



**HAL**  
open science

## Environment, public debt and epidemics

Marion Davin, Mouez Fodha, Thomas Seegmuller

► **To cite this version:**

Marion Davin, Mouez Fodha, Thomas Seegmuller. Environment, public debt and epidemics. 2021.  
halshs-03222251v2

**HAL Id: halshs-03222251**

**<https://shs.hal.science/halshs-03222251v2>**

Preprint submitted on 10 May 2021

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

## Environment, public debt and epidemics

Marion Davin  
Mouez Fodha  
Thomas Seegmuller

WP 2021 - Nr 28

# Environment, public debt and epidemics\*

Marion Davin<sup>†</sup>, Mouez Fodha<sup>‡</sup> and Thomas Seegmuller<sup>§</sup>

May 7, 2021

## 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 and public debt can be used to tackle the effects of epidemics. We show that fiscal policies can promote the convergence to a stable steady state with no epidemics. 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.

*JEL classification:* E6, I18, Q59

*Keywords:* Epidemics, pollution, overlapping generations, public debt.

---

\*This work was supported by the French National Research Agency Grant ANR-17-EURE-0020, ANR-15-CE33-0001-01 and ANR-16-CE03-0005, and by the Excellence Initiative of Aix-Marseille University - A\*MIDEX.

<sup>†</sup>CEE-M, Univ Montpellier, CNRS, INRAE, SupAgro, Montpellier, France. E-mail: marion.davin@umontpellier.fr

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

<sup>§</sup>Corresponding author. 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 Covid-19 epidemic is one of the most serious threat to health in the last decades. To tackle the crisis, countries have put in place extraordinary measures. “Health is priceless. The government will provide all the financial means necessary to bring assistance, to take care of the sick, to save lives. Whatever it takes” declared President Macron in March 2020. This “whatever it takes” policy is indisputably associated to an increase in public debt, to support economic activity and health care system.

The aim of this paper is to study the interplay between the health and economic impacts of epidemics, and to analyze the effects of non-pharmaceutical public interventions, i.e. in the absence of vaccines,<sup>1</sup> in particular health public spending and fiscal policy with public debt.

The recent contributions about the economic consequences of epidemics assess the strategies to fight the disease. They highlight the role of confinement, social distancing and the speed at which a vaccine develops (Acemoglu *et al.*, 2020; Alvarez *et al.*, 2020; Eichenbaum *et al.*, 2020; Toxvaerd, 2021). In our work, we are rather interested in the consequences of the fiscal policy and public debt on the epidemic control, which are often overlooked in the literature, despite their importance, as underlined by the OECD (OECD, 2020).

We develop a three-period overlapping generations (OLG) model with young inactive agents, working adults and old retirees. The dynamics of epidemics are formalized by introducing a SIS (susceptible–infected–susceptible) model, as in Bosi and Desmarchelier (2020), Goenka and Liu (2012, 2020) or Goenka *et al.* (2014).

In our model, the following features are highlighted. First, the impacts of the epidemic depend on the age of the person affected: the older agent bears a 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; U.S. Department of Health & Human Services<sup>2</sup>) and hence, they are more affected by saturated health’s system capacities entailed by the epidemics.

Secondly, we consider a link between epidemics and environmental issues. The degradation of the environmental quality increases the rate of contagion of the epidemic. On this point, we rely on two transmission channels highlighted by empirical works: (1) the loss of biodiversity increases the risk of transmission of an animal virus to humans (IPBES report, 2020), which is known as

---

<sup>1</sup>It follows the terminology of Flaxman *et al.* (2020).

<sup>2</sup><https://www.cdc.gov/coronavirus/2019-ncov/need-extra-precautions/older-adults.html>

the dilution effect (Civitello *et al.*, 2015); (2) air pollution (like PM 2.5 and 10) accelerates the spread of the virus between humans as shown by empirical works interested in the propagation factors of Covid-19 (Austin *et al.*, 2020; Andree, 2020; Bourdrel *et al.*, 2021 ; Cole *et al.*, 2020; Isphording and Pestel, 2021; Persico and Johnson, 2021; Rohrer *et al.*, 2020).

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. Taking into account public debt lever is particularly relevant to examine the economic consequences of important epidemics. For example, the OECD points out the sharp increase in public debt expected in OECD area because of the Covid-19 crisis.<sup>3</sup>

In our model, which takes neither the vaccine nor herd immunity into account, once the epidemic appears, it cannot disappear spontaneously, without any public intervention. 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. We show that if public debt increases, it enables the financing of these health expenses. Such a strategy can allow to rule out the steady state with epidemic and converge to a steady state without epidemic. 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, it could however be used to reduce the number of infected people and increase capital per capita in the long run equilibrium with epidemic. 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.

---

<sup>3</sup>According to OECD (2020), “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.”

Finally, when the economy converges to the steady state with epidemic, 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 transfer 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.

There exists a huge literature interested in the analysis of the interactions between economics and epidemics. Basically, these 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. 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 greatly differ from those explored in this paper.

With respect to the different consequences of epidemics according to the age of individuals, the seminal contribution of Chakraborty *et al.* (2016) studies the dynamics of poverty and health in an overlapping generations model, applied to sub-Saharan Africa countries, where poor health is the result of infectious diseases. Survival depends on whether or not people contract an infectious disease early in life and prematurely die from it. The Covid-19 epidemic has generalized this intergenerational issue since older individuals have a much higher probability of developing a severe form of the disease (Williamson *et al.*, 2020). Hence, Acemoglu *et al.* (2020) assume that infection and fatality rates vary between young, adult and old agents. Similarly, Glover *et al.* (2020) consider a two-sector economy and suppose that individuals differ by age (young or retired) and health status, while Brotherhood *et al.* (2020) consider an economy populated by a continuum of young and old agents. These works, like most of the articles dealing with the Covid-19 epidemic in homogeneous agent settings (Alvarez *et al.*, 2020; Eichenbaum *et al.*, 2020; Toxvaerd, 2021), focus on containment and social distancing strategies. Generally, the results are conditioned by the date on which a medical solution, such as a vaccine, appears.

