change. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

premium. The feedback parameter governs the intensity of the Central Bank's reaction to changes in the spread relative to the steady-state risk premium. When the risk premium is larger than its steady-state, the Central Bank expands its credit with the larger the ω, and the greater the credit expansion. We impose a zero lower bound condition for ψ_t to avoid negative values. Our baseline simulations treat $\bar{\psi}_t$ as a constant equal to zero. We then relax this assumption and take $\bar{\psi}_t$ as a deterministic, exogenous shock to study the ability of our "*epi loans*" to alleviate the negative effects of the epidemic.

The Central Bank also conducts conventional monetary policy by setting nominal interest rates, i_t, following a Taylor rule of the form:

$$1 + i_t = \left(1 + i_{t-1}\right)^{\phi_i} \left(\frac{1}{\beta} \left(\frac{\Pi_t}{\Pi_{ss}} \right)^{\phi_\pi} \left(\frac{Y_t}{Y_{ss}} \right)^{\phi_y} \right)^{1-\phi_i}, \tag{39}$$

where Π_{ss} is the steady-state of inflation, and Y_{ss} is the steady-state level of GDP obtained without the epidemic. The Taylor rule determines how the Central Bank fixes the nominal interest rate. Parameters ϕ_y and ϕ_π measure the Central Bank's response to the output gap and changes in inflation. Parameter ϕ_i measures the persistence of the Taylor rule over time. As there is a possibility for negative interest rates, we have also included a zero lower bound condition for i to ensure positive nominal interest rates. This condition reinforces the use of unconventional monetary policy; Keynes (1936), Fuhrer and Madigan (1997), Eggertsson and Woodford (2003), and McKay and Wieland (2021) (among others) emphasized that conventional monetary policy may be insufficient to fight an economic recession in a "liquidity trap".

Finally, we have the following Fisher relation that links nominal interest rates, fixed by the Central Bank, to the gross real interest rate, fixed by the market:

$$1 + i_t = R_{t+1} \mathbb{E}_t \Pi_{t+1} \tag{40}$$

Market clearing conditions established that production is divided between consumption, investment, government expenditures in goods, and expenses associated with financial intervention from the government.

$$Y_t = C_t + I_t + f\left(\frac{I_t}{I_{t-1}} \right) I_t + G_t + \tau \psi_t Q_t K_{t+1} \tag{41}$$

Eq. (41) closes the model.

3. Parameter calibration and simulation analysis

Details on model aggregation and calculation of the steady-state values are given in Appendix B. Each period corresponds to a quarter. Baseline parameter values are summarized in Table 3.

Economic parameters were chosen to illustrate the effects of an epidemic on a model economy. Calibration of our baseline economic parameters follows Smets and Wouters (2007) and Gertler and Karadi (2011) for the US economy. Parameters' values are consistent with

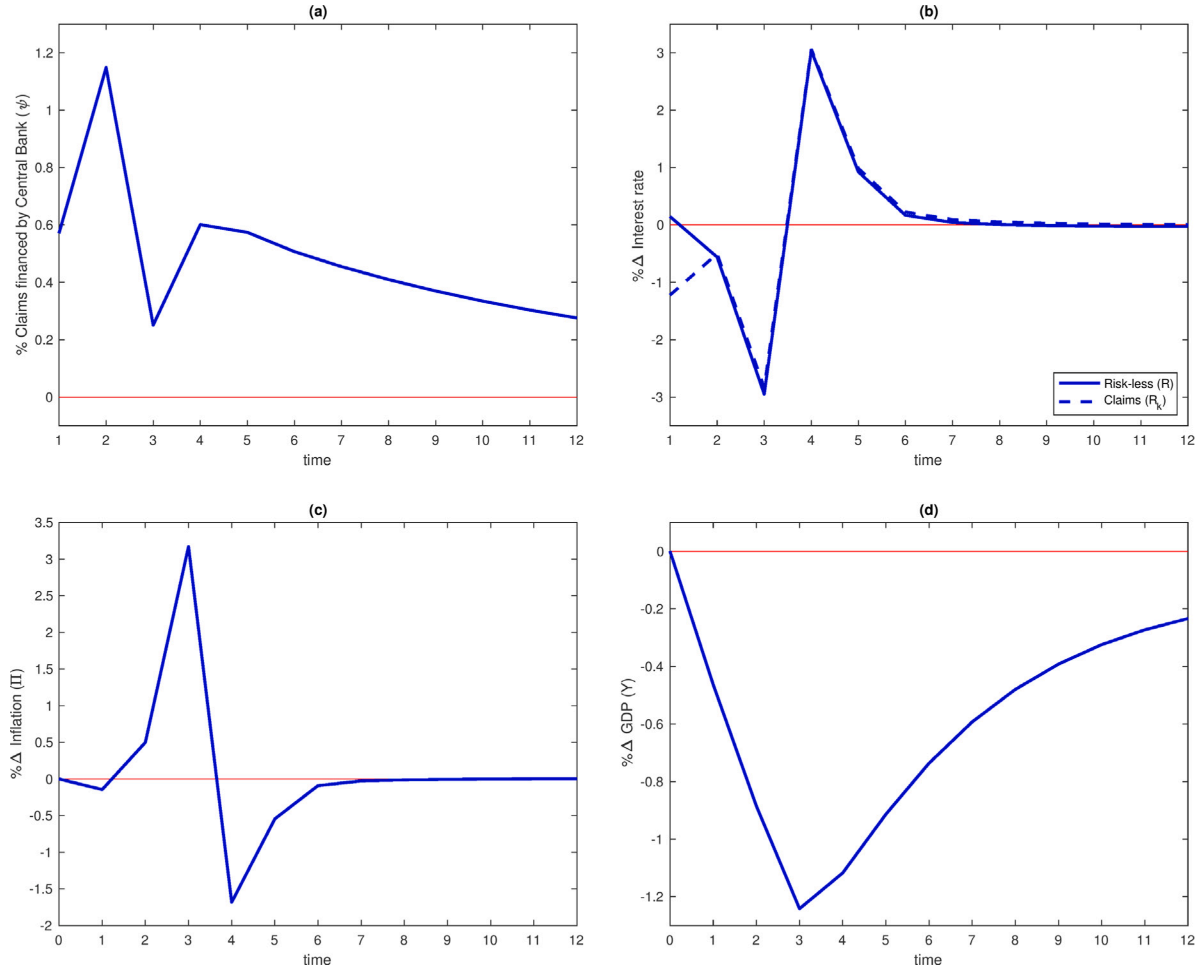

Fig. 4. Baseline results for the fraction of total credits financed by the Central Bank (a), interest rates (b), inflation (c), and GDP (d). Reported values are the percent deviation from the no-disease case (i.e., the long-term steady-state value without disease). For comparison, the red line corresponds to a zero percent change. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

long-run growth and microeconomic observations. These values are widely used as calibrations in DSGE models (Galí, 2015), most recently by Bianchi (2020) and Lim and McNelis (2018). Furthermore, Smets and Wouters (2007) and Gertler and Karadi (2011) are quite influential papers in the field; thus, calibrating our model in this way allows our results to be more comparable to other conventional DSGE models.

Specifically, the discount factor β is set to ensure a 4% annual interest rate, with the elasticity of substitution among final goods taken to yield a steady-state price markup of 31%. The output of elasticity of capital α is calibrated assuming a "labor share" of approximately $2/3$, and the bankers' survival rate is fixed at 0.975, which assumes that bankers remain bankers on average for 10 years. Following Gertler and Karadi (2011), the private banks' parameters, λ and ϵ, are fixed to meet the following targets: a risk premium steady-state of 100 basis points and a steady-state leverage ratio of 4.[7] We assume the disease has a basic reproductive number (R_0) of 2 with a recovery time of 25 days. Baseline epidemiological parameters were chosen to illustrate a full epidemic cycle within three years, with a peak infection of about 15% of the total population; they are *not* meant to represent a specific disease. Our baseline results should be interpreted as a stylized exercise only.

