COVID-19 and a Green Recovery?*

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Abstract

Preliminary evidence indicates that pollution increases the severity and likelihood of COVID-19 infections similar to many other infectious diseases. This paper models the interaction of pollution and disease preventive actions, either pharmaceutical or non-pharmaceutical interventions, on transmission of infectious diseases in a neoclassical growth framework. There are two externalities – households do not take into account how their actions affect disease transmission, and productive activity results in pollution which increases the likelihood of infections. The disease dynamics are modeled to be of *SIS* type. We study the difference in health and economic outcomes between the decentralized economy, where households do not internalize externalities, and socially optimal outcomes, and characterize the taxes and subsidies that decentralize the latter. Thus, we examine the question whether there are sufficient incentives to reduce pollution, at both private and public levels, once its effects on disease transmission is considered. In competitive outcomes, pollution than a competitive outcome, despite increase in abatement, as the effect of higher productivity and larger labor supply dominates. The results question the hopes of a Green Recovery.

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

JEL Classification: 115, 116, Q53, H23, E22, C61

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1 Introduction

Preliminary evidence from China, Italy, USA and other countries has shown that the presence of pollution, especially particulate matter, *PM*, nitrogen oxides, *NOx*, and ground level ozone increases the severity and likelihood of COVID-19 infections (Conticini, et al., 2020, Martelleti and Martelleti, 2020, Wu, et al., 2020, Yongjian, et al., 2020. Some studies 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 The COVID-19 pandemic has raised expectations of a "Green recovery" where understanding the role of pollution will promote 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. Thus, it studies the interaction of two mechanisms to control COVID-19 or other infectious diseases - preventive health expenditures, which prevent infections, and a pollution abatement policy, which reduces disease transmission by reducing pollution.

The interaction of these two instruments of policy are analyzed in a dynamic general equilibrium framework by extending the economic epidemiological model of Goenka and Liu, 2020, Goenka, et al., 2014, and 2020b, The disease dynamics are of *SIS* type 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. Health investment and abatement are 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. There are two externalities in the model: one being the pollution externality, where the pollution resulting from production increases disease transmission, and second is the disease externality, that is, 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, 2020, 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. The model is complex to solve analytically and we study the extent to which outcomes are affected by the externalities by a numerical analysis of the model. As 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 understand the qualitative properties of the model. We also characterize the dynamic Pigovian taxes that will decentralize the efficient outcomes. Analogous to the first set of numerical exercises we study how the taxes will change as

¹Pollution also increases the incidence of non-communicable diseases such as asthma, COPD, and other respiratory diseases, as well as cardiovascular disease. See Goenka, et al., 2020a, for a dynamic model 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.

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 expenditure, and, thus, better health outcomes, pollution is higher than in the decentralized equilibrium. While there is more abatement undertaken the effect of the higher output - due to increased labor supply and output due to both better disease control and higher productivity- dominates. Thus, there is no "disease dividend" from controlling the disease for 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 a 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 one are Bosi and Desmarchelier, 2018 and 2021, and Goenka and Liu, 2020. These papers use *SIS* dynamics in a growth model and model the disease externality. Bosi and Desmarchelier, 2018,model pollution affecting disease transmission directly as the current paper but not optimal control of either pollution or the disease as they treat these as pure externalities. Goenka and Liu, 2020, model a negative effect of capital stock which can be interpreted as pollution and address the control of disease incidence through accumulation of disease control capability, that is, health capital, ³ but not the abatement of pollution. directly. Bosi and Desmarchelier, 2021, 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. The current 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 a social planner internalizes the externalities, Section 5 contains the numerical analysis of the steady state equilibria, Section 6 the dynamic Pigovian taxes which decentralize the efficient outcomes, and Section 7 concludes.

2 Model

Epidemiology We use the *SIS* model to study the spread of the disease. While *SIR* dynamics was the most common modeling choice to model COVID-19 (see Ferguson, et al., 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. Using data from China, Long, et al., 2020, found evidence consistent with a steep decline in 2–3 months. Similar results were found by Ibarrando, et al., 2020, Isho, et al.,

 $^{^{3}}$ As do Goenka, et al., 2014.

