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Marion Davin, Mouez Fodha, Thomas Seegmuller

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Abstract

We study whether fiscal policies, especially public debt, can help to curb the macroeconomic and health consequences of epidemics. Our approach is based on three main features: we introduce the dynamics of epidemics in an overlapping generations model to take into account that old people are more vulnerable; people are more easily infected when pollution is high; public spending in health care and public debt can be used to tackle the effects of epidemics. We show that fiscal policies can promote the convergence to a stable disease-free steady state. When public policies are not able to permanently eradicate the epidemic, public debt and income transfers could reduce the number of infected people and increase capital and GDP per capita. As a prerequisite, pollution intensity should not be too high. Finally, we define a household subsidy policy which eliminates income and welfare inequalities between healthy and infected individuals.

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†Corresponding author. CEE-M, Univ Montpellier, CNRS, INRAE, SupAgro, Montpellier, France. E-mail: marion.davin@umontpellier.fr

‡University Paris 1 Panthéon-Sorbonne and Paris School of Economics. PjSE, 48 Boulevard Jourdan, 75014 Paris, France. E-mail: mouez.fodha@univ-paris1.fr.

§Aix Marseille Univ, CNRS, AMSE, Marseille, France. 5-9 Boulevard Bourdet, CS 50498, 13205 Marseille Cedex 1, France. E-mail: thomas.seegmuller@univ-amu.fr.

1 Introduction

The recent Covid-19 epidemic is one of the most serious threat to health in the last decades. It has revealed how managing a pandemic to limit health and economic costs is challenging. In a context in which environmental factors have been found to influence the transmissions of viral pathogens and the disease emergence, there is a need to well understand the interplay between the health and economic impacts of epidemics. This is the aim of this paper.

Without restricting our attention to a particular epidemic, we analyze the effects of public interventions to control the disease, in particular health public spending and fiscal policy with public debt. We consider fiscal policy and public debt in the discussion as the management of sanitary crisis usually goes along with extraordinary public measures. The OECD points out the sharp increase in public debt expected in OECD area because of the Covid-19 crisis.[1] For other diseases like HIV or malaria, debt relief are frequently mentioned as instrument to help endemic countries to control epidemics (Snow *et al.*, 2010; Abah, 2020). This illustrates that debt management is expected to play a crucial role to try to control or eradicate this type of disease.

Another important aspect to consider when examining the interplay between epidemics and economics is the environmental issue. Growing evidence suggests that environmental factors has an effect on the rate of spread of epidemics. Air pollution, such as CO2 and PM, is a factor in accelerating virus transmission between humans. Pollution particles behave as vehicles for virus transport, especially in epidemics where the mode of transmission is mainly via aerosols (Domingo and Rovira, 2020; Rohrer *et al.*, 2020; Bourdrel *et al.*, 2021). Moreover, there is an indirect effect of pollution on the rate of propagation, through the consequences of pollution on the health of individuals, particularly the most fragile. Pollution makes individuals more vulnerable and therefore less resistant because they are more immunocompromised.

In line with Augier and Yaly (2013), Chakraborty *et al.* (2010, 2014), Momota *et al.* (2005), we develop an overlapping generations (OLG) model with epidemics, that we enriched with an environmental dimension and the introduction of a government. This model offers the interest to consider heterogeneity of agents according to their age and is relevant to analyze the role of public debt. There are three-period lived households with young inactive agents, working adults and old retirees. The dynamics of epidemics are formalized by introducing a SIS (suscep-

[1] According to OECD (2020b), "For the OECD area as a whole, outstanding central government debt is expected to increase from USD 47 trillion in 2019 to USD 52.7 trillion at the end of 2020. This is USD 3.5 trillion higher than the pre-Covid estimate. As a result of both the rapid increase in borrowing needs and the decline in GDP across OECD economies, the central government marketable debt-to-GDP ratio for the OECD area is projected to increase by 13.4 percentage points to around 86% in 2020, the largest increase in a single year since 2007."

tible–infected–susceptible) model, as in Bosi and Desmarchelier (2020), Goenka and Liu (2012, 2020) or Goenka *et al.* (2014).

In our model, we first consider that the impacts of the epidemic depend on the age of the person affected: the older agent bears a premature risk of death while the adult agent will be sick, without fatal consequences, but will have to take time off work. Indeed, empirical studies show that the mortality of elderly patients is higher than that of young and middle-aged patients during epidemics, because of their higher vulnerability. Taking the recent example of Covid-19, elderly patients are more likely to progress to severe disease (see Liu *et al.*, 2020; Williamson *et al.*, 2020) and hence, they are more affected by saturated health's system capacities entailed by the epidemics. Secondly, in line with arguments previously mentioned, we consider that degradation of the environmental quality increases the rate of contagion of the epidemic. Finally, health policy consisting in public spending to prevent, detect, control and treat quickly the epidemics, contributes to push down the contagion rate. The government finances its expenditures through taxation of income and production, but also through the issuance of public debt. In line with Geoffard and Philipson (1997),[2] we assume that health policy reduce the transmission of the virus but does not allow to have full immunity.

The stable steady state is indeed characterized by the presence of the virus. However, the government can increase health expenditures to slow down the spread of the virus. Such a strategy can allow to rule out the endemic steady state and converge to a disease-free steady state. These results are conditioned by the pollution intensity of production: the higher the pollution intensity, the more difficult it is to fight the epidemic. If the public policy is not able to remove entirely the epidemic, the economy converges towards an endemic steady state. It could however be used to reduce the number of infected people and increase capital per capita in the long run endemic equilibrium. The complexity of the interactions between fiscal policy and the fight against epidemics is highlighted. In fact, on the one hand, any increase in public debt leads to a crowding-out effect on productive capital. The latter implies a drop in production, wages, savings and tax revenues, which curbs the expected effects of increased public spending, and reduces the effectiveness of public policy. At the same time, it also slows down pollution and plays a positive role in the fight against the virus. On the other hand, the increase in debt allows an increase in public spending, and thus a slowing of the epidemic, which has a stimulus effect on the economy through the increase in the number of workers, savings and capital (crowding-in effect). The final outcome depends on the relative magnitude of these two channels. We show that the crowding-in effect dominates if the rate of pollution emission is not too high.

[2]Even if public funds are directed towards the development of a vaccine, Geoffard and Philipson (1997) underline the difficulties for policies to increase demand for vaccine and hence to achieve eradication with vaccine.

At the endemic steady state, there are income inequalities between infected and healthy people. We show that it is possible to design an appropriate redistribution income policy to address welfare disparities. This policy consists of a differentiated transfer of income for workers and the sick. It complements the public policy to combat the virus. We emphasize that such intervention can be costly for healthy people when public budget for transfers is not sufficiently important.

Our results underline the importance to maintain a high level of public health spending and a high public budget for transfer to control an epidemic and address its economic consequences. This implies a sufficient level of public debt. Our conclusions are thus in line with the proposal of Douglas and Raudla (2020) for the U.S. economy, who argue that the States should suspend their balanced budget rules and norms, and run deficits in their operating budgets to maintain services and meet additional obligations due to the pandemic of Covid-19.

Our paper relates to the large literature interested in the analysis of the interactions between economics and epidemics. On the one hand, there is an important literature characterized by age specific effect of epidemics (Boucekkine *et al.,* 2009; Boucekkine and Laffargue 2010; Fabbri *et al.,* 2021[3]). On the other hand, some models are based on mathematical frameworks developed by epidemiologists (see Hethcote, 2000, for an interesting survey); they expanded with the HIV epidemic (see for example Geoffard and Philipson, 1997). This literature has obviously been revived and adapted to the specificities of the Covid-19 (see for example Acemoglu *et al.*, 2021; Alvarez *et al.*, 2021; Goenka *et al.,* 2021; Gori *et al.*, 2021; Hritonenko *et al.*, 2021). Nonetheless, no study considers simultaneously the differentiated effects of the consequences of the virus according to the age of the infected persons, and the role played by the environment in the spread and incidence of the virus. Moreover, public actions examined in the literature dealing with epidemics and economics, such as confinement, social distancing and the speed at which a vaccine develops, greatly differ from those explored in this paper.

We highlight in this study the direct consequences of public finance on epidemics and *vice-versa*. We show that in the absence of full immunity, health care spending can play a major role in the fight against the epidemic and public debt can push up GDP per capita. Our paper thus complement the literature taking into account the costs and benefits of public policies, and the specific impacts of the epidemic by age groups. In this way, we provide new intuitions about the effects of public debt and pollution on economic aggregates and epidemics propagation.

The rest of this paper is organized as follows. Section 2 presents the model with epidemics. Section 3 defines the intertemporal equilibrium. Section 4 analyzes the existence of steady states and Section 5 the convergence to the steady states. Section 6 focuses on the role of fiscal policy.

[3]This last paper is based on M'Kendrick (1925) setting.

Section 7 presents transfer schemes to address inequalities among agents. Finally, Section 8 concludes, while technical details are relegated to an Appendix.

2 An OLG model with pollution and epidemics

We consider a discrete time ($t = 0, 1, ...$) overlapping generations (OLG) model. The dynamics of epidemics follow a SIS (Susceptible-Infected-Susceptible) model. There are three types of agents, households, a government, and firms which generate pollution.

2.1 Environmental quality

Environmental quality decreases with pollution P_t, which is a flow that proportionally raises with production Y_t:

$$P_t = \alpha Y_t$$

where $\alpha > 0$ is the pollution rate.

This global pollution index encompasses the degradation imposed by human activity on the quality of the environment. It reflects both the level of pollutant emissions and changes in biodiversity. It can be represented by Ecological Footprint measurements or by Environmental Performance Index (Wendling *et al.*, 2020).

2.2 Population and epidemics

As in Bosi and Desmarchelier (2020), Goenka and Liu (2012, 2020) or Goenka *et al.* (2014), the dynamics of epidemics are driven by a SIS model, with susceptible and infected people. It differs from the SIR (Susceptible-Infectious-Recovered) model recently used in economics (see e.g. Acemoglu *et al.*, 2021; Eichenbaum *et al.*, 2020) which also introduced the category of recovered people. [4]

We consider a model with three period-lived agents: childhood, adulthood, and old age. The population size of a generation is constant and equal to N. When young, an agent has contact with other generations what makes her susceptible or infected at the beginning of her adult life. Thus, at the beginning of each period, there are also N adults and N old consumers, which inherit their type from the previous period. Let H_t^i be healthy susceptible people and I_t^i be infected people, with the superscript $i = a$ for adult people and $i = o$ for old people. At time t, we have $2N = H_t^a + H_t^o + I_t^a + I_t^o$. We assume that susceptibility to infection does not depend

[4] In our OLG framework, considering the SIS rather than the SIR model does not alter the results.

on age. The number of adult and old agents being susceptible and infected are thus the same, i.e. $H_t^a = H_t^o = H_t$ and $I_t^a = I_t^o = I_t$.

Nevertheless, the health impacts of the infection are assumed to be heterogeneous, i.e. age-dependent.[5] This is consistent with the fact that older infected people really face a more important probability to die and to develop severe illness, because of physiological changes that make them more vulnerable. While the size of adult population is not affected by the proportion of infected people, epidemics shorten the probability to enter the old age. The more infected old people there are in the population, the higher the probability to die prematurely. We do not consider the individual effect of infection on mortality, but we rather focus on the negative externality associated with epidemics for all elderly population. The general intuition is that a high number of infected people in the economy tends to reduce the efficiency of medical services or can be source of congestion effects in the healthcare sector, and hence entails a negative externality on the old vulnerable population. Several studies have underlined the negative indirect effects associated with important epidemics as they tend to disrupt and suspend health services (Chang *et al.*, 2004; Rust *et al.*, 2009; Kontis *et al.*, 2020). As an example, this is also typically what we observe since the beginning of the Covid-19 crisis. Elderly patients are more likely to progress to severe disease (Liu *et al.*, 2020; Williamson *et al.*, 2020) and thus, they are more affected by saturated health's system capacities.

