Balancing mitigation policies during pandemics: economic, health, and environmental implications

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Abstract

The strategies implemented to contain the spread of COVID-19 have clearly shown the existence of a nontrivial relation between epidemiological and environmental outcomes. On the one hand, health regulations and recommendations contribute to deteriorate the environmental quality through increased waste and emissions. On the other hand, increased pollution exposes individuals to a higher chance of severe symptoms increasing their probability of death due to respiratory diseases. In order to understand how balancing the different goals in the design of effective containment policies we develop a normative approach to account for their consequences on the economy, health and the environment by analyzing the working mechanisms of social distancing in a pollution-extended macroeconomic-epidemiological framework with health-environment feedback effects. By limiting social contacts and thus disease incidence, social distancing favors health and environmental outcomes at the cost of a deterioration in macroeconomic conditions. By calibrating the model to the Italian COVID-19 experience during the first epidemic wave, we characterize how the optimal social distancing policy depends on the main environmental factors, showing that social distancing alone is not enough to reverse the growth pattern of both disease prevalence and pollution and thus it is optimal to reduce the disease spread even if this generates a deterioration in environmental conditions. We also extend our baseline model to account for the role of strategic interactions between two-neighbor economies in which both pollution and disease prevalence are transboundary. In this context we show that free-riding induces sizeable efficiency losses, quantifiable in about 15% excess disease prevalence and 5% excess pollution at the end of the epidemic management program.

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1 Introduction

Sustainable development has become a very popular topic lately and in its broader definition it demands policies promoting improvements in economic, health and environmental issues (WCED, 1987; UN, 2005; UNEP, 2012). The ongoing COVID-19 pandemic has shown more clearly than ever that economy, environment and health are all interrelated and that exogenous communicable-disease-induced shocks may generate devastating effects on economic activities, health conditions and environmental outcomes at once. Indeed, since the initial outbreak of the disease in China in late 2019, it has thus far (at the time of writing, in December 2021) generated more than 275 million cases and more than 5 million deaths at world level (Dong

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et al., 2020). A broad variety of policy measures have been implemented everywhere in the world in order to contain the spread of the disease, including traditional preventive and treatment measures but also lockdowns, quarantines, social distancing, limitations on mobility (Cheng et al., 2020). Such containment strategies, forcing individuals to work from home and imposing the closure of unnecessary businesses, have resulted in dramatic consequences for economic activities, in terms of drastic reductions in household income, substantial increases in unemployment rates, and increases in social inequalities (Brodeur et al., 2021; Crossley et al., 2021). However, apart from such short-run economic effects, mitigation policies are also likely to generate important long-run consequences on the environment, since the growing use of plastic-material in the manufacturing of single-use medical and personal protection equipment and in the single-use packaging for food has resulted in a massive increase in waste and emissions, ending up in polluting the soil and oceans (EEA, 2021; Peng et al., 2021). Considering also that pollution generates sizeable implications on morbidity and mortality especially when interacting with respiratory diseases (Cui, 2003; Wu et al., 2020), it is essential to understand not only the health and economic consequences of disease control strategies but also their environmental impacts in order to design effective policies aiming to minimize their social cost and support policymakers in one of the most difficult periods of the recent economic history.

The recent COVID-19 experience has pointed out the existence of a nontrivial relation between epidemiological and environmental outcomes. By modifying the production and the delivery needs of specific products the public health regulations implemented to contain the spread of the disease have contributed to deteriorate the environmental quality through increased waste and emissions. Several studies show that one of the most important effects of public health recommendations during the COVID-19 pandemic consists of changing individuals' purchasing habits, which has shifted towards plastic-intensive products (OECD, 2020b, EEA, 2021). Indeed, the needs of the frontline health workers and private citizens to wear protective equipment (such as face masks, gloves, and aprons) along with those of staying-home workers to increase their reliance on e-commerce and take-away food deliveries in order to minimize their mobility have resulted in a massive increase in the production, transport and consumption of plastic (EEA, 2021; Filho et al., 2021). Moreover, prolonged periods of stay-at-home conditions have increased the production of household waste (such as cleaning and disinfecting material, used or unused medical waste, but also food waste) which have put under stress recycling facilities and the health of the environment (OECD, 2020a; Hantoko et al., 2021). The increased use of plastic-based products during the COVID-19 pandemic has important environmental and climate impacts, related to resource extraction, production, transport, waste handling and littering, resulting in increased pollution on streets, in rivers, on beaches, along coasts and in the sea (Adyel, 2020; Canning-Clode et al., 2020).²