2020, Riperger, et al., 2020,, and Ward, et al., 2021. On the other hand, Wajnberg, et al., 2020, and Sekine, et al., 2020, found evidence suggesting longer immunity. More recent evidence suggests that even vaccination does not confer immunity to infections (Al Qahtani, et al. (2021) present evidence on effectivity of four different vaccines in a population study of vaccinations in Bahrain, and Gazit, et al. (2021) present evidence from the vaccination program in Israel). As a modeling strategy Goenka, et al., 2021b, and Kissler, et al.,2020, used an *SIRS* model to model COVID-19. As we are concerned about the disease–pollution nexus in the medium to longer– run in this paper, we abstract from the temporary immunity phase (i.e., the state R).⁴ The population (N_t) is divided in two classes: (1) susceptible, healthy and who can catch the disease, S_t , and (2) infective, those infected and capable of transmitting the disease I_t with $S_t + I_t = N_t$. The disease dynamics are given by the following system of equations:

$$dS_t/dt = bN_t - dS_t - \alpha S_t I_t/N_t$$

$$dI_t/dt = \bar{\alpha} S_t I_t/N_t - \gamma I_t - dI_t$$

$$S_t, I_t, N_t \ge 0, \forall t;$$

$$S_0, I_0, N_0 > 0 \text{ with } N_0 = S_0 + I_0,$$

where b is the exogenous birth rate, and d is the exogenous death rate. We assume that $d \le b$ so that the population does not face extinction. The key epidemiology variables are the contact rate, $\bar{\alpha}$, that is, the average number of adequate contacts of a person to catch the disease per unit time, and the recovery rate from the disease, γ . The dynamics is represented in the transfer diagram, Figure 1.

In the paper, the contact rate in the pure epidemiology model without any interventions is denoted by the parameter $\bar{\alpha}$, We will subsequently, endogenize it by making it dependent on health expenditures and pollution. We treat γ as exogenous to keep the model tractable. Antivirals and anti-inflammatory drugs are now known to reduce the severity of the COVID-19 but these were inexpensive and widely available prior to the outbreak. ⁵ One of the features of COVID-19 is disease–related mortality. The case fatality rate (CFR) is estimated to be about 1.4%, that is, 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 paper we abstract from it.⁶

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

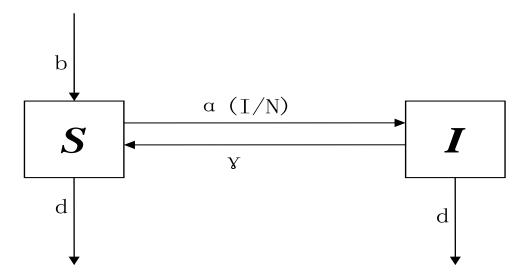
$$dN_t/dt = (b-d)N_t, \ b \ge 0, \ d \ge 0, \ b-d \ge 0.$$

⁴This 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, which 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.

⁵Goenka, et al., 2014, endogenized the recovery rate in a model without pollution.

⁶Goenka, et al., 2020b, modeled mortality in an *SIS* model, Goenka, et al., 2021a, in an *SIR* model and Goenka, et al., 2021b, in an *SIRS* model. All of these did not consider the role of pollution.

Figure 1. Transfer diagram of SIS dynamics



Note: The figure shows transfer between the two health states: Susceptibles, *S*, and Infective, *I*. *b* is the exogenous birth rate, *d* the exogenous death rate, α the contact rate, and γ the recovery rate. *N* is the total population size.

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:

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

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

$$(s^*,i^*)=\left(rac{b+\gamma}{arlpha},rac{arlpha-(b+\gamma)}{arlpha}
ight).$$

The disease-free steady state always exists, and the disease-endemic steady state exists only if $\bar{\alpha} > (b + \gamma)$. If this inequality is satisfied, then the disease-free steady state is unstable and the disease-free steady state is stable, otherwise the disease-free steady state is stable. There is a trans-critical bifurcation when $\bar{\alpha} = (b + \gamma)$. The basic reproduction number, $R_0 = \frac{\bar{\alpha}}{b + \gamma}$, 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 $f(k,l) : \mathbb{R}^2_+ \to \mathbb{R}_+$ is \mathscr{C}^2 , homogeneous of degree one,

and

1. $f_1 > 0, f_{11} < 0, f_2 > 0, f_{22} < 0,$

2.
$$\lim_{k_t \to 0} f_1 = \infty$$
, $\lim_{k_t \to \infty} f_1 = 0$ and $f(0, l) = f(k, 0) = 0$.

Profit maximization implies the following marginal conditions:

$$R = f_1(k, 1-i)$$
(1)

$$W = f_2(k, 1-i)$$

Production also results in a flow of pollution. By pollution we primarily mean PM and NO_x , which have been shown to affect the transmission of COVID-19 (and increase other infections such as acute lower respiratory infections, including pneumonia, bronchitis, and influenza (We treat this as a flow, that is, it does not accumulate, as shown by evidence. ⁷ In the decentralized economy, this is treated as an externality. The evolution of pollution is as follows.