The interplay between environmental degradation and viruses appears in papers analysing the infectivity of the disease, i.e. the dilution effect. Bosi and Desmarchelier (2020) assume that pollution externalities deteriorate biodiversity, which in turn affects the dilution effect. They show that the magnitude of the dilution effect and pollution strongly influence the effects of the public policies and the persistence of economic fluctuations. Augeraud-Véron *et al.* (2020) focus specifically on the conservation of biodiversity and the likelihood of the spread of zoonoses. They show that the lockdown is more severe in societies valuing more human life, and the biodiversity conservation is more relevant for more patient and altruistic economies. In Brock and Xepapadeas (2020), containment policies such as social distancing could stop the epidemic in the short run. In the long run, policies that target the consumption patterns and climate change help to reduce the potential of the epidemic.

Our contribution highlights the direct consequence of public finance on epidemics and *vice-versa*. We show that in the absence of vaccine, 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 complements 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 and the Susceptible-Infected-Susceptible assumptions. 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. 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 ( $t = 0, 1, \dots$ ) 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.*, 2020; Eichenbaum *et al.*, 2020) which also introduced the category of recovered people. In our OLG framework, considering the SIS rather than the SIR model does not alter the results.

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 his 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 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.<sup>4</sup> 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 longevity of the elder, meaning that their longevity or survival probability decreases with the proportion of infected people. The more infected people there are in the population, the higher the probability to die prematurely when old. 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

---

<sup>4</sup>We consider an OLG model precisely to take into account that adult and old people do not face the same consequences of being infected.



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; U.S. Department of Health & Human Services) 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  $\theta_t$  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  $\gamma I_t$  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 ( $\theta_t$ ).

The dynamics of infected people are exactly the opposite:<sup>5</sup>

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

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  $\gamma$  is constant, as in Goenka and Lui (2020). On the other hand, we assume that the transmission rate  $\theta_t$  is increasing with pollution  $P_t$ . Many evidences support this assumption. Recently, Cole *et al.* (2020) highlight that  $PM_{2.5}$  and  $NO_2$  concentrations have a positive link with Covid-19 cases, hospital admissions and deaths in Germany, while Coker *et al.* (2020) shows that an increase in  $PM_{2.5}$  concentration is associated to an increase of mortality in the Northern Italian regions. The transmission rate  $\theta_t$  is also decreasing with public expenditures in health care  $G_t$ , which represent masks, tests, emergency services, research for treatments, among the others. The higher  $G_t$ , the

---

<sup>5</sup>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.

higher public health response capacity and the lower the spread of epidemics. 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  $\gamma$ .

### 2.3 Households

The economy is populated by overlapping generations of agents living for three periods. When young, an agent does neither consume nor work but, as we already mentioned, has contact with other generations such that he 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  $j \in \{1, \dots, N\}$  born at period  $t - 1$  are represented by the following utility function over consumption when adult  $c_{jt}$  and old  $d_{jt+1}$ :

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

where  $\beta(h_{t+1}) \in (0, 1]$  measures the longevity when old or the survival probability. This utility function introduces a heterogeneity between adult and old agents facing the virus. To be more specific, whereas an infected adult has no chance to die, the probability to not live through the entire old-age increases with the proportion of infected people in the population 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  $\beta'(h_{t+1}) < \beta'(0)$ . With this specification, we formalize the idea that 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. Thus, the sensitivity of  $\beta$  to  $h$  captures also the limited resilience of healthcare institutes.

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

$$\sigma_{jt} + c_{jt} = \Omega_{jt} + \tau_{jt} \quad (4)$$

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

where  $\sigma_{jt}$  represents savings of individual  $j$ . Since each household supplies one unit of labor if she is healthy,  $\Omega_{jt}$  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.  $\tau_{jt} \geq 0$  is a lump-sum subsidy/tax which is

specific to each individual and can for instance be used by the government to (partially) cover the loss of labor income. 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.<sup>6</sup>

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

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

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.

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

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

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

The parameter  $\underline{h} \in (0, 1)$  is a threshold for epidemics, representing massive epidemic outbreak which implies quarantine and containment. As a result, a virulent epidemic causes a double penalty on the production side. On the one hand, it reduces the amount of people able to work, on the other hand, it reduces the productivity of the population not infected.

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  $(1 - \tau_t^f) Y_t - r_t K_t - w_t L_t$ , such that we get:

$$r_t = (1 - \tau_t^f) A(h_t) s a_t^{s-1} \equiv r(a_t) \quad (7)$$

$$w_t = (1 - \tau_t^f) A(h_t) (1 - s) a_t^s \equiv w(a_t). \quad (8)$$

---

<sup>6</sup>For simplification, we also assume complete depreciation of capital.