At the beginning of the period, we have $2N = 2H_t + 2I_t$ of adult and old people, which is equivalent to $1 = h_t + i_t$, where $h_t = H_t/N$ represents the share of healthy people and $i_t = I_t/N$ represents the share of infected people. Note that i_t can also be interpreted as the likelihood of being infected.

Let $\theta_t > 0$ be the transmission rate of the epidemics among healthy agents and $\gamma \in (0, 1)$ the rate of recovery among infected people. The rate θ_t can also be interpreted as the average number of contacts per unit time such that an infective transmits the disease. The dynamics of healthy people are given by:

$$H_{t+1} = H_t + \gamma I_t - \theta_t \frac{I_t}{N} H_t$$

where γI_t represents cured people and $\theta_t \frac{I_t}{N} H_t$ new people infected, which is given by healthy people (H_t) times the risk of meeting infected individual ($\frac{I_t}{N}$) times the transmission rate (θ_t).

The dynamics of infected people are exactly the opposite:[6]

$$I_{t+1} = I_t - \gamma I_t + \theta_t \frac{I_t}{N} H_t$$

[5] We consider an OLG model precisely to take into account that adult and old people do not face the same consequences of being infected.

[6] Note that we assume that the proportion of people which die prematurely during the old age is too negligible in the society to affect the dynamics of epidemics.

These last two equations rewrite:

$$h_{t+1} = \gamma i_t + (1 - \theta_t i_t)\, h_t \tag{1}$$

$$i_{t+1} = (1 - \gamma)\, i_t + \theta_t i_t h_t. \tag{2}$$

On the one hand, to keep things as simple as possible, the rate of recovery γ is constant, as in Goenka and Liu (2020). On the other hand, we assume that the transmission rate θ_t is increasing with pollution P_t. Many evidences support that air pollution raises the transmission of respiratory viral infections (see Domingo and Rovira, 2020 for a survey). The recent Covid crisis confirm the relevance of the assumption. For example, Cole *et al.* (2020) highlight that $PM_{2.5}$ and NO_2 concentrations have a positive link with Covid-19 cases, hospital admissions.

Climate variability has also lead to a proliferation of vector-borne diseases, like malaria, resulting in an increase in transmission (Barreca and Shimshack, 2012; Rohr and Cohen, 2020). The transmission rate θ_t is also decreasing with public expenditures in health care G_t. The higher G_t, the higher public health response capacity and the lower the spread of epidemics. We assume that health policy does not allow to build full immunity, in line with the arguments of Geoffard and Philipson (1997). According to them, even when vaccines are available, it is difficult for policies to increase demand for it and hence to achieve eradication. Moreover, many viruses, such as malaria or HIV, have not found a cure and policies to control the spread are limited to subsidies and protective spending. As a result, G_t could be masks, tests, emergency services, controls, information campaign, or vaccine among the others.

Let $g_t \equiv G_t/N$ be public expenditures per adult and $p_t \equiv P_t/N$ pollution per adult. For tractability, we assume:

Assumption 1 $\theta_t = \theta(G_t/P_t) = \theta(g_t/p_t) > 0$ *for all* g_t/p_t*, with* $\theta'(g_t/p_t) < 0$*. In addition,* $\theta_t i_t < 1$.

The inequality $\theta_t i_t < 1$ means that the probability for a healthy people to be infected is less than one. Using (1), it also implies that $h_t > 0$ whatever the value of γ.

2.3 Households

The economy is populated by overlapping generations (OLG) of agents living for three periods. As we aim at analyzing the role of public policies, the non-Ricardian properties of the OLG model are convenient. Finally, it is a tractable way to consider the coexistence of heterogeneous agents at each period of time. We will have adult savers which will live at the same time that elderly who spend their remunerated savings. Considering such an OLG model where the

dynamics of infected are driven by a SIS model means that the length of a period is quite long with respect to the period of being infected for many diseases. Nonetheless, some papers already consider infectious disease and overlapping generations model. For example, in the OLG models considered by Augier and Yaly (2013), Chakraborty *et al.* (2010, 2014), Momota *et al.* (2005), the dynamics of epidemics is also driven by a stock variable. Their results highlight interesting mechanisms, confirming that this framework remains well adapted for the study of population-epidemic dynamics interactions. As it is well-known, more complicated frameworks could rationalize heterogeneous behaviors between workers and retirees, for instance models with infinitely-lived agents facing some financial constraints.[7] We could also note that the behavior of agents will be summarized by their saving behavior which is closely related to what we get in a standard Solow model.

When young, an agent does neither consume nor work but, as we already mentioned, has contact with other generations such that she becomes susceptible or infected at the next period. In contrast, an agent is active and consume at the adult age, and is retired and consumes at the old age. The preferences of the household $j \in \{1, ..., N\}$ born at period $t-1$ are represented by the following utility function over consumption when adult c_{jt} and old d_{jt+1}:

$$\ln c_{jt} + \beta(h_{t+1}) \ln d_{jt+1} \tag{3}$$

where $\beta(h_{t+1}) \in (0, 1]$ measures the survival probability. It means that either the adult survives at the old age or he dies at the beginning of the old age. This utility function means that, whereas an infected adult has no chance to die at the adult age, the probability to die at the beginning of the old age increases with the proportion of infected people among old agents or equivalently the survival probability increases with the proportion of non-infected susceptible people h_{t+1} at old age, i.e. $\beta'(h_{t+1}) > 0$. We also assume that $\beta(h_{t+1})$ is strictly concave, which implies that $\beta'(h_{t+1}) < \beta'(0)$. This specification is similar to Momota *et al.* (2005), that consider the negative effect of the prevalence of the disease on the probability to be alive in the second period of life. The sensitivity of β to h can also be interpreted as the limited resilience of healthcare institutes. The possible saturation of the health system when the proportion of infected becomes high leads to a decrease of the survival probability of the elderly population.

The budget constraints faced by an individual $j \in \{1, ..., N\}$ are given by:

$$\sigma_{jt} + c_{jt} = \Omega_{jt} + \tau_{jt} \tag{4}$$

$$d_{jt+1} = \frac{r_{t+1}}{\beta(h_{t+1})} \sigma_{jt} \tag{5}$$

where σ_{jt} represents savings of individual j. Since each household supplies one unit of labor if she

[7] See for instance Woodford (1986) and Kocherlakota (1992).

is healthy, Ω_{jt} is the labor income, which is equal to the real wage paid by the firm to workers w_t if individual j is healthy and 0 if she is infected. This means that at periods of epidemics, a share of adults will not work. We note that this is not specific to our framework since it also occurs in OLG models with some labor market imperfections and unemployment (Coimbra *et al.*, 2005; Kaas and von Thadden, 2004; Ono, 2007). Moreover, this is not crucial for our results. What is important is that, at the equilibrium, the income used for aggregate savings will increase with the number of healthy people.

$\tau_{jt} \gtreqless 0$ is a lump-sum subsidy/tax which is specific to each individual and can be used by the government to (partially) cover the loss of labor income. We therefore have in mind that for infected people who will not work, τ_{jt} can be interpreted as a health insurance or a paid sick leave. As it is empirically established, paid sick leave is implemented by governments in a large number of countries (Scheil-Adlung and Sandner, 2010). Replacement rates vary up to 100 % of wages. There is also evidence that paid sick leave has been used as a policy response to protect income through the Covid-19 crisis (OECD, 2020a). Indeed, data suggest that paid sick leave has grown significantly in most countries in the outbreak of the pandemic.

The return of savings is $r_{t+1}/\beta(h_{t+1})$, with r_{t+1} the marginal productivity of capital, because there is perfect annuity on the asset markets.[8]

We deduce that the consumptions and savings of individual j are equal to:

$$c_{jt} = \frac{1}{1+\beta(h_{t+1})}(\Omega_{jt}+\tau_{jt}) \tag{6}$$

$$d_{jt+1} = \frac{r_{t+1}}{1+\beta(h_{t+1})}(\Omega_{jt}+\tau_{jt}) \tag{7}$$

$$\sigma_{jt} = \frac{\beta(h_{t+1})}{1+\beta(h_{t+1})}(\Omega_{jt}+\tau_{jt}) \tag{8}$$

In our model, pollution affects the consequences of the epidemic through two channels, a direct and an indirect one. On the one hand, the direct effect is measured by the effect of pollution on the transmission rate $\theta(g/p)$. The higher the pollution, the higher the rate of spread, and the higher the number of infected individuals. On the other hand, it mechanically lowers the number of healthy individuals h, which reduces the life expectancy of the oldest people $\beta(h)$ and the saving rate $\beta(h)/(1+\beta(h))$.

2.4 Firms

Markets are perfectly competitive and production is performed by a representative firm. Output Y_t is produced with labor L_t and capital K_t according to a constant returns to scale technology.

$$Y_t = A(h_t)\,F(K_t, L_t)$$

[8]For simplification, we also assume complete depreciation of capital.

The total factor productivity $A(h_t)$ experiences a large decrease if the proportion of infected people becomes higher than a threshold:

$$\begin{cases} A(h_t) = 1 & if \quad h_t > \underline{h} \\ A(h_t) = A < 1 & otherwise \end{cases}$$

$A(h_t)$ acts as an externality in production and the parameter $\underline{h} \in (0,1)$ is a threshold for epidemics which plays a crucial role on this externality. If the proportion of infected people is positive but sufficiently low ($h_t > \underline{h}$), nothing happens on the productivity. On the contrary, if the proportion of infected people is too high ($h_t \leqslant \underline{h}$), the number of working people is too low to maintain efficient labor, because of costly work disorganization for instance. Moreover, this too low working people can create an inefficient utilization of capital (some units should close or some machines are no more used because not enough people are working). These different arguments allow us to think that the total factor productivity becomes lower when prevalence of disease is sufficiently high. This is in line with Cole and Neumayer (2006) that find a negative impact of poor health, i.e health problems associated to great burden, on total factor productivity.

For tractability, we use a Cobb-Douglas technology:

$$Y_t = A(h_t) L_t f(a_t) = A(h_t) L_t a_t^s$$

with $s \in (0, 1/2)$ the capital share in total income and $a_t = K_t/L_t$ the capital-labor ratio.

Production, at the origin of pollution flow, is taxed by the government at a rate $\tau_t^f \geq 0$. Hence, firms choose inputs by maximizing its profit $\left(1-\tau_t^f\right) Y_t - r_t K_t - w_t L_t$, such that we get:

$$r_t = \left(1-\tau_t^f\right) A(h_t)\, s a_t^{s-1} \equiv r(a_t) \tag{9}$$

$$w_t = \left(1-\tau_t^f\right) A(h_t)\,(1-s) a_t^s \equiv w(a_t). \tag{10}$$

2.5 Public sector

To limit the adverse economic effects of an epidemic, public authorities generally implement a policy mix aiming at improving the health situation and mitigating the effects on the economic activity. In this perspective, we consider that the government can fight the disease and improve health by financing public health expenditures G_t. These spending have a direct effect on epidemics, since they reduce the rate of transmission θ_t, without allowing to have full immunity. In addition, the government can also limit the economic costs of epidemics by paying some lump-sum subsidies τ_{jt} to adult households. We define $\tau_t = \sum_{j=1}^{N} \tau_{jt}/N$ the average subsidy.

To finance these expenditures, the government levies a tax on production, at the rate $\tau_t^f \geq 0$, or can issue debt B_t. Since capital and public debt are perfectly substitutable assets, they face

the same return. Hence, debt reimbursement from the previous period is given by $r_t B_{t-1}$. The intertemporal budget constraint for the government therefore satisfies for all $t \geqslant 0$:

$$B_t = r_t B_{t-1} + G_t + \tau_t N - \tau_t^f Y_t \tag{11}$$

with $B_{-1} \geq 0$ given.