Model simulation proceeds in three steps. First, we solve for the steady-state equilibrium values of all state variables in the economic block of the model (Appendix B). Second, we calculate the trajectories of the number of susceptible, infected, and recovered individuals given the epidemic parameters and an initial proportion of infected of 0.1%. The epidemic dynamics are solved using a first-order Euler approximation for a time horizon of three years. While epidemic models can exhibit complex non-linearities, a first-order Euler approximation is sufficient to accurately solve the basic SIR model forms (Brauer and Castillo-Chavez, 2012; Lenhart and Workman, 2007). Even at fine time scales, economic models usually operate at larger time scales than epidemiological ones (e.g., monthly or quarterly versus daily); therefore, to align the time scales of the two models, we sample every 90th value of the disease simulation to provide quarterly observations of disease incidence. Finally, we used the trajectory of infected individuals as a deterministic, permanent shock to the real economy. In this way, agents possess perfect foresight regarding the future states of the epidemic when computing their optimal solutions. We initialize the model's economic block from a set of initial conditions (steady-state, equilibrium values, and only susceptible individuals), perturb the system by "infecting" 0.1 percent of the population, and measure the return of both economic and epidemic blocks to their steady-state

---

[7] The steady-state of this variable depends on λ and ϵ. Like Gertler and Karadi (2011), we calibrate these parameters by fixing the steady-state value ($\phi = 4$) and solve for the values of the parameters that result in this value. As pointed out by a reviewer, one could solve in the opposite direction, assuming a change in λ and ϵ to hit a value of 4 for the steady-state value of ϕ.

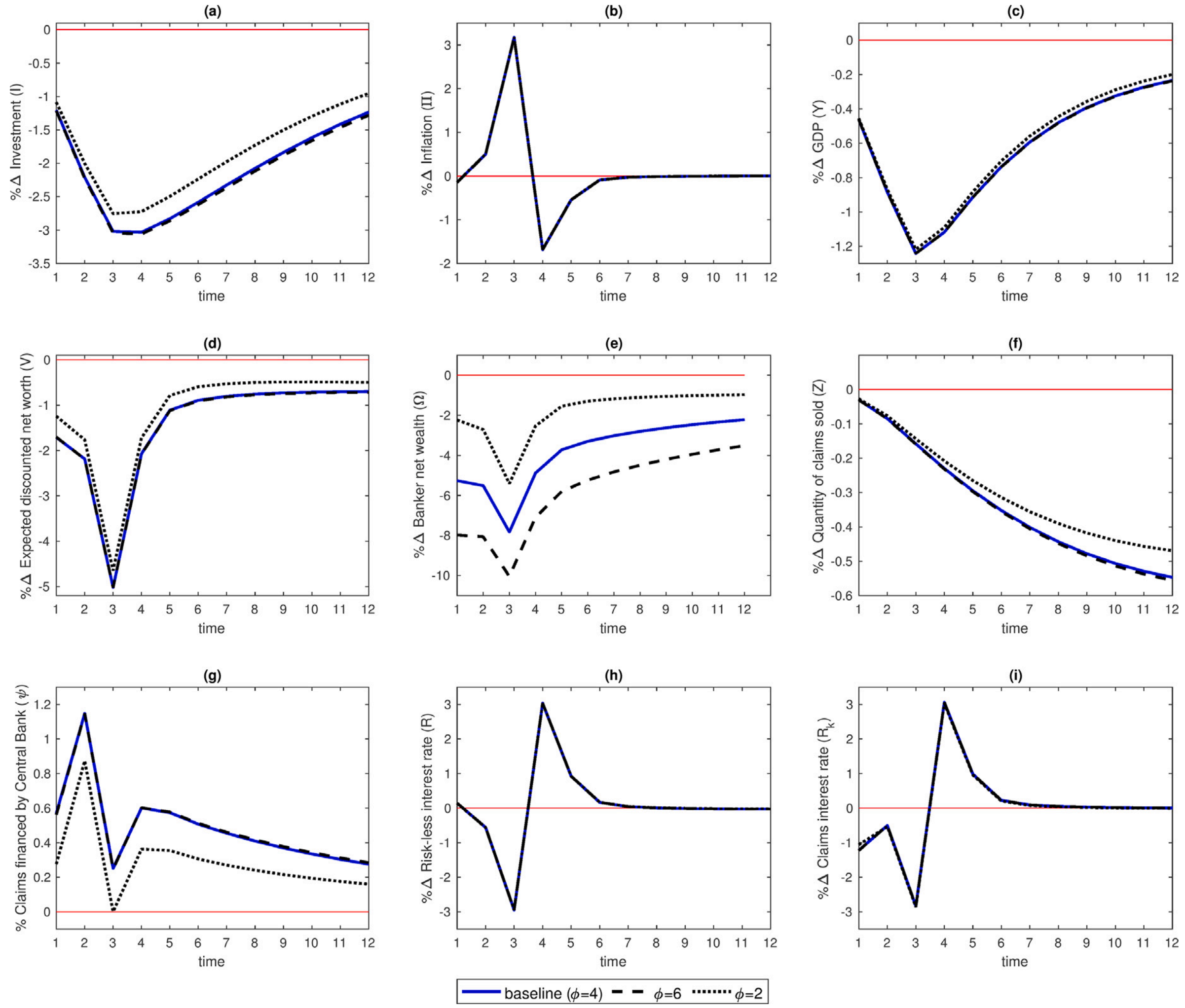

Fig. 5. Model sensitivity to the steady-state leverage ratio (ϕ). Results are reported as the percent change from the no-disease case. Line style and color indicate the trajectories with different steady-state leverage parameters: $\phi = 2$ (dotted, black), $\phi = 4$ (solid, blue; baseline), and $\phi = 6$ (dashed, black). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

values. Trajectories are obtained using a Levenberg–Marquardt mixed complementarity problem solver (Kanzow and Petra, 2004), which allows us to consider the inequality constraints placed on the share of claims financed by the Central Bank ψ_t and the nominal interest rate i_t.

To test the effectiveness of unconventional monetary policy to mitigate the epidemic crisis, we first establish a baseline model scenario with an epidemic and study the economic consequences of changes in the epidemic structure. We then implement unconventional monetary policy by testing the sensitivity of the model to the steady-state leverage ratio for private banks, the intensity of the reaction of the Central Bank to changes in the spread, and our "*epi loans*" policy. All model simulations were conducted in Dynare 4.6.1. All source codes and simulation data can be found on the Open Science Framework (osf.io/j7m65).

4. Results and discussion

This section is divided into three parts. First, we present our model's baseline results and the different pathways by which the epidemic affects the economy. Second, we evaluate the potential of monetary policies to remedy the economic burden of the epidemic. Finally, we apply our framework to a case study of COVID-19 in the US. Recall that, in the first two subsections, we fit the epidemic parameters not to represent a specific disease but to provide a characteristic illustration of the effects of an epidemic on the model economy.

For each result, we compare the trajectories of our economic variables to those in the absence of disease (or the "no-disease" case), corresponding to their steady-state equilibrium values. Our units are in percent changes from the no-disease case instead of absolute, standalone terms. When changing economic parameters in our policy analyses, we re-calculate the trajectories of the no-disease case to correspond to the new set of parameters.

4.1. Baseline results

Our baseline results are summarized in Figs. 3 and 4. For brevity, we focus on a set of core variables when discussing our results. By assumption, the epidemic decreases the quantity of available labor—only healthy individuals are allowed to work. The epidemic reaches its maximum severity in period 3. This effect on the labor market echoes throughout the economy, with declines in household consumption, non-financial intermediary capital, and capital producer investment following the labor trajectory. The first is a consequence of lost wages and equality in the market clearing condition. The latter two follow declines in production due to a lower workforce. Once the epidemic passes its peak, labor increases, and with it, all other variables converge toward their respective steady-state values, except for capital and claims, which need longer to adjust.