Apart from the effects of public health regulations and recommendations on pollution, pollution in turn affects epidemiological outcomes as well. By magnifying the health risk factors increased pollution exposes individuals to a higher chance of severe symptoms increasing their probability of death. Indeed, several studies show that pollution increases people's vulnerability to the effects of respiratory infections, such as SARS and COVID-19 (Cui, 2003; Wu et al., 2020). It is well known that high pollution levels lead to several health problems especially to lung and respiratory diseases, such as triggering new cases of asthma, exacerbating previously-existing respiratory illness, and provoking the development or progression of chronic illnesses including lung cancer, chronic obstructive pulmonary disease, and emphysema (Pope et al., 1995;

¹During the first epidemic wave the extensive use of lockdowns has resulted in a significant reduction in economic activities, and thus also in emissions and pollution concentrations (Venter et al., 2020). However, as soon as the restrictive measures have been relaxed pollution has returned back to its pre-COVID level in a just few months (ESA, 2021). Therefore, it seems convenient from a normative perspective to focus on the possible long-run environmental effects of mitigation policies.

²Just to give a sense of the magnitude of the problem, the number of plastic facemasks used on a daily basis at the world level is estimated to exceed 7 billion (Hantoko et al., 2021). And during the height of the epidemic in Wuhan the city has dealt with 240 tons of medical waste a day, compared to around 40 tons a day before the outbreak (Zuo, 2020). The increased consumption of face masks only during the first wave (April-September 2020) has led to the emission of 2.4-5.7 million tonnes of CO2 above the business-as-usual level in Europe, equivalent to an increase of 118% (EEA, 2021).

Katsouyanni et al., 1996; Kunzli et al., 2000). And pre-existing medical conditions, including those involving lung and respiratory impairments, increase the likelihood of severe illness and death from COVID-19 (CDC, 2021; Lacedonia et al., 2021). In particular, recent estimates show that a person exposed for decades to high levels of fine particulate matter is 15% more likely to die from COVID-19 than someone exposed to one unit less of the fine particulate pollution (Wu et al., 2020; OECD, 2020). Therefore, not only the health regulations implemented in the fight of COVID-19 increase pollution but also pollution increases the mortality associated with COVID-19, which requires to carefully account for the existence of such a bilateral relation between epidemiological and environmental outcomes in the design of effective containment policies.

However, optimally designing disease control policies is not simple at all since the effectiveness of the different measures implemented in a given economy largely depends on those implemented in other economies as well. Several papers discuss that because of the growing level of globalization, international trade, technological progress and migration, localized epidemic shocks tend to spread fast on a geographical level eventually achieving a pandemic scale (Kimball, 2006; Tatem et al., 2006; Baker et al., 2021). Such a geographical interrelation between epidemiological outcomes at single country level suggests that trying to limit the spread of an infectious disease without accounting for the policy actions in other economies is pointless and only international coordination may effectively allow for disease eradication (Barrett, 2003; La Torre et al., 2022). Even in the case of COVID-19, a growing number of works document that the fast spread of the disease both within and between countries is driven by mobility and trade patterns, justifying the introduction of travel bans and other policies aiming to reduce individuals' mobility at different geographical levels in order to limit the diffusion of the illness (Tayoun et al., 2020; Chang et al., 2021). This requires to critically understand the extent to which uncoordinated mitigation efforts may allow for disease containment, especially in light of the fact that the unpopularity of the most widely spread policy tools in the fight of COVID-19 (i.e., social distancing) may give rise to free-riding opportunities. Therefore, apart from introducing environmental considerations in the analysis of disease control measures, it is essential to account for strategic interactions between multiple policymakers in order to quantify the effects of free-riding on mitigation efforts.