## 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  $\theta_t$ .<sup>7</sup> In addition, the government can also limit the economic costs of epidemics by paying some lump-sum subsidies  $\tau_{jt}$  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 emission is given by  $r_t B_{t-1}$ . The intertemporal budget constraint for the government therefore satisfies for all  $t \geq 0$ :

$$B_t = r_t B_{t-1} + G_t + \tau_t N - \tau_t^f Y_t \quad (9)$$

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:

$$\sum_{j=1}^N \Omega_{jt} = w_t L_t = w_t H_t \quad (10)$$

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  $Y_t/N = A(h_t) a_t^s h_t$ , with  $a_t = k_t/h_t$ . Then, using (7), the government budget constraint (9) rewrites:

$$g_t = b_t - r(a_t) b_{t-1} - \tau_t + \tau_t^f A(h_t) a_t^s h_t \quad (11)$$

Equilibrium on the asset market is ensured by  $k_{t+1} + b_t = \sum_{j=1}^N \sigma_{jt}/N$ . We use (6), (8), (10), and  $k_{t+1} = a_{t+1} h_{t+1}$  to get:

$$a_{t+1} h_{t+1} + b_t = \frac{\beta(h_{t+1})}{1 + \beta(h_{t+1})} (w(a_t) h_t + \tau_t) \quad (12)$$

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

$$h_{t+1} = h_t + (1 - h_t) [\gamma - \theta [g_t / (\alpha A(h_t) a_t^s h_t)] h_t] \quad (13)$$

---

<sup>7</sup> $G_t$  could be masks, tests, emergency services, controls, information campaign...

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

By inspection of equation (11), we now define which policy parameters will be considered as fixed and will be used to conduct comparative statics.

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

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:

$$g_t = b(1 - r(a_t)) - \tau + \tau^f A(h_t)a_t^s h_t \equiv g(a_t, h_t) \quad (14)$$

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 (12) and (13) for all  $t \geq 0$ , i.e.

$$h_{t+1} = h_t + (1 - h_t) [\gamma - \theta[g(a_t, h_t)/(\alpha A(h_t)a_t^s h_t)]h_t] \quad (15)$$

$$a_{t+1}h_{t+1} + b = \frac{\beta(h_{t+1})}{1 + \beta(h_{t+1})} [(1 - \tau^f)(1 - s)A(h_t)a_t^s h_t + \tau] \quad (16)$$

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:

$$(1 - h) [\gamma - \theta[g(a, h)/(\alpha A(h)a^s h)]h = 0 \quad (17)$$

$$ah + b = \frac{\beta(h)}{1 + \beta(h)} [(1 - \tau^f)(1 - s)A(h)a^s h + \tau] \quad (18)$$

with

$$g(a, h) = b[1 - (1 - \tau^f)A(h)sa^{s-1}] - \tau + \tau^f A(h)a^s h \quad (19)$$

and

$$\theta[g(a, h)/(\alpha A(h)a^s h)](1 - h) < 1 \quad (20)$$

By direct inspection of equation (17), 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 (17), a steady state with  $h < 1$  satisfies:

$$\gamma = \theta[g(a, h)/(\alpha A(h)a^s h)]h \quad (21)$$

Using (19), we easily get:

$$\frac{g}{p} = \frac{g(a, h)}{\alpha A(h)a^s h} = \frac{b - \tau}{\alpha A h a^s} - \frac{b(1 - \tau^f)s}{\alpha h a} + \frac{\tau^f}{\alpha} \equiv \eta(h, a) \quad (22)$$

with  $A = 1$  if  $h > \underline{h}$ . Therefore, equation (21) rewrites:

$$\gamma = \theta(\eta(h, a))h = \theta \left[ \frac{b - \tau}{\alpha A h a^s} - \frac{b(1 - \tau^f)s}{\alpha h a} + \frac{\tau^f}{\alpha} \right] h \equiv Z(h, a) \quad (23)$$

In the following, we assume that there is a primary deficit independently of the subsidy  $\tau$ . 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 (19), 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 (18):

$$F(h, a) \equiv ah + b - \frac{\beta(h)}{1 + \beta(h)} [(1 - \tau^f)(1 - s)A(h)a^s h + \tau] = 0 \quad (24)$$

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

**Lemma 1** Equation (23) implicitly defines a function  $h = H_1(a)$  which is increasing for  $\underline{a} < a < \hat{a}$  and decreasing for  $a > \hat{a}$ , with

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

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

Equation (24) 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 steady states with no infected people. A solution  $h = 1$  satisfies equation (17). It also implies that  $A(h) = 1$ . Therefore, equation (18) rewrites:

$$F(1, a) = a - \frac{\beta(1)}{1 + \beta(1)}(1 - \tau^f)(1 - s)a^s + b - \frac{\beta(1)}{1 + \beta(1)}\tau = 0 \quad (25)$$

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

**Proposition 1** Under Assumptions 1-3 and

$$A \frac{b}{b - \tau} < \frac{\beta(1)}{1 + \beta(1)} \frac{1 - s}{s} \quad (26)$$

there exists  $b_1 > 0$  such that for all  $b < b_1$ , there is a unique steady state with no infected people,  $(a_1, 1)$ , which always satisfies inequality (20).

**Proof.** See Appendix B. ■

Assuming that  $b$  is not too large, the crowding-out effect of public debt is not too high. Moreover, inequality (26) 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 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  $\hat{h} < \underline{h}$  holds. From (23),  $\hat{h}$  is a solution of:

$$h = \frac{\gamma}{\theta(\eta(h, \hat{a}))} \quad (27)$$

Substituting  $\hat{a} = \left[ \frac{b(1-\tau^f)A}{b-\tau} \right]^{\frac{1}{1-s}}$  in (22), we get:

$$\eta(h, \hat{a}) = \frac{(1-s)}{\alpha h (1-\tau^f)^{\frac{s}{1-s}} A^{\frac{1}{1-s}}} \left( \frac{b-\tau}{b} \right)^{\frac{1}{1-s}} b + \frac{\tau^f}{\alpha} \quad (28)$$

which is a decreasing function of  $h$ .