Assumption 2. The level of pollution is a function of output y and abatement q - that is, P(y,q), where $P: \mathbb{R}^2_+ \to \mathbb{R}_+$ is \mathscr{C}^2 . We assume $P_1(y,q) > 0$ and $P_2(y,q) < 0$.

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

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

where the \bar{Y} denotes that the output is taken as given such 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 $l = 1 - i.^8$ We are assuming for simplicity that all infected workers do not work (see Goenka and Liu, 2020, and Goenka, et al., 2021a, 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 the treatment of COVID-19 other than the available therapies that reduce the severity of infection, we treat γ as exogenous.⁹ The contact rate depends on preventive health expenditure h and pollution P. In this paper we treat 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 - labeled in this paper as health expenditures or infection preventing activities.¹⁰ Thus, the contact rate is a function $\alpha(h, P)$ as

⁷See the Introduction for references that motivate our modeling of pollution.

⁸We assume labor supply by healthy workers is inelastic. If labor supply is elastic then under standard assumption of preferences u(c,l) > 0 the qualitative features of the model are unaffected. (see Goenka and Liu, 2012, for an SIS model with elastic labor supply. If we drop this assumption, then labor supply can be an independent source of nonlinear dynamics and cycles.

⁹In Goenka, et al., 2014, we modeled γ as a function of *h*.

¹⁰Bosi and Desmarchellier, 2018, have $\alpha(P)$; Goenka, et al., 2014, modeled this as $\alpha(h)$; Goenka and Liu, 2020, have $\alpha(h,k,e)$, where k, h, e are stocks of physical, health and human capital in an endogenous growth model. In the last paper, $\frac{\partial \alpha}{\partial k} > 0$ consistent with the interpretation of a pollution increasing transmission.

opposed to the parameter $\bar{\alpha}$ in the pure epidemiology model.

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

• $\alpha_1(h,P) < 0$ and $\alpha_2(h,P) > 0$, with $\alpha(0,0) = \bar{\alpha}$.

Thus, in the model the only role of pollution is to increase the contact rate. Pollution, indeed, has other effects on the economy: it can create production and consumption externalities, and increase the noninfectious mortality rate. We abstract from these effects as these have been modeled in dynamic general equilibrium models and we focus on the interaction of pollution, infectious disease transmission and preventive health measures.¹¹

Households: We assume that the economy is populated by a continuum of nonatomic identical households who are the representative decision-making agents. The size of the population in each household grows over time at the rate of $b - d \ge 0$, where b is the birth rate and d the death rate. We treat the demographic parameters, b and d, as exogenous. 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 such 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*, and of infected individuals is *i*. 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, and Goenka and Liu, 2020, the household considers itself small relative to the population and believes that the disease status within the household does not affect the proportion of infected in the entire population. In particular, the household takes as given the proportion of the population that is infected, denoted as Π , and believes that the probability for the healthy individuals to contract the disease is $\alpha \Pi$, rather than αi . Consequently, the disease transmission dynamics perceived by the households is now given as follows:

$$\dot{i} = \alpha(h, P(\bar{Y}, q))\Pi(1 - i) - \gamma i - bi.$$
(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" considering only 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 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 individual's productivity. We 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, that is, L = S, and dynamics of *L* inherit the dynamics of (1 - i).

¹¹Goenka, et. al., 2020a, studied the optimal policies when pollution increases mortality through noncommunicable diseases.

The representative household's preferences are given as:

$$\int_0^\infty e^{-\rho t} u(C) N_t dt = \int_0^\infty e^{-(\rho - b + d)t} u(C) N_0 dt,$$
(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, $u : \mathscr{R}_{++} \to \mathscr{R}$ is \mathscr{C}^2 with $u_c > 0$ and $u_{cc} < 0$, *jointly with the limit conditions:* $\lim_{c\to 0^+} u_c = \infty$ and $\lim_{c\to +\infty} u_c = 0$.

Households take the interest rate, R, and wage, W, as given. They rent out physical capital, K, 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

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

The evolution of the capital stock is given by:

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

where $\delta \in (0, 1]$ is the depreciation rate of physical capital. The equation (6) can be written as

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

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

$$\max_{\{c,h,q\}} \int_{0}^{\infty} e^{-(\rho-b+d)t} u(C) N_{0} dt$$

$$\dot{k} = Rk + W(1-i) - c - h - q - \delta k - (b-d)k$$

$$\dot{i} = \alpha(h, P(\bar{Y}, q)) \Pi(1-i) - \gamma i - bi$$
(7)

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

3 Decentralized economy

We define 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 the infected in the population, *i*, and $\bar{Y} = Y$.