In order to address these issues, we extend a macroeconomic-epidemiological framework to an environmental dimension to assess the extent to which pollution considerations may impact the intensity of mitigation strategies. Our work is thus related to the growing economic epidemiology literature which aims to analyze how health policies may impact economic activities both at microeconomic and macroeconomic levels (Philipson, 2000; Gersovitz and Hammer, 2003; Goenka and Liu, 2012; La Torre et al., 2020). In particular, a huge number of works has analyzed the consequences of different policies on the trade-off between economic and health objectives in the context of COVID-19, placing particular emphasis on social distancing and lockdown (Acemoglu et al., 2020; Alvarez et al., 2020; Gori et al., 2021; La Torre et al., 2021b). Several works have also examined the role of strategic interactions between different players, in terms of individual agents, individual demographic groups or individual economies, in determining the relation between the spread of COVID-19 and macroeconomic outcomes (Cui et al., 2020; Bouveret and Mandel, 2021; La Torre et al., 2021a). Most of these works discuss the macroeconomic implications of COVID-19 and the related mitigation measures, abstracting completely from their environmental impacts. To the best of our knowledge, very limited are the papers accounting for the possible environmental issues associated with disease-control strategies, and all these works abstract completely from strategic interactions (Brock and Xepapadeas, 2020; Augeraud-Veron et al., 2021). Brock and Xepapadeas (2020) discuss the importance to take into account environmental issues in the analysis of disease containment strategies to distinguish between short-run epidemic management objectives and long-run climate mitigation goals, but they do not derive the optimal policy. Augeraud-Veron et al. (2021) discuss how the optimal policy depends on biodiversity conservation which by decreasing the probability of an epidemic shock acts as a preventive measure of disease containment showing that biodiversity conservation is larger the more forward looking the society; however, they abstract from pollution and bidirection feedback epidemiological-environmental effects. Different from these

works, we explicitly account for the two-ways health-environment relation driven by emissions and mortality effects, discussing in particular how the optimal policy depends on environmental conditions. Moreover, we analyze the implications of strategic interactions between two-neighbor economies to understand the role of transboundary epidemiological and pollution externalities on free-riding opportunities and the optimal policy.

Specifically, we analyze a pollution-extended macroeconomic-epidemiological framework in which the spread of the disease deteriorates economic activities and increases the stock of pollution which in turn raises the disease-induced mortality rate. Disease dynamics are described by a susceptible-infected-susceptible (SIS) model with vital dynamics, which represents a simple but general enough setting to capture the implications of epidemiological factors on the economy and the environment. Mitigation policies, in the form of social distancing by reducing disease incidence, favor epidemiological and environmental outcomes at the cost of a deterioration in macroeconomic conditions. The social planner needs to balance these conflicting goals optimally determining the intensity of the policy measure over a finite time horizon, representing the duration of the epidemic management program. We calibrate the model's parameters according to Italian data related to the first epidemic wave, occurred between February to July 2020 in order to exemplify the relevance of our analysis in real world situations. We characterize how the optimal social distancing policy depends on the main environmental factors, showing that social distancing alone is not enough to reverse the growth pattern of both disease prevalence and pollution. Indeed, the optimal policy allows for a reduction of disease prevalence only at a cost of a deterioration in environmental outcomes, suggesting that placing too much emphasis on epidemic management as done in the policy arena risks to leave us with a high environmental bill which will require massive efforts in the near future to improve environmental conditions in order to achieve long-run sustainability. We also extend our baseline model to account for the role of strategic interactions between two neighbor economies in which not only pollution is transboundary but also disease prevalence is. We show that free-riding induces important efficiency losses, quantifiable in about 15% excess disease prevalence and 5% excess pollution at the end of the epidemic management program. This suggests that policy coordination is essential in order to effectively mitigate the consequences of infectious diseases. To the best of our knowledge, ours is the first attempt to quantify how environmental conditions may depend on and affect the optimal management of the macroeconomic-epidemiological trade-off.

