

June 2021

“Covid-19 and a Green Recovery?”

Aditya Goenka, Lin Liu and Manh-Hung Nguyen

Covid-19 and a Green Recovery?*

Aditya Goenka[†] Lin Liu[‡] Manh-Hung Nguyen[§]

June 9, 2021

Abstract

Preliminary evidence indicates that pollution increases severity and likelihood of Covid-19 infections as is the case for many other infectious diseases. This paper models the interaction of pollution and preventive actions on transmission of infectious diseases in a neoclassical growth framework where households do not take into account how their actions affects disease transmission and production activity results in pollution which increases likelihood of infections. Household can take private actions for abatement of pollution as for controlling disease transmission. Disease dynamics follow *SIS* dynamics. We study the difference in health and economic outcomes between the decentralized economy, where households do not internalize the externalities, and the socially optimal outcomes, and characterize the taxes and subsidies that will decentralize the socially optimal outcomes. Thus, we examine the question whether there are sufficient incentives to reduce pollution, both at the private and public levels, once its effects on disease transmission is taken into account.

Keywords: Covid-19, pollution, environmental policy, infectious disease, Green Recovery, dynamic Pigovian taxes.

JEL Classification: *I15, I16, Q53, H23, E22, C61*

*Manh-Hung Nguyen acknowledges support from ANR under grant ANR-17-EURE-0010 (Investissements d'Avenir program). We thank participants at the *Economic Modelling* Workshop especially Angus Chu, Guido Cozzi, Pietro Peretto and Akos Valentinyi for helpful comments. Any errors are ours.

[†]Department of Economics, University of Birmingham, Email: a.goenka@bham.ac.uk

[‡]Management School, University of Liverpool, Email: lin.liu@liverpool.ac.uk

[§]Toulouse School of Economics, INRAE, University of Toulouse Capitole, Email: manh-hung.nguyen@tse-fr.eu

1 Introduction

Preliminary evidence from China, Italy, USA and other countries has shown that the presence of pollution, *PM* (particulate matter), *NOx* (nitrogen oxides) and ground level ozone increases the severity and likelihood of Covid-19 infections (Conticini, et al. (2020), Martelletti and Martelletti (2020), Wu, et al. (2020), Yongjian, et al. (2020)). Some studies also suggest that higher pollution increases aerosol transmission (Setti, et al. (2020), Qin, et al. (2020)). These pollutants are also known to increase other infections especially acute lower respiratory infections including pneumonia, bronchitis, and influenza (Cienciwicki and Jaspers (2007), Horne, et al. (2018), Huang, et al. (2016), Kampa and Castanas (2008), Kelly and Fussell (2011), Lian, et al. (2014), Mehta, et al. (2013), Tasci, et al. (2018)).¹ Thus, to control Covid-19 or other infectious diseases there are two mechanisms - preventive health expenditures which prevent infections and a pollution abatement policy which by reducing pollution reduces disease transmission. The Covid epidemic has raised expectations of a “Green recovery” where the understanding the role of pollution will increase the impetus for less polluting technologies. This paper adds to the thinking of how likely is it that pollution abatement will result once the additional channel of pollution affecting transmission of Covid-19 and other infectious diseases is taken into account.

This paper studies the interaction of these two instruments in a dynamic general equilibrium analysis by extending the economic epidemiological model of Goenka, Liu and Nguyen (2014) and Goenka and Liu (2019) to model the interaction of pollution with health where the disease dynamics are of *SIS* type used to model Covid-19. Pollution is modeled as a flow (consistent with evidence on *PM* and *NOx* ((Varotsos *et al.* (2005), Windsor and Toumi (2001), Zeka *et al.* (2005))) which increases with productive activity. The contact rate in the *SIS* model² is increased by pollution and decreased by preventive health expenditures. The health investment is chosen to maximize discounted welfare along with current consumption and investment in physical capital. Thus, the interaction between pollution and health is endogenized and depends on optimal decisions. In the model, there are two externalities. One is the pollution externality where the pollution resulting from production increases disease transmission. The second is the disease externality, i.e. a household in deciding its optimal plans does not take into account the effect of their decisions on the evolution of the infectious disease (see Geoffard and Philipson (1996), Gersovitz and Hammer (2004), and Goenka and Liu (2019) for modeling of disease externalities). We study the decentralized dynamic equilibrium and contrast it with the social planner’s efficient outcome that internalizes both these externalities.

We show that there can be two steady states: a disease free steady state (which is essentially the neoclassical steady state) and a disease endemic steady state if in equilibrium, the disease is infective enough. As the model is complex to solve analytically we study to what extent the outcomes are affected by the externalities through numerically analyzing the model. Since there is insufficient information so far to calibrate the effect of diseases and preventive health expenditures on disease evolution we fix the other parameters and vary the elasticity of pollution on the contact rate, the elasticity of pollution abatement and the TFP to get a sense of the qualitative properties of the model. We also characterize the dynamic Pigovian taxes that will decentralize the efficient

¹Pollution also increases incidence non-communicable diseases such as asthma, COPD, and other respiratory diseases as well cardiovascular diseases. See Goenka, et al. (2020) for dynamic models studying nexus of pollution and non-communicable diseases but this is not the focus of this paper.

²This modeling choice is discussed in detail in the following section

outcomes. Analogous to the first set of numerical exercises we study how the taxes will change as we vary the two elasticities and the TFP. Several interesting results emerge. First, even if there are two externalities, two instruments are insufficient to decentralize the efficient outcome since pollution affects the economy through multiple channels. Second, the subsidy on health expenditure and pollution abatement are the same even though their effects on disease transmission need not be. The intuition is that in equilibrium, the marginal benefit of pollution abatement and health expenditure are equal as they both effect the economy only through the effect on the contact rate. Third, in the efficient outcome even if there is more abatement and health expenditures and better health outcomes, pollution is higher than in the decentralized. While there is more abatement undertaken the effect of the higher output - due to increased labor supply and output mainly due to higher labor - dominates. Thus, there is no “disease dividend” from controlling the disease in controlling pollution. This is consistent with the evidence that emission levels have gone up and even exceeded pre-lockdown levels in many regions that have controlled Covid infections (see Myllyvirta (2020) for study of emissions in China). Fourth, countries with higher TFP will have higher pollution as the incentive to tax capital reduces. Thus, faster growing economies will have lower incentive to control pollution. The last two implications are consistent with the evidence that the Green Recovery that was hoped after the Covid outbreak may be evanescent (see Harvey (2020)).

The papers most close to this paper are Bosi and Desmarchelier (2018, 2019) and Goenka and Liu (2020). These papers use *SIS* dynamics in a growth model and model the disease externality. While Bosi and Desmarchelier (2018) has pollution affecting disease transmission directly as the current paper, Goenka and Liu (2019) have health capital (as does Goenka, Liu and Nguyen (2014)) and a negative effect of capital stock which can be interpreted as pollution. Bosi and Desmarchelier (2018) treat pollution as a pure externality, while the Goenka and Liu (2020) also considers health interventions. Bosi and Desmarchelier (2019) extend their earlier paper by making utility depend on pollution and by considering abatement of pollution which is modeled as a stock rather than flow. This paper studies the effect of both pollution and health and includes health responses as well as abatement activity.

The plan of the paper is as follows. Section 2 develops the model, Section 3 analyzes the decentralized equilibria, Section 4 the centralized equilibria where the social planner internalizes the externalities, Section 5 does the numerical analysis of the steady state equilibria, Section 6 the dynamic Pigovian taxes which decentralize the planning outcomes, and Section 7 concludes.