The left-hand side of (27) 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  $\underline{h} > \frac{\gamma}{\theta(\eta(\underline{h}, \hat{a}))}$ , the value  $\hat{h}$  which solves (27) belongs to  $(0, \underline{h})$  and a steady state with infected people is always marked by epidemic outbreak. Let us assume:

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

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

**Proposition 2** *Under Assumptions 1-4,*

$$\frac{b}{b-\tau} < \frac{\beta(\gamma/\theta(\tau^f/\alpha))}{1 + \beta(\gamma/\theta(\tau^f/\alpha))} \frac{1-s}{s} \quad \text{and} \quad \theta(\tau^f/\alpha) < 1 + \gamma \quad (29)$$

*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  $\underline{a} < a_2 < \hat{a}$  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:  $\underline{a} < a_2 < a_1 < \hat{a}$ .*

**Proof.** See Appendix C. ■

We notice that conditions (29) and  $b < b_2$  both ensure the existence of a steady state with epidemic outbreak that coexists with the one with no infected people.<sup>8</sup> A level of debt not too high associated to a high enough saving rate maintain 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 depends 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

<sup>8</sup>Note that this inequality (29) is more stringent than inequality (26).



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.

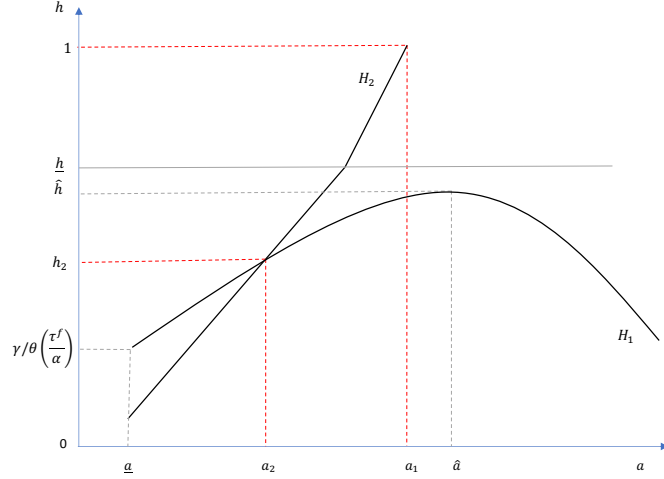


Figure 1: Steady states with epidemics outbreak  $(a_2, h_2)$  and no infected people  $(a_1, 1)$

We pay a particular attention to the effect of a variation in the productivity parameter  $A$  on the steady state with epidemics. A decrease or a sufficiently low level of  $A$  can be seen as the economic impacts of a virulent pandemic, that may require containment measures costly for economic activity for example.<sup>9</sup> In such a period of containment, there is not only a lower productivity or efficiency of labor, but also a lower capital utilization. Therefore, we analyze now in more details the effect of a variation of  $A$  on  $h_2$  and  $a_2$ :

**Proposition 3** *Under Assumptions 1-4, inequality (29),  $\theta(\tau^f/\alpha) < 1 + \gamma$ ,  $\alpha > \underline{\alpha}$  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  $\bar{\alpha}_A$  and  $\underline{\alpha}_A (\leq \bar{\alpha}_A)$  such that a slight increase of  $A$  implies an increase of  $a_2$  for  $\alpha > \max\{\underline{\alpha}, \bar{\alpha}_A\}$  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  $\theta$ . Through this direct effect of  $A$ , pollution decreases more than public spending, which means that the transmission rate  $\theta$  goes down.

<sup>9</sup>Note that we do not explicitly introduce the reduction of social interactions due to a containment even if it could be taken into account through a variation of  $\theta$ .

A lower productivity also implies a fall in income that reduces savings and hence capital-labor ratio if  $\alpha$  is high enough. Indeed, in this case, the positive effect of a fall in  $A$  on savings, that goes through the reduction of  $\theta$ , 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  $\theta$  goes up through this channel. These two competing effects on  $\theta$  explain that the effect of  $A$  on the proportion of healthy people  $h_2$  is not so clear-cut.

The steady state with infected people depends also on pollution intensity of production, captured by  $\alpha$ , as it modifies the transmission rate of epidemics. When  $\alpha$  goes up, so does  $\theta$ : 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 be considered as a tool accompanying an appropriate fiscal policy.

## 5 Convergence to epidemic or non-epidemic 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 (15) and (16) rewrite:

$$h_{t+1} = h_t + (1 - h_t) [\gamma - \theta(\eta(h_t, a_t))h_t] \quad (30)$$

$$a_{t+1}h_{t+1} + b = \frac{\beta(h_{t+1})}{1 + \beta(h_{t+1})} [(1 - \tau^f)(1 - s)Aa_t^s h_t + \tau] \quad (31)$$

where  $A = 1$  if  $h_t > \underline{h}$  and  $\eta(h_t, a_t)$  is given by equation (22). 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:

$$dh_{t+1} = J_{hh}dh_t + J_{ha}da_t \quad (32)$$

$$da_{t+1} = J_{ah}dh_t + J_{aa}da_t \quad (33)$$

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

$$J_{hh} = 1 - \gamma + \theta(\eta)h - (1-h) \left[ \theta(\eta) + h\theta'(\eta) \frac{\partial \eta}{\partial h} \right] \quad (34)$$

$$J_{ha} = -(1-h)h\theta'(\eta) \frac{\partial \eta}{\partial a} \quad (35)$$

$$J_{ah} = \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)A \frac{a^s}{h} - \left[ \frac{a}{h} - \frac{\beta'(h)}{h(1+\beta(h))^2}((1-\tau^f)(1-s)Aa^s h + \tau) \right] \\ \left[ 1 - \gamma + \theta(\eta)h - (1-h) \left[ \theta(\eta) + h\theta'(\eta) \frac{\partial \eta}{\partial h} \right] \right] \quad (36)$$

$$J_{aa} = \frac{\beta(h)}{1+\beta(h)}(1-\tau^f)(1-s)sAa^{s-1} + \left[ \frac{a}{h} - \frac{\beta'(h)}{h(1+\beta(h))^2}((1-\tau^f)(1-s)Aa^s h + \tau) \right] \\ (1-h)h\theta'(\eta) \frac{\partial \eta}{\partial a} \quad (37)$$