The paper proceeds as follows. Section 2 presents our pollution-extended macroeconomic-epidemiological framework where disease prevalence determines and is affected by both economic and environmental outcomes. Section 3 characterizes the optimal solution of the epidemic management problem from a normative perspective, presenting some numerical experiments based on our Italian data calibration. Section 4 introduces strategic interactions between multiple policymakers to explore the implications of free-riding opportunities on the optimal policy and the eventual effectiveness of the epidemic management program. Section 5 presents concluding remarks and directions for future research. Appendix A focuses on the early-epidemic stage problem to explicitly derive the optimal solution which allows us to test the reliability of our numerical algorithm.

2 The Model

We consider a pollution-extended macroeconomic-epidemiological framework in which the spread of an infectious disease drives output production and emissions, and social distancing which reduces output further but also decreases disease incidence and emissions is used to manage the epidemic. The epidemiological side is described by a SIS model in which disease incidence determines emissions which in turn drive the disease-induced mortality, while in the macroeconomic one disease prevalence affects output. This gives rise to feedback effects between health and macroeconomic outcomes. A similar setting has been recently analyzed in La Torre et al. (2021b) to determine the optimal social distancing policy, abstracting completely from pollution considerations and feedback health-environment effects.

2.1 The Epidemiological Framework

Before introducing our macroeconomic-epidemiological setup, we briefly review the basic SIS model with vital dynamics, having its origin in the seminal works by Kermack and McKendrick (1927) and Busenberg and van den Driessche (1990), and extend it to account how the disease spread affects and is affected by pollution. The SIS model is one of the simplest and most discussed frameworks in mathematical epidemiology, widely applicable to a range of diseases not conferring permanent immunity, such as the seasonal flu, some sexually transmitted diseases and some vector-borne diseases (Hethcote, 2008). Since individuals do not acquire permanent immunity from COVID-19 either through recovery or through vaccination, it is also well suited to characterize in a simplified way the spread of COVID-19 (WHO, 2020; La Torre et al., 2021b).

The population, N_t , which grows because of natality at rate b>0 and shrinks because of mortality at rate d > 0, is composed by healthy individuals who are susceptible to the disease, S_t , and the infectives who have already contracted the disease and can transmit it by getting in contact with suceptibles, I_t . Thus, at any moment in time we have that $N_t = S_t + I_t$, and the interactions between susceptibles and infectives determine the evolution of the two subpopulation groups. Infectives spontaneously recover at the rate $\delta > 0$ but suffer the excess mortality induced by the infection at rate $\bar{\mu} > 0$, and susceptibles become infective by interacting with infectives which occurs at the rate $\alpha > 0$, measuring the number of social contacts required to give rise to a new infection (i.e., the product between the number of contacts between infectives and susceptibles per unit of time and the probability that one contact leads to disease transmission). In order to control the spread of the disease policymakers implement social distancing measures (i.e., lockdowns) to limit the social contacts by a percentage $0 < u_t < 1$ reducing thus disease transmission and disease incidence. Individual activities aiming to minimize infection (i.e. purchasing plastic face masks and gloves) generate some pollution (in excess to the normal pollution trend), P_t , which increases the disease-induced mortality as follows: $\bar{\mu} = \bar{\mu}_t = \mu(1 + \frac{P_t}{N_t})$, where $\mu > 0$ measures the magnitude of such environmental effects on mortality. Pollution accumulates according to the difference between emissions and natural absorption: emissions are proportional to disease incidence at a rate $\theta > 0$ quantifying the dirtiness of individuals' preventive response to the infection, while the pollution decay rate is $\eta > 0$. This implies that the dynamics of susceptibles, infectives, population and pollution can be described through a dynamic system as follows:

$$\dot{S}_t = bN_t - dS_t + \delta I_t - \alpha (1 - u_t) \frac{I_t}{N_t} S_t, \tag{1}$$

$$\dot{I}_t = \alpha (1 - u_t) \frac{I_t}{N_t} S_t - \delta I_t - dI_t - \mu \left(1 + \frac{P_t}{N_t} \right) I_t, \tag{2}$$