3 Intertemporal equilibrium

On the labor market, we recall that each adult agent supplies inelastically one unit of labor, but only healthy people are able to work. This means that at equilibrium, we have $L_t = H_t$. We deduce that:

$$\sum_{j=1}^{N} \Omega_{jt} = w_t L_t = w_t H_t \tag{12}$$

We define debt and capital per adult as $b_t \equiv B_t/N$ and $k_t \equiv K_t/N$. Production per adult is given by $Y_t/N = A\left(h_t\right) a_t^s h_t$, with $a_t = k_t/h_t$. Then, using (9), the government budget constraint (11) rewrites:

$$g_t = b_t - r(a_t) b_{t-1} - \tau_t + \tau_t^f A(h_t) a_t^s h_t \tag{13}$$

Equilibrium on the asset market is ensured by $k_{t+1} + b_t = \sum_{j=1}^{N} \sigma_{jt}/N$. We use (8), (10), (12), and $k_{t+1} = a_{t+1} h_{t+1}$ to get:

$$a_{t+1} h_{t+1} + b_t = \frac{\beta(h_{t+1})}{1 + \beta(h_{t+1})} (w(a_t) h_t + \tau_t) \tag{14}$$

Since $p_t = \alpha Y_t/N$, equation (1), that describes the dynamics of healthy people, rewrites:

$$h_{t+1} = h_t + (1 - h_t) \left[\gamma - \theta[g_t/(\alpha A(h_t) a_t^s h_t)] h_t\right] \tag{15}$$

with $\theta[g_t/(\alpha A(h_t) a_t^s h_t)](1 - h_t) < 1$ under Assumption 1.

By inspection of equation (13), we now define which policy parameters will be considered as fixed and will be used to conduct comparative statics.

Assumption 2 *$b_t = b \geqslant 0$, $\tau_t = \tau \geqslant 0$, with $b > \tau$, and $\tau_t^f = \tau^f \in (0, 1)$ are constant for all $t \geqslant 0$.*

The subsidy to adult and the tax rate are considered as constant, as well as debt per capita. This implicitly means that debt sustainability is not an issue or debt is always fixed at a level which is sustainable. Moreover, Assumption 2 enforces that debt per capita is high enough with respect to the average subsidy supporting adults income.

Then, public spending will vary to satisfy the government budget constraint:

$$g_t = b(1 - r(a_t)) - \tau + \tau^f A(h_t) a_t^s h_t \equiv g(a_t, h_t) \tag{16}$$

which is an increasing function in a_t and h_t. Public expenditures are thus pro-cyclical. In case of strong epidemics, we could *a priori* observe a decrease in a_t and/or h_t, and therefore of public spending. Then, public debt could be used to maintain a sufficient level of public spending if debt emission is higher than debt reimbursement.

Considering this last equation and Assumption 2, an intertemporal equilibrium is a sequence (h_t, a_t) satisfying equations (14) and (15) for all $t \geqslant 0$, i.e.

$$h_{t+1} = h_t + (1 - h_t)\left[\gamma - \theta[g(a_t, h_t)/(\alpha A(h_t) a_t^s h_t)] h_t\right] \tag{17}$$

$$a_{t+1} h_{t+1} + b = \frac{\beta(h_{t+1})}{1 + \beta(h_{t+1})}[(1 - \tau^f)(1 - s) A(h_t) a_t^s h_t + \tau] \tag{18}$$

with $\theta[g(a_t, h_t)/(\alpha A(h_t) a_t^s h_t)](1 - h_t) < 1$.

Note that both h_t and $a_t = k_t/h_t$ are predetermined variables, with initial conditions $h_0 = H_0/N > 0$ and $a_0 = K_0/H_0 > 0$.

Our model highlights multiple important interplays between the real side of the economy and epidemics. On the one side, epidemic affects labor supply, and therefore labor income. On the other side, economic activity affects the evolution of epidemic by determining the amount of public health spending, but also the level of pollution. In addition, our model also emphasizes relevant properties characterizing the consequences of epidemics: the productivity of workers lowers in case of epidemic outbreak and we observe a damaging effect on longevity, what affects negatively the saving rate.

4 Steady states with and without epidemic outbreak

A steady state corresponds to a long-term equilibrium in which the share of infected people $1 - h$ and the capital-labor ratio a are stationary. A steady state is a solution (h, a) satisfying:

$$(1 - h)\left[\gamma - \theta[g(a, h)/(\alpha A(h) a^s h)] h\right] = 0 \tag{19}$$

$$ah + b = \frac{\beta(h)}{1 + \beta(h)}[(1 - \tau^f)(1 - s) A(h) a^s h + \tau] \tag{20}$$

with

$$g(a, h) = b[1 - (1 - \tau^f) A(h) s a^{s-1}] - \tau + \tau^f A(h) a^s h \tag{21}$$

and

$$\theta[g(a, h)/(\alpha A(h) a^s h)](1 - h) < 1 \tag{22}$$

By direct inspection of equation (19), we distinguish two types of steady states. Some are characterized as states with infected people, $h < 1$, and the others as states with only healthy -susceptible- people, $h = 1$.

We focus first on states with infected people. From (19), a steady state with $h < 1$ satisfies:

$$\gamma = \theta[g(a,h)/(\alpha A(h)a^s h)]h \tag{23}$$

Using (21), we easily get:

$$\frac{g}{p} = \frac{g(a,h)}{\alpha A(h)a^s h} = \frac{b-\tau}{\alpha A h a^s} - \frac{b(1-\tau^f)s}{\alpha h a} + \frac{\tau^f}{\alpha} \equiv \eta(h,a) \tag{24}$$

with $A = 1$ if $h > \underline{h}$. Therefore, equation (23) rewrites:

$$\gamma = \theta(\eta(h,a))h = \theta\left[\frac{b-\tau}{\alpha A h a^s} - \frac{b(1-\tau^f)s}{\alpha h a} + \frac{\tau^f}{\alpha}\right] h \equiv Z(h,a) \tag{25}$$

In the following, we assume that there is a primary deficit independently of the subsidy τ. Whatever the public support to workers, we consider that the amount of public health spending exceeds tax revenues at least when there is epidemic outbreak, $g > \tau^f A h a^s$. Using the steady state value for g given in equation (21), this corresponds to the inequality $(b-\tau)a^{1-s} > b(1-\tau^f)sA$, which also ensures $\eta(h,a) > 0$. We thus have:

Assumption 3 $a > \left[\frac{b(1-\tau^f)sA}{b-\tau}\right]^{\frac{1}{1-s}} \equiv \underline{a}$.

A steady state with $h < 1$ also satisfies equation (20):

$$F(h,a) \equiv ah + b - \frac{\beta(h)}{1+\beta(h)}[(1-\tau^f)(1-s)A(h)a^s h + \tau] = 0 \tag{26}$$

with $A(h) = 1$ if $h > \underline{h}$ and $A(h) = A$ if $h < \underline{h}$.

Lemma 1 *Equation (25) implicitly defines a function $h = H_1(a)$ which is increasing for $\underline{a} < a < \widehat{a}$ and decreasing for $a > \widehat{a}$, with*

$$\widehat{a} \equiv \left[\frac{b(1-\tau^f)A}{b-\tau}\right]^{\frac{1}{1-s}} > \underline{a}$$

The maximum value taken by this function is given by $\widehat{h} = H_1(\widehat{a})$. We also have that $H_1(\underline{a}) = H_1(+\infty) = \gamma/\theta(\tau^f/\alpha)$ and $H_1(a) \geqslant \gamma/\theta(\tau^f/\alpha)$.

Equation (26) implicitly defines a function $h = H_2(a)$ which is strictly increasing for all $a > \underline{a}$. Moreover, we have $H_2'(a) = -\frac{\partial F}{\partial a}/\frac{\partial F}{\partial h}$ which is increasing in A meaning that when h crosses $\underline{h}$, A becomes equal to 1 and the slope increases.

Proof. See Appendix A. ■

Before examining in details existence and uniqueness of a steady state with $h < 1$, we focus on disease-free steady states. A solution $h = 1$ satisfies equation (19). It also implies that $A(h) = 1$. Therefore, equation (20) rewrites:

$$F(1, a) = a - \frac{\beta(1)}{1+\beta(1)}(1-\tau^f)(1-s)a^s + b - \frac{\beta(1)}{1+\beta(1)}\tau = 0 \tag{27}$$

and allows to identify directly the existence of steady states with $h = 1$.

Proposition 1 *Under Assumptions 1-3 and*

$$A\frac{b}{b-\tau} < \frac{\beta(1)}{1+\beta(1)}\frac{1-s}{s} \tag{28}$$

there exists $b_1 > 0$ such that for all $b < b_1$, there is a unique disease-free steady state, $(a_1, 1)$, which always satisfies inequality (22).

Proof. See Appendix B. ■

Assuming that b is not too large, the crowding-out effect of public debt is not too high. Moreover, inequality (28) ensures that savings allowing to finance public debt and to invest in productive capital is sufficiently high. Under these conditions, there exists a steady state in which all the population is healthy and the level of capital is high.

We now analyze endemic steady states, characterized by infected people $h < 1$. Based on Lemma 1, steady states with $h < 1$ are solution satisfying $h = H_1(a) = H_2(a)$. As previously mentioned, when the number of infected people is too high ($h < \underline{h}$), the economy observes an epidemic outbreak that reduces the productivity of factor $A(h)$. We thus examine the existence of steady states with such properties ($h < \underline{h} < 1$) which will coexist with the steady state without infected, $(a_1, 1)$.

Since $H_1(a)$ is single peaked, a steady state with $h < 1$ is characterized by $h < \bar{h}$ if the sufficient condition $\widehat{h} < \underline{h}$ holds. From (25), $\widehat{h}$ is a solution of:

$$h = \frac{\gamma}{\theta(\eta(h, \widehat{a}))} \tag{29}$$

Substituting $\widehat{a} = \left[\frac{b(1-\tau^f)A}{b-\tau}\right]^{\frac{1}{1-s}}$ in (24), we get:

$$\eta(h, \widehat{a}) = \frac{(1-s)}{\alpha h (1-\tau^f)^{\frac{s}{1-s}} A^{\frac{1}{1-s}}} \left(\frac{b-\tau}{b}\right)^{\frac{1}{1-s}} b + \frac{\tau^f}{\alpha} \tag{30}$$

which is a decreasing function of h.

The left-hand side of (29) is of course increasing in h from 0 to 1, while the right-hand side is decreasing in h from a positive value when $h = 0$. Therefore, if $\underline{h} > \frac{\gamma}{\theta(\eta(\underline{h},\widehat{a}))}$, the value $\widehat{h}$ which solves (29) belongs to $(0, \underline{h})$ and a steady state with infected people is always marked by epidemic outbreak. Let us assume:

Assumption 4 $\underline{h}\theta(\eta(\underline{h}, \widehat{a})) > \gamma$.

We show the following result (see also Figure 1):

Proposition 2 *Under Assumptions 1-4,*

$$\frac{b}{b-\tau} < \frac{\beta(\gamma/\theta(\tau^f/\alpha))}{1+\beta(\gamma/\theta(\tau^f/\alpha))}\frac{1-s}{s} \quad \textit{and} \quad \theta(\tau^f/\alpha) < 1+\gamma \tag{31}$$

there exist $b_2 > 0$ and $\underline{\alpha}$ such that if $b < b_2$ and $\alpha > \underline{\alpha}$, there is a unique steady state (a_2, h_2), with $\underline{a} < a_2 < \widehat{a}$ and $h_2 < \underline{h} < 1$, characterized by a positive share of infected people and with epidemic outbreak.

This steady state coexists with the one with no infected people, $(a_1, 1)$, and is characterized by a lower capital-labor ratio. We have the following ranking: $\underline{a} < a_2 < a_1 < \widehat{a}$.