2 Model

Epidemiology We use the *SIS* model to study the spread of the disease. While this was the most common modeling choice to model Covid-19 (see Ferguson (2020)) it is not well understood for how long is disease related immunity conferred for coronaviruses such as Covid-19. The evidence is preliminary, but there is emerging evidence that subsequent immunity may not be long lasting. Long, et al. (2020) using data from China find evidence consistent with steep decline in 2-3 months. Similar results were found in Ibarra, et al. (2020), Isho, et al. (2020), Ripberger, et al. (2020), Ward, et al. (2020). On the other hand Wajnberg, et al. (2020) and Sekine, et al. (2020) find evidence suggesting longer immunity. As a modeling strategy Kissler, et al. (2020) use an *SIRS* model for medium run projections. As we are concerned about the medium to longer run in

this paper we abstract from the temporary immunity phase (i.e. the state R).³ The population (N_t) is divided in two classes: susceptible, healthy and who can catch the disease (S_t) and infective, those infected and capable of transmitting the disease (I_t). with $S_t + I_t = N_t$.

$$\begin{aligned} dS_t/dt &= bN_t - dS_t - \alpha S_t I_t / N_t \\ dI_t/dt &= \alpha S_t I_t / N_t - \gamma I_t - dI_t \\ S_t, I_t, N_t &\geq 0, \forall t; \\ S_0, I_0, N_0 &> 0 \text{ with } N_0 = S_0 + I_0, \end{aligned}$$

where b is the exogenous birth rate, d is exogenous death rate, we assume that $d \leq b$. One of the features of Covid-19 is disease related mortality. The case fatality rate (CFR) is estimated to be around 1.4%, i.e. the percentage of the individuals who are known to be infected die from the disease. This has deep implications for thinking about the control of Covid-19. Our paper studies the trade-offs between controlling pollution and disease prevention and in this we abstract from it. See Goenka, Liu and Nguyen (2020a) on modeling mortality in a *SIS* model without pollution.⁴ The key epidemiology variables are the contact rate, α , i.e. the average number of adequate contacts of a person to catch the disease per unit time and γ , the recovery rate from the disease. In this paper we endogenize α by making it depend on health expenditures and on pollution. We treat γ as exogenous to keep the model tractable. Antivirals and anti-inflammatory drugs are now known to reduce severity of the Covid illness but these are inexpensive and widely available prior to the outbreak. Goenka, Liu and Nguyen (2014) endogenize the recovery rate in a model without pollution.

The proportions of susceptible and infective is given by $s_t = \frac{S_t}{N_t}$, $i_t = \frac{I_t}{N_t}$. The population growth rate is given by

$$dN_t/dt = (b - d)N_t, \quad b \geq 0, \quad d \geq 0, \quad b - d \geq 0.$$

We assume $b - d \geq 0$ so that the population is not declining. As $s + i = 1$ and $\dot{s} + \dot{i} = 0$ we can describe the epidemiology dynamics by the following equation, the law of motion of the infectives:

$$\dot{i} = \alpha i(1 - i) - (b + \gamma)i.$$

There are two steady states in the pure-epidemiology model, the disease free steady state

$$(s^*, i^*) = (1, 0)$$

and the disease endemic steady state

$$(s^*, i^*) = \left(\frac{b + \gamma}{\alpha}, \frac{\alpha - (b + \gamma)}{\alpha} \right).$$

³This is also consistent with many of the other infectious diseases that are the main sources of disease related mortality, in particular malaria, tuberculosis, dengue, and influenza also do not have disease related immunity. While an individual may have immunity to a particular strain of influenza for a short period, the virus mutates and there is no lasting immunity. HIV/AIDS is a disease of *SI* class and its epidemiology is not captured by either *SIS* or *SIR* models.

⁴Goenka, Liu and Nguyen (2020b) model the effect of disease mortality in a *SIR* model.

The disease free steady state always exists, and the disease endemic steady state exists only if $\alpha > (b + \gamma)$. If this inequality is satisfied, then the disease free steady state is unstable and the disease endemic steady state is stable, otherwise the disease free steady state is stable. There is a trans-critical bifurcation when $\alpha = (b + \gamma)$. The basic reproduction number, $R_0 = \frac{\alpha}{b + \gamma}$ in this model.

Production and Pollution

There are many perfectly competitive firms that maximize profit by choosing physical capital, k , and labor, l , as inputs taking the real interest rate, R , and the wage rate, W , as given. The assumptions on the production function, $f(k, L)$ are standard and as follows.

Assumption 1. *The production function $f(k, l) : \mathbb{R}_+^2 \rightarrow \mathbb{R}_+$ is \mathcal{C}^2 , homogeneous of degree one, and*

1. $f_1 > 0, f_{11} < 0, f_2 > 0, f_{22} < 0$,
2. $\lim_{k \rightarrow 0} f_1 = \infty, \lim_{k \rightarrow \infty} f_1 = 0$ and $f(0, l) = f(k, 0) = 0$.

Profit maximization implies the following marginal conditions:

$$\begin{aligned} R &= f_1(k, 1 - i) \\ W &= f_2(k, 1 - i) \end{aligned} \tag{1}$$

Production also results in a flow of pollution. By pollution we mean primarily PM and NO_x which have been shown to affect transmission of Covid-19 (Conticini, et al. (2020), Martelletti and Martelletti (2020), Setti, et al. (2020), Qin, et al. (2020), Wu, et al. (2020), and Yongjian, et al. (2020)) and increase other infections such as acute lower respiratory infections including pneumonia, bronchitis and influenza (Cienciwicki and Jaspers (2007), Horne, et al. (2018), Huang, et al. (2016), Kampa and Castanas (2008), Kelly and Fussell (2011), Lian, et al. (2014), Mehta, et al. (2013), Tasci, et al. (2018)). We treat this as a flow, i.e. it does not accumulate, as evidence suggests that it does not accumulate (Varotsos *et al.* (2005), Windsor and Toumi (2001), Zeka *et al.* (2005)). In the decentralized economy, this is treated as an externality. The evolution of pollution is given below.

Assumption 2. *The level of pollution is a function of output y and abatement q - that is, $P(y, q)$ where $P : \mathbb{R}_+^2 \rightarrow \mathbb{R}_+$ is \mathcal{C}^2 . We assume*

- $P_1(y, q) > 0$ and $P_2(y, q) < 0$.

In the paper we will model both the private choice of abatement as well as the optimal amount of abatement. In the decentralized economy, the pollution level is given as:

$$P = P(\bar{Y}, q) = P(f(k, 1 - i), q), \tag{2}$$

where the \bar{Y} denotes that the output is taken as given so that pollution is an externality.

Labor supply: We assume that the labor force (l) consists of healthy people: $l = s$. Then l inherits the dynamics of $l = 1 - i$.⁵ We are assuming for simplicity that all infected workers do not work (see Goenka and Liu (2020) and Goenka, Liu and Nguyen (2020a) for further discussion of this assumption).

Economic epidemiology model

In this paper we endogenize the contact rate, α . As there are no special therapies for treatment of Covid-19 other than available therapies that reduce severity of the infection, we treat γ as exogenous.⁶ The contact rate depends on preventive health expenditure h and pollution P . In this paper we are treating health expenditures as only a flow of expenditures that does not accumulate as opposed to a stock of capital. This is consistent with the modeling in Eichenbaum, et al. (2020) where NPIs that reduce infections act as a tax on consumption - what we label as health expenditures or infection preventing activities.⁷ Thus, the contact rate is the function $\alpha(h, P)$.

Assumption 3. *The contact rate function: $\alpha(h, P) : \mathbb{R}_+ \rightarrow \mathbb{R}_+$ is a \mathcal{C}^2 function with*

- $\alpha_1(h, P) < 0$ and $\alpha_2(h, P) > 0$.

Thus, in the model the only role of pollution is to increase the contact rate. Pollution, indeed, has other affects in the economy: it can create production and consumption externalities, and increase the non-infectious mortality rate. We abstract from these effects as these have been modeled in dynamic general equilibrium models and we want to concentrate on the interaction of pollution, infectious disease transmission and preventive health measures.⁸

Households: We assume the economy is populated by a continuum of non-atomic identical households who are the representative decision-making agents. The size of the population in each household grows over time at the rate of $b - d \geq 0$, where b is the birth rate and d is the death rate. We treat the demographic parameters, b and d , as exogenous and abstract from the fertility-mortality nexus. Within each household, an individual is either susceptible (healthy and not yet infected by the disease) or infective (infected and capable of transmitting it to others).

Each household is assumed to be sufficiently large so that the proportion of the household in each disease status is identical to the corresponding population proportion. Thus, within a household, the proportion of healthy individuals is s , proportion of infected individuals is i , and proportion of recovered individuals is r . Each household understands and anticipates how the disease evolves and is fully forward-looking with regard to its possible future states as well as its present situation. However, following Gersovitz and Hammer (2004) the household considers itself small relative to the population and believes that the disease status within the household does

⁵We take labor supply by the healthy workers as inelastic. If labor supply is elastic then under standard assumption of preferences $u(c, l) > 0$ the qualitative features of the model are not affected. (see Goenka and Liu (2013) for a SIS model with elastic labor supply. If we drop this assumption then labor supply can be an independent source of non-linear dynamics and cycles.

⁶In Goenka, Liu and Nguyen (2014) we modelled γ as a function of h .