$$\dot{N}_t = (b-d)N_t - \mu \left(1 + \frac{P_t}{N_t}\right)I_t, \tag{3}$$

$$\dot{P}_t = \theta \alpha (1 - u_t) \frac{I_t}{N_t} S_t - \eta P_t. \tag{4}$$

Disease incidence, $\alpha(1-u_t)\frac{I_t}{N_t}S_t$, is determined by biological factors, α , public policy, u_t and social interactions between individuals (either on the workplace or for daily life activities), $\frac{I_t}{N_t}S_t$. The latter term states that the patterns of social contacts and human interactions are stable over time independently of the spread of the disease, and thus disease transmission and incidence ultimately depend on the share of the infectives, $\frac{I_t}{N_t}$, rather than the total number of infectives, I_t . Apart from affecting the speed of the epidemic, disease incidence determines also the speed of pollution accumulation which in turn drives disease dynamics. In particular, disease-induced mortality depends on the amount of per-capita pollution $\frac{P_t}{N_t}$ which quantifies the impact at the single individual level of the environmental externality.

The above system can be recast in terms of susceptible and infective shares, $s_t = \frac{S_t}{N_t}$ and $i_t = \frac{I_t}{N_t}$

respectively, and per capita pollution, $p_t = \frac{P_t}{N_t}$, as follows:

$$\dot{s}_t = b(1 - s_t) + \delta i_t - [\alpha(1 - u_t) - \mu(1 + p_t)(1 - i_t)]i_t s_t, \tag{5}$$

$$\dot{i}_t = \alpha(1 - u_t)i_t s_t - i_t [b + \delta + \mu(1 + p_t)(1 - i_t)], \tag{6}$$

$$\dot{p}_t = \theta \alpha (1 - u_t) i_t s_t - [\eta + b - d - \mu (1 + p_t) i_t] p_t. \tag{7}$$

Since $s_t = 1 - i_t$, the above system can be recast in terms of the following planar system:

$$\dot{i}_t = \alpha (1 - u_t)(1 - i_t)i_t - [b + \delta + \mu(1 + p_t)(1 - i_t)]i_t, \tag{8}$$

$$\dot{p}_t = \theta \alpha (1 - u_t)(1 - i_t)i_t - [\eta + b - d - \mu(1 + p_t)i_t]p_t. \tag{9}$$

As extensively discussed in mathematical epidemiology, the long-run disease outcome depends on the relative intensity of the effective speed of disease transmission, $\alpha(1-u_t)$, and the effective speed of recovery, $b+\delta+\mu(1+p_t)$. Only if the latter exceeds the former it may be possible to achieve disease eradication in the long run, and since the effective speed of transmission depends on social distancing public policy may be effectively used to promote eradication. Social distancing by reducing disease incidence limiting the number of possible contacts between susceptibles and infectives allows to decrease both disease prevalence and pollution, improving eventually both epidemiological and environmental outcomes.

2.2 The Macroeconomic Framework

After having described a pollution-extended SIS model, we now introduce our macroeconomic setup in which the public policy (i.e., social distancing) intensity is optimally determined. Specifically, we consider a short time horizon framework in which the social planner decides the policy measures to reduce the spread of a communicable disease in order to minimize the social cost associated with the epidemic management program. The short time horizon suggests that saving and capital accumulation are irrelevant, thus we simply assume that individuals entirely consume their income as follows: $c_t = y_t$, where $c_t = \frac{C_t}{N_t}$ denotes per capita consumption and $y_t = \frac{Y_t}{N_t}$ per capita income (while C_t and Y_t are aggregate consumption and income). Output is produced through a linear production function by the number of susceptibles as follows: $Q_t = S_t = N_t - I_t$, but since only a certain share of the social contacts, $1 - u_t$, is allowed to regularly occur output net of social distancing is given by: $Y_t = (1 - u_t)Q_t$, which in per capita terms reads as: $y_t = (1 - u_t)(1 - i_t)$. The effects of social distancing on health and environment are exactly as discussed before, and thus disease prevalence and pollution dynamics are given by (8) and (9), respectively.