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

- 1. Households solve the maximization problem (7).
- 2. Firms maximize profits, given by equation (1).
- 3. The capital market, labor market and goods market clear.
- 4. Pollution satisfies (2) with $\overline{Y} = f(k, 1-i)$.
- 5. As 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:

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

+ $\mu_1 i + \mu_2 h$

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

Incorporating the equilibrium conditions:

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

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 transversality conditions to the household problem incorporating the equilibrium conditions):

$$\dot{k} = f(k, 1-i) - c - h - q - \delta k - (b - d)k$$
(8)

$$\dot{i} = \alpha(h, P(f(k, 1-i), q))i(1-i) - \gamma i - bi$$
(9)

$$u'(c) = \lambda_1 \tag{10}$$

$$\lambda_1 = \lambda_2 \alpha_1(h, P(f(k, 1-i), q))i(1-i) \tag{11}$$

$$\lambda_1 = \lambda_2 \alpha_2(h, P(f(k, 1-i), q)) P_2(f(k, 1-i), q) i(1-i)$$
(12)

$$\hat{\lambda}_1 = \lambda_1 [\rho + \delta + b - d - f_1(k, 1 - i)]$$
(13)

$$\dot{\lambda}_2 = \rho \lambda_2 + \lambda_1 f_2(k, 1-i) + \lambda_2 [\alpha(h, P(f(k, 1-i), q))i + \gamma + b]$$
(14)
$$\mu_1 > 0, i > 0, \mu_1 i = 0.$$

$$\mu_1 \ge 0, h \ge 0, \mu_1 h = 0,$$

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

As the economy is a neoclassical economy with a bounded capital stock and *i* lies in a bounded interval, the transversality conditions are satisfied. In the subsequent discussion we suppress the transversality conditions.

Note that the optimization problem has nonconvex constraints on the state variable i and the usual Arrow and Mangasarian condition do not apply. Goenka, et al., 2014, showed the existence and sufficiency for the *SIS* model and we study the first order conditions which will characterize the dynamical system of the economy.¹²

3.1 Steady states

We now characterize the steady state competitive equilibria for the decentralized economy. As in the pure epidemiology model there are two equilibria that depend on the basic reproduction number, that is, 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 $q^* = 0$. The economic variables k^* and c^* are determined by

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

$$f(k,1) = c + \delta k + (b - d)k.$$

In the disease-free steady state pollution plays no role in the determination of equilibrium as the only effect in the model is through disease incidence, and the economy is in the neoclassical steady state. As a result there is neither pollution abatement nor health expenditure. Let us denote the equilibrium contact rate $\alpha(h^*, P^*) = \alpha^*$. If $\alpha^* < b + \gamma$, or the equilibrium endogenous reproduction number $R_0^* \equiv \frac{\alpha^*}{b+\gamma} < 1$, the disease is eradicated and the steady state is locally stable. While if

¹²The sufficiency conditions have been extended to account for mortality in Goenka, et al., 2020b, and for the *SIR* model with mortality in Goenka, et al., 2021a, and *SIRS* model with mortality in Goenka, et al., 2021b.

 $\alpha^* > b + \gamma$, that if $R_0^* > 1$ the disease free steady state is unstable. In the disease–free steady state, as there is no disease incidence, the equilibrium health and abatement expenditures. Thus, in equilibrium, $\alpha^*(0,0) = \bar{\alpha}$.

The disease–endemic steady state is a solution to the system of equations (8 - 14). It exists when the contact rate is high enough such that $s^* < 1$, i.e. $\alpha(h^*, P^*) > b + \gamma$ or the equilibrium endogenous reproduction number $R_0^* > 1$.

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

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

$$\tag{15}$$

$$\alpha_1(h, P(f(k, 1-i), q)) = \alpha_2(h, P(f(k, 1-i), q))P_2(f(k, 1-i), q)$$
(16)

$$f_1(k, 1-i) = \rho + \delta + b - d$$
 (17)

$$-f_2(k,1-i)\alpha_1(h,P(f(k,1-i),q))i(1-i) = \rho + \alpha(h,P(f(k,1-i),q))$$
(18)

$$f(k, 1-i) = c + h + q + \delta k + (b-d)k$$
(19)

Proof. From equation (9) $\dot{i} = 0$, 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 (11) and (12), 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 the impact on the contact rate α .