Proof. See Appendix C. ■

We notice that conditions (31) and $b < b_2$ both ensure the existence of a steady state with epidemic outbreak that coexists with the one with no infected people.[9] A level of debt not too high associated to a high enough saving rate maintains a sufficient level of saving. The inequality $\alpha > \underline{\alpha}$, which means a sufficiently high pollution rate, ensures that $\theta(\eta(h, a))$ does not strongly depend on the capital-labor ratio. This ensures the uniqueness of the steady state because $H_1(a)$ is not too steep (see Figure 1).

We note that the existence of both steady states requires a level of debt not too significant. This is due to the fact that one channel through which debt intervenes on the equilibrium goes through savings. Part of savings is devoted to finance public debt. Therefore, if debt is too large, the level of savings is no more sufficient to sustain a positive level of capital, which may rule out the existence of any steady state.

We pay a particular attention to the effect of a variation in the productivity parameter A on the endemic steady state. A decrease or a sufficiently low level of A can be seen as the economic impacts of a virulent pandemic, which induce a strong disorganization of labor. In such a period, there is not only a lower productivity or efficiency of labor, but also of capital, because some machines have to be stopped for instance. Therefore, we analyze now in more details the effect of a variation of A on h_2 and a_2, i.e. what happens if the loss of productivity is even more severe:

[9] Note that this inequality (31) is more stringent than inequality (28).

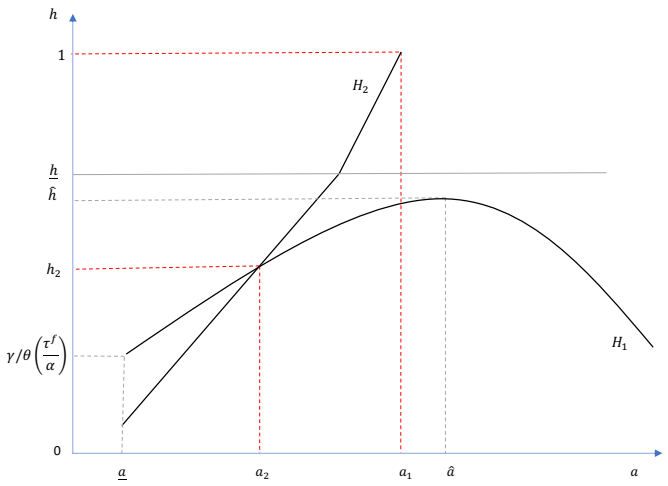

Figure 1: Steady states with epidemics outbreak (a_2, h_2) and no infected people $(a_1, 1)$

Proposition 3 *Under Assumptions 1-4, inequality (31),* $\theta(\tau^f/\alpha) < 1+\gamma$, $\alpha > \underline{\alpha}$ *and* $b < b_2$, *following a slight increase of the productivity A, the sign of the variation of* h_2 *is indeterminate.*

Moreover, there exist $\overline{\alpha}_A$ *and* $\underline{\alpha}_A (\leqslant \overline{\alpha}_A)$ *such that a slight increase of A implies an increase of* a_2 *for* $\alpha > \max\{\underline{\alpha}, \overline{\alpha}_A\}$ *and a decrease of* a_2 *for* $\underline{\alpha}_A > \alpha > \underline{\alpha}$ *and A low enough.*

Proof. See Appendix D. ■

The fall in the productivity parameter A implies opposite effects on the transmission rate θ. Through this direct effect of A, pollution decreases more than public spending, which means that the transmission rate θ goes down.

A lower productivity also implies a fall in income that reduces savings and hence capital-labor ratio if α is high enough. Indeed, in this case, the positive effect of a fall in A on savings, that goes through the reduction of θ, is not sufficient to compensate for the negative income effect. Since a lower capital-labor ratio also means lower public spending relative to pollution, the transmission rate θ goes up through this channel. These two competing effects on θ explain that the effect of A on the proportion of healthy people h_2 is not so clear-cut.

The endemic steady state depends also on pollution intensity of production, captured by α, as it modifies the transmission rate of epidemics. When α goes up, so does θ: epidemic spreads more easily. Examining Figure 1, the curve $H_1(a)$ shifts downward, whereas $H_2(a)$ is not affected: both a_2 and h_2 fall. A higher pollution intensity is thus damaging for the economy as it increases the transmission rate of epidemics and therefore the number of infected people. This negatively affects both the labor income and the saving rate $(\beta(h)/(1+\beta(h)))$, which reduces

capital investment along the steady state with epidemic outbreak. Therefore, any environmental policy or technological progress that aims to reduce the pollution rate is useful. It decreases the proportion of infected people, through a lower transmission rate. But interestingly, it will also raise capital and production. We are not able to quantify the precise impact of such policy measures on economic variables, but this type of environmental improvement can at least been considered as a tool accompanying an appropriate fiscal policy.

5 Convergence to endemic or disease-free steady state

We analyze the issue of convergence by studying the local stability properties of the two steady states $(a_1, 1)$ and (a_2, h_2). Using Assumption 1, equations (17) and (18) rewrite:

$$h_{t+1} = h_t + (1-h_t)\left[\gamma - \theta(\eta(h_t, a_t))h_t\right] \tag{32}$$

$$a_{t+1}h_{t+1} + b = \frac{\beta(h_{t+1})}{1+\beta(h_{t+1})}[(1-\tau^f)(1-s)Aa_t^s h_t + \tau] \tag{33}$$

where $A = 1$ if $h_t > \underline{h}$ and $\eta(h_t, a_t)$ is given by equation (24). We also keep in mind that this two-dimensional dynamic system involves two predetermined variables, h_t and $a_t = k_t/h_t$. Differentiating these two equations in the neighborhood of a steady state, we get:

$$dh_{t+1} = J_{hh}dh_t + J_{ha}da_t \tag{34}$$

$$da_{t+1} = J_{ah}dh_t + J_{aa}da_t \tag{35}$$

where the terms J_{ii} are the elements of the associated Jacobian matrix given by:

$$J_{hh} = 1 - \gamma + \theta(\eta)h - (1-h)\left[\theta(\eta) + h\theta'(\eta)\frac{\partial \eta}{\partial h}\right] \tag{36}$$

$$J_{ha} = -(1-h)h\theta'(\eta)\frac{\partial \eta}{\partial a} \tag{37}$$

$$J_{ah} = \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)A\frac{a^s}{h} - \left[\frac{a}{h} - \frac{\beta'(h)}{h(1+\beta(h))^2}((1-\tau^f)(1-s)Aa^s h + \tau)\right] \left[1 - \gamma + \theta(\eta)h - (1-h)\left[\theta(\eta) + h\theta'(\eta)\frac{\partial \eta}{\partial h}\right]\right] \tag{38}$$

$$J_{aa} = \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1} + \left[\frac{a}{h} - \frac{\beta'(h)}{h(1+\beta(h))^2}((1-\tau^f)(1-s)Aa^s h + \tau)\right] (1-h)h\theta'(\eta)\frac{\partial \eta}{\partial a} \tag{39}$$

with $\eta = \eta(h, a)$. Substituting $h = 1$ in these equations, we easily obtain:

Proposition 4 *Under Assumptions 1-4, inequality (28) and $b < b_1$, the steady state with no infected people, $(a_1, 1)$, is a saddle because $\theta(\eta_1) > \gamma$, where $\eta_1 \equiv \eta(1, a_1)$.*

Proof. See Appendix E. ■

Under Assumption 4, the steady state with no epidemics is a saddle. Hence, since the two dynamic variables are predetermined, the economy cannot converge toward a steady state without infected people. Indeed, the dynamics of the capital-labor ratio, which is governed by the equilibrium between capital investment and savings, is stable. However, under Assumption 4, the transmission rate exceeds the recovery rate at the disease-free steady states. Then, the evolution of healthy people is unstable, which explains that one cannot converge to this steady state with no epidemics.

We focus now on the dynamics around the steady state (a_2, h_2) characterized by $h_2 < 1$:

Proposition 5 *Under Assumptions 1-4, inequality (31), $\alpha > \underline{\alpha}$ and $b < b_2$, there exits $\widetilde{\beta}'(1) > 0$ such that the steady state (a_2, h_2) is stable for $\beta'(1) > \widetilde{\beta}'(1)$.*

Proof. See Appendix F. ■

The endemic steady state is the only one which is stable. This means that the economy should converge to this steady state rather than toward the steady state with no infected people. As a direct implication, under our current assumptions, epidemics will persist and will not collapse even in the long run.

We notice that the condition $\alpha > \underline{\alpha}$ is the one that ensures the uniqueness of the steady state with epidemic outbreak (see Proposition 2).[10] Of course, it promotes the stability of the steady state. Convergence toward the steady state with epidemic outbreak requires $\beta'(1)$ high enough, i.e. the longevity is sufficiently sensitive to the share of healthy people.[11] Taking into account the dynamic equations (32) and (33), consider an increase in h_t and a_t. This induces an important increase in labor income, savings and hence future capital. At the same time, these raises may favor a higher transmission rate θ_t, which dampens the positive dynamics for h_t. When agent's longevity is highly sensitive to an improvement of the health condition in the economy, the increase in savings is dampened which prevents capital accumulation to be explosive. Rather, the economy converges to a stationary long-run equilibrium with epidemic outbreak.

6 The role of fiscal policy

The fiscal instruments seem to play a crucial role on the existence of both steady states and their stability properties, through their impacts on the transmission rate which depends on public

[10] It excludes any bifurcation (pitchfork) associated to an eigenvalue which would cross the value one.

[11] This is a sufficient condition which excludes the occurrence of a flip bifurcation whatever the value of $\theta'(\eta)$. It does not mean that a flip bifurcation could occur otherwise. For instance, if $\theta'(\eta)$ is sufficiently weak in absolute value, such a bifurcation is always excluded. In any case, we are not interesting in the existence of endogenous cycles in this paper.

spending. The question is therefore to know if a modification of the fiscal policy could improve the health and macroeconomic situation described until now. We investigate first whether following an appropriate choice of the level of debt b and transfer τ, the existence of the steady state with epidemic outbreak (a_2, h_2) can be ruled out and the economy may rather converge to the steady state without epidemics.

Considering that Assumption 4 is not satisfied, i.e. $\underline{h}\theta(\eta(\underline{h}, \widehat{a})) < \gamma$, we show in the following proposition that the steady state with epidemic outbreak may be ruled out while the steady state with no infected people becomes stable.

Proposition 6 *Under Assumptions 1-3,* $\underline{h}\theta(\eta(\underline{h}, \widehat{a})) < \gamma$, $\alpha > \underline{\alpha}$ *and*

$$\frac{b}{b-\tau} < \min\left\{\frac{\beta(\gamma/\theta(\tau^f/\alpha))}{1+\beta(\gamma/\theta(\tau^f/\alpha))}\frac{1-s}{s}; \frac{\beta(1)}{1+\beta(1)}\frac{1-s}{A}\right\} \tag{40}$$

there exits $b_b > 0$ *such that for* $b < b_b$, *the steady state* (a_2, h_2) *does no more exist while the steady state* $(a_1, 1)$ *is stable.*

Proof. See Appendix G. ■

This proposition shows that for an appropriate choice of debt b and transfer τ, the economy can enter in a configuration where there is only one existing steady state, the one without epidemics and infected people. Figure 2 illustrates the proposition.