Regarding financial intermediaries, the epidemic primarily affects their expected discounted terminal wealth (V). Both components of

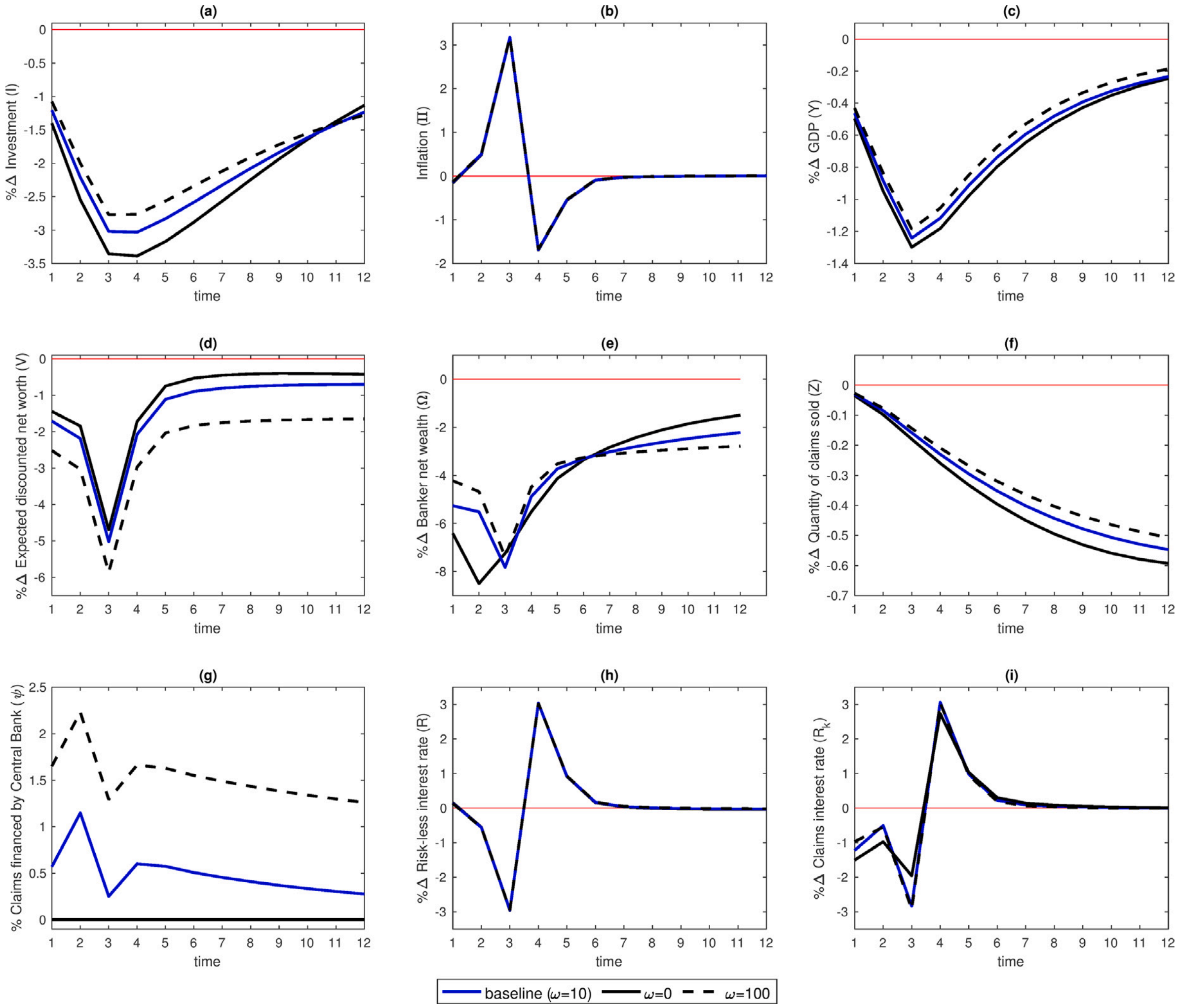

Fig. 6. Model sensitivity to the feedback parameter (ω). Note that results are reported as the percent change from the no-disease case. Line style and color indicate the trajectories with a different feedback parameter: $\omega = 10$ (solid, blue; baseline) and $\omega = 100$ (dashed, black). The solid black line represents the trajectory, assuming that no unconventional policy is performed ($\psi_t = 0, \forall t$). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

wealth – net worth (Ω) and claim issuing (QZ) – are affected because a decrease in capital translates to a decrease in claims demand ($K_{t+1} = Z_t$), which negatively affects claim prices (Q) compared to the no-disease case. We observe significant declines in GDP compared to the no-disease case, reaching a maximum loss in the 3rd quarter.

We observe a slight decrease in prices compared to the no-disease case at the beginning of the recession, which increases as the crisis worsens.[8] This model holds the standard relationships between supply and demand and prices. If the price decreases (increases) at equilibrium, then the demand (supply) side dominates as the DSGE framework shifts back to equilibrium. When the decrease in labor is mild at the start of the epidemic, consumers lose wage revenue, and demand decreases; however, at the beginning, this reduction in labor does not significantly affect production, thus supply decreases less than demand. Prices then decrease. As the epidemic intensifies and affects a larger workforce, supply decreases much more than demand, and we observe an increase in prices.[9]

The risk-less real interest rate follows movements in demand and inflation. Initially, the risk-less real interest rate is higher than its steady-state because even though the nominal interest rate decreases following the Taylor rule, this decrease is not enough to overcome reductions in the inflation rate (Eq. (40)) and the real interest rate increases. Nonetheless, when the epidemic worsens, the risk-less interest rate does not increase as fast as inflation because of the decrease in the output gap, and so it decreases. Capital real rates follow the movement of gross real risk-less interest rates.

Particularly interesting is that the Central Bank increases its share of total credits it finances (ψ_t) from the beginning of the crisis, regardless of the inflation rate. The Central Bank follows its rule given in Eq. (38). When the spread (i.e., the difference between the return on claims and interest on debt) gets far from its steady-state, the Central Bank

---

[8] We have defined the gross inflation rate as $\Pi_t = \frac{P_t}{P_{t-1}}$. In the no-disease case, we assume this variable to be equal to 1, meaning that when calculating a percentage deviation of Π_t concerning the no-disease, steady-state value, we obtain the nominal inflation rate ($\frac{P_t - P_{t-1}}{P_{t-1}}$).

[9] In a perfectly competitive market, we expect to see a larger than observed movement in prices; however, price movements are milder than that of a perfectly competitive framework because of sticky prices.