From $\dot{\lambda_1} = 0$, we have

$$f_1(k,1-i) = \rho + \delta + b - d_2$$

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

From $\dot{\lambda_2} = 0$, we have

$$\lambda_1 f_2(k, 1-i) = -\lambda_2
ho - \lambda_2 [lpha(h, P)i + \gamma + b]_2$$

which implies the marginal benefit of disease control (one unit of reduction in *i* - the proportion of the infected) equal to its marginal cost. The left-hand side 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 right-hand side is the marginal cost of controlling the disease. 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 individuals, there are more infections.

Substituting into equation (11) we have

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

Note, that in equilibrium from (15) we have $R_0^* > 1$. Thus, in the competitive equilibrium the disease is endemic. However, this depends not only on the disease characteristics, that is, the function α , but also on the deeper economic parameters that 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 regarding what will happen once the reproduction number, R_0^* , 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 depth. The paper Goenka and Liu, 2012 and Goenka, et. al., 2014, studied these issues comprehensively in a model without pollution in discrete time and continuous time respectively.

4 The Centralized Economy

In the centralized economy, the social planner considers both pollution and disease externalities, that is, the true law of motion for the disease and effect of output on pollution are taken into account. Therefore, the social planner solves the following maximization problem:

$$\max_{\{c,h,q\}} \max_{\{c,h,q\}} \int_0^\infty e^{-(\rho-b+d)t} u(C) N_0 dt$$

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

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

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

$$\dot{k} = f(k, 1-i) - c - h - q - \delta k - (b-d)k$$
(20)

$$\dot{i} = \alpha(h, P(f(k, 1-i), q))i(1-i) - \gamma i - bi$$
(21)

$$u'(c) = \lambda_1 \tag{22}$$

$$\lambda_1 = \lambda_2 \alpha_1(h, P(f(k, 1-i), q))i(1-i)$$
(23)

$$\lambda_1 = \lambda_2 \alpha_2(h, P(f(k, 1-i), q)) P_2(f(k, 1-i), q) i(1-i)$$
(24)

$$\lambda_{1} = \lambda_{1}[\rho + \delta + b - d - f_{1}(k, 1 - i)] + [-\lambda_{2}\alpha_{2}(h, P)P_{1}(y, q)f_{1}(k, 1 - i)(1 - i)i]$$
(25)
$$\dot{\lambda}_{2} = \rho\lambda_{2} + \lambda_{1}f_{2}(k, 1 - i) + \lambda_{2}[\alpha(h, P(f(k, 1 - i), q))i + \gamma + b] +$$

$$+[\lambda_2 \alpha_2(h, P)P_1(y, q)f_2(k, 1-i)(1-i)i] + [-\lambda_2 \alpha(h, P)(1-i)]$$
(26)

An additional term in (25) exists as compared to (13) in a decentralized economy, that arises

from taking into account the effect of output on pollution and hence, disease transmission; and two additional terms in (26) as compared to (14) that arise from considering the effects of infections on labor supply, and hence, output, pollution, and disease transmission, and the second that 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 identical to the one in the 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

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

$$\tag{27}$$

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

$$P_2(f(k, 1-i), q) = \alpha_2(h, P(f(k, 1-i), q))P_2(f(k, 1-i), q)$$
(28)

$$f_1(k, 1-i) = \rho + \delta + b - d + \left[-\frac{P_1(f(k, 1-i), q)f_1(k, 1-i)}{P_2(f(k, 1-i), q)}\right]$$
(29)

$$-f_2(k,1-i)\alpha_1(h,P(f(k,1-i),q))i(1-i) + [\alpha(h,P(f(k,1-i),q))(1-i)] =$$
(30)

$$= \rho + \alpha(h, P(f(k, 1-i), q)) + [\alpha_2(h, P(f(k, 1-i), q))P_1(f(k, 1-i), q)f_2(k, 1-i)(1-i)i]$$

$$f(k, 1-i) = c + h + q + \delta k + (b-d)k$$
(31)

Proof. The proof is similar to the one in the decentralized economy and, thus, omitted here. \Box

Compared with the decentralized economy, there are two differences: one is equation (29), which states that the marginal social benefit of physical capital should be equal to the marginal social cost. In the centralized economy, a social planner takes into account the negative externality of pollution on disease transmission, thus, there is an additional cost to physical capital investment. When physical capital increases, output increases, which leads to more pollution and a higher effective contact rate for disease transmission. This is captured by the term $\left[-\frac{P_1(f(k,1-i),q)f_1(k,1-i)}{P_2(f(k,1-i),q)}\right]$. The other difference is equation (30), which states that the marginal social benefit of disease controlling is equal to the marginal social cost. In the centralized economy, there are two additional terms in equation (30). When the social planner accounts for the negative externality of pollution, there is an additional cost of 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 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, a social planner takes into account the disease externality, that is, there is an additional benefit from controlling disease, as the social planner considers that behavior will 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 considers both negative pollution externalities and positive disease controlling externalies. To compare the abatement and health

expenditures in the centralized and decentralized economies, we use numerical methods because the system of nonlinear equations is too complex to compare directly.