The social cost is the weighted sum of two terms: the discounted sum ($\rho > 0$ is the time discount rate) of the instantaneous losses associated with the epidemic management program during its duration and the discounted final damage associated with the remaining level of disease prevalence and pollution at the end of the epidemic management program. The instantaneous loss function is the weighted average between two terms capturing the social loss and the environmental loss associated with the epidemic management program. The social loss is assumed to depend on the spread of the disease, the output lost due to social distancing and the lives lost due to the epidemic, $\Delta_t = \mu(1+p_t)i_t$, and to take a quadratic non-separable form as follows: $\ell_1(i_t, u_t q_t, \Delta_t) = \frac{i_t^2(1+u_t^2q_t^2+\mu^2(1+p_t)^2)}{i_t^2(1+p_t)^2}$, penalizing deviations from the disease-free status, from the no-production-loss scenario and from the no-lives-loss outcome. The environmental loss is assumed to be quadratic in the pollution stock: $\ell_2(p_t) = \frac{p_t^2}{2}$. The relative weight of the environmental loss with respect to the social loss is captured by $\omega > 0$. The final damage function is the weighted average between two terms capturing the social damage and the environmental damage. The social damage is assumed to depend on the share of infectives and the lives lost due to the epidemic at the end of the epidemic management

³The relative size of these two factors determines the magnitude of the "basic reproduction number", \mathcal{R}_0 , measuring the average number of secondary infections produced by a typical infectious individual introduced into a completely susceptible population (Hethcote, 2000; 2008).

program, and to take a quadratic non-separable form as follows: $\vartheta_1 = \frac{i_T^2[1+\mu^2(1+p_T)^2]}{2}$. The environmental damage is assumed to depend only on the amount of pollution at the end of the epidemic management program, and to take a quadratic form as follows: $\vartheta_2 = \frac{p_T^2}{2}$. The relative weight of the final damage in terms of the instantaneous losses is given by $\frac{\phi}{T} > 0$, which measures the concerns for long-run socio-environmental outcomes proxying sustainability concerns, and depends on the degree of sustainability concern, $\phi > 0$, and the final time period, T. This means that, independently of the degree of sustainability concern, the weight attached to long-run outcomes critically depends on today's distance from the long-run date: if $T \to 0$ the short and long run coincide and thus an infinitely large weight is attached to the final damage; if $T \to \infty$ the long run is infinitely far away and thus the weight attached to the final damage is null; for positive but finite values of T a positive and finite value is attached to the final damage giving rise to a clear trade off between the discounted sum of the instantaneous losses (which are minimized with $T \to 0$) and the discounted final damage (which is minimized with $T \to \infty$).

Therefore, given the initial conditions $i_0 > 0$ and $p_0 > 0$, the social planner problem reads as follows:

$$\min_{u_t} \quad \mathcal{C} = \int_0^T \left\{ \frac{i_t^2 [1 + u_t^2 (1 - i_t)^2 + \mu^2 (1 + p_t)^2]}{2} + \omega \frac{p_t^2}{2} \right\} e^{-\rho t} dt + \frac{\phi}{T} \left\{ \frac{i_T^2 [1 + \mu^2 (1 + p_T)^2]}{2} + \omega \frac{p_T^2}{2} \right\} e^{-\rho T} \right.$$

$$s.t. \quad \dot{i}_t = \alpha (1 - u_t) (1 - i_t) i_t - i_t [b + \delta + \mu (1 + p_t) (1 - i_t)], \tag{10}$$

$$\dot{p}_t = \theta \alpha (1 - u_t) (1 - i_t) i_t - [\eta + b - d - \mu (1 + p_t) i_t] p_t.$$

The problem above clearly shows the impact of social distancing on economy, health and environment. A higher policy intensity deteriorates macroeconomic conditions increasing the economic loss due to the epidemic management program, but at the same time by reducing disease incidence it improves epidemiological and environmental outcomes decreasing the infection and pollution losses. An optimal policy requires to carefully balance these conflicting needs, and while most papers in literature have focused on the macroeconomic-epidemiological trade off we will emphasize the role played by environmental factors and considerations. In particular, we will analyze how the optimal policy and health-economic-environmental outcomes depend on the degree of sustainability concern (ϕ) , the weight attached to the environmental loss (ω) and the degree of dirtiness of individual response to contagion (θ) .