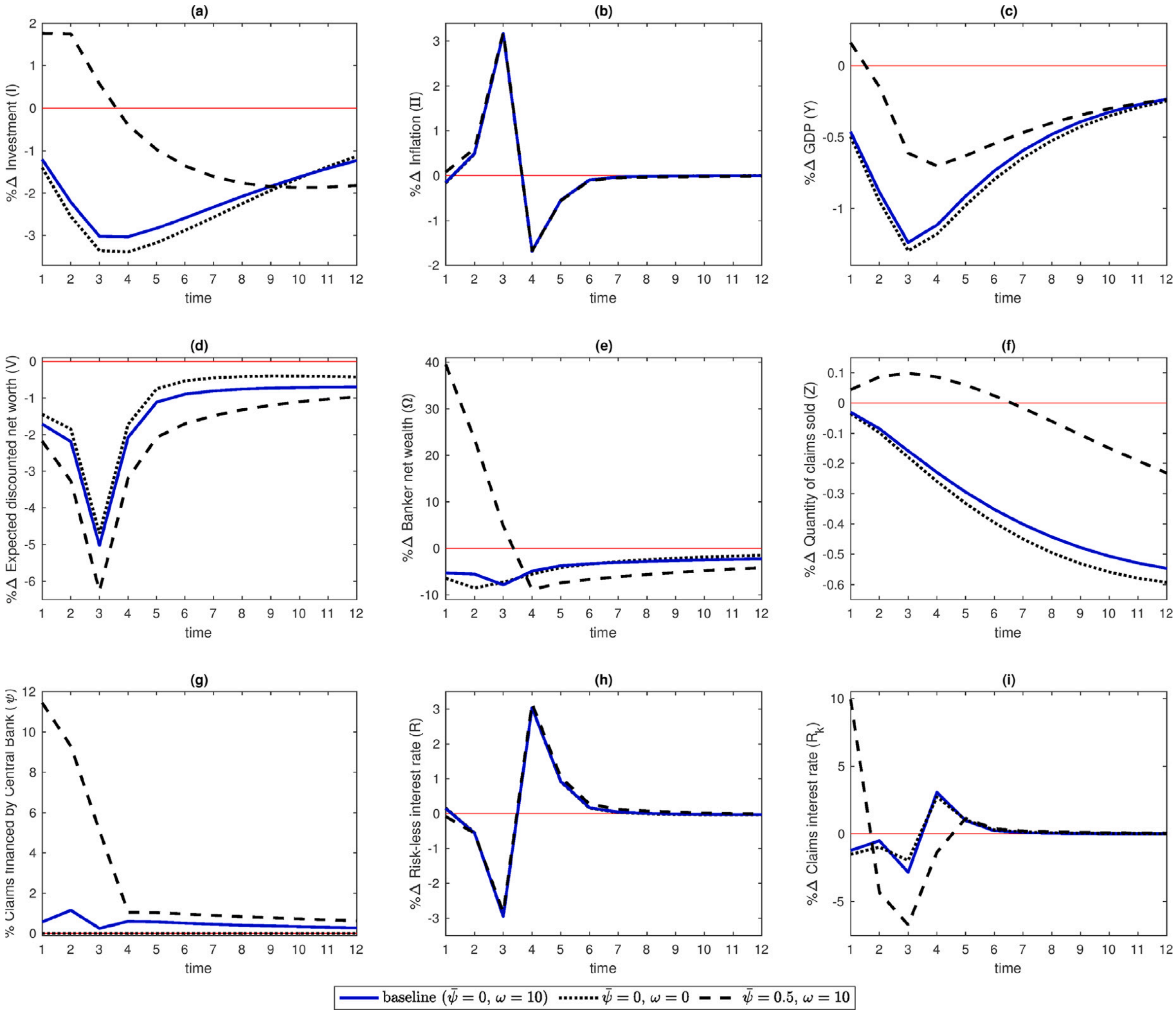

Fig. 7. *Epi loans* ($\bar{\psi}_t$). Results are reported as the percent change from the no-disease case. The solid blue line indicates the baseline model. The black, dashed line indicates a model implementing "*epi loans*" ($\bar{\psi}_t = 0.5$), and the black, dotted line indicates a scenario with no unconventional monetary policy ($\psi_t = 0$). Note that the Central Bank administers "*epi loans*" from period 1 until the peak of the epidemic (period 3). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

intervenes to compensate for losses in investment and production that follow labor declines.

4.2. Can monetary policy help fight the adverse effects of an epidemic?

We individually vary a set of economic parameters to answer this research question, holding all the other parameters at their baseline values. We concentrate our analysis on financial parameters only, specifically focusing on three policy instruments. It is important to notice that a change in these parameters could be understood as a form of *forward guidance* policy, as we are in a model with rational expectations; thus, any change in the parameters is known by all agents in the economy who act accordingly. In this model, changing the economic parameters never provokes a change in labor because we take labor as exogenously determined by the epidemic.

We start by first considering a change in the steady-state leverage ratio for private banks (ϕ in Eq. (15)), defined as the total loans that a private bank can issue compared to its net worth (Fig. 5). We assume that the Central Bank has the power to extend leverage ratio relief for private banks, as was the case for the Federal Reserve in January 2020 (FED, 2020b) and the European Central Bank in September 2020 June 2021 (ECB, 2020, 2021).[10] We find that the higher the leverage ratio, the higher the injection of funds from the Central Bank into the economy (ψ_t). This effect is observed because, with a higher leverage ratio at the steady-state, a greater probability arises for banks to issue claims. As this occurs, the spread in the interest rates increases, leading the Central Bank to insert money into the economy. We also find a compositional shift in bankers' wealth, with income increasing due to issuing a larger quantity of claims; however, we do not observe a marked change in GDP compared to the baseline scenario.

Second, we test the sensitivity of Central Bank claims funding in Eq. (38) to a change in the spread via the feedback parameter ω (Fig. 6). As the Central Bank responds more intensively to changes in the spread, it injects more funds into the economy at the beginning of the epidemic (when the difference in the spread is the highest) and then drops off in the later stages. Volatility in the spread variation is greater with ω, which affects the quantity and composition of bankers' wealth,

---

[10] A change in this value implies a change in the proportional transfer to new bankers (ϵ) and the fraction of claims income that can be diverted (λ). No other parameter is affected (Appendix B).

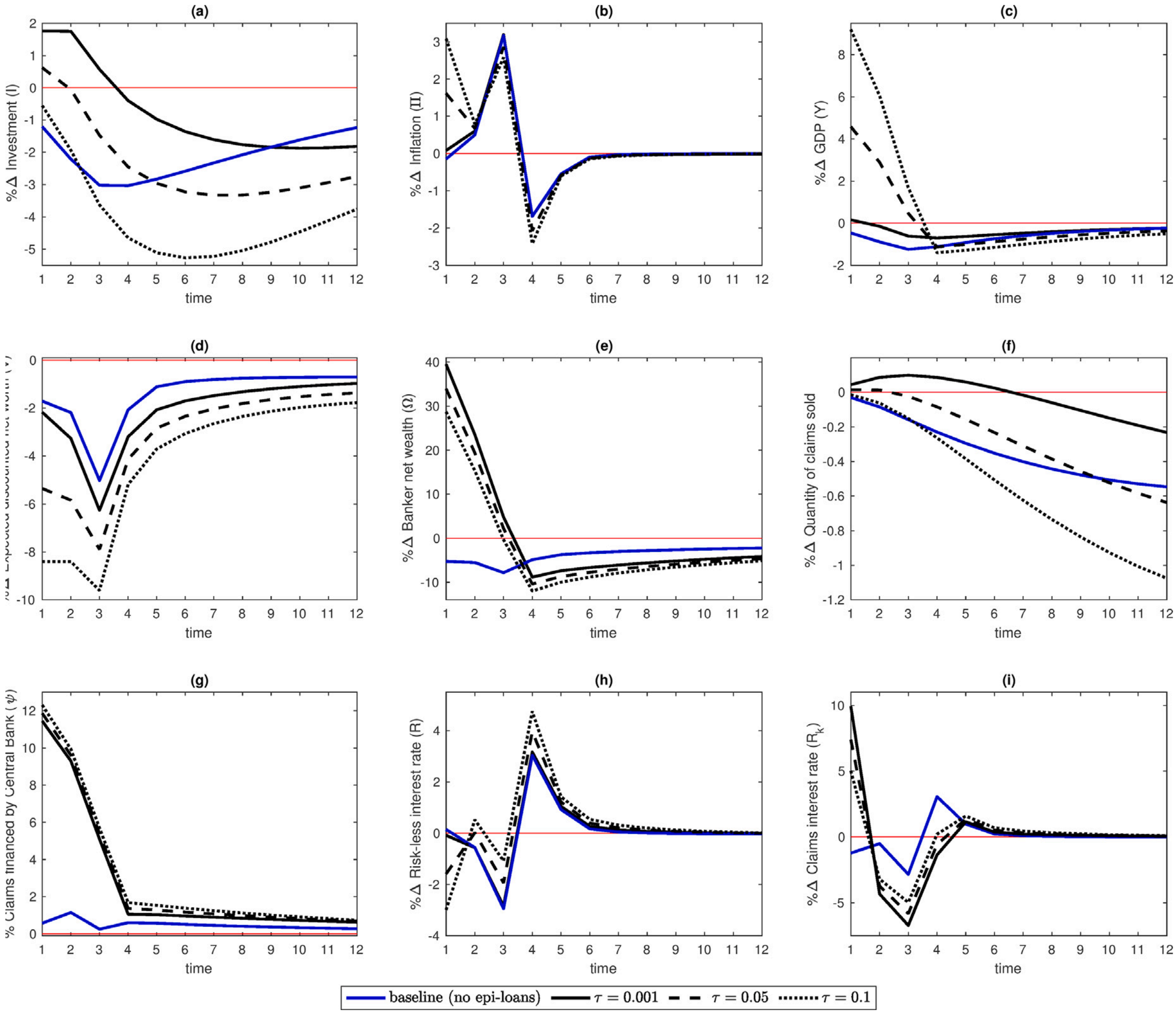

Fig. 8. Model sensitivity to the inefficiency cost (τ). Results are reported as the percent change from the no-disease case. The solid blue line indicates the baseline model. Others line style and color indicate the trajectories using "*epi loans*" with a different feedback parameter: $\tau = 0.001$ (solid, black), $\tau = 0.05$ (dashed, black), and $\tau = 0.1$ (dotted, black). The "*epi loans*" shock is the same at each case ($\bar{\psi}_t = 0.5$). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

with higher wealth stemming from a smaller decrease in net worth. The injection of funds from the Central Bank slightly incentivizes investment after a disease shock, which is why we find only a limited effect on GDP loss. The trajectories of GDP are almost the same whether the Central Bank implements an unconventional monetary policy or not. This result is important—contrary to Gertler and Karadi (2011), a credit policy focusing on the feedback parameter is no longer enough to moderate reductions in GDP.[11]