5 Model Simulations

As the model is too complex for closed form solutions or conducting comparative statics analytically, in this section, we parameterize the model and conduct comparative statics numerically to understand the interaction of the different components. We focus on examining the parameters where pollution can affect 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 parameter is important because it tries to ascertain whether economies that are more productive will have different responses and outcomes. The analysis here focuses on the equilibrium steady states before and after the change as the study wants 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 consistent with the literature. The production function is assumed to be a Cobb–Douglas function: $y = f(k, 1-i) = Ak^{\beta}(1-i)^{1-\beta}$ 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 as b = 3% and the death rate d = 1.5%. The recovery rate is $\gamma = 0.2$.

We do not have enough empirical evidence to suggest a 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 is large enough to generate an endemic steady state in the simulation. We assume that the contact rate function is a power function as follows: $\alpha(h) = \varepsilon_0(h + \varepsilon_1)^{\varepsilon_2}(P + \varepsilon_3)^{\varepsilon_4}$ 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 that $P(y,q) = \phi_0 f(k, 1 - i) - \phi_1(q + \phi_2)^{\phi_3}$ with $\phi_0 = 1, \phi_1 = 1, \phi_2 = 0$ and $\phi_3 = 0.5$ in the baseline specification.

Figure 2, 3, and 4 are the simulation results for varying the elasticity of pollution on the contact rate function (ε_4), the elasticity of abatement on pollution (ϕ_3), and TFP (A), respectively. The parameters are chosen such that the disease–endemic steady state exists for both the decentralized and centralized economies. Therefore, we can focus on how changes in those parameters change the 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 the three figures, if we compare the decentralized with the centralized economy, the pictures are similar. The social planner can achieve lower contact rates in equilibrium with higher health expenditures. As a result, the labor force is larger and, hence, and output is higher. The capital stock is lower in the planning solution. While the abatement is higher in the planning solution, paradoxically pollution is also higher because the output is higher. Although there is greater abatement, the net effect of the higher output dominates and pollution is higher. Thus, even when the planner considers the deleterious effect of pollution on health outcomes, the efficient outcome has higher pollution.

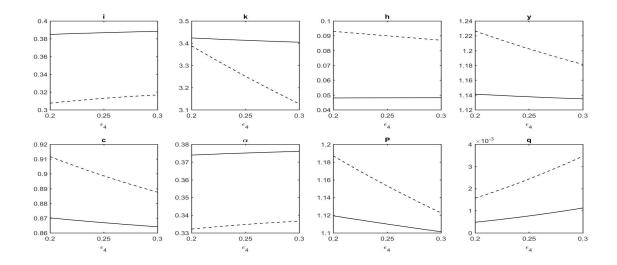


Figure 2. The simulation results - varying the elasticity of pollution on contact rate

Note: The figure shows changes in the disease endemic steady state when we change the elasticity of pollution on contact rate. There are total eight panels - *i* (the proportion of the infected), *k* (physical capital), *h* (health expenditure), *y* (output), *c* (consumption), α (contact rate), *P* (pollution), and *q* (abatement). The solid line is the decentralized economy (without disease and pollution externalities). The dashed line is the centralized economy (with disease and pollution externalities).

Whether an economy has a 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 abatement are not affected by a change in TFP.

In the pure epidemiology model, a disease-free steady state always exists, and disease-endemic steady state only exists when the contact rate is high enough, that is, $\bar{\alpha} > b + \gamma$ or $R_0^* = \frac{\bar{\alpha}}{b+\gamma} > 1$. When there is only the disease-free steady state, it is stable. When both disease-free and diseaseendemic steady states co-exist, the disease-free steady state is unstable and the disease-endemic steady state is stable. The same applies to the economic epidemiological models here for both the decentralized and centralized economies. The only difference is that the contact rate, α^* , or reproduction number, R_0^* , are 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 can achieve lower contact rates in equilibrium, it is more likely to have the disease eradicated in the centralized economy with the same parameter values. In other words, 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. Thus, 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 the decentralized and the centralized economies. The simulation results are shown in Figure 5. The cut-off points of disease–endemic steady state is $\varepsilon_0 = 0.08$ for the decentralized economy and $\varepsilon_0 = 0.12$ for the centralized economy. When the contact rate parameter ε_0 is small enough (below 0.08) such that $\alpha < b + \gamma$, there is a disease-free steady state in both the decentralized and centralized economies. 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, because