⁷Bosi and Desmarchellier (2018) have $\alpha(P)$, Goenka, Liu and Nguyen (2014) model this as $\alpha(h)$, Goenka and Liu (2019) have $\alpha(h, k, e)$ where k, h, e are stocks of physical, health and human capital in an endogenous growth model.

⁸Goenka, Jafarey, and Pouliot (2020) study optimal policies when pollution increases mortality through non-communicable diseases.

not affect the proportion of infectives in the entire population. In particular, the household takes as given the proportion of the population that is infected, denoted as Π , and thinks the probability for the healthy individuals to contract disease is $\alpha\Pi$, rather than αi . As a result, the disease transmission dynamics perceived by the households is now given as follows:

$$\dot{i} = \alpha(h, P(\bar{Y}, q))\Pi(1 - i) - \gamma i - bi. \quad (3)$$

This captures the idea that the household is small relative to the population and does not take into account the externality on disease transmission. It is competitive “disease taking” looking only at private benefits/costs and not social benefits/costs. This distinguishes the competitive model from the social optimum where this externality is taken into account. Furthermore, the household taking the level of output as given, \bar{Y} chooses the amount of *private* abatement.

There is a two-way interaction between the economy and the disease. On the one hand, diseases have direct adverse effects on the economy by reducing the labor force participation. Being infected with a disease affects the productivity of an individual. As make the simplifying assumption that an infected individual is incapacitated by the disease or that the productivity falls to zero, For each household labor supply L is given by the proportion of the healthy individuals, i.e. $L = S$ and dynamics inherits the dynamics of $(1 - i)$.

The representative household’s preferences are given as:

$$\int_0^\infty e^{-\rho t} u(C) N_t dt = \int_0^\infty e^{-(\rho - b + d)t} u(C) N_0 dt, \quad (4)$$

where ρ is the discount factor with $\rho > b - d$, and the initial size of household is assumed to be one. The assumptions on the felicity function are given below. We further assume there is full insurance within each household and all individuals have the same consumption irrespective of their health status. This is indeed optimal, if the household welfare aggregator is concave.

Assumption 4. *The household’s felicity function, $u : \mathcal{R}_{++} \rightarrow \mathcal{R}$ is \mathcal{C}^2 with $u_c > 0$ and $u_{cc} < 0$, jointly with the limit conditions: $\lim_{c \rightarrow 0^+} u_c = \infty$ and $\lim_{c \rightarrow +\infty} u_c = 0$.*

Households take the interest rate R and wage W as given, rent out physical capital K and choose how much to consume, c , how much to invest in capital, v , spend in disease prevention activities, h , and in pollution abatement, q . Thus, the budget constraint is:

$$C + v + h + q = Rk + Wl. \quad (5)$$

The evolution of the capital stock is given by:

$$\dot{k} = v + \delta k - k(b - d) \quad (6)$$

$$(7)$$

where $\delta_k \in (0, 1]$ is the depreciation rates of physical capital.

The equation (6) can be written as

$$\dot{k} = Rk + Wl - c - h - q - \delta k - k(b - d)$$

Given Π, \bar{Y}, R, W the representative household maximizes the intertemporal utility. Therefore, each household solves the following maximization problem:

$$\begin{aligned} \max_{\{c, h, q\}} \int_0^{\infty} e^{-(\rho - b + d)t} u(C) N_0 dt & \quad (8) \\ \dot{k} = Rk + W(1 - i) - c - h - q - \delta k - (b - d)k & \\ \dot{i} = \alpha(h, P(\bar{Y}, q))\Pi(1 - i) - \gamma i - bi & \end{aligned}$$

The control variables are c, h, q , the state variables are k, i .

3 Decentralized economy

We define the competitive equilibrium in the decentralized economy. It is a standard definition with the condition that the perceived proportion of infected by households, Π is equal to the actual proportion of infected in the population, i , and $\bar{Y} = Y$.

Definition 1. A competitive equilibrium is a feasible allocation $\{c, k, h, q, i\}$ and given $\{\Pi, \bar{Y}, R, W\}$

1. Households solve the maximization problem (8).
2. Firms maximize profits, given by equation (1).
3. The capital market, labor market and goods market clear.
4. Pollution satisfies (2) with $\bar{Y} = f(k, 1 - i)$.
5. Since each household is representative of the population, in equilibrium, $\Pi = i$.

We now characterize the dynamical system that defines the dynamic general equilibrium of the economy.

The current value Lagrangian for the optimization household problem is:

$$\begin{aligned} \mathcal{L} = u(c) + \lambda_1 [Rk + W(1 - i) - c - h - q - \delta k - (b - d)k] & + \lambda_2 [\alpha(h, P(\bar{Y}, q))\Pi(1 - i) - \gamma i - bi] \\ + \mu_1 i + \mu_2 h & \end{aligned}$$

Incorporating the equilibrium conditions

$$\begin{aligned}
R &= f_1(k, 1-i) \\
W &= f_2(k, 1-i) \\
P &= P(\bar{Y}, q) = P(f(k, 1-i), q) \\
\Pi &= i
\end{aligned}$$

we can write the conditions for an equilibrium in the economy.

The equilibrium in the decentralized economy is determined by the following equations (the first order conditions and the transversality conditions to the household problem incorporating the equilibrium conditions):

$$\dot{k} = f(k, 1-i) - c - h - q - \delta k - (b-d)k \quad (9)$$

$$\dot{i} = \alpha(h, P(f(k, 1-i), q))i(1-i) - \gamma i - bi \quad (10)$$

$$u'(c) = \lambda_1 \quad (11)$$

$$\lambda_1 = \lambda_2 \alpha_1(h, P(f(k, 1-i), q))i(1-i) \quad (12)$$

$$\lambda_1 = \lambda_2 \alpha_2(h, P(f(k, 1-i), q))P_2(f(k, 1-i), q)i(1-i) \quad (13)$$

$$\dot{\lambda}_1 = \lambda_1[\rho + \delta + b - d - f_1(k, 1-i)] \quad (14)$$

$$\dot{\lambda}_2 = \rho \lambda_2 + \lambda_1 f_2(k, 1-i) + \lambda_2[\alpha(h, P(f(k, 1-i), q))i + \gamma + b] \quad (15)$$

$$\mu_1 \geq 0, i \geq 0, \mu_1 i = 0,$$

$$\mu_2 \geq 0, h \geq 0, \mu_2 h = 0,$$

$$\lim_{t \rightarrow \infty} e^{-\theta t} \lambda_1 k = \lim_{t \rightarrow \infty} e^{-\theta t} \lambda_2 i = 0.$$

As the economy is a neo-classical economy with a bounded capital stock and i lies in a bounded interval, the transversality conditions are satisfied, and in the subsequent discussion we suppress these.

Note that the optimization problem has non-convex constraints on the state variable i and the usual Arrow and Mangasarian condition do not apply. Goenka, Liu and Nguyen (2014) have however, shown existence and sufficiency for the *SIS* model and we concentrate on studying the first order conditions which will characterize the dynamical system of the economy.⁹

3.1 Steady states

We know characterize the steady state competitive equilibria for the decentralized economy. As in the pure epidemiology model there are two equilibria which depend on the basic reproduction number, i.e. determined by the contact rate. However, unlike the pure epidemiology model it is endogenous depending on the abatement and disease preventive activities.

Proposition 1. *There always exists a unique disease free steady state with $i^* = 0$, $h^* = 0$ and*

⁹The sufficiency conditions have been extended to account for mortality in Goenka, Liu and Nguyen (2020a) and for the *SIR* model with mortality in Goenka, Liu and Nguyen (2020b).

$q^* = 0$. The economic variables k^* and c^* are determined by

$$\begin{aligned} f_1(k, 1) &= \rho + \delta + b - d \\ f(k, 1) &= c + \delta k + (b - d)k. \end{aligned}$$

In the disease free steady state pollution plays no role in the determination of equilibrium, and the economy is in the neoclassical steady state. In the disease free steady state, there is no abatement as the only effect of pollution in this model is on disease transmission. As there is no disease prevalence, there is also no health preventive expenditure. Let us call this $\bar{\alpha}$. If $\bar{\alpha} < b + \gamma$ the disease is eradicated and the steady state is locally stable, and if $\bar{\alpha} > b + \gamma$ the disease free steady state is unstable.

The disease endemic steady state is a solution to the system of equations (9 - 15). It will exist if when the contact rate is high enough so that $s^* < 1$, i.e. $\alpha(h^*, P^*) > b + \gamma$ or the equilibrium endogenous contact rate $R_0^* > 1$.