Finally, we evaluate the use of "*epi loans*" to mitigate the effects of the epidemic (Fig. 7), which takes the form of an exogenous shock on variable $\bar{\psi}_t$ in Eq. (38) and affects the share of total claims the Central Bank finances (ψ_t). We assume that the Central Bank (with a cost) administers liquidity directly to the real economy in the form of claims that are transformed (one-to-one) into capital, and it does so from the beginning of the epidemic to its peak (period 3). This shock is deterministic and forms a *forward guidance* policy as the Central Bank announces, in addition to reacting to the spread in interest rates, it will issue a fixed share of claims (50% in our simulation) over a certain period (from period 1 to period 3). Instead of giving money directly to households with no expectation of being repaid, the Central Bank increases its share of total claims issued, and firms subsequently purchase capital without passing through private banks. Thus our "*epi loans*" directly affect demand by incentivizing investment and should be thought of as expanding Central Bank intermediation rather than expanding the money supply.

With this policy, we observe a smaller reduction in GDP compared to two other cases, one with ($\omega = 10$, the baseline) and another without the use of unconventional monetary policy ($\psi_t = 0, \forall t$, Fig. 7). For the first periods of the epidemic, we even observe an absence of a recession, which should not be surprising given that any increase in ψ_t will increase GDP, which is government expenditure. It is important to note that when the disease is mild, and the production sector is not greatly affected, the application of "*epi loans*" is enough to increase total demand, thereby increasing investment, capital, claims demand, and claims sold by private banks. However, when the epidemic affects more people, the use of "*epi loans*", although helpful, is no longer enough to counteract the recession. We then observe a reduction in the expected discounted terminal wealth of banks. As a side effect, particularly at the beginning of the epidemic, we observe a stronger

---

[11] We simulate a model with a negative exogenous $AR(1)$ shock at the capital level in Appendix E, which, according to Gertler and Karadi (2011), represents a financial crisis shock. As shown, losses in GDP provoked by a $AR(1)$ capital shock could be lessened by this type of policy, which is not necessarily the case for a SIR labor shock.

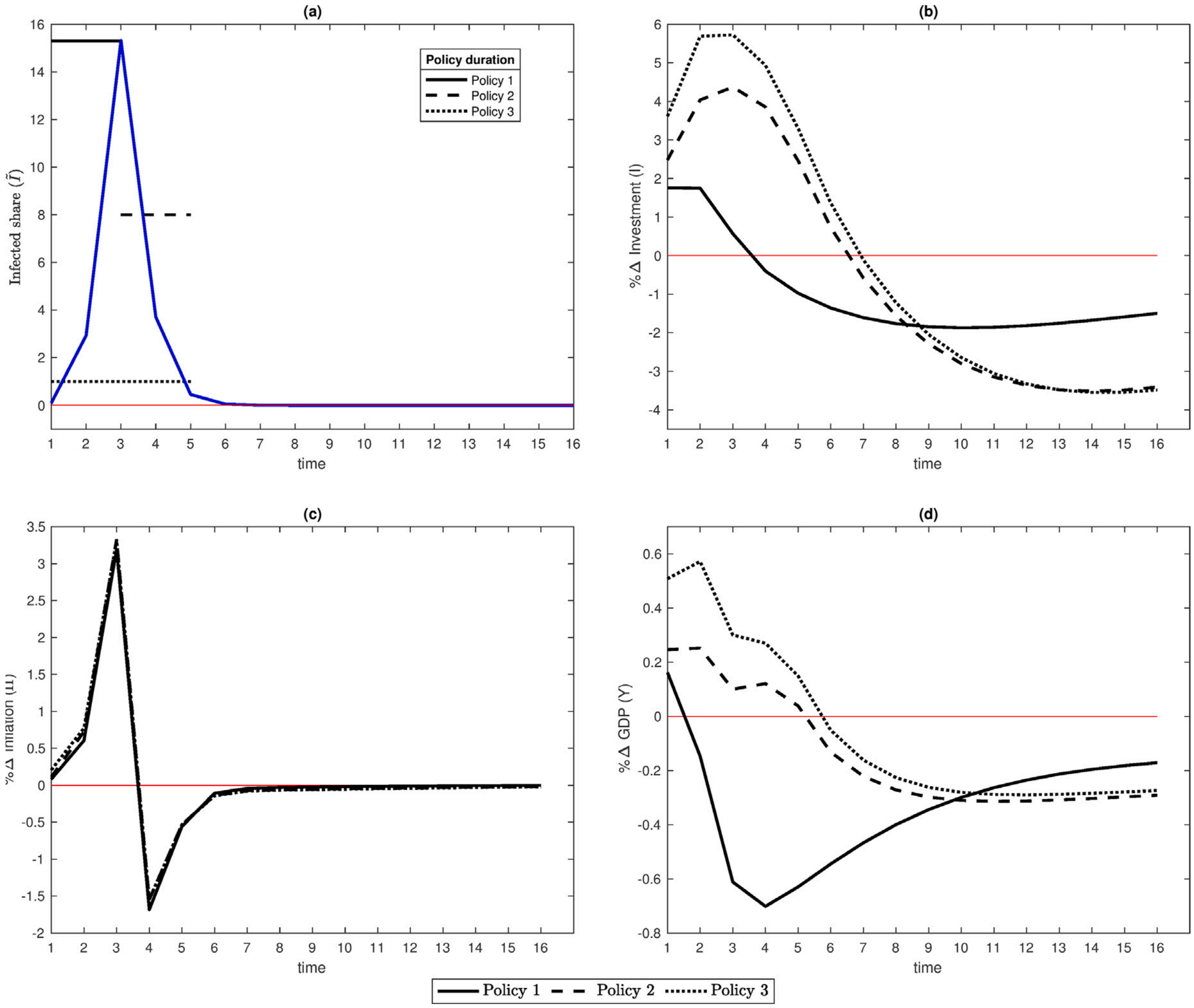

Fig. 9. Model sensitivity to different "*epi loans*" policies. Results are reported as the percent change from the no-disease case. The solid line (Policy 1) indicates a policy that applies "*epi loans*" from periods 1 to 3. The dashed line (Policy 2) indicates a policy that applies "*epi loans*" from periods 3 to 5. The dotted line (Policy 3) indicates a policy that applies "*epi loans*" from periods 1 to 5. The "*epi loans*" shock is the same in each case ($\bar{\psi}_t = 0.5$).

increase in inflation compared to the no-disease case. By increasing demand, we drive up prices; however, the peak of inflation does not change when applying an "*epi loans*" policy. Our results support Sharma et al. (2021), Céspedes et al. (2020), Kiley (2020), and Cardani et al. (2021).

A key aspect of the capability of "*epi loans*" to combat the recession is their capacity to incentivize investment in physical capital. One way to measure this capacity in our model is through the parameter τ, which represents the inefficiency cost of "*epi loans*". At equilibrium, this efficiency cost influences the market clearing condition, Eq. (41). We test the model's responsiveness to this parameter by comparing the performances of "*epi loans*" at different inefficiency cost levels (Fig. 8). At the beginning of the epidemic crisis, greater inefficiency provokes a stronger increase in GDP because the larger the inefficiency cost, the more a government must spend to implement "*epi loans*", and increased government expenses lead to the larger GDP. Nonetheless, an increase in GDP due to higher inefficiency costs does not translate to a similar increase in investment; instead, investment decreases with higher inefficiency costs because when inefficiency costs are high, the government increases its expenditures, which are paid in part with lump-sum taxes. Our Ricardian agents anticipate this, save more and decrease consumption and investment. This situation increases volatility on the spread in the interest rates; thus, the Central Bank takes a higher share of claims, thereby decreasing the number of claims sold by private banks.