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

**Proposition 4** *Under Assumptions 1-4, inequality (26) 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 steady state without epidemics. 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 (29),  $\alpha > \underline{\alpha}$  and  $b < b_2$ , there exists  $\tilde{\beta}'(1) > 0$  such that the steady state  $(a_2, h_2)$  is stable for  $\beta'(1) > \tilde{\beta}'(1)$ .*

**Proof.** See Appendix F. ■

The steady state with epidemics 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). 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. Taking into

account the dynamic equations (30) and (31), 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  $\theta_t$ , 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 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  $\tau$ , 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*

$$\frac{b}{b - \tau} < \min \left\{ \frac{\beta(\gamma/\theta(\tau^f/\alpha))}{1 + \beta(\gamma/\theta(\tau^f/\alpha))} \frac{1 - s}{s}; \frac{\beta(1)}{1 + \beta(1)} \frac{1 - s}{A} \right\} \quad (38)$$

*there exists  $b_b > 0$  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  $\tau$ , 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.

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 equilibrium without epidemics. The results of Proposition 6 requires that the transmission rate is sufficiently low,  $\underline{h}\theta(\eta(\underline{h}, \widehat{a})) < \gamma$ , i.e.  $\eta(\underline{h}, \widehat{a})$  not too low. By direct inspection of (28), we observe that it is possible if either  $b$  or  $(b - \tau)/b$

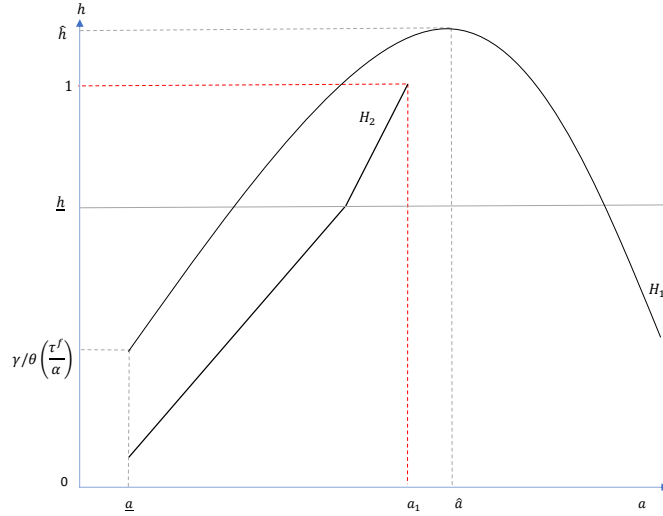


Figure 2: Steady state with no infected people  $(a_1, 1)$

are not too close to 0, taking into account that the pollution rate  $\alpha$  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 steady state with epidemics. In such a configuration, the steady state without epidemics 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 steady state without epidemic. 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 steady state without epidemic.

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 steady state without epidemic. In such a context, the question is rather to know what can improve the features of the stable steady state with epidemics. Accordingly, we examine now how, under Assumption 4, the government can manage its policy instruments, in particular its

public debt  $b$  and tax  $\tau^f$ , 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 (29),  $\theta(\tau^f/\alpha) < 1 + \gamma$ ,  $\alpha > \underline{\alpha}$ ,  $\beta'(1) > \tilde{\beta}'(1)$  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  $\tilde{\alpha} > 0$ ,  $\bar{\alpha} > 0$  and  $b_4 > 0$  such that a slight increase of debt implies a decrease of  $a_2$  for  $\alpha > \max\{\underline{\alpha}, \tilde{\alpha}\}$  and an increase of  $a_2$  for  $\underline{\alpha} < \alpha < \bar{\alpha} (\leq \tilde{\alpha})$  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  $\beta$ . 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  $\alpha$  is high, but there is a positive effect of debt on the capital-labor ratio if  $b$  is not too high and  $\alpha$  has an intermediate value. Of course, when longevity is highly sensitive to the health condition, the critical value of  $\alpha$  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

(23) and (24), we can also easily conjecture that a decrease of the average lump-sum subsidy  $\tau$  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 steady state with epidemics:

**Proposition 8** *Under Assumptions 1-4, inequality (29),  $\theta(\tau^f/\alpha) < 1 + \gamma$ ,  $\alpha > \underline{\alpha}$ ,  $\beta'(1) > \tilde{\beta}'(1)$  and  $b < b_2$ , there exists  $b_5 > 0$  such that following a slight increase of the tax rate on production  $\tau^f$ ,  $h_2$  increases for  $b < b_5$ .*

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

**Proof.** See Appendix I. ■

The effects of a variation of  $\tau^f$  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  $\alpha$  takes intermediate values and the average subsidy  $\tau$  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  $\alpha$  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  $\tau^f$  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  $\tau^f$  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  $\tau^f$  thus depends on the saving rate. The condition to have a policy favorable for  $a_2$  requires  $\tau$  close to  $b$ . This implies a low level of government spending  $g$  and hence a high transmission rate  $\theta$ . In such a case, saving rate is sufficiently low, so does the negative effect of  $\tau^f$ .

## 7 Transfers to address inequalities between healthy and infected agents

In our model, epidemics create inequality. Indeed, at the steady state with epidemics, 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.