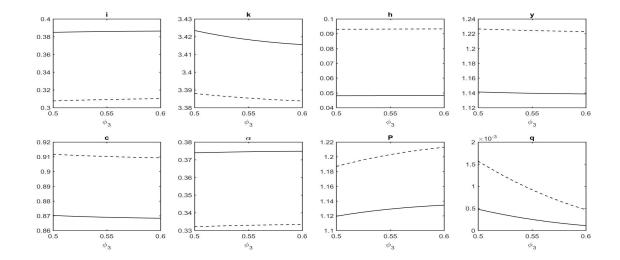


Figure 3. The simulation results - varying the elasticity of abatement on pollution

Note: The figure shows changes in the disease endemic steady state when we change the elasticity of abatement on pollution. There are total eight panels -*i* (the proportion of the infected), *k* (physical capital), *h* (health expenditure), *y* (output), *c* (consumption), α (contact rate), *P* (pollution), and *q* (abatement). The solid line is the decentralized economy (without disease and pollution externalities). The dashed line is the centralized economy (with disease and pollution externalities).

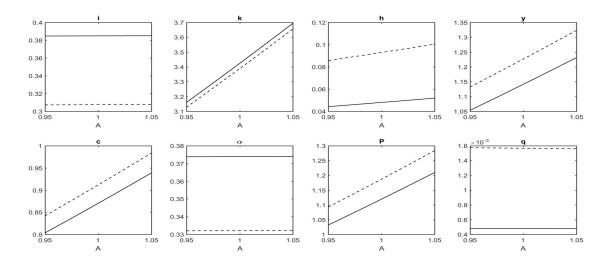


Figure 4. The simulation results - varying productivity

Note: The figure shows changes in the disease endemic steady state when we change productivity in the production function. *i* (the proportion of the infected), *k* (physical capital), *h* (health expenditure), *y* (output), *c* (consumption), α (contact rate), *P* (pollution), and *q* (abatement). The solid line is the decentralized economy (without disease and pollution externalities). The dashed line is the centralized economy (with disease and pollution externalities).

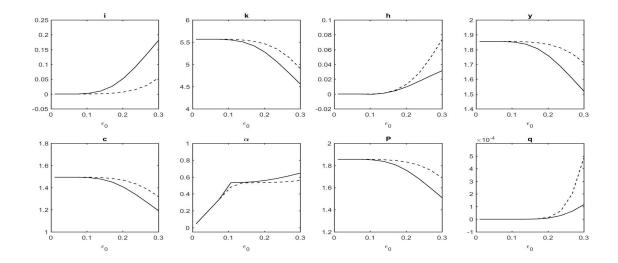


Figure 5. The simulation results - disease free and disease endemic steady states

Note: The figure shows changes in the disease free and disease endemic steady states when we change the contact rate parameter ε_0 . *i* (the proportion of the infected), *k* (physical capital), *h* (health expenditure), *y* (output), *c* (consumption), α (contact rate), *P* (pollution), and *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).

as $\alpha < b + \gamma$ there is only one disease–free steady state, and a disease–endemic steady state does not exist. In contrast, in the decentralized economy, a disease–endemic steady state exists.¹³ For a disease–endemic steady state in the decentralized economy, the fraction of the infected is positive and physical capital, output and consumption are lower, then that in the centralized economy. When ε_0 is more than 0.12, for both the decentralized and centralized economies, as the contact rate is high enough, disease endemic steady state exists.¹⁴

6 Public Policy

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

¹³Note that there is also a disease–free steady state (not shown in the figure).

¹⁴Note that there is also a disease –free steady state (not shown in the figure).

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

$$\max_{\{c,h,q\}} \int_0^\infty e^{-\rho t} u(c) dt$$

$$\dot{k} = (1 - \tau_k) Rk + (1 - \tau_l) W(1 - i) - c - (1 - \tau_h) h - (1 - \tau_q) q - \delta k - (b - d) k$$

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

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

$$\tau_k Rk + \tau_l W(1-i) = \tau_h h + \tau_q q.$$
(32)