Furthermore, when the Central Bank stops the "*epi loans*" policy, the larger the inefficiency cost, the deeper the recession. Moreover, as total demand increases with the inefficiency cost, we observe more substantial inflation with a higher inefficiency cost. This last result highlights that if "*epi loans*" are not correctly used, they may lead to a worse recession.

One might wonder about the optimal path of the trajectory of "*epi loans*", e.g., when and how much should be administered. Solving for the optimal trajectory exceeds the scope of this paper and would likely depend on a suite of epidemiological and economic parameters in addition to the inefficiency cost. Nonetheless, as a first step, we test two alternative timings of our "*epi loans*" policy in Fig. 9. In each case, the number of exogenous claims purchased by the Central Bank ($\bar{\psi}_t$) is the same (at 50%). In our initial policy (Policy 1), claims were purchased by the Central Bank from the start of the epidemic to its peak (periods 1 to 3). In our first alternative (Policy 2), claims are purchased for the same number of quarters as our initial policy but start at the peak of the epidemic (period 3 to 5). In our second alternative policy (Policy 3), claims are purchased from the beginning of the epidemic to the end (periods 1 to 5). Comparing the three policies, an early and lasting reaction of the Central Bank (Policy 3) postpones the recession, though

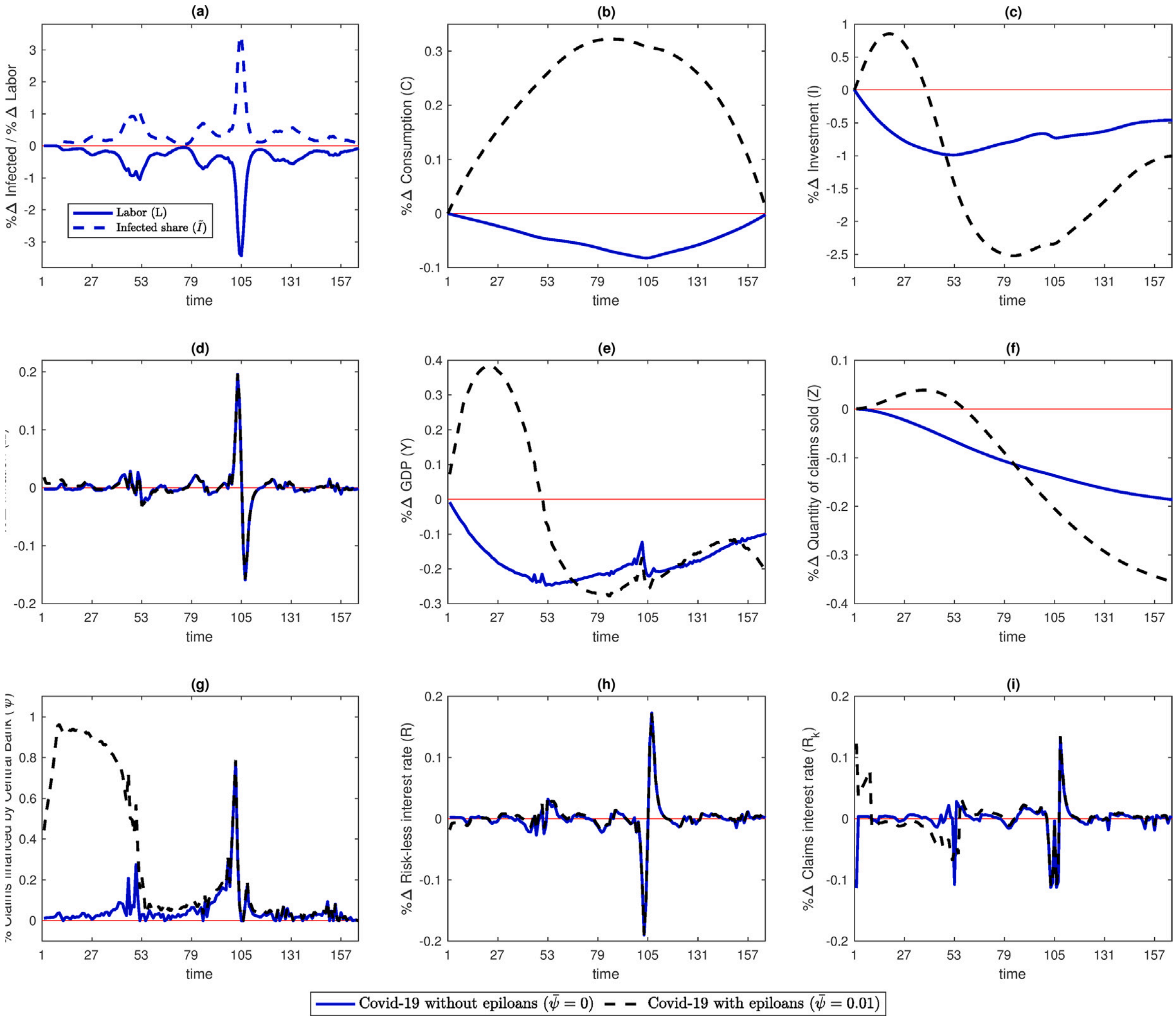

Fig. 10. Covid-19 case study. Results are reported as the percent change from the no-disease case. The solid blue line indicates the variable's trajectories that follow a COVID-19 infection. The dashed black line indicates the trajectories of the variables when implementing "*epi loans*" ($\bar{\psi}_t = 0.01$) from period 8 to 53. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

it leads to a deeper recession than the other policies once the "*epi loans*" are stopped. Regarding inflation, little difference is shown between the three policies.; however, in terms of cumulative GDP throughout the simulation, Policy 3 results in the fewest losses, followed by Policies 1 and 2. We leave a proper study of the optimal policy for future research.

4.3. Case study: Covid-19 in the United States

As an illustration of our framework, we calibrate the model to the US during COVID-19 to study the effects of the epidemic on the country's economy and evaluate the hypothetical use of "*epi loans*" for mitigating its effects. Notably, lockdowns, vaccinations, remote work, and other characteristics of the COVID-19 pandemic are not captured in a pure SIR model or the current baseline macroeconomic framework. We expect the inclusion of these components – through channels of consumption and investment, for example – to deepen the recession compared to our current model.

We used weekly data of the number of new COVID-19 cases from the Center for Disease Control and Prevention between January 29, 2020, to March 22, 2023. We transformed the series to percentage shares of the workforce by dividing by the mean of the Civilian labor force level in 2019. Economic parameters were calibrated using data from the Federal Reserve Bank of Saint Louis to represent the US economy in 2019. For instance, the discount factor β was set to ensure a 2.16% annual interest rate, which was the 2019 average value. As Moody's seasoned BAA corporate bond yield in 2019 was 4.37%, we calibrated the annual steady-state spread to 2.21%. The ratio of assets to equity in 2019 was around 9 for global systemic important banks, bank holding companies, and intermediate holding companies; the leverage ratio was 1.94 for corporate firms and 3.47 for non-corporate firms. Using the same distribution of entities as in Gertler and Karadi (2011), we calibrated the leverage ratio to 3. Other economic parameters remained unchanged from our baseline values. Finally, to match the time scale of the disease (weekly) with the time scale of the economic model, we adjusted all economic parameters to be on a weekly time scale. A complete table of recalibrated parameters can be found in Appendix F.