Using (4)-(6), we have adult and old consumption for agent  $j$ :

$$c_{jt} = \frac{1}{1 + \beta(h_{t+1})} (\Omega_{jt} + \tau_{jt}) \quad (39)$$

$$d_{jt+1} = \frac{r_{t+1}}{1 + \beta(h_{t+1})} (\Omega_{jt} + \tau_{jt}) \quad (40)$$

Substituting these two expressions in the utility function (3), we get the agents  $j$  welfare :

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

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

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

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

where  $\tau_H$  is the subsidy distributed to each healthy individual and  $\tau_I$  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:

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

Given the values of the average subsidy  $\tau$  and the share of healthy agents  $h_2$ , the transfer system,  $\tau_H$  and  $\tau_I$ , is constrained and should satisfy:

$$\tau = h_2 \tau_H + (1 - h_2) \tau_I \quad (43)$$

Note that using (42), we have  $\tau_I = \tau_H + w(a_2)$ . Substituting  $\tau_I$  in equation (43), we obtain  $\tau = \tau_H + (1 - h_2)w(a_2) < \tau_H + w(a_2)$ . Since  $\tau \geq 0$ , 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 (29),  $\theta(\tau^f/\alpha) < 1 + \gamma$ ,  $\alpha > \underline{\alpha}$ ,  $\beta'(1) > \tilde{\beta}'(1)$  and  $b < b_2$ , all individuals have the same welfare at the steady state with epidemics  $(a_2, h_2)$  if  $\tau_H$  and  $\tau_I$  satisfy equations (42) and (43).*



This proposition shows that once the policy parameters  $b$ ,  $\tau^f$  and  $\tau$  are fixed to determine the levels of  $a_2$  and  $h_2$ , the two levels of subsidy  $\tau_H$  and  $\tau_I$  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  $\tau$  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  $\tau$  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 steady state with epidemics 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 steady state with epidemics. 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 state without epidemics. 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 remove entirely 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 an effective vaccine against the

viruses does not exist or is not sufficiently available, 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 (23) and (24). Using (22), we obtain the following derivatives:

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

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

Under Assumption 3, we have  $\partial \eta / \partial h < 0$  and there exists  $\hat{a} \equiv \left[ \frac{b(1 - \tau^f)A}{b - \tau} \right]^{\frac{1}{1-s}} > \underline{a}$  such that  $\partial \eta / \partial a > 0$  for  $a < \hat{a}$  and  $\partial \eta / \partial a < 0$  for  $a > \hat{a}$ . Therefore, equation (22) implicitly defines a function  $h = H_1(a)$  which is increasing for  $\underline{a} < a < \hat{a}$  and decreasing for  $a > \hat{a}$ . The maximum value taken by this function is given by  $\hat{h} = H_1(\hat{a})$ . 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  $H_1(a) \geq \gamma / \theta(\tau^f / \alpha)$ .

Then, differentiating Equation (24), 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 (24) 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 (24) 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 (25), 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  $\tau$  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:

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

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 (24), we have:

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

Under Assumption 4, we have  $F(\hat{h}, \hat{a}) > 0$ , which is equivalent to  $H_2(\hat{a}) > H_1(\hat{a}) = \hat{h}$ . Therefore, there exists a steady state with  $h < 1$  if  $H_2(\underline{a}) < H_1(\underline{a})$ . From (24), we have:

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

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  $F(1, \hat{a}) > 0$ . Since  $\partial F(h, a)/\partial a > 0$ , this implies that  $a_1 < \hat{a}$ .

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 (24) and (A. 4), we have:

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

Using now (23) 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:

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

Note that  $h$  and  $a$  have finite and strictly positive values. We deduce that  $H_2'(a) > H_1'(a)$  for  $\alpha$  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 (20) is satisfied at the steady state  $(a_2, h_2)$  if  $\theta(\eta(h_2, a_2))(1-h_2) < 1$ . Using (21), 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 (23) and (24) with  $h < 1$ , the effect of a variation of  $A$  on the steady state with epidemics 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:

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

This is strictly positive for all  $a < \hat{a}$  if  $\frac{b-\tau}{b} \hat{a}^{1-s} < \frac{\beta(h)}{1+\beta(h)} (1-\tau^f)^2 (1-s) s A^2$ . 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  $\frac{b-\tau}{b} \underline{a}^{1-s} > \frac{\beta(h)}{1+\beta(h)} (1-\tau^f)^2 (1-s) s A^2$ . This is equivalent to  $\frac{b}{b-\tau} s > \frac{\beta(h)}{1+\beta(h)} (1-s)$ , but is incompatible with inequality (29). 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:

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

This is positive if and only if:

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

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

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

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

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 (34)-(37), 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  $1 > \underline{h} > \hat{h} = \gamma/\theta(\eta(\hat{h}, \hat{a}))$ . This implies that  $\theta(\eta(\hat{h}, \hat{a})) > \gamma$ . Since  $\hat{h} < 1$  and  $\hat{a} > a_1$ , we deduce that  $\theta(\eta_1) = \theta(\eta(1, a_1)) > \theta(\eta(\hat{h}, \hat{a})) > \gamma$ . 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 (32)-(33).

Using (34)-(37) and (23), we get:

$$T = 1 + \gamma - \theta(\eta) + \frac{\beta(h)}{1 + \beta(h)}(1 - \tau^f)(1 - s)SAa^{s-1} \quad (\text{F. 16})$$

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

$$D = \frac{\beta(h)}{1 + \beta(h)}(1 - \tau^f)(1 - s)SAa^{s-1} \left[ 1 + \gamma - \theta(\eta) - (1 - h)h\theta'(\eta) \left( \frac{\partial \eta}{\partial h} - \frac{a}{sh} \frac{\partial \eta}{\partial a} \right) \right] \quad (\text{F. 17})$$

We note that equation (23) 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 (23) and (24), we have:

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

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 (23), (24), (A. 1),(A. 2), (F. 16) and (F. 17), we obtain:

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

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 (20), we note that  $\theta(\eta)(1 - h) < 1$ . Since  $a_2$  and  $h_2$  have finite values, we easily deduce that there is  $\tilde{\beta}'(1) > 0$  such that  $1 + T + D > 0$  and  $(a_2, h_2)$  is a sink for  $(\beta'(h) >) \beta'(1) > \tilde{\beta}'(1)$ .