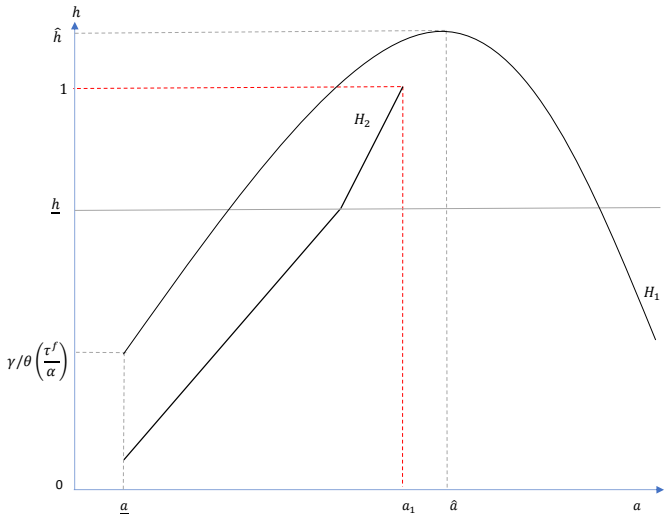

Figure 2: Steady state with no infected people $(a_1, 1)$

In addition, this unique steady state is stable because the transmission rate is now lower than the rate of recovery, which implies that the share of infected people converges to zero. This means that the economy might converge toward the long-run disease-free equilibrium.

For an agent who was already healthy, converging to such a steady state instead of being at an endemic one is a source of higher welfare because his income raises. This can be dampened by the decrease of interest rate which reduces the return of debt used to consume when old and by the decrease of the share of income consumed $1/(1+\beta(h))$ (see equations (6) and (7)). For an infected people, which becomes healthy at a disease-free steady state, the increase of income is even larger because her labor supply increases. As a result, moving from the endemic to the disease free steady state improves welfare as long as the income gains are sufficiently important.

The results of Proposition 6 requires that the transmission rate is sufficiency low, $\underline{h}\theta(\eta(\underline{h},\widehat{a})) < \gamma$, i.e. $\eta(\underline{h},\widehat{a})$ not too low. By direct inspection of (30), we observe that it is possible if either b or $(b-\tau)/b$ are not too close to 0, taking into account that the pollution rate α is not too high. Debt has to be positive but not too important to remove the unfavorable situation in which the economy converges to a steady state with epidemics. Of course, as already mentioned after Proposition 2, if the level of debt is too high, the two steady states are ruled out and the economy surely collapses because it is not possible to sustain investment in capital that does not converge to zero. Another unfavorable policy corresponds to the situation where, in contrast to Proposition 6, Assumption 4 holds but the level of debt rules out the existence of the endemic steady state. In such a configuration, the disease-free steady state still exists but is unstable, which means that the public policy is not able to improve the health and economic situations.

Focusing on the results of Proposition 6, public debt has a crowding-out effect on capital because part of savings is used to finance it. At the steady state with epidemic outbreak, h is of course lower than at the disease-free steady state. This means that both the labor income and the saving rate $\beta(h)/(1+\beta(h))$ are lower. As a result, for a given level of debt, the share of savings devoted to finance public assets is larger at the steady state with epidemic outbreak. This explains that there are levels of debt such that savings can no more sustain a positive level of capital with epidemic outbreak, whereas it is still possible at the disease-free steady state.

If the government has not the degree of freedom to set its fiscal instruments such that Assumption 4 will be violated, public policy cannot be managed to achieve a transition toward the disease-free steady state. In such a context, the question is rather to know what can improve the features of the stable endemic steady state. Accordingly, we examine now how, under Assumption 4, the government can manage its policy instruments, in particular its public debt b and tax τ^f, to increase the steady state values with epidemic outbreak, namely a_2 and h_2. Let us focus first on a variation of debt b:

Proposition 7 *Under Assumptions 1-4, inequality (31), $\theta(\tau^f/\alpha) < 1+\gamma$, $\alpha > \underline{\alpha}$, $\beta'(1) > \widetilde{\beta}'(1)$ and $b < b_2$, there exists $b_3 > 0$ such that following a slight increase of debt, h_2 increases if $b < b_3$.*

Moreover, there exists $\widetilde{\alpha} > 0$, $\overline{\alpha} > 0$ *and* $b_4 > 0$ *such that a slight increase of debt implies a decrease of* a_2 *for* $\alpha > \max\{\underline{\alpha}, \widetilde{\alpha}\}$ *and an increase of* a_2 *for* $\underline{\alpha} < \alpha < \overline{\alpha} (\leqslant \widetilde{\alpha})$ *and* $b < b_4$.

Proof. See Appendix H. ■

Debt increase affects positively h as it allows to increase public spending. This tends to reduce the transmission rate of epidemics. However, at the same time, it might reduce the capital-labor ratio and, hence, increases the cost of debt, which affects negatively the amount of public spending devoted to health. This last effect on the transmission rate is dampened by a negative effect of the capital-labor ratio reduction on pollution. When debt is not too important, the possible negative effect is not too high and the net effect of the debt variation on h is positive.

The debt increase entails competing effects on the capital-labor ratio. On the one hand, it has a negative effect on capital accumulation through a standard crowding-out effect. On the other hand, a higher level of debt allows to increase public health spending, to reduce the transmission rate of epidemics and hence to increase the share of healthy people. This tends to favor capital accumulation in the economy as it increases the labor income and the saving rate, which depends positively on the share of healthy people through the longevity β. Hence, we highlight a new mechanism through which debt can finally have a crowding-in effect on capital.

As regards the crowding-in effect, it is dampened if the pollution rate is sufficiently high. In such a case, the increase of the share of healthy people through the reduction of the transmission rate is too low. To summarize, the crowding-out effect dominates when α is high, but there is a positive effect of debt on the capital-labor ratio if b is not too high and α has an intermediate value. Of course, when longevity is highly sensitive to the health condition, the critical value of α under which the crowding-in effect of the debt dominates is larger.

Note that the existence of $b_4 > 0$ requires $\tau > 0$. A positive average transfer allows to define a policy favorable to capital accumulation. The increase in h entailed by an increase in government debt leads to a higher positive effect on savings when households' incomes are subsidized. The increase in savings generated by a higher longevity is more important when the government makes high income transfers.

When a debt increase affects positively the share of healthy people and the capital stock, the positive effect on production is clear cut, and we can conjecture a positive effect on welfare. Using (25) and (26), we can also easily conjecture that a decrease of the average lump-sum subsidy τ will have the same effect than an increase of public debt.

We now address the effect of a variation of the product tax rate on the endemic steady state:

Proposition 8 *Under Assumptions 1-4, inequality (31),* $\theta(\tau^f/\alpha) < 1 + \gamma$, $\alpha > \underline{\alpha}$, $\beta'(1) > \widetilde{\beta}'(1)$

and $b < b_2$, there exists $b_5 > 0$ such that following a slight increase of the tax rate on production τ^f, h_2 increases for $b < b_5$.

Moreover, there exist $\widetilde{\alpha}_\tau > 0$ and $\overline{\alpha}_\tau > 0$ such that a slight increase of τ^f implies a decrease of a_2 for $\alpha > \max\{\underline{\alpha}, \widetilde{\alpha}_\tau\}$ and an increase of a_2 for $\underline{\alpha} < \alpha < \overline{\alpha}_\tau (\leqslant \widetilde{\alpha}_\tau)$ and τ close to b.

Proof. See Appendix I. ■

The effects of a variation of τ^f and b on the steady state with epidemics are very similar. On the one hand, an increase of the tax rate increases the amount of public health funds available, g, which reduces the transmission rate of epidemics, even if the capital-labor ratio becomes lower. This raises the share of healthy people, and therefore labor. It improves aggregate savings and hence capital accumulation, through higher labor income and saving rate. On the other hand, a higher tax reduces available income, which has a negative effect on the amount of savings. The first effect of the tax rate, which is positive, is the dominant one if the pollution rate α takes intermediate values and the average subsidy τ is sufficiently high and close to the level of debt b to ensure a high enough income.

On the contrary, the second effect, which is negative, dominates when the pollution rate α is high enough. In this case, pollution is important, the transmission rate higher and the labor force h smaller. The fall in savings entailed by the tax τ^f is highly costly since the variation of healthy worker is quite weak and this policy instrument will not be efficient to increase a_2.

Although the effect of a variation of τ^f and b are similar, it is important to note that both instruments are not perfect substitute. While public debt decreases the share of saving allocated to private assets without affecting directly the amount of saving, production tax has a direct effect on saving. The negative effect of τ^f thus depends on the saving rate. The condition to have a policy favorable for a_2 requires τ close to b. This implies a low level of government spending g and hence a high transmission rate θ. In such a case, saving rate is sufficiently low, so does the negative effect of τ^f.

7 Transfers to address inequalities between healthy and infected agents

In our model, epidemics create inequality. Indeed, at the endemic steady state, we can distinguish two groups of individuals that have heterogeneous income profile. People are either healthy at adult age, able to work and hence earn a wage, or are infected at adult age and receive only the lump sum subsidy. Government can put in place a transfer program for redistribution motives. We address this question in the section.

Substituting the two expressions of consumptions (6) and (7) in the utility function (3), we get the agents j welfare:

$$W_{jt} \equiv \beta(h_{t+1}) \ln r_{t+1} + (1 + \beta(h_{t+1}))[\ln(\Omega_{jt} + \tau_{jt}) - \ln(1 + \beta(h_{t+1}))] \tag{41}$$

As Ω_{jt} is equal to the labor income for healthy and zero for infected, the subsidies τ_{jt} can be used to correct inequality and ensure the same welfare for both kind of agents.

Using (41), at the endemic steady state (a_2, h_2), the difference between the welfare of a healthy $(j = H)$ and an infected $(j = I)$ individual is given by:

$$W_H - W_I = (1 + \beta(h_2)) \left[\ln(w(a_2) + \tau_H) - \ln \tau_I\right]$$

where τ_H is the subsidy distributed to each healthy individual and τ_I the one distributed to each infected people. Then, the welfare is the same for all people if $W_H = W_I$, which is equivalent to:

$$\tau_I - \tau_H = w(a_2) = (1 - \tau^f)A(1 - s)a_2^s \tag{42}$$

Given the values of the average subsidy τ and the share of healthy agents h_2, the transfer system, τ_H and τ_I, is constrained and should satisfy:

$$\tau = h_2\tau_H + (1 - h_2)\tau_I \tag{43}$$

Note that using (42), we have $\tau_I = \tau_H + w(a_2)$. Substituting τ_I in equation (43), we obtain $\tau = \tau_H + (1 - h_2)w(a_2) < \tau_H + w(a_2)$. Since $\tau \geqslant 0$, this means that the income received by each agent is identical and positive.

This result is summarized in the following proposition:

Proposition 9 *Under Assumptions 1-4, inequality (31), $\theta(\tau^f/\alpha) < 1 + \gamma$, $\alpha > \underline{\alpha}$, $\beta'(1) > \widetilde{\beta}'(1)$ and $b < b_2$, all individuals have the same welfare at the endemic steady state (a_2, h_2) if τ_H and τ_I satisfy equations (42) and (43).*

This proposition shows that once the policy parameters b, τ^f and τ are fixed to determine the levels of a_2 and h_2, the two levels of subsidy τ_H and τ_I can be used to equalize the levels of income and welfare of healthy and infected people. Two subsidies different from zero are required to be able to fix the redistribution policy independently of the level of the average subsidy τ and of the stationary levels a_2 and h_2. An income redistribute policy in favor of infected $\tau_I > \tau_H$ is a way to avoid inequality generated by epidemics. Note that such policy could come at an additional cost for healthy people. When public budget for transfer τ is low enough, healthy individuals becomes a taxpayer ($\tau_H < 0$). To tackle inequality due to epidemics without reducing income profile of healthy people, government should devote a large amount of fund to transfer policy. This has to come with an increase in debt.

To sum up, we show that in case of epidemics, the fiscal policy can be used first to rule out any endemic steady state and promote the convergence to a stable long-run equilibrium with no epidemics. If the implementation of such a policy is not possible, we argue that a government should try to improve the situation at the stable endemic steady state. We show that this is possible by raising debt or increasing the fiscal pressure if associated public spending are used to improve health care. Another novelty of this paper is to underline that the effectiveness of these policies strongly depends on the level of pollution, which is linked to the production technology. Finally, if the economy converges to the steady state where healthy and infected individuals coexist, it is possible to redistribute income between individuals with an appropriate choice of subsidies to rule out welfare inequalities.