Proposition 2. *There exists a disease endemic steady state where i^*, k^*, h^*, q^* and c^* are determined by:*

$$i = 1 - \frac{b + \gamma}{\alpha(h, P(f(k, 1 - i), q))} \quad (16)$$

$$\alpha_1(h, P(f(k, 1 - i), q)) = \alpha_2(h, P(f(k, 1 - i), q))P_2(f(k, 1 - i), q) \quad (17)$$

$$f_1(k, 1 - i) = \rho + \delta + b - d \quad (18)$$

$$-f_2(k, 1 - i)\alpha_1(h, P(f(k, 1 - i), q))i(1 - i) = \rho + \alpha(h, P(f(k, 1 - i), q)) \quad (19)$$

$$f(k, 1 - i) = c + h + q + \delta k + (b - d)k \quad (20)$$

Proof. From equation (10) $\dot{i} = 0$, we have either $i^* = 0$ or $i^* = 1 - \frac{b + \gamma}{\alpha(h, P)}$. Note that this implies $\alpha(h, P)i = \alpha(h, P) - (b + \gamma)$.

From equation (12) and (13), we have

$$\alpha_1(h, P) = \alpha_2(h, P)P_2(y, q).$$

In equilibrium the marginal benefit of health expenditure h should be the same as the marginal benefit of pollution abatement q as both health expenditure and pollution abatement only affect the economy through their impact on the contact rate α .

From $\dot{\lambda}_1 = 0$, we have

$$f_1(k, 1 - i) = \rho + \delta + b - d,$$

which is the standard marginal benefit of physical capital equals to its marginal cost.

From $\dot{\lambda}_2 = 0$, we have

$$\lambda_1 f_2(k, 1 - i) = -\lambda_2 \rho - \lambda_2 [\alpha(h, P)i + \gamma + b],$$

which implies the marginal benefit of disease controlling (one unit of reduction in i - the proportion of the infected) equals to its marginal cost. The L.H.S of the above equation is the marginal benefit, as with one unit of reduction in the proportion of the infected i , the labor force increases by one unit and generates the marginal product $f_2(k, 1 - i)$. The R.H.S is the marginal cost of disease controlling. Note that the shadow value of the infected - λ_2 is negative. When there is one unit of reduction in the proportion of the infected, the proportion of the susceptible increases by one unit. As there are more susceptible around, there are more infections.

Then, we substitute into equation (12), and we have

$$-f_2(k, 1 - i)\alpha_1(h, P)i(1 - i) = \rho + \alpha(h, P).$$

□

Note, that in equilibrium from (16) we have $R_0^* > 1$. Thus, in the competitive equilibrium the disease is endemic. Whether this is the case depends on not only the disease characteristics, i.e. the function α , but also the deeper economic parameters which determine whether it is in the interest of the households to take sufficient actions to bring the contact rate below the threshold. There is a tension on what will happen once the contact rate drops below 1, whether the economy will converge to a disease free steady state. It depends on whether the disease free steady state is stable or not and depends on the bifurcation when the contact rate is 1. In this paper, we focus on the difference between the private and public actions and do not investigate this issue in further depth. The paper Goenka and Liu (2013) and Goenka, Liu and Nguyen (2014) study these issues in further depth in a model without pollution in discrete time and continuous time respectively.

4 The Centralised Economy

In the centralized economy, the social planner takes into account both pollution externalities and disease externalities, i.e. the true law of motion for the disease and affect of output on pollution are taken into account. Therefore, the social planner solves the following maximization problem:

$$\begin{aligned} \max_{\{c, h, q\}} \max_{\{c, h, q\}} \int_0^{\infty} e^{-(\rho - b + d)t} u(C) N_0 dt \\ \dot{k} = f(k, 1 - i) - c - h - q - \delta k - (b - d)k \\ \dot{i} = \alpha(h, P(f(k, 1 - i), q))i(1 - i) - \gamma i - bi \end{aligned}$$

The equilibrium in the centralized economy is determined by the following equations (suppressing the transversality conditions):

$$\dot{k} = f(k, 1-i) - c - h - q - \delta k - (b-d)k \quad (21)$$

$$\dot{i} = \alpha(h, P(f(k, 1-i), q))i(1-i) - \gamma i - bi \quad (22)$$

$$u'(c) = \lambda_1 \quad (23)$$

$$\lambda_1 = \lambda_2 \alpha_1(h, P(f(k, 1-i), q))i(1-i) \quad (24)$$

$$\lambda_1 = \lambda_2 \alpha_2(h, P(f(k, 1-i), q))P_2(f(k, 1-i), q)i(1-i) \quad (25)$$

$$\dot{\lambda}_1 = \lambda_1[\rho + \delta + b - d - f_1(k, 1-i)] + [-\lambda_2 \alpha_2(h, P)P_1(y, q)f_1(k, 1-i)(1-i)i] \quad (26)$$

$$\begin{aligned} \dot{\lambda}_2 = & \rho \lambda_2 + \lambda_1 f_2(k, 1-i) + \lambda_2 [\alpha(h, P(f(k, 1-i), q))i + \gamma + b] + \\ & + [\lambda_2 \alpha_2(h, P)P_1(y, q)f_2(k, 1-i)(1-i)i] + [-\lambda_2 \alpha(h, P)(1-i)] \end{aligned} \quad (27)$$

Note that there is an additional term in (26) as compared to (14) in the decentralized economy which arises from taking the effect of output on pollution and hence, disease transmission; and two additional terms in (27) as compared to (15) that arise from taking into account the effects of infections on labor supply, and hence output, pollution and disease transmission and the second which internalizes the disease externality.

4.1 Steady states

There always exists a unique disease free steady state with $i^* = 0$, $h^* = 0$ and $q^* = 0$. This is exactly the same as the one in decentralized economy as it is the neoclassical steady state with $h^* = q^* = 0$.

As in the decentralized economy there is also a disease endemic steady state.

Proposition 3. *There exists a disease endemic steady state where i^*, k^*, h^*, q^* and c^* are determined by:*

$$i = 1 - \frac{b + \gamma}{\alpha(h, P(f(k, 1-i), q))} \quad (28)$$

$$\alpha_1(h, P(f(k, 1-i), q)) = \alpha_2(h, P(f(k, 1-i), q))P_2(f(k, 1-i), q) \quad (29)$$

$$f_1(k, 1-i) = \rho + \delta + b - d + \left[-\frac{P_1(f(k, 1-i), q)f_1(k, 1-i)}{P_2(f(k, 1-i), q)} \right] \quad (30)$$

$$-f_2(k, 1-i)\alpha_1(h, P(f(k, 1-i), q))i(1-i) + [\alpha(h, P(f(k, 1-i), q))(1-i)] = \quad (31)$$

$$\begin{aligned} & = \rho + \alpha(h, P(f(k, 1-i), q)) + [\alpha_2(h, P(f(k, 1-i), q))P_1(f(k, 1-i), q)f_2(k, 1-i)(1-i)i] \\ f(k, 1-i) & = c + h + q + \delta k + (b-d)k \end{aligned} \quad (32)$$

Proof. The proof is the similar as the one in decentralized economy and thus omitted here. \square

Compared with the decentralized economy, there are two differences. One difference is equation (30), which says the marginal social benefit of physical capital should equal to the marginal social cost. In the centralized economy, the social planner takes into account the negative externality of pollution on disease transmission, and thus there is an additional cost to physical capital

investment. When physical capital increases, output increases, which leads to more pollution and higher contact rate for disease transmission. This is captured by the term $[-\frac{P_1(f(k,1-i),q)f_1(k,1-i)}{P_2(f(k,1-i),q)}]$. The other difference is equation (31), which says the marginal social benefit of disease controlling equals to the marginal social cost. In the centralized economy, there are two additional terms in equation (31). When the social planner takes into account the negative externality of pollution there is additional cost of an increase in labor input or reduction in the proportion of the infected. When the proportion of the infected decreases, the proportion of the susceptible or the labor force increases, which leads to more output and pollution, and then higher contact rate for disease transmission. This is captured by the term $[\alpha_2(h, P(f(k, 1 - i), q))P_1(f(k, 1 - i), q)f_2(k, 1 - i)(1 - i)i]$. Moreover, the social planner takes into account the disease externality. That is, there is additional benefit from controlling disease, as the social planner further takes into account the behavior is going to affect the proportion of the infected. This is captured by the term $\alpha(h, P(f(k, 1 - i), q))(1 - i)$. Thus, compared with the decentralized economy, the centralized economy takes into account both negative pollution externalities and positive disease controlling externality. To compare the abatement and health expenditures in the centralized and decentralized economies we have to use numerical methods as the system of non-linear equations are too complex to compare directly.