Fig. 10 summarizes our baseline case study results. The disease data show the different waves of COVID-19, the strongest beginning in January 2022 (week 104). As in our generic case, rises and falls in economic variables trajectories follow the disease. At its worst, we observe a 0.25% decrease in GDP; the recession only starts to diminish once the last wave passes. Consumption and investment both decline, though investment is more severely affected, which can be explained by high habit consumption in the short term, which prevents consumption from being significantly reduced. Furthermore, investment costs are high in the short term, meaning that capital (and claims sold) cannot adjust

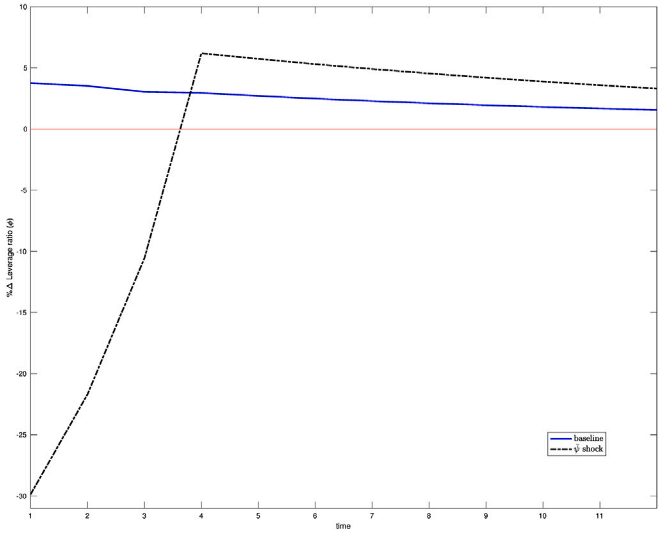

Fig. 11. Banks' leverage ratio. Results are reported as the percent change from the no-disease case. The solid blue line indicates the baseline model. The black, dotted line indicates a model implementing "*epi loans*" ($\bar{\psi}_t = 0.5$). Note that the Central Bank administers "*epi loans*" from period 1 until the peak of the epidemic (period 3). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

quickly and stays low even when the disease starts to diminish. Inflation does not change, which can be explained by high price rigidity in the short term.

Overall, we find decreases in GDP of 0.25% in the first year (2021) and 0.22% in the second year (2022) compared to the steady-state, which was calculated using a calibration for 2019. Although not directly comparable, empirically, the US annual growth rate in 2020 was minus 1.02% compared to 2019. This result differs significantly from other studies, such as Angelini et al. (2020), Chudik et al. (2021), and Bodenstein et al. (2022), who found decreases in GDP post COVID-19 between 1.5% to 2.5%, 15%, and 20% to 30% respectively.

Regarding the use of "*epi loans*", we assume that the Central Bank exogenously finances 1% of total claims ($\bar{\psi}_t$) and that the Central Bank purchases those claims from period 8 for one year. We use this formulation of "*epi loans*" as the Federal Reserve Board implemented similar measures like the Primary Dealer Credit Facility from March 17, 2020, to March 31, 2021 (FED, 2020a). As before, this policy helps with the recession while it is applied but may provoke a stronger recession when stopped.

5. Conclusion

We use a financial DSGE-SIR model to study the economy's response to an epidemic shock. We summarize our findings into three primary contributions.

First, due to the epidemic, the economy will likely experience a deep recession. With our baseline calibration, we observed significant declines in GDP compared to the no-disease case. Second, we found that, except for increasing the share of claims from the Central Bank, none of our unconventional monetary policies can negate the adverse economic effects of the crisis. Nonetheless, as a last resort lender, the Central Bank could use an unconventional monetary policy to exogenously increase its share of total claims issued ("*epi loans*"), which firms will then use to buy capital. This policy has the potential to lessen total losses in GDP, partially mitigating the economic recession without being extremely inflationary, a side effect which has worried economists since the first use of unconventional monetary policies after the subprime crisis (e21-Staff, 2010). This result is encouraging, as many industrialized countries have given billions in stimulus to combat the COVID-19 crisis.

This last conclusion raises an interesting question: could this unconventional monetary policy – while ameliorating the effects of the epidemic – weaken the financial sector? Examining the trajectory of banks' leverage ratio (Fig. 11) shows that when the Central Bank stop applying a strong, unconventional monetary policy ("*epi loans*" in this case), the risk taken by the financial intermediates could be higher; an increase in this variable means that the proportion of worth that a banker lends increases. This should not be considered problematic in our model, as claims are transformed one-to-one into capital; however, in a world where banks' investments could be directed into a shadow banking system, the increase in financial risk, fed by a QE policy, might lead to the creation of financial bubbles and affect the vulnerability of the financial system (Gertler et al., 2012; Hallett et al., 2017; Faia and Karau, 2019; Blot et al., 2020).

Our general framework can be calibrated to specific diseases and countries. As a first attempt, we recalibrated our model to present a case study of COVID-19 in the US; however, future studies will need to consider the dynamics of the epidemic and economy carefully. Who and where are people getting infected, and is everyone equally susceptible? Are deaths a critical component that must be considered? Is the movement of individuals between regions or countries a significant factor in disease spread? What management strategies are in place to prevent the propagation of the epidemic? Answers to these questions imply structural changes to our model, including age structure, heterogeneous mixing, heterogeneous agents (some non-Ricardian), and parameter values, a more realistic labor market and agent decisions, non-constant populations sizes, metapopulation dynamics, and the implementation of management strategies such as treatments, vaccination, or lockdowns. We leave this to future work.

Declaration of competing interest

None

Data availability

All source code and simulation data can be found on the Open Science Framework (https://osf.io/j7m65).

Appendix A

Supplementary material related to this article can be found online at https://doi.org/10.1016/j.econmod.2023.106431.

References

Albertazzi, U., Becker, B., Boucinha, M., 2018. Portfolio Rebalancing and the Transmission of Large-Scale Asset Programmes: Evidence from the Euro Area. Working Paper Series 2125, European Central Bank.

Angelini, E., Darracq-Paries, M., Zimic, S., Damjanovic, M., 2020. ECB-BASIR: A Primer on the Macroeconomic Implications of the Covid-19 Pandemic. Working Paper Series 2431, European Central Bank.

Atkeson, A., 2020. What Will Be the Economic Impact of COVID-19 in the US? Rough Estimates of Disease Scenarios. Working Paper 26867, National Bureau of Economic Research.

Baumeister, C., Benati, L., 2013. Unconventional monetary policy and the great recession: Estimating the macroeconomic effects of a spread compression at the zero lower bound. Int. J. Central Bank. 9 (2), 165–212.

Bianchi, F., 2020. The great depression and the great recession: A view from financial markets. J. Monetary Econ. 114, 240–261.

Bichara, D., Kang, Y., Castillo-Chavez, C., Horan, R., Perrings, C., 2015. SIS and SIR epidemic models under virtual dispersal. Bull. Math. Biol. 77, 2004–2034.

Blot, C., Hubert, P., Labondance, F., 2020. Monetary policy and asset prices in the euro area since the global financial crisis. Rev. Écon. Politique 130 (2), 257–281.

Bodenstein, M., Corsetti, G., Guerrieri, L., 2022. Social distancing and supply disruptions in a pandemic. Quant. Econ. forthcoming.

Boissay, F., Rungcharoenkitkul, P., 2020. Macroeconomic Effects of Covid-19: An Early Review. BIS Bulletins 7, Bank for International Settlements.

Brauer, F., Castillo-Chavez, C., 1994. Ecological Time Series. Raven Pressr, New York, pp. 410–477, Chapter Basic Models in Epidemiology.

Brauer, F., Castillo-Chavez, C., 2012. Mathematical Models in Population Biology and Epidemiology. Springer-Verlag, New York.

Calvo, G., 1983. Staggered prices in a utility-maximizing framework. J. Monetary Econ. 12 (3), 383–398.

Cardani, R., Croitorov, O., Giovannini, M., Pfeiffer, P., Ratto, M., Vogel, L., 2021. The Euro Area's Pandemic Recession: A DSGE-Based Interpretation. European Economy - Discussion Papers 2015 - 153, Directorate General Economic and Financial Affairs (DG ECFIN), European Commission.

Céspedes, L.F., Chang, R., Velasco, A., 2020. The Macroeconomics of a Pandemic: A Minimalist Model. Working Paper 27228, National Bureau of Economic Research.

Chowell, G., Fenimore, P., Castillo-Garsow, M., Castillo-Chavez, C., 2003. SARS outbreaks in Ontario, Hong Kong, and Singapore: The role of diagnosis and isolation as a control mechanism. J. Theoret. Biol. 224, 1–8.

Christiano, L.J., Eichenbaum, M., Evans, C.L., 2005. Nominal rigidities and the dynamic effects of a shock to monetary policy. J. Polit. Econ. 113 (1), 1–45.

Christiano, L.J., Eichenbaum, M.S., Trabandt, M., 2018. On DSGE models. J. Econ. Perspect. 32 (3), 113–140.