8 Conclusion

In this paper, we examine the interplay between epidemics, pollution and fiscal policies in a macroeconomic framework. The health impacts of the infection depend on the pollution intensity and on the age profile of the agent. To take into account these features, we study the dynamics of epidemics in an OLG model where fiscal instruments are used to fight the health and macroeconomic consequences of the disease. We emphasize situations in which the transmission rate of epidemics exceeds the recovery rate, implying that the economy cannot achieve a disease-free state. Public debt and income transfers may address such unfavorable situations. We determine some fiscal policies that allow the economy to converge to a state without infected people and with a higher level of capital. In a context in which public policy is not able to eradicate the epidemic, it could however be used to reduce the number of infected people and increase capital per capita in the long run. In an economy where a public health policy is not sufficient to eradicate the epidemics, fiscal policy with public debt is an appropriate tool for living with the epidemic, keeping the number of infected people at a stationary, and potentially low, level. It requires, however, a not too high pollution intensity. This result thus highlights the potential role of environmental policies to prevent and fight efficiently infectious diseases. Finally, a redistribution using subsidies to households is recommended to reduce or even rule out welfare inequalities.

Appendix

A Proof of Lemma 1

A steady state with $h < 1$ satisfies equations (25) and (26). Using (24), we obtain the following derivatives:

$$\frac{\partial \eta}{\partial h} = \frac{b(1-\tau^f)sA - (b-\tau)a^{1-s}}{\alpha A h^2 a} \quad \text{(A. 1)}$$

$$\frac{\partial \eta}{\partial a} = s\frac{b(1-\tau^f)A - (b-\tau)a^{1-s}}{\alpha A h a^2} \quad \text{(A. 2)}$$

Under Assumption 3, we have $\partial\eta/\partial h < 0$ and there exists $\widehat{a} \equiv \left[\frac{b(1-\tau^f)A}{b-\tau}\right]^{\frac{1}{1-s}} > \underline{a}$ such that $\partial\eta/\partial a > 0$ for $a < \widehat{a}$ and $\partial\eta/\partial a < 0$ for $a > \widehat{a}$. Therefore, equation (24) implicitly defines a function $h = H_1(a)$ which is increasing for $\underline{a} < a < \widehat{a}$ and decreasing for $a > \widehat{a}$. The maximum value taken by this function is given by $\widehat{h} = H_1(\widehat{a})$. We also have $\eta(h, \underline{a}) = \eta(h, +\infty) = \tau^f/\alpha$. We deduce that $H_1(\underline{a}) = H_1(+\infty) = \gamma/\theta(\tau^f/\alpha)$ and $H_1(a) \geqslant \gamma/\theta(\tau^f/\alpha)$.

Then, differentiating Equation (26), we obtain:

$$\frac{\partial F}{\partial h} = a - \frac{\beta'(h)}{(1+\beta(h))^2}[(1-\tau^f)(1-s)Aa^s h + \tau] - \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)Aa^s \quad \text{(A. 3)}$$

$$\frac{\partial F}{\partial a} = h\left[1 - \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1}\right] \quad \text{(A. 4)}$$

Using (26) and (A. 3), we have:

$$\frac{\partial F}{\partial h}h = \frac{\beta(h)}{1+\beta(h)}\tau - b - \frac{\beta'(h)h}{(1+\beta(h))^2}[(1-\tau^f)(1-s)Aa^s h + \tau] < 0$$

Using the last equation, we note that:

$$\begin{aligned}\frac{\partial F}{\partial a} &> h\left[1 - \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sA\underline{a}^{s-1}\right] \\ &= h\left[1 - \frac{\beta(h)}{1+\beta(h)}(1-s)\left(1-\frac{\tau}{b}\right)\right] > 0\end{aligned}$$

meaning that $\frac{\partial F}{\partial a} > 0$ for all $a > \underline{a}$. Therefore, equation (26) implicitly defines a function $h = H_2(a)$ which is strictly increasing for all $a > \underline{a}$. We note that $H_2'(a) = -\frac{\partial F}{\partial a}/\frac{\partial F}{\partial h}$ is increasing in A, which means that when h crosses $\underline{h}$, A becomes equal to 1 and the slope increases.

B Proof of Proposition 1

Under Assumption 3, we have $\frac{\beta(1)}{1+\beta(1)}\tau - b < 0$. Using (27), we can easily deduce the following:

1. If $\frac{\beta(1)}{1+\beta(1)}\tau - b$ is not too negative, there are two steady states with $a > 0$ and $h = 1$;
2. If $\frac{\beta(1)}{1+\beta(1)}\tau - b$ is sufficiently negative, there is no steady state with $a > 0$ and $h = 1$.

Note that in the last case, the crowding-out effect of public debt is too high. Even if all the population is healthy, the transfer τ is too low to maintain a sufficient level of saving allowing to finance public debt and to invest in productive capital.

Assuming that b is not too large, we are in the configuration with two steady states. To show the existence of the steady state with the highest level of capital, we note that $\partial^2 F(1,a)/\partial a^2 > 0$ and $F(1,+\infty) > 0$. Therefore, if:

$$A\frac{s}{1-s}\frac{b}{b-\tau} < \frac{\beta(1)}{1+\beta(1)} \tag{B. 5}$$

there exist $b_1 > 0$ such that $F(1,\underline{a}) < 0$ for all $\tau < b < b_1$.

C Proof of Proposition 2

We examine the conditions for the existence of steady states with infected people and epidemic outbreak, i.e $h < \underline{h} < 1$. From (26), we have:

$$F(h,\widehat{a}) = h\widehat{a}\left(1 - \frac{\beta(h)}{1+\beta(h)}\frac{A(h)}{A}\frac{(b-\tau)(1-s)}{b}\right) + b - \tau\frac{\beta(h)}{1+\beta(h)} \tag{C. 6}$$

Under Assumption 4, we have $F(\widehat{h},\widehat{a}) > 0$, which is equivalent to $H_2(\widehat{a}) > H_1(\widehat{a}) = \widehat{h}$. Therefore, there exists a steady state with $h < 1$ if $H_2(\underline{a}) < H_1(\underline{a})$. From (26), we have:

$$\begin{aligned} F(\gamma/\theta(\tau^f/\alpha),\underline{a}) &= \frac{\gamma}{\theta(\tau^f/\alpha)}\left(\underline{a} - \frac{\beta(\gamma/\theta(\tau^f/\alpha))}{1+\beta(\gamma/\theta(\tau^f/\alpha))}(1-\tau^f)(1-s)A\underline{a}^s\right) \\ &\quad + b - \tau\frac{\beta(\gamma/\theta(\tau^f/\alpha))}{1+\beta(\gamma/\theta(\tau^f/\alpha))} \end{aligned} \tag{C. 7}$$

Thus, when the following inequality:

$$\underline{a} < \frac{\beta(\gamma/\theta(\tau^f/\alpha))}{1+\beta(\gamma/\theta(\tau^f/\alpha))}(1-\tau^f)(1-s)A\underline{a}^s \Leftrightarrow \frac{b}{b-\tau}\frac{s}{1-s} < \frac{\beta(\gamma/\theta(\tau^f/\alpha))}{1+\beta(\gamma/\theta(\tau^f/\alpha))}$$

is satisfied, there exists $b_2 > 0$ such that for $b < b_2$, we have $F(\gamma/\theta(\tau^f/\alpha),\underline{a}) < 0$. As $F(\gamma/\theta(\tau^f/\alpha),\underline{a}) < 0$ is equivalent to $H_2(\underline{a}) < H_1(\underline{a}) = \gamma/\theta(\tau^f/\alpha)$, we have sufficient conditions for the existence of a steady state with $h < 1$. Note that $F(\gamma/\theta(\tau^f/\alpha),\underline{a}) < 0$ implies $F(1,\underline{a}) < 0$ because $\partial F(a,h)/\partial h < 0$, which ensures also the existence of the steady state $(a_1, 1)$.

Furthermore, we note that the steady state $(a_1, 1)$ solves $H_2(a_1) = 1$, which is equivalent to $F(1,a_1) = 0$. Since $H_2(a)$ is an increasing function whatever the value of A, it is clear that a steady state with $h < 1$ is always characterized by a lower level of capital-labor ratio a than a steady state with $h = 1$. Using (C. 6), we further have $F(1,\widehat{a}) > 0$. Since $\partial F(h,a)/\partial a > 0$, this implies that $a_1 < \widehat{a}$.

Finally, we note that there is a unique steady state with $h < 1$ if we have $H_2'(a) > H_1'(a)$ at each equilibrium satisfying $H_2(a) = H_1(a)$. Using (26) and (A. 4), we have:

$$H_2'(a) = \frac{h^2\left[1 - \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1}\right]}{b - \frac{\beta(h)}{1+\beta(h)}\tau + \frac{\beta'(h)h}{(1+\beta(h))^2}[(1-\tau^f)(1-s)Aa^s h + \tau]} \quad \text{(C. 8)}$$

Using now (25) and (A. 2), we obtain:

$$H_1'(a) = \frac{-\theta'(\eta)s\frac{b(1-\tau^f)A-(b-\tau)a^{1-s}}{\alpha A a^2}}{\theta(\eta) - \theta'(\eta)\frac{(b-\tau)a^{1-s}-b(1-\tau^f)sA}{\alpha A h a}} \quad \text{(C. 9)}$$

The inequality $H_2'(a) > H_1'(a)$ is thus equivalent to:

$$\begin{aligned} &\alpha A a \gamma > \theta'(\eta)[(b-\tau)a^{1-s} - b(1-\tau^f)sA] \\ &-\theta'(\eta)s\frac{b(1-\tau^f)A-(b-\tau)a^{1-s}}{ah\frac{\partial F}{\partial a}}\left[b - \frac{\beta(h)}{1+\beta(h)}\tau + \frac{\beta'(h)h}{(1+\beta(h))\beta(h)}(ah+b)\right] \quad \text{(C. 10)} \end{aligned}$$

Note that h and a have finite and strictly positive values. We deduce that $H_2'(a) > H_1'(a)$ for α high enough. It means that there exists $\underline{\alpha}$ such that for $\alpha > \underline{\alpha}$, there is a unique steady state (a_2, h_2) such that $h < 1$.

Finally, inequality (22) is satisfied at the steady state (a_2, h_2) if $\theta(\eta(h_2, a_2))(1 - h_2) < 1$. Using (23), this is equivalent to $\theta(\eta(h_2, a_2)) < 1 + \gamma$. Using Lemma 1, this is always satisfied because $\theta(\tau^f/\alpha) < 1 + \gamma$.

D Proof of Proposition 3

Focusing on equations (25) and (26) with $h < 1$, the effect of a variation of A on the endemic steady state outbreak (a_2, h_2) is given by:

$$I\begin{pmatrix} da \\ dh \end{pmatrix} + \begin{pmatrix} \partial Z/\partial A \\ \partial F/\partial A \end{pmatrix} dA = \begin{pmatrix} 0 \\ 0 \end{pmatrix}$$

with

$$I = \begin{pmatrix} \partial Z/\partial a & \partial Z/\partial h \\ \partial F/\partial a & \partial F/\partial h \end{pmatrix}$$

which implies that:

$$\frac{da}{dA} = -\frac{1}{\text{Det}I}\left(\frac{\partial F}{\partial h}\frac{\partial Z}{\partial A} - \frac{\partial Z}{\partial h}\frac{\partial F}{\partial A}\right)$$

$$\frac{dh}{dA} = -\frac{1}{\text{Det}I}\left(-\frac{\partial F}{\partial a}\frac{\partial Z}{\partial A} + \frac{\partial Z}{\partial a}\frac{\partial F}{\partial A}\right)$$

with $-1/\text{Det}I > 0$.