## G Proof of Proposition 6

Under  $\alpha > \underline{\alpha}$ , inequality (38) 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  $h\theta(\eta(h, \hat{a})) < \gamma$ , the peak of  $H_1(a)$  is above  $\underline{h}$ . We show now that it can be higher than  $h = 1$ . Indeed,  $\hat{h} = H_1(\hat{a}) > H_2(\hat{a}) (> 1)$  is equivalent to:

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

Under inequality (38), 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  $a \in (\underline{a}, \hat{a})$ . 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 (23) and (24) 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

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

The steady state with epidemics 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  $\text{Det}I < 0$ .

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

$$\begin{aligned} \frac{da}{db} &= -\frac{1}{\text{Det}I} \left( \frac{\partial F}{\partial h} \frac{\partial Z}{\partial b} - \frac{\partial Z}{\partial h} \right) \\ \frac{dh}{db} &= -\frac{1}{\text{Det}I} \left( -\frac{\partial F}{\partial a} \frac{\partial Z}{\partial b} + \frac{\partial Z}{\partial a} \right) \end{aligned}$$

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

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:

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

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



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

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

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  $\bar{\alpha} > 0$  and  $\tilde{\alpha} (\geq \bar{\alpha})$  such that this inequality is satisfied for  $\alpha < \bar{\alpha}$  and is not satisfied for  $\alpha > \tilde{\alpha}$ .<sup>10</sup>

$H_2'(a) > H_1'(a)$  is equivalent to  $\alpha > \underline{\alpha}$ , or (C. 10). Inequalities (H. 22) and (C. 10) are both satisfied for  $\underline{\alpha} < \alpha < \bar{\alpha}$ . 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

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

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

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

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

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  $\tau^f$  on the steady state with epidemics outbreak  $(a_2, h_2)$  is given by:

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

<sup>10</sup> $\bar{\alpha}$  and  $\tilde{\alpha}$  may not be equal because  $a$ ,  $h$  and  $\eta$  depend on  $\alpha$ .

which implies that:

$$\begin{aligned}\frac{da}{d\tau^f} &= -\frac{1}{\text{Det}I} \left( \frac{\partial F}{\partial h} \frac{\partial Z}{\partial \tau^f} - \frac{\partial Z}{\partial h} \frac{\partial F}{\partial \tau^f} \right) \\ \frac{dh}{d\tau^f} &= -\frac{1}{\text{Det}I} \left( -\frac{\partial F}{\partial a} \frac{\partial Z}{\partial \tau^f} + \frac{\partial Z}{\partial a} \frac{\partial F}{\partial \tau^f} \right)\end{aligned}$$

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

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

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

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

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

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:

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

This is positive if and only if:

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

There exists  $\bar{\alpha}_\tau > 0$  and  $\tilde{\alpha}_\tau (\geq \bar{\alpha}_\tau)$  such that this inequality is satisfied for  $\alpha < \bar{\alpha}_\tau$  and is not satisfied for  $\alpha > \tilde{\alpha}_\tau$ .<sup>11</sup>

$H'_2(a) > H'_1(a)$  is equivalent to  $\alpha > \underline{\alpha}$ , or (C. 10). Inequalities (H. 22) and (I. 27) are both satisfied for  $\underline{\alpha} < \alpha < \bar{\alpha}_\tau$ . This interval is non empty if:

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

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:

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

<sup>11</sup> $\bar{\alpha}_\tau$  and  $\tilde{\alpha}_\tau$  may not be equal because  $a$ ,  $h$  and  $\eta$  depend on  $\alpha$ .

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

## References

- [1] Acemoglu, D., V. Chernozhukov, I. Werning and M. D. Whinston (2020): “Optimal targeted lockdowns in a multi-group SIR model”, *American Economic Review: Insights*, forthcoming.
- [2] Alvarez, F. E., D. Argente and F. Lippi (2020): “A simple planning problem for COVID-19 lockdown”, NBER Working Paper 26981.
- [3] Andree, B. P. J. (2020): “Incidence of COVID-19 and connections with air pollution exposure: evidence from the Netherlands”, Policy Research Working Paper Series 9221, The World Bank.
- [4] Augeraud-Véron, E., G. Fabbri and K. Schubert (2020): “Prevention and mitigation of epidemics: biodiversity conservation and confinement policies”, *Journal of Mathematical Economics*, **93**, 102484.
- [5] Austin, W., S. Carattini, J. G. Mahecha and M. Pesko (2020): “Covid-19 mortality and contemporaneous air pollution”, CESifo Working Paper Series 8609, CESifo.
- [6] Bosi, S. and D. Desmarchelier (2020): “Biodiversity, infectious diseases and the dilution effect”, *Environmental Modeling and Assessment*, **25**, 277–292.
- [7] Bourdrel T., Annesi-Maesano I., Alahmad B. et al. (2021): “The impact of outdoor air pollution on COVID-19: a review of evidence from in vitro, animal, and human studies”, *Eur Respir Rev* **30:200242**.
- [8] Brock, W. and A. Xepapadeas (2020): “The economy, climate change and infectious diseases: links and policy implications”, *Environmental and Resource Economics*, **76**, 811-824.
- [9] Brotherhood, L., P. Kircher, C. Santos and M. Tertilt (2020): “An economic model of the Covid-19 epidemic: the importance of testing and age-specific policies”, CEPR Discussion Paper 14695.
- [10] Chakraborty, S., C. Papageorgiou and F. Sebastian (2016): “Health cycles and health transitions”, *Macroeconomic Dynamics*, **20**, 189-213.