5 Model Simulations

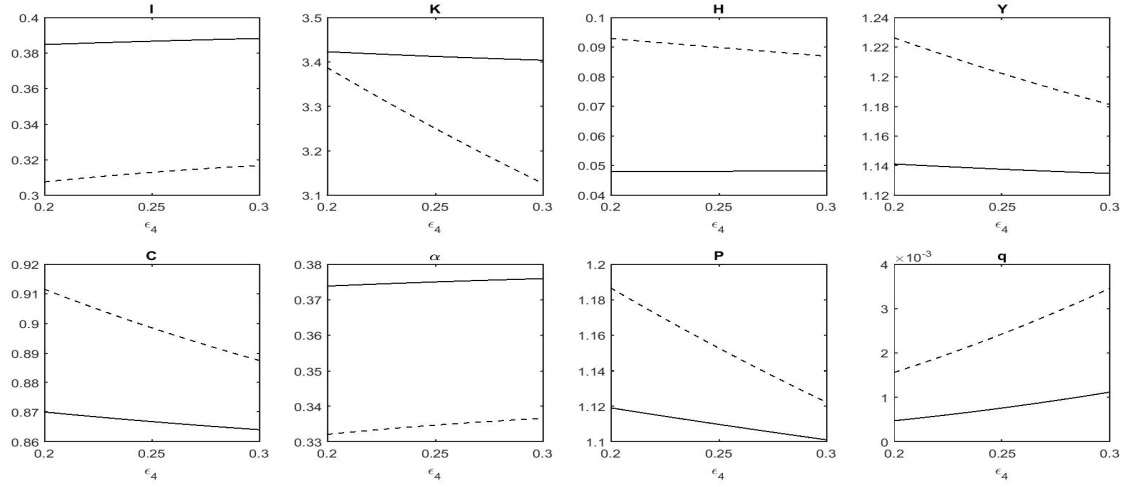
As the model is too complex for closed form solutions or doing comparative statics analytically, in this section, we calibrate the model and do comparative statics numerically to understand the interaction of the different components. We focus on examining the parameters where pollution can affect the disease transmission. That is, we vary the elasticity of pollution on the disease contact rate, the elasticity of pollution abatement, and the productivity in the economy. The last is important as it tries to ascertain whether more productive economies will have different responses and outcomes. The analysis here focuses on the equilibrium steady states before and after the change as we want to capture the medium to longer term effects when investment and returns to labor and capital have adjusted.

The following functional forms and parameters are chosen in line with the literature. The production function is assumed to be Cobb Douglas: $y = f(k, 1 - i) = Ak^\beta(1 - i)^{1-\beta}$ with $A = 1$ and $\beta = 0.36$. Physical capital depreciates at the rate $\delta = 0.05$ and discount rate $\rho = 0.055$. The utility function is of the CES form $U(c) = \frac{c^{1-\sigma}}{1-\sigma}$ and we choose $\sigma = 1$, that is, the utility function is log utility. We set the birth rate $b = 3\%$ and death rate $d = 1.5\%$. The recovery rate is $\gamma = 0.2$.

We do not have enough empirical evidence in suggesting for functional form for contact rate function $\alpha(h, P)$ and pollution function $P(y, q)$. Therefore, the contact rate function is chosen in line with the assumption on $\alpha(h, P)$ and large enough to generate an endemic steady state in the simulation. We assume contact rate function is a power function: $\alpha(h) = \varepsilon_0(h + \varepsilon_1)^{\varepsilon_2}(P + \varepsilon_3)^{\varepsilon_4}$ with $\varepsilon_0 = 0.2, \varepsilon_1 = 0, \varepsilon_2 = -0.2, \varepsilon_3 = 0$ and $\varepsilon_4 = 0.2$ in the baseline specification. The pollution function is also chosen in line with the assumptions. We assume $P(y, q) = \phi_0 f(k, 1 - i) - \phi_1(q + \phi_2)^{\phi_3}$ with $\phi_0 = 1, \phi_1 = 1, \phi_2 = 0$ and $\phi_3 = 0.5$ in the baseline specification.

Figure 1, 2 and 3 are the simulation results for varying the elasticity of pollution on contact rate

Figure 1. The simulation results - varying the elasticity of pollution on contact rate

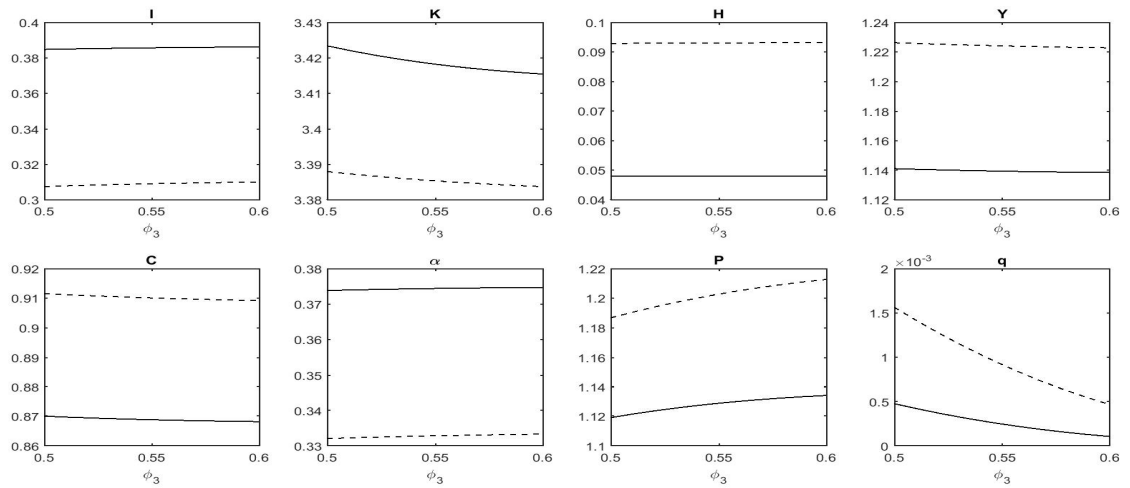


Note: The figure shows changes in the disease endemic steady state when we change the elasticity of pollution on contact rate. There are total 8 panels - I (the proportion of the infected), K (physical capital), H (health expenditure), Y (output), C (consumption), α (contact rate), P (pollution), q (abatement). The solid line is the decentralized economy (without disease externality and without pollution externality). The dashed line is the centralized economy (with disease externality and pollution externality).

function (ϵ_4), the elasticity of abatement on pollution (ϕ_3) and the productivity (A), respectively. The parameters are chosen such that disease endemic steady states exist for both the decentralized and centralized economy. So we can focus on how changes in those parameters change economic variables. The solid line is the decentralized economy (where the disease and pollution externalities have not been internalized). The dashed line is the centralized economy (where the disease externality and pollution externalities have been internalized). Across all the three figures, if we compare the decentralized economy with the centralized economy, the pictures are very similar. The social planner when taking the externalities into account is able to achieve lower contact rates in equilibrium with higher health expenditures. As a result, the labor force is larger and output higher. The capital stock is lower in the planning solution. While the abatement is higher in the planning solution, paradoxically pollution is also higher. This is being driven by the fact that in the planning solution, the output is higher and even though there is greater abatement, the net effect of the higher output dominates and pollution is higher. Thus, even when the planner takes into account the deleterious effect of pollution on health outcomes, the efficient outcome has higher pollution. If an economy has higher TFP or not does not change the qualitative effects, except that a more productive economy will have better economic outcomes as well as higher pollution. The health outcomes and the abatement is not affected by a change in TFP.