We first examine the effect of A on h_2, examining the sign of dh/dI, which is given by the sign of:

$$-\frac{\partial F}{\partial a}\frac{\partial Z}{\partial A}+\frac{\partial Z}{\partial a}\frac{\partial F}{\partial A} = h\theta'(\eta)\frac{b}{\alpha A^2 a^{2-s}}\left[\frac{b-\tau}{b}a^{1-s}-\frac{\beta(h)}{1+\beta(h)}(1-\tau^f)^2(1-s)sA^2\right]$$

This is strictly positive for all $a<\widehat{a}$ if $\frac{b-\tau}{b}\widehat{a}^{1-s}<\frac{\beta(h)}{1+\beta(h)}(1-\tau^f)^2(1-s)sA^2$. This is equivalent to $\frac{b}{b-\tau}<\frac{\beta(h)}{1+\beta(h)}s(1-s)$, which is never satisfied. This is negative for all $a>\underline{a}$ if $\frac{b-\tau}{b}\underline{a}^{1-s}>\frac{\beta(h)}{1+\beta(h)}(1-\tau^f)^2(1-s)sA^2$. This is equivalent to $\frac{b}{b-\tau}s>\frac{\beta(h)}{1+\beta(h)}(1-s)$, but is incompatible with inequality (31). As a result, we have no clear-cut conclusion concerning the effect of A on h.

We then examine the effect of a variation of the productivity on the stationary capital-labor ratio, da/dA. It is given by the sign of the following expression:

$$\begin{aligned}\frac{\partial F}{\partial h}\frac{\partial Z}{\partial A}-\frac{\partial Z}{\partial h}\frac{\partial F}{\partial A} &= \theta'(\eta)\frac{b-\tau}{\alpha A^2 a^s h}\left[b-\frac{\beta(h)}{1+\beta(h)}\tau+\frac{\beta'(h)h}{(1+\beta(h))\beta(h)}(ah+b)\right]\\ &+ \left[\gamma+\theta'(\eta)\frac{b(1-\tau^f)sA-(b-\tau)a^{1-s}}{\alpha Aa}\right](1-\tau^f)(1-s)a^s\frac{\beta(h)}{1+\beta(h)}\end{aligned}$$

This is positive if and only if:

$$\begin{aligned}&\alpha Aa\gamma>\theta'(\eta)[(b-\tau)a^{1-s}-b(1-\tau^f)sA]\\ &-\theta'(\eta)\frac{b-\tau}{(1-\tau^f)(1-s)Aa^{2s-1}h}\left[\frac{1+\beta(h)}{\beta(h)}b-\tau+\frac{\beta'(h)h}{\beta(h)^2}(ah+b)\right]\end{aligned} \quad \text{(D. 11)}$$

There exist $\overline{\alpha}_A\geqslant 0$ and $\underline{\alpha}_A(\leqslant\overline{\alpha}_A)$ such that this inequality is satisfied for $\alpha>\overline{\alpha}_A$ and is not satisfied for $\alpha<\underline{\alpha}_A$.

Using (C. 10) and (D. 11), $\underline{\alpha}_A>\underline{\alpha}$ is equivalent to:

$$\frac{b-\tau}{b}\left[ah+\frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^s(1-h)\right]>\frac{\beta(h)}{1+\beta(h)}s(1-s)(1-\tau^f)^2A^2 \quad \text{(D. 12)}$$

Using $a>\underline{a}$, this requires:

$$\left[\frac{b(1-\tau^f)s}{b-\tau}\right]^{\frac{s}{1-s}}\left[h+\frac{\beta(h)}{1+\beta(h)}(1-s)(1-h)\frac{b-\tau}{b}\right]>\frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)A^{\frac{1-2s}{1-s}} \quad \text{(D. 13)}$$

which is satisfied if A is low enough.

E Proof of Proposition 4

Substituting $a=a_1$ and $h=1$ in equations (36)-(39), we note that $J_{ha}=0$, which means that the two eigenvalues are given by:

$$J_{hh} = 1-\gamma+\theta(\eta_1) \quad \text{(E. 14)}$$

$$J_{aa} = \frac{\beta(1)}{1+\beta(1)}(1-\tau^f)(1-s)sAa_1^{s-1} \quad \text{(E. 15)}$$

where $\eta_1 \equiv \eta(1, a_1)$. Using (A. 4) and the result that $\partial F/\partial a > 0$ for all $h > 0$, we easily deduce that $J_{aa} \in (0, 1)$.

Using Assumption 4, we have $1 > \underline{h} > \widehat{h} = \gamma/\theta(\eta(\widehat{h}, \widehat{a}))$. This implies that $\theta(\eta(\widehat{h}, \widehat{a})) > \gamma$. Since $\widehat{h} < 1$ and $\widehat{a} > a_1$, we deduce that $\theta(\eta_1) = \theta(\eta(1, a_1)) > \theta(\eta(\widehat{h}, \widehat{a})) > \gamma$. This means that $J_{hh} > 1$.

F Proof of Proposition 5

The stability of the steady state (a_2, h_2) is given by the roots of the characteristic polynomial $P(\lambda) = \lambda^2 - T\lambda + D = 0$, where $T = J_{hh} + J_{aa}$ and $D = J_{hh}J_{aa} - J_{ha}J_{ah}$ are the trace and the determinant of the Jacobian matrix obtained from the linearized system (34)-(35).

Using (36)-(39) and (25), we get:

$$\begin{aligned} T &= 1 + \gamma - \theta(\eta) + \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1} \\ &\quad -(1-h)h\theta'(\eta)\left[\frac{\partial \eta}{\partial h} - \frac{a}{h}\frac{\partial \eta}{\partial a} + \frac{\partial \eta}{\partial a}\frac{\beta'(h)}{h(1+\beta(h))^2}((1-\tau^f)(1-s)Aa^s h + \tau)\right] \qquad \text{(F. 16)} \\ D &= \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1} \\ &\quad \left[1 + \gamma - \theta(\eta) - (1-h)h\theta'(\eta)\left(\frac{\partial \eta}{\partial h} - \frac{a}{sh}\frac{\partial \eta}{\partial a}\right)\right] \qquad \text{(F. 17)} \end{aligned}$$

We note that equation (25) with $h < 1$ implies that $\theta(\eta) > \gamma$. Using (A. 4), $\partial F/\partial a > 0$, $\theta'(\eta) < 0$, $\partial \eta/\partial h < 0$ and $\partial \eta/\partial a > 0$ at the steady state (a_2, h_2), we deduce that $D < 1$.

Using (F. 16) and (F. 17), and also (25) and (26), we have:

$$\begin{aligned} P(1) &= 1 - T + D \\ &= (1-h)\left[1 - \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1}\right]\left[\theta(\eta) + h\theta'(\eta)\frac{\partial \eta}{\partial h}\right] \\ &+ (1-h)\frac{\theta'(\eta)}{h}\frac{\partial \eta}{\partial a}\left[b - \frac{\beta(h)}{1+\beta(h)}\tau + \frac{\beta'(h)h}{\beta(h)(1+\beta(h))}(ah+b)\right] \qquad \text{(F. 18)} \end{aligned}$$

Substituting (A. 1) and (A. 2), $1 - T + D > 0$ is equivalent to $H_2'(a) > H_1'(a)$, where $H_2'(a)$ and $H_1'(a)$ are given by (C. 8) and (C. 9) respectively. This is satisfied for $\alpha > \underline{\alpha}$.

Using (25), (26), (A. 1),(A. 2), (F. 16) and (F. 17), we obtain:

$$\begin{aligned} &P(-1) = 1 + T + D \\ &= [2 - \theta(\eta)(1-h)]\left[1 + \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1}\right] \\ &+(1-h)\theta'(\eta)\left[(1-s)\frac{b-\tau}{\alpha Aha^s} + \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1}\frac{(1-s)b(1-\tau^f)}{\alpha ha}\right. \\ &\left.-s\frac{b(1-\tau^f)A - (b-\tau)a^{1-s}}{\alpha Aha^2}\frac{\beta'(h)}{\beta(h)(1+\beta(h))}(ah+b)\right] \qquad \text{(F. 19)} \end{aligned}$$

Note that on the one hand, the second term (third line) on the right-hand side of this equation is negative because $\theta'(\eta) < 0$. On the other hand, the third term (fourth line) on the right-hand side of equation (F. 19) is positive because $\theta'(\eta) < 0$ and $\partial\eta/\partial a > 0$. Moreover, using inequality (22), we note that $\theta(\eta)(1-h) < 1$. Since a_2 and h_2 have finite values, we easily deduce that there is $\widetilde{\beta}'(1) > 0$ such that $1 + T + D > 0$ and (a_2, h_2) is a sink for $(\beta'(h) >)\beta'(1) > \widetilde{\beta}'(1)$.

G Proof of Proposition 6

Under $\alpha > \underline{\alpha}$, inequality (40) and $b < b_2$, we have $F(\gamma/\theta(\tau^f/\alpha), \underline{a}) < 0$ or equivalently $H_2(\underline{a}) < H_1(\underline{a})$ (see the proof of Proposition 2).

Since $\underline{h}\theta(\eta(\underline{h}, \widehat{a})) < \gamma$, the peak of $H_1(a)$ is above $\underline{h}$. We show now that it can be higher than $h = 1$. Indeed, $\widehat{h} = H_1(\widehat{a}) > H_2(\widehat{a})(> 1)$ is equivalent to:

$$F(\widehat{h}, \widehat{a}) = \widehat{a}\widehat{h}\left[1 - \frac{\beta(\widehat{h})}{1+\beta(\widehat{h})}\frac{1-s}{A}\frac{b-\tau}{b}\right] + b - \tau\frac{\beta(\widehat{h})}{1+\beta(\widehat{h})} < 0 \qquad \text{(G. 20)}$$

Under inequality (40), this is satisfied if $b(> \tau)$ is lower than an upper bound $b_2' > 0$. We note $b_b > 0$ as being $b_b \equiv \min\{b_2; b_2'\}$. Under $\alpha > \underline{\alpha}$, $H_2'(a) > H_1'(a)$ if $H_2(a) = H_1(a)$, which implies that $H_2(a)$ and $H_1(a)$ do not cross for all $a \in (\underline{a}, \widehat{a})$. Therefore, there are no steady state with $h < 1$.

This also means that $H_1(a_1) > H_2(a_1) = 1$. This implies that $\theta(\eta(a_1, 1)) < \gamma$. Using the proof of Proposition 4, we deduce that the steady state $(a_1, 1)$, which still exists, is stable.

H Proof of Proposition 7

Focusing on equations (25) and (26) with $h < 1$, we have:

$$I\begin{pmatrix} da \\ dh \end{pmatrix} + \begin{pmatrix} \partial Z/\partial b \\ \partial F/\partial b \end{pmatrix} db = \begin{pmatrix} 0 \\ 0 \end{pmatrix}$$

with

$$I = \begin{pmatrix} \partial Z/\partial a & \partial Z/\partial h \\ \partial F/\partial a & \partial F/\partial h \end{pmatrix}$$

Thus,

$$\begin{pmatrix} da \\ dh \end{pmatrix} = -I^{-1}\begin{pmatrix} \partial Z/\partial b \\ \partial F/\partial b \end{pmatrix} db$$

with

$$I^{-1} = \frac{1}{\mathrm{Det}I}\begin{pmatrix} \partial F/\partial h & -\partial Z/\partial h \\ -\partial F/\partial a & \partial Z/\partial a \end{pmatrix}$$

The endemic steady state outbreak (a_2, h_2) is characterized by $a_2 < \hat{a}$ and $H_2' = -\frac{\partial F/\partial a}{\partial F/\partial h} > H_1' = -\frac{\partial Z/\partial a}{\partial Z/\partial h}$. Using Lemma 1, at the point (a_2, h_2), we have $\partial Z/\partial a < 0$, $\partial Z/\partial h > 0$, $\partial F/\partial a > 0$ and $\partial F/\partial h < 0$. We thus deduce that $DetI < 0$.