- [11] Chang, H.J., N. Huang, C. H. Lee, Y. J. Hsu, C. J. Hsieh and Y. J. Chou (2004): “The impact of the SARS epidemic on the utilization of medical services: SARS and the fear of SARS”, *American Journal of Public Health*, **94**, 562-564
- [12] Civitello, D.J., J. Cohen, H. Fatima, N. T. Halstead, J. Liriano, T. A. McMahon, N. Ortega, E. L. Sauer, T. Sehgal, S. Young and J. R. Rohr (2015): “Biodiversity inhibits parasites: Broad evidence for the dilution effect”, *Proc. Natl. Acad. Sci. U.S.A.*, **112**, 8667-8671.
- [13] Coker, E. S., L. Cavalli, E. Fabrizi, G. Guastella, E. Lippo, M. L. Parisi, N. Pontarollo, M. Rizzati, A. Varacca and S. Vergalli (2020): “The effects of air pollution on Covid-19 related mortality in Northern Italy ”, *Environmental and Resource Economics*, **76**, 611-634.
- [14] Cole, M. A., C. Ozgen and E. Strobl (2020): “Air pollution exposure and Covid-19 in Dutch municipalities”, *Environmental and Resource Economics*, **76**, 581-610.
- [15] Douglas, J. W. and R. Raudla (2020): “What is the remedy for state and local fiscal squeeze during the COVID-19 recession? More debt, and that is okay”, *American Review of Public Administration*, **50**, 584-589.
- [16] Eichenbaum, M. S., S. Rebelo and M. Trabandt (2020): “The macroeconomics of epidemics”, NBER Working Paper 26882.
- [17] Flaxman, S., S. Mishra, and A. Gandy, *et al.* (2020): “Estimating the effects of non-pharmaceutical interventions on COVID-19 in Europe”, *Nature*, **584**, 257-261.
- [18] Geoffard P. Y. and T. Philipson (1997): “Disease eradication: private versus public vaccination”, *American Economic Review*, **87**, 222-230.
- [19] Glover, A., J. Heathcote, D. Krueger and J.-V. Ríos-Rull (2020): “Health versus wealth: on the distributional effects of controlling a pandemic”, NBER Working Paper 27046.
- [20] Goenka, A. and L. Liu (2012): “Infectious diseases and endogenous fluctuations”, *Economic Theory*, **50**, 125-149.
- [21] Goenka, A. and L. Liu (2020): “Infectious diseases, human capital and economic growth”, *Economic Theory*, **70**, 1-47.
- [22] Goenka, A., L. Liu and M. H. Nguyen (2014): “Infectious diseases and economic growth”, *Journal of Mathematical Economics*, **50**, 34-53.
- [23] Hethcote, H. (2000): “The mathematics of infectious diseases”, *SIAM Review*, **42**, 599-653.

- [24] IPBES (2020): “Workshop report on biodiversity and pandemics of the intergovernmental platform on biodiversity and ecosystem services.” Daszak, P., das Neves, C., Amuasi, J., Hayman, D., Kuiken, T., Roche, B., Zambrana-Torrel, C., Buss, P., Dundarova, H., Feferholtz, Y., Foldvari, G., Igbiosa, E., Junglen, S., Liu, Q., Suzan, G., Uhart, M., Wannous, C., Woolaston, K., Mosig Reidl, P., O’Brien, K., Pascual, U., Stoett, P., Li, H., Ngo, H. T., IPBES secretariat, Bonn, Germany.
- [25] Ipsfording, I. E. and N. Pestel (2021): “Pandemic meets pollution: poor air quality increases deaths by Covid-19”, *Journal of Environmental Economics and Management*, **108**, 102448.
- [26] Kontis, V., J. E. Bennett, T. Rashid *et al.* (2020): “Magnitude, demographics and dynamics of the effect of the first wave of the COVID-19 pandemic on all-cause mortality in 21 industrialized countries”, *Nat Med*, **26**, 1919–1928.
- [27] Liu, K., Y. Chen, R. Lin and K. Han (2020): “Clinical features of COVID-19 in elderly patients: a comparison with young and middle-aged patients”, *Journal of Infection*, **80**, e14-e18.
- [28] OECD (2020): Sovereign Borrowing Outlook for OECD Countries 2020, Special Covid-19 Edition.
- [29] Persico, C. and K. R. Johnson (2021): “The effects of increased pollution on COVID-19 cases and deaths”, *Journal of Environmental Economics and Management*, forthcoming.
- [30] Rohrer, M., A. Flahault and M. Stoffel (2021): “Peaks of fine particulate matter may modulate the spreading and virulence of COVID-19”, *Earth Systems and Environment*, **4**, 789-796.
- [31] Rust, G., M. Melbourne, B. I. Truman, E. Daniels, Y. Fry-Johnson and T. Curtin (2009): “Role of the primary care safety net in pandemic influenza”, *American Journal of Public Health*, **99**, S316-S323.
- [32] Toxvaerd, F. (2021): “Equilibrium social distancing”, Cambridge Working Papers in Economics, Faculty of Economics, University of Cambridge.
- [33] Wendling, Z. A., J. W. Emerson, A. de Sherbinin, D. C. Esty, *et al.* (2020): “2020 environmental performance index”, New Haven, CT: Yale Center for Environmental Law & Policy. epi.yale.edu
- [34] Williamson, E. J., A. J. Walker, K. Bhaskaran *et al.* (2020): “Factors associated with COVID-19-related death using OpenSAFELY”, *Nature*, **584**, 430-436.