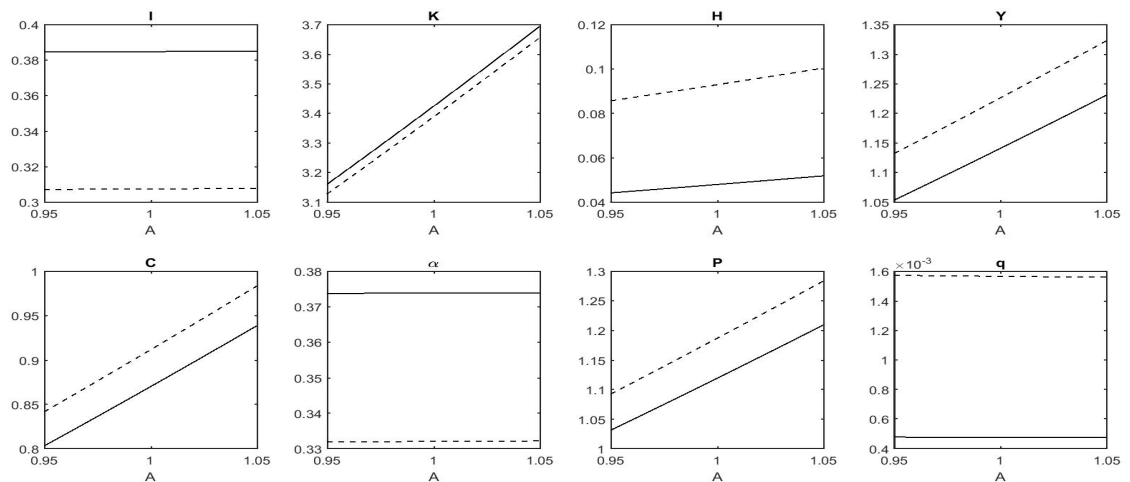
In the pure epidemiology model, disease free steady state always exists, and disease endemic steady state only exists when contact rate is high enough, that is, $\alpha > b + \gamma$ or $R_0 = \frac{\alpha}{b+\gamma} > 1$. When there is only one disease free steady state, it is stable. When both disease free and disease endemic steady states co-exist, disease free steady state is unstable and disease endemic steady state is stable. The same applies to the economic epidemiological models here for both decentralized economy and centralized economy. The only difference is that the contact rate α or R_0

Figure 2. The simulation results - varying the elasticity of abatement on pollution



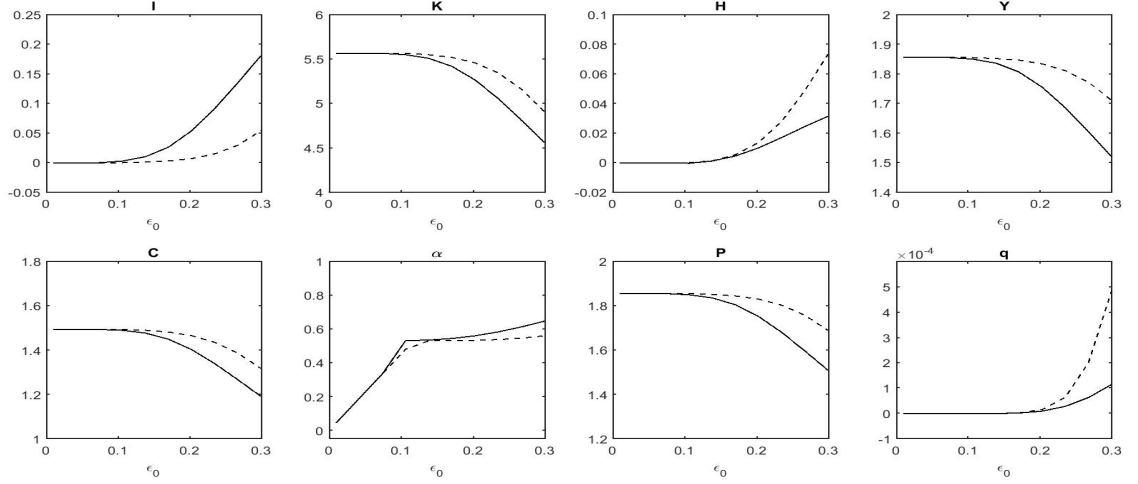
Note: The figure shows changes in the disease endemic steady state when we change the elasticity of abatement on pollution. There are total 8 panels - I (the proportion of the infected), K (physical capital), H (health expenditure), Y (output), C (consumption), α (contact rate), P (pollution), q (abatement). The solid line is the decentralized economy (without disease externality and without pollution externality). The dashed line is the centralized economy (with disease externality and pollution externality).

Figure 3. The simulation results - varying productivity



Note: The figure shows changes in the disease endemic steady state when we change productivity in the production function. There are total 8 panels - I (the proportion of the infected), K (physical capital), H (health expenditure), Y (output), C (consumption), α (contact rate), P (pollution), q (abatement). The solid line is the decentralized economy (without disease externality and without pollution externality). The dashed line is the centralized economy (with disease externality and pollution externality).

Figure 4. The simulation results - disease free and disease endemic steady states



Note: The figure shows changes in the disease free and disease endemic steady states when we change the contact rate parameter ϵ_0 . There are total 8 panels - I (the proportion of the infected), K (physical capital), H (health expenditure), Y (output), C (consumption), α (contact rate), P (pollution), q (abatement). The solid line is the decentralized economy (without disease externality and without pollution externality). The dashed line is the centralized economy (with disease externality and pollution externality).

is endogenous, and thus the cut-off points of the contact rate for the existence of disease endemic steady state are different in the decentralized and centralized economies. Since the social planner takes the externalities into account, and is able to achieve lower contact rates in equilibrium, with the same parameter values it is more likely to have disease eradicated in the centralized economy. That is, the cut-off point of the contact rate parameter for the existence of disease endemic steady state in the centralized economy is higher than the one in the decentralized economy. To show this, we have chosen the recovery rate $\gamma = 0.5$ such that when we vary the contact rate parameter ϵ_0 from 0.01 to 0.3, there are changes in steady states in both decentralized and centralized economy. The simulation results is shown in Figure 4. We can see that the cut-off points to have disease endemic steady state is $\epsilon_0 = 0.08$ for the decentralized economy and $\epsilon_0 = 0.12$ for the centralized economy. When the contact rate parameter ϵ_0 is small enough (below 0.08) such that $\alpha < b + \gamma$, we can see there is disease free steady state in both decentralized and centralized economy, The proportion of the infected is zero and all variables are the same in both economies. When ϵ_0 is between 0.08 and 0.12, that is, the contact rate rises, in the centralized economy, as $\alpha < b + \gamma$ there is only one disease free steady state, and disease endemic steady state does not exist. In contrast, in the decentralized economy, disease endemic steady state exists.¹⁰ For the disease endemic steady state in the decentralized economy, the fraction of the infected is positive and physical capital, output and consumption are lower, compared with the centralized economy. When ϵ_0 is above 0.12, for both decentralized and centralized economies, as the contact rate is high enough, disease endemic steady state exists.¹¹

¹⁰Note that there is also disease free steady state (not shown in the figure).

¹¹Note that there is also disease free steady state (not shown in the figure).

6 Public Policy

We study the dynamic Pigovian taxes that will decentralize the efficient competitive equilibrium. Even though there are two externalities: the disease externality and the pollution externality we need four taxes and subsidies to decentralize the efficient outcome. The reason is that these the externalities affect the economy in a complex way: pollution not only affects the contact rate, as the private abatement is not at the efficient level, but also through the change in labor supply through disease incidence the marginal product of labor. Thus, there are two wedges introduced by the pollution externality. The disease externality affects the private health expenditures. A fourth tax is needed to meet the balanced budget or self-financing nature of the tax-subsidy policy.

Thus, we introduce tax policies - capital income tax τ_k and labor income tax τ_l , and subsidies - health expenditure subsidy τ_h and pollution abatement subsidy τ_q . The decentralized economy with public polices is as the follows:

$$\begin{aligned} & \max_{\{c,h,q\}} \int_0^{\infty} e^{-\rho t} u(c) dt \\ & \dot{k} = (1 - \tau_k)Rk + (1 - \tau_l)W(1 - i) - c - (1 - \tau_h)h - (1 - \tau_q)q - \delta k - (b - d)k \\ & \dot{i} = \alpha(h, P(\bar{Y}, q))\Pi(1 - i) - \gamma i - bi \end{aligned}$$

Then, we incorporate equilibrium conditions, which is the same as those in decentralized economy. Moreover, there is a balance budget constraint:

$$\tau_k Rk + \tau_l W(1 - i) = \tau_h h + \tau_q q. \quad (33)$$

The equilibrium in the decentralized economy with public policies is determined by the following equations:

$$\begin{aligned} \dot{k} &= f(k, 1 - i) - c - h - q - \delta k - (b - d)k \\ \dot{i} &= \alpha(h, P(f(k, 1 - i), q))i(1 - i) - \gamma i - bi \\ u'(c) &= \lambda_1 \\ \lambda_1 &= \lambda_2 \alpha_1(h, P(f(k, 1 - i), q))i(1 - i) \frac{1}{1 - \tau_h} \\ \lambda_1 &= \lambda_2 \alpha_2(h, P(f(k, 1 - i), q))P_2(f(k, 1 - i), q)i(1 - i) \frac{1}{1 - \tau_q} \\ \dot{\lambda}_1 &= \lambda_1 [\rho + \delta + b - d - (1 - \tau_k)f_1(k, 1 - i)] \\ \dot{\lambda}_2 &= \rho \lambda_2 + \lambda_1 (1 - \tau_l)f_2(k, 1 - i) + \lambda_2 [\alpha(h, P(f(k, 1 - i), q))i + \gamma + b] \end{aligned}$$