The effect of a variation in debt on the endemic steady state outbreak (a_2, h_2) is given by:

$$\frac{da}{db} = -\frac{1}{\text{Det}I}\left(\frac{\partial F}{\partial h}\frac{\partial Z}{\partial b} - \frac{\partial Z}{\partial h}\right)$$

$$\frac{dh}{db} = -\frac{1}{\text{Det}I}\left(-\frac{\partial F}{\partial a}\frac{\partial Z}{\partial b} + \frac{\partial Z}{\partial a}\right)$$

with $-1/DetI > 0$.

We first examine the effect of a debt variation on h, examining the sign of dh/db. We have:

$$\begin{aligned} -\frac{\partial F}{\partial a}\frac{\partial Z}{\partial b} + \frac{\partial Z}{\partial a} &= -h\left[1 - \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1}\right]\theta'(\eta)\frac{a^{1-s} - (1-\tau^f)sA}{\alpha Aa} \\ &+ \theta'(\eta)s\frac{b(1-\tau^f)A - (b-\tau)a^{1-s}}{\alpha Aa^2} \end{aligned}$$

Thus, we have $dh/db > 0$ if and only if:

$$ah\left[1 - \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1}\right](a^{1-s} - (1-\tau^f)sA) > sb(1-\tau^f)A - s(b-\tau)a^{1-s}$$

Since the left-hand side of this inequality is positive, there is $b_3 > 0$ such that it is satisfied if $b < b_3$.

We then examine the effect of a debt variation on the stationary capital-labor ratio, i.e. da/db. Using the proof of Lemma 1, we have:

$$\begin{aligned} \frac{\partial F}{\partial h}\frac{\partial Z}{\partial b} - \frac{\partial Z}{\partial h} &= -\theta'(\eta)\frac{\partial \eta}{\partial h}h - \theta(\eta(h,a)) \\ &+ \theta'(\eta)\frac{\partial \eta}{\partial b}\left[\frac{\beta(h)}{1+\beta(h)}\tau - b - \frac{\beta'(h)h}{(1+\beta(h))^2}[(1-\tau^f)(1-s)Aa^s h + \tau]\right] \end{aligned}$$

with, under Assumption 3:

$$\frac{\partial \eta}{\partial b} = \frac{a^{1-s} - (1-\tau^f)sA}{\alpha hAa} > 0 \quad ; \quad \frac{\partial \eta}{\partial h}h = \frac{b(1-\tau^f)sA - (b-\tau)a^{1-s}}{\alpha hAa} < 0$$

We thus have $-\frac{\partial \eta}{\partial h}h = \frac{\partial \eta}{\partial b}b - \frac{\tau a^{1-s}}{\alpha hAa}$

$$\begin{aligned} \frac{\partial F}{\partial h}\frac{\partial Z}{\partial b} - \frac{\partial Z}{\partial h} &= \theta'(\eta)\left(\frac{\partial \eta}{\partial b}\left[\frac{\beta(h)}{1+\beta(h)}\tau - \frac{\beta'(h)h}{(1+\beta(h))^2}[(1-\tau^f)(1-s)Aa^s h + \tau]\right] - \frac{\tau a^{1-s}}{\alpha hAa}\right) \\ &- \theta(\eta(h,a)) \end{aligned}$$

$$\begin{aligned} \frac{\partial F}{\partial h}\frac{\partial Z}{\partial b} - \frac{\partial Z}{\partial h} &= -\theta(\eta(h,a)) - \theta'(\eta)\frac{\tau a^{1-s} + (1-\tau^f)sA\tau\beta(h)}{\alpha Aha(1+\beta(h))} \\ &- \theta'(\eta)\frac{a^{1-s} - (1-\tau^f)sA}{\alpha Aha}\frac{\beta'(h)h}{(1+\beta(h))^2}[(1-\tau^f)(1-s)Aa^s h + \tau] \quad \text{(H. 21)} \end{aligned}$$

We have $da/db > 0$ if this last expression is strictly positive. $\frac{\partial F}{\partial h}\frac{\partial Z}{\partial b} - \frac{\partial Z}{\partial h} > 0$ is equivalent to:

$$\alpha A a \gamma < -\theta'(\eta)\frac{\tau a^{1-s} + (1-\tau^f)sA\tau\beta(h)}{1+\beta(h)} - \theta'(\eta)[a^{1-s} - (1-\tau^f)sA]\frac{\beta'(h)h(ah+b)}{\beta(h)(1+\beta(h))} \quad \text{(H. 22)}$$

Therefore, there exist $\overline{\alpha} > 0$ and $\widetilde{\alpha}(\geqslant \overline{\alpha})$ such that this inequality is satisfied for $\alpha < \overline{\alpha}$ and is not satisfied for $\alpha > \widetilde{\alpha}$.[12]

$H_2'(a) > H_1'(a)$ is equivalent to $\alpha > \underline{\alpha}$, or (C. 10). Inequalities (H. 22) and (C. 10) are both satisfied for $\underline{\alpha} < \alpha < \overline{\alpha}$. This interval is non empty if:

$$\frac{\beta'(h)h}{\beta(h)(1+\beta(h))}(ah+b)B_1 > B_2 \quad \text{(H. 23)}$$

with

$$B_1 \equiv a^{1-s} - (1-\tau^f)sA - s\frac{b(1-\tau^f)A - (b-\tau)a^{1-s}}{ah\partial F/\partial a} \quad \text{(H. 24)}$$

$$B_2 \equiv s\frac{b(1-\tau^f)A - (b-\tau)a^{1-s}}{ah\partial F/\partial a}\left(b - \frac{\beta(h)}{1+\beta(h)}\tau\right) - \frac{\tau a^{1-s} + (1-\tau^f)sA\tau\beta(h)}{1+\beta(h)} - [(b-\tau)a^{1-s} - b(1-\tau^f)sA] \quad \text{(H. 25)}$$

Using $a > \underline{a}$, $B_1 > 0$ and $B_2 < 0$ if:

$$\left[(1-\tau^f)sA\right]^{\frac{1}{1-s}} h^2 \frac{\tau}{b}\left(\frac{b}{b-\tau}\right)^{\frac{2-s}{1-s}}\left[1 - \frac{\beta(h)}{1+\beta(h)}(1-s)\frac{b-\tau}{b}\right] > b(1-s)$$

There exists $b_4 > 0$ such that this is satisfied if $b < b_4$.

I Proof of Proposition 8

The effect of a variation in τ^f on the endemic steady state outbreak (a_2, h_2) is given by:

$$I\begin{pmatrix} da \\ dh \end{pmatrix} + \begin{pmatrix} \partial Z/\partial\tau^f \\ \partial F/\partial\tau^f \end{pmatrix} d\tau^f = \begin{pmatrix} 0 \\ 0 \end{pmatrix}$$

which implies that:

$$\frac{da}{d\tau^f} = -\frac{1}{\mathrm{Det}I}\left(\frac{\partial F}{\partial h}\frac{\partial Z}{\partial \tau^f} - \frac{\partial Z}{\partial h}\frac{\partial F}{\partial \tau^f}\right)$$

$$\frac{dh}{d\tau^f} = -\frac{1}{\mathrm{Det}I}\left(-\frac{\partial F}{\partial a}\frac{\partial Z}{\partial \tau^f} + \frac{\partial Z}{\partial a}\frac{\partial F}{\partial \tau^f}\right)$$

with $-1/\mathrm{Det}I > 0$.

We first examine the effect of τ^f on h, examining the sign of $dh/d\tau^f$, which is given by the sign of:

$$-\frac{\partial F}{\partial a}\frac{\partial Z}{\partial \tau^f} + \frac{\partial Z}{\partial a}\frac{\partial F}{\partial \tau^f} = -h^2\left[1 - \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1}\right]\theta'(\eta)\left(\frac{bs}{\alpha ha} + \frac{1}{\alpha}\right) + \theta'(\eta)s\frac{b(1-\tau^f)A - (b-\tau)a^{1-s}}{\alpha A a^2}Aa^s h(1-s)\frac{\beta(h)}{1+\beta(h)}$$

[12] $\overline{\alpha}$ and $\widetilde{\alpha}$ may not be equal because a, h and η depend on α.

Therefore, we have $dh/d\tau^f > 0$ if and only if:

$$\left[1 - \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1}\right]\left(\frac{bs}{a}+h\right) > s\frac{b(1-\tau^f)Aa^{s-1}-(b-\tau)}{a}(1-s)\frac{\beta(h)}{1+\beta(h)}$$

There exists an upper bound $b_5 > 0$ such that this inequality is satisfied for $b < b_5$.

We then examine the effect of a tax variation on the stationary capital stock, $da/d\tau^f$. It is given by the sign of the following expression:

$$\begin{aligned}\frac{\partial F}{\partial h}\frac{\partial Z}{\partial \tau^f} - \frac{\partial Z}{\partial h}\frac{\partial F}{\partial \tau^f} &= h\theta'(\eta)\left(\frac{bs}{\alpha h a}+\frac{1}{\alpha}\right)\left[\frac{\beta(h)}{1+\beta(h)}\tau - b - \frac{\beta'(h)h}{(1+\beta(h))^2}[(1-\tau^f)(1-s)Aa^s h+\tau]\right] \\ &- \left[\theta'(\eta)\frac{b(1-\tau^f)sA-(b-\tau)a^{1-s}}{\alpha h A a}+\theta(\eta)\right]Aa^s h(1-s)\frac{\beta(h)}{1+\beta(h)} \qquad \text{(I. 26)}\end{aligned}$$

This is positive if and only if:

$$\begin{aligned}&-\frac{\theta'(\eta)}{1-s}\left(\frac{bs}{a^s}+ha^{1-s}\right)\left[\frac{1+\beta(h)}{\beta(h)}b-\tau+\frac{\beta'(h)h}{\beta(h)^2}(ah+b)\right] \\ &-\theta'(\eta)[b(1-\tau^f)sA-(b-\tau)a^{1-s}] > \alpha\gamma Aa \qquad \text{(I. 27)}\end{aligned}$$

There exists $\overline{\alpha}_\tau > 0$ and $\widetilde{\alpha}_\tau (\geqslant \overline{\alpha}_\tau)$ such that this inequality is satisfied for $\alpha < \overline{\alpha}_\tau$ and is not satisfied for $\alpha > \widetilde{\alpha}_\tau$.[13]

$H_2'(a) > H_1'(a)$ is equivalent to $\alpha > \underline{\alpha}$, or (C. 10). Inequalities (H. 22) and (I. 27) are both satisfied for $\underline{\alpha} < \alpha < \overline{\alpha}_\tau$. This interval is non empty if:

$$\begin{aligned}&\left[\frac{\beta'(h)h}{(1+\beta(h))\beta(h)}(ah+b)+b-\frac{\beta(h)}{1+\beta(h)}\tau\right] \\ &\left[\frac{1+\beta(h)}{\beta(h)}\left(\frac{bs}{(1-s)a^s}+\frac{ha^{1-s}}{1-s}\right)-s\frac{b(1-\tau^f)A-(b-\tau)a^{1-s}}{ah\partial F/\partial a}\right] > 0 \qquad \text{(I. 28)}\end{aligned}$$

Since the first term into brackets is strictly positive, this inequality is satisfied if the second term into brackets is strictly positive. Using $a > \underline{a}$, this requires:

$$h^2\frac{1+\beta(h)}{\beta(h)}\left(\frac{bs}{1-s}+\frac{h\underline{a}}{1-s}\right)\left[1-\frac{\beta(h)}{1+\beta(h)}(1-s)\left(1-\frac{\tau}{b}\right)\right] > (1-s)(b-\tau) \qquad \text{(I. 29)}$$

Taking into account the expression of $\underline{a}$ given in Assumption 3, inequality (I. 29) is satisfied if τ is sufficiently close to b.

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