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

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

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:

$$i = 1 - \frac{b + \gamma}{\alpha(h, P(f(k, 1 - i), q))}$$
(33)

$$\alpha_1(h, P(f(k, 1-i), q))\frac{1}{1-\tau_h} = \alpha_2(h, P(f(k, 1-i), q))P_2(f(k, 1-i), q)\frac{1}{1-\tau_q}$$
(34)

$$(1 - \tau_k)f_1(k, 1 - i) = \rho + \delta + b - d$$
(35)

$$-\frac{1-\tau_l}{1-\tau_h}f_2(k,1-i)\alpha_1(h,P(f(k,1-i),q))i(1-i) = \rho + \alpha(h,P(f(k,1-i),q))$$
(36)

$$f(k, 1-i) = c + h + q + \delta k + (b-d)k$$
(37)

Next, we show how tax and subsidy policies are designed, such that the decentralized economy can replicate the allocations from the centralized economy. A comparison of equation (28) with

(34) shows that the subsidy for health expenditure τ_h should be equal to the subsidy for pollution abatement τ_q :

$$\tau_h = \tau_q. \tag{38}$$

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

Then, by comparing equation (29) with (35), we can derive capital income tax as follows:

$$\tau_k = -\frac{P_1(f(k, 1-i), q)}{P_2(f(k, 1-i), q)}.$$
(39)

As $P_1 > 0$ and $P_2 < 0$, the capital income tax is $\tau_k > 0$. If we compare equation (30) with (36), we get

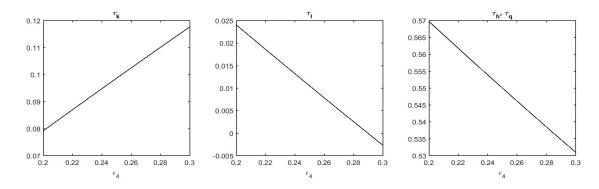
$$\frac{\tau_h - \tau_l}{1 - \tau_h} = \frac{\alpha_2(h, P(f(k, 1 - i), q))P_1(f(k, 1 - i), q)f_2(k, 1 - i)i - \alpha(h, P(f(k, 1 - i), q))}{f_2(k, 1 - i)\alpha_1(h, P(f(k, 1 - i), q))i}.$$
 (40)

Thus, we have the following proposition.

Proposition 5. The tax and subsidy policies $(\tau_k, \tau_l, \tau_h, and \tau_q)$ are determined by equation (32), (38), (39) and (41), where the economic variables *i*,*k*,*h*,*q* and *c* are the equilibrium allocations from the centralized economy.

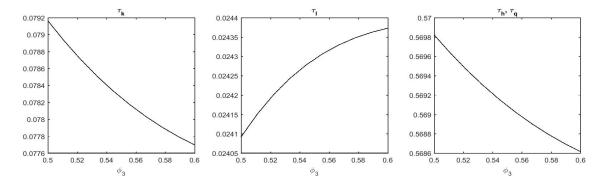
To understand the taxes and subsidies, we conduct a similar exercise as in the previous section of varying the elasticity of pollution, the elasticity of abatement and the TFP. Interestingly, for each of the exercises, the subsidy on health and abatement decreases and the primary mechanisms are the direct taxes on labor and capital income. The effects of increasing elasticity of pollution and contact rate and on abatement rate is different. As the 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 the elasticity of abatement increases, i.e. abatement is more effective, capital income is taxed at a lower rate. However, labor income taxes are increased to maintain budget balance. Similarly, as the TFP increases, capital income tax is reduced and therefore, labor income taxes are increased to compensate for the reduced tax on capital income. Thus, we should expect a different mix of labor-capital income taxes for economies depending on their TFPs and thus, growth rates.

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



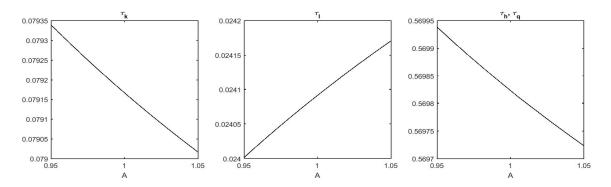
Note: The figure shows changes in tax and subsidy policies when we change the elasticity of pollution on contact rate.

Figure 7. 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

Figure 8. 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 examines the interaction of pollution with transmission of diseases. A decentralized economy has both the pollution and disease transmission externalities. As expected, the level of pollution abatement and preventive health expenditures are lower than those in the centralized economy. While the centralized outcomes can be decentralized through dynamic Pigovian taxes, higher productivity increases the level of pollution while achieving the efficient outcome. The control of the disease interacts with higher productivity, and although more is spent on abatement, the net effect is that pollution goes up. This is consistent with the recent evidence that during lockdown pollution levels decreased as mobility and activity decreased. However, this was temporary and pollution levels have gone up since then (Kumari and Toshniwal (2020)). Thus, the expectation of a Green Recovery may be illusory.

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