Chudik, A., Mohaddes, K., Pesaran, M.H., Raissi, M., Rebucci, A., 2021. A counterfactual economic analysis of Covid-19 using a threshold augmented multi-country model. J. Int. Money Finance 119, 102477.

Costa Junior, C.J., Garcia-Cintado, A.C., Junior, K.M., 2021. Macroeconomic policies and the pandemic-driven recession. Int. Rev. Econ. Finance 72, 438–465.

Daszak, P., Cunningham, A., Hyatt, A., 2001. Anthropogenic environmental change and the emergence of infectious diseases in wildlife. Acta Trop. 78, 103–116.

Dedola, L., Karadi, P., Lombardo, G., 2013. Global implications of national unconventional policies. J. Monetary Econ. 60 (1), 66–85.

e21-Staff, 2010. An open letter to ben bernanke. Economics21.org.

ECB, 2020. ECB allows temporary relief in banks? leverage ratio after declaring exceptional circumstances due to pandemic.

ECB, 2021. ECB extends leverage ratio relief for banks until March 2022.

Eggertsson, G.B., Woodford, M., 2003. The zero bound on interest rates and optimal monetary policy. Brook. Pap. Econ. Act. 2003 (1), 139–211.

Eichenbaum, M.S., Rebelo, S., Trabandt, M., 2020a. Epidemics in the Neoclassical and New Keynesian Models. Working Paper 27430, National Bureau of Economic Research.

Eichenbaum, M.S., Rebelo, S., Trabandt, M., 2020b. The Macroeconomics of Testing and Quarantining. Working Paper 27104, National Bureau of Economic Research.

Eichenbaum, M.S., Rebelo, S., Trabandt, M., 2021. The macroeconomics of epidemics. Rev. Financ. Stud. 34 (11), 5149–5187.

Faia, E., Karau, S., 2019. Systemic Bank Risk and Monetary Policy. CEPR Discussion Papers 13456, C.E.P.R. Discussion Papers.

FED, 2020a. Federal Reserve Board announces establishment of a Primary Dealer Credit Facility (PDCF) to support the credit needs of households and businesses.

FED, 2020b. Regulators temporarily change the supplementary leverage ratio to increase banking organizations' ability to support credit to households and businesses in light of the coronavirus response.

Fenichel, E., Castillo-Chavez, C., Ceddia, M., Chowell, G., Hickling, P.G.P.G., Holloway, G., Horan, R., Morin, B., Perrings, C., Springborn, M., Velazquez, L., Villalobosi, C., 2011. Adaptive human behavior in epidemiological models. Proc. Natl. Acad. Sci. 108, 6306–6311.

Friedman, M., 1969. The Optimum Quantity of Money and Other Essays. Adline Publishing Company, Chicago, pp. 1–50, Chapter The Optimum Quantity of Money.

Fuhrer, J.C., Madigan, B.F., 1997. Monetary policy when interest rates are bounded at zero. Rev. Econ. Stat. 79 (4), 573–585.

Galí, J., 2015. Monetary Policy, Inflation, and the Business Cycle: An Introduction to the New Keynesian Framework and Its Applications, second ed. In: Economics Books, vol. 10495, Princeton University Press.

Gelain, P., Ilbas, P., 2017. Monetary and macroprudential policies in an estimated model with financial intermediation. J. Econom. Dynam. Control 78 (C), 164–189.

Gertler, M., Karadi, P., 2011. A model of unconventional monetary policy. J. Monetary Econ. 58 (1), 17–34, Carnegie-Rochester Conference Series on Public Policy: The Future of Central Banking April 16-17, 2010.

Gertler, M., Kiyotaki, N., 2010. In: Friedman, B.M., Woodford, M. (Eds.), Chapter 11 - Financial Intermediation and Credit Policy in Business Cycle Analysis. In: Handbook of Monetary Economics, vol. 3, Elsevier, pp. 547–599.

Gertler, M., Kiyotaki, N., Queralto, A., 2012. Financial crises, bank risk exposure and government financial policy. J. Monetary Econ. 59, S17–S34, Supplement issue:October 15-16 2010 Research Conference on 'Directions for Macroeconomics: What did we Learn from the Economic Crises' Sponsored by the Swiss National Bank (http://www.snb.ch).

Hallett, A., Fiedler, S., Kooths, S., Stolzenburg, U., Blot, C., Creel, J., Hubert, P., Labondance, F., Ragot, X., 2017. Extending Quantitative Easing: Additional Risks for Financial Stability? Compilation of Notes. In: Monetary Dialogue, Publications Office of the European Union, Luxembourg.

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

Horan, R., Fenichel, E., 2007. Economics and ecology of managing emerging infectious animal diseases. Am. J. Agric. Econ. 89, 1232–1238.

Horan, R., Wolf, C., 2005. The economics of managing infectious wildlife disease. Am. J. Agric. Econ. 87, 537–551.

IMF, 2020. World Economic Outlook: A Long and Difficult Ascent. World Economic Outlook 1, International Monetary Fund.

Jones, K., Patel, N., Levy, M., Storeygard, A., Balk, D., J.L. Gittleman, P.D., 2008. Global trends in emerging infectious diseases. Nature 451, 990–993.

Kanzow, C., Petra, S., 2004. On a semismooth least squares formulation of complementarity problems with gap reduction. Optim. Methods Softw. 19, 507–525.

Keynes, J.M., 1936. The General Theory of Employment, Interest and Money. Macmillan, London, p. 403.

Kiley, M.T., 2020. Pandemic Recession Dynamics: The Role of Monetary Policy in Shifting a U-Shaped Recession to a V-Shaped Rebound. Finance and Economics Discussion Series 2020–083, Board of Governors of the Federal Reserve System (U.S.).

Krueger, D., Uhlig, H., Xie, T., 2020. Macroeconomic Dynamics and Reallocation in an Epidemic: Evaluating the "Swedish Solution. Working Paper 27047, National Bureau of Economic Research.

Lenhart, S., Workman, J., 2007. Optimal Control Applied to Biological Models. Chapman and Hall, London.

LePan, N., 2020. A visual history of pandemics.

Lim, G., McNelis, P.D., 2018. Unconventional monetary and fiscal policies in interconnected economies: Do policy rules matter? J. Econom. Dynam. Control 93, 346–363, Monetary and Fiscal Policy Stabilization amid a Debt Crisis.

McKay, A., Wieland, J.F., 2021. Lumpy durable consumption demand and the limited ammunition of monetary policy. Econometrica 89 (6), 2717–2749.

Merton, R.C., 1973. An intertemporal capital asset pricing model. Econometrica 41 (5), 867–887.

Morin, B., Perrings, C., Kinzig, A., Levin, S., 2015. The social benefits of private infectious disease-risk mitigation. Theor. Ecol. 8, 467–479.

Morin, B., Perrings, C., Levin, S., Kinzig, A., 2014. Disease risk mitigation: The equivalence of two selective mixing strategies on aggregate contact patterns and resulting epidemic spread. J. Theoret. Biol. 363, 262–270.

Qianying, C., Andrew, F., Dong, H., Feng, Z., 2016. Financial crisis, US unconventional monetary policy and international spillovers. J. Int. Money Finance 67, 62–81.

Sharma, D., Bouchaud, J.-P., Gualdi, S., Tarzia, M., Zamponi, F., 2021. V–, U–, L– or W–shaped economic recovery after Covid-19: Insights from an Agent Based Model. PLOS ONE 16 (3), 1–22.

Smets, F., Wouters, R., 2007. Shocks and frictions in US business cycles: A Bayesian DSGE approach. Amer. Econ. Rev. 97 (3), 586–606.

Toxvaerd, F., 2020. Equilibrium Social Distancing. Cambridge working papers in economics, Faculty of Economics, University of Cambridge.

Toxvaerd, F., Rowthorn, R., 2020. On the Management of Population Immunity. Cambridge working papers in economics, Faculty of Economics, University of Cambridge.

Wu, T., Kinzig, C.P.A., Collins, J., Minteer, B., Daszak, P., 2017. Economic growth, urbanization, globalization, and the risks of emerging infectious diseases in China: A review. Ambio 46, 18–29.