Proposition 4. *In the decentralized economy with public policies, there exists a disease endemic*

steady state where i^*, k^*, h^*, q^* and c^* are determined by:

$$i = 1 - \frac{b + \gamma}{\alpha(h, P(f(k, 1 - i), q))} \quad (34)$$

$$\alpha_1(h, P(f(k, 1 - i), q)) \frac{1}{1 - \tau_h} = \alpha_2(h, P(f(k, 1 - i), q)) P_2(f(k, 1 - i), q) \frac{1}{1 - \tau_q} \quad (35)$$

$$(1 - \tau_k) f_1(k, 1 - i) = \rho + \delta + b - d \quad (36)$$

$$-\frac{1 - \tau_l}{1 - \tau_h} f_2(k, 1 - i) \alpha_1(h, P(f(k, 1 - i), q)) i (1 - i) = \rho + \alpha(h, P(f(k, 1 - i), q)) \quad (37)$$

$$f(k, 1 - i) = c + h + q + \delta k + (b - d)k \quad (38)$$

Next, we are going to show how we design tax and subsidy policies, such that the decentralized economy can replicate the allocations from the centralized economy. If we compare equation (29) with (35), it is easy to see that the subsidy for health expenditure τ_h should be equal to the subsidy for pollution abatement τ_q :

$$\tau_h = \tau_q. \quad (39)$$

Note that this is independent of elasticities of pollution and health expenditures on the contact rate.

Then, by comparing equation (30) with (36), we can derive capital income tax:

$$\tau_k = -\frac{P_1(f(k, 1 - i), q)}{P_2(f(k, 1 - i), q)}. \quad (40)$$

Since $P_1 > 0$ and $P_2 < 0$, the capital income tax $\tau_k > 0$. If we compare equation (31) with (37), we get

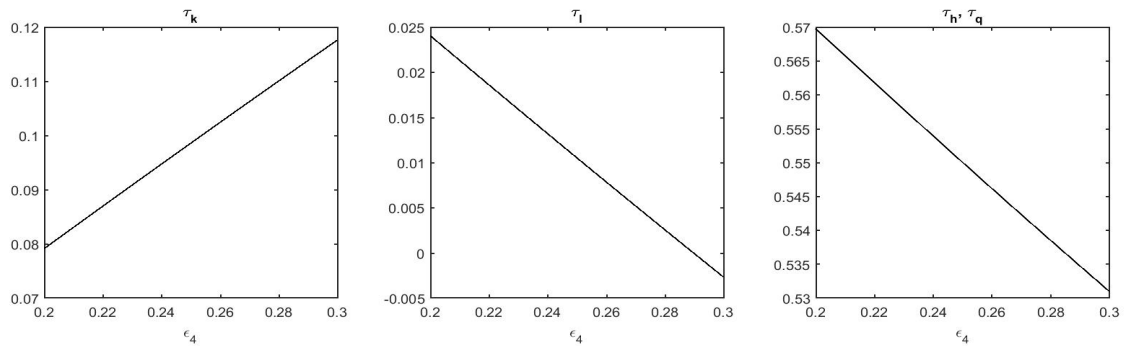
$$\frac{\tau_h - \tau_l}{1 - \tau_h} = \frac{\alpha_2(h, P(f(k, 1 - i), q)) P_1(f(k, 1 - i), q) f_2(k, 1 - i) i - \alpha(h, P(f(k, 1 - i), q))}{f_2(k, 1 - i) \alpha_1(h, P(f(k, 1 - i), q)) i}. \quad (41)$$

Thus, we have the following proposition.

Proposition 5. *The tax and subsidy policies $(\tau_k, \tau_l, \tau_h, \tau_q)$ are determined by equation (33), (39), (40) and (41), where the economic variables i, k, h, q and c are the equilibrium allocations from the centralized economy.*

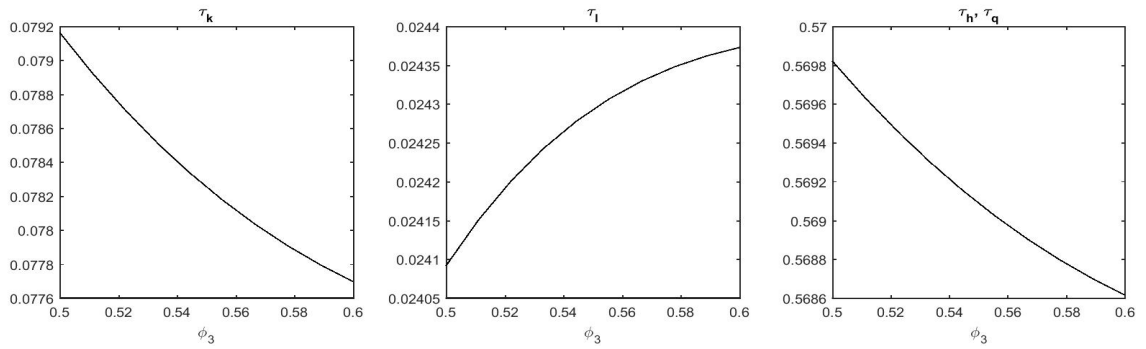
To understand the taxes and subsidies we do a similar exercise as in the previous section of varying the elasticity of pollution, the elasticity of abatement and the TFP. Interestingly, for each of the exercises, the subsidy on health and abatement decrease and the primary mechanisms are the direct taxes on labor and capital income. The effect of increasing elasticity of pollution and contact rate and on abatement rate is different. As elasticity of pollution on contact rate increases, the capital income is taxed at a higher rate and labor income at a lower rate. However, when elasticity of abatement increases, i.e. abatement is more effective, capital income is taxed at a lower rate but to maintain budget balance, labor income taxes are increased. As TFP increases,

Figure 5. Tax and subsidy policies - varying the elasticity of pollution on contact rate



Note: The figure shows changes in tax and subsidy policies when we change the elasticity of pollution on contact rate.

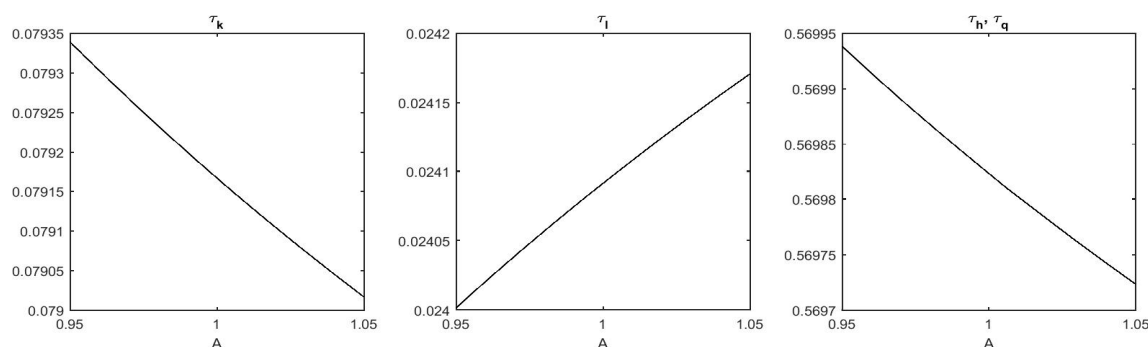
Figure 6. Tax and subsidy policies - varying the elasticity of abatement on pollution



Note: The figure shows changes in tax and subsidy policies when we change the elasticity of abatement on pollution

in a similar way, capital income is taxed is taxed less and to compensate labor income taxes are increased. Thus, we should expect a different mix of labor-capital income taxes for economies depending on their TFPs and thus, growth rates.

Figure 7. Tax and subsidy policies - varying productivity



Note: The figure shows changes in tax and subsidy policies when we change productivity in the production function.

7 Conclusion

This paper studies the interaction of pollution with transmission of diseases. In a decentralized economy there is the pollution externality and a disease transmission externality. As one would expect, the level of abatement of pollution and preventive health expenditures are lower than the centralized situation where the externalities are internalized. While the centralized outcomes can be decentralized through dynamic Pigovian taxes, with higher productivity the level of pollution in fact goes up in the efficient outcome. The control of the disease interacts with higher productivity, and even though more is spent on abatement, the net effect is that pollution goes up. This is consistent with the recent evidence where during lockdown as mobility and activity declined, so did pollution levels. However, this was temporary and since then pollution levels have gone up (Kumari and Toshniwal (2020)). Thus, expectation of a Green Recovery may be illusory.

References

- [1] Bosi, S. and Desmarchelier, D. (2018) Pollution and infectious diseases, *International Journal of Economic Theory*, 18, 351-372.
- [2] Bosi, S. and Desmarchelier, D. (2019) Pollution effects on disease transmission and economic stability, *International Journal of Economic Theory*.
- [3] Cadotte, M.C. (2020) Early evidence that government Covid-19 policies reduce air pollution, University of Toronto.
- [4] Ciencewicki, J. and Jaspers, I. (2007) Air pollution and respiratory viral infection, *Inhalation Toxicology*, 19:14, 1135-1146.
- [5] Conticini, E., Frediani, B., Faro, D. (2020) Can atmospheric pollution be considered a co-factor in extremely high level of SARS-Cov-2 lethality in Northern Italy? *Environmental Pollution*, 261, 114465.

- [6] Eichenbaum, M.S., Rebelo, S., and Trabandt, M. (2020) The macroeconomics of epidemics, *NBER Working Paper* No. 26882.
- [7] Ferguson, N., et al. (2020) Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand, Imperial College Covid-19 Response Team, DOI: <https://doi.org/10/25561/77482>.
- [8] Geoffard, P-Y. and Philipson, T. (1996) “Rational Epidemics and Their Public Control,” *International Economic Review*, 37(3): 603-24.
- [9] Gersovitz, M. and Hammer, J.S., (2004): “The economical control of infectious diseases”, *Economic Journal*, 1-27.
- [10] Goenka, A., Jafarey, S. and Pouliot, W. (2020) Pollution, mortality and time consistent abatement taxes, *Journal Mathematical Economics*, 88, 1-15.
- [11] Kumari, P. and Toshniwal, D. (2020) Impact of lockdown on air quality over major cities across the globe during COVID-19 pandemic, *Urban Climate* 34, December, 100719
- [12] Goenka, A., Liu, L. (2013) Infectious diseases and endogenous fluctuations, *Economic Theory*, 125-149.
- [13] Goenka, A., Liu, L. (2020) Human capital, infectious diseases and economic growth, *Economic Theory*, 70, 1-47.
- [14] Goenka, A., Liu, L., Nguyen, M.H. (2014) Infectious diseases and economic growth, *Journal of Mathematical Economics* 50, 34-53.
- [15] Goenka, A., Liu, L., Nguyen, M.H. (2020a) Modeling optimal quarantines under infectious disease related mortality, TSE Working paper 20-1136.
- [16] Goenka, A., Liu, L., Nguyen, M.H. (2020b) *SIR* Economic Epidemiological Models with Disease Induced Mortality, TSE Working paper 20-1150.
- [17] Harvey, F. (2020) Revealed Covid recovery plans threaten global climate hopes, *The Guardian*, 9/11/2020. <https://www.theguardian.com/environment/2020/nov/09/revealed-covid-recovery-plans-threaten-global-climate-hopes>.
- [18] Horne, B.D., et al. (2018) Short-term elevation of fine particulate matter air pollution and acute lower respiratory infection, *American Journal of Respiratory Critical Care and Medicine*, 198, 759-766.
- [19] Huang, L., et al. (2016) Acute effects of air pollution on influenza-like illness in Nanjing, China: A population-based study, *Chemosphere* 147, 180-187.
- [20] Ibarra, F.J., et al. (2020) Rapid decay of Anti-SARS-Cov-2 antibodies in persons with mild Covid-19, *New England Journal of Medicine*, letter, DOI: 10.1056/NEJMc2025179.
- [21] Isho, B., et al. (2020) Persistence of serum and saliva antibody responses to SARS-Cov-2 spike antigens in COVID-19 patients, *Science Immunology*, 5(52), October.

- [22] Kampa, M. and Castanas, E. (2008) Human health effects of air pollution, *Environmental Pollution*, 151, 362-367.
- [23] Kelly, F.J. and Fussell, J.C. (2011) Air pollution and airway disease, *Clinical and Experimental Allergy* 41, 1059-1071.
- [24] Kissler, S.M., Tedijanto, C., Goldstein, E., Grad, Y.H., and Lipsitch, M. (2020) Projecting the transmission dynamics of SARS-CoV-2 through the postpandemic period, *Science*, 368, 860-868.
- [25] Liang, Y., et al. (2014) $PM_{2.5}$ in Beijing - temporal pattern and its association with influenza, *Environmental Health* 13: 102.
- [26] Long, Q., Tang, X., Shi, Q. et al. (2020) Clinical and immunological assessment of asymptomatic SARS-CoV-2 infections, *Nature Medicine* 26, 1200-1204. <https://doi.org/10.1038/s41>.
- [27] Martelletti, L. and Martelletti, P. (2020) Air pollution and the novel Covid-19 disease: A putative disease risk factor, *SN Comprehensive Clinical Medicine*, 2: 282-387.
- [28] Mehta, S., Shin, H., Burnett, R., North, T., and Cohen, A.J. (2013) Ambient particulate air pollution and acute lower respiratory infections: a systematic review and implications for estimating global burden of disease, *Air Quality, Atmosphere and Health* 6, 69-83.
- [29] Myllyvirta, L. (2020) China's air pollution overshoots pre-crisis levels for the first time, CREA, May. <https://energyandcleanair.org/wp/wp-content/uploads/2020/05/China-air-pollution-rebound-final.pdf>
- [30] Qin, N., et al. (2020) Longitudinal survey of microbiome associated with particulate matter in a megacity, *Genome Biology*, 21, 1-11.
- [31] Ripperger, T.J., et al. (2020) Orthogonal SARS-CoV-2 serological assays enable surveillance of low-prevalence communities and reveal durable humoral immunity, *Immunity*, November.
- [32] Setti, L., et al. (2020) Airborne transmission route of Covid-19: Why 2 meters/6 feet of inter-personal distance may not be enough, *Int. J. Environ. Res. Public Health*, 17(8) 2392.
- [33] Sekine, et al. (2020) Robust T cell immunity in convalescent individuals with asymptomatic or mild COVID-19, *bioRxiv* 2020.06.29.174888; doi: <https://doi.org/10.1101/2020.06.29.174888>
- [34] Tasci, S.S., Kavalci, C. and Kayipmaz, A.E. (2018) Relationship of meteorological and air pollution parameters with Pneumonia in elderly patients, *Emergency Medicine International*, Article ID4183203.
- [35] Varotsos, C., Ondov, J., Efstathiou, M. (2005) Scaling properties of air pollution in Athens, Greece and Baltimore, Maryland, *Atmospheric Environment* 39: 4041-4047

- [36] Wajnberg, A., et al. (2020) Humoral immune response and prolonged PCR positivity in a cohort of 1343 SARS-CoV 2 patients in the New York City region, *medRxiv* 2020.04.30.20085613; doi: <https://doi.org/10.1101/2020.04.30.20085613>
- [37] Ward, H., et al. (2020) Declining prevalence of antibody positivity to SARS-CoV-2: a community study of 365,000 adults, October. <https://www.imperial.ac.uk/media/imperial-college/institute-of-global-health-innovation/MEDRXIV-2020-219725v1-Elliott.pdf>
- [38] Windsor, H.L. and Tuomi, R. (2001) Scaling and persistence of UK pollution, *Atmospheric Environment* 35: 4545-4556.
- [39] World Bank (2018) “Data on real interest rates,” <https://data.worldbank.org/indicator/fr.inr.rinr> (Accessed 8/06/2020).
- [40] Wu, X., et al. (2020) Exposure to air pollution and Covid-19 mortality in the US: A nationwide cross-sectional study, MedRxiv preprint doi:<https://doi.org/10.1101/2020.04.05.20054502>.
- [41] Yongjian, Z., et al. (2020) Association between short-term exposure to air pollution and Covid-19 infection: Evidence from China, *Science of the Total Environment* 727, 138794.
- [42] Zeka, A., Zanobetti, A., and Schwartz, J. (2005) Short term effects of particulate matter on cause specific mortality: Effects of lags and modification by city characteristics, *Occupational and Environmental Medicine* 62(10): 718-725.