3 The Optimal Policy and Dynamics

We now analyze the optimal policy and dynamics associated with problem (10). By defining the variable $\xi_t = (1 - i_t)i_t u_t$ the above model can be simplified as follows:

$$\min_{\xi_{t}} \quad \mathcal{C} = \int_{0}^{T} \left\{ \frac{i_{t}^{2} + \xi_{t}^{2} + \mu^{2} i_{t}^{2} (1 + p_{t})^{2} + \omega p_{t}^{2}}{2} \right\} e^{-\rho t} dt + \frac{\phi}{T} \left\{ \frac{i_{T}^{2} + \mu^{2} i_{T}^{2} (1 + p_{T})^{2} + \omega p_{T}^{2}}{2} \right\} e^{-\rho T}
s.t. \quad \dot{i}_{t} = \alpha (1 - i_{t}) i_{t} - \alpha \xi_{t} - i_{t} (b + \delta) - \mu i_{t} (1 + p_{t}) (1 - i_{t}),$$

$$\dot{p}_{t} = \theta \alpha (1 - i_{t}) i_{t} - \theta \alpha \xi_{t} - (\eta + b - d) p_{t} + \mu (1 + p_{t}) i_{t} p_{t}.$$
(11)

The current Hamiltonian associated with problem (11), $\mathcal{H}(i_t, \xi_t, p_t, \lambda_t, \eta_t)$, where λ_t and η_t denote the costate variables associated with the share of infectives and the pollution stock respectively, reads as:

$$\mathcal{H} = \frac{i_t^2 + \xi_t^2 + \mu^2 i_t^2 (1 + p_t)^2 + \omega p_t^2}{2} + \lambda_t \left[\alpha (1 - i_t) i_t - \alpha \xi_t - i_t (b + \delta) - \mu i_t (1 + p_t) (1 - i_t) \right] + \eta_t \left[\theta \alpha (1 - i_t) i_t - \theta \alpha \xi_t - (\eta + b - d) p_t + \mu (1 + p_t) i_t p_t \right].$$
(12)

The optimality conditions follow:

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\begin{cases} \frac{\partial H}{\partial \xi_t} = \xi_t - \alpha \lambda_t - \theta \alpha \eta_t = 0, \\ i_t = \alpha (1 - i_t) i_t - \alpha \xi_t - i_t (b + \delta) - \mu i_t (1 + p_t) (1 - i_t), \\ \dot{p}_t = \theta \alpha (1 - i_t) i_t - \theta \alpha \xi_t - (\eta + b - d) p_t + \mu (1 + p_t) i_t p_t, \\ -\dot{\lambda}_t + \rho \lambda_t = i_t + \mu^2 (1 + p_t)^2 i_t + \lambda_t \left[ \alpha (1 - 2i_t) - (b + \delta) - \mu (1 + p_t) (1 - 2i_t) \right] + \eta_t [\theta \alpha (1 - 2i_t) + \mu (1 + p_t) p_t], \\ -\dot{\eta}_t + \rho \eta_t = \mu^2 i_t^2 (1 + p_t) + \omega p_t - \lambda_t \mu i_t (1 - i_t) + \eta_t [-(\eta + b - d) + \mu i_t (1 + 2p_t)], \\ \lambda_T = \frac{\phi}{T} [\mu^2 i_T^2 (1 + p_T)^2], \\ \eta_T = \frac{\phi}{T} \left[ \mu^2 i_T^2 (1 + p_T) + \omega p_T \right], \\ i_{t=0} = i_0, \\ \eta_T = 0. \end{cases}
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By exploiting the fact that $\xi_t = \alpha \lambda_t + \theta \alpha \eta_t$ and after some simple algebra, it is possible to obtain the following system of backward-forward differential equations which determines the optimal social distancing policy:

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$$\begin{cases}
\dot{i}_{t} = -\alpha^{2} \lambda_{t} - \alpha^{2} \theta \eta_{t} - i_{t}(b+\delta) + i_{t}(1-i_{t})[\alpha - \mu(1+p_{t})], \\
\dot{p}_{t} = \theta \alpha(1-i_{t})i_{t} - \theta \alpha^{2}(\lambda_{t} + \theta \eta_{t}) - (\eta + b - d)p_{t} + \mu(1+p_{t})i_{t}p_{t}, \\
-\dot{\lambda}_{t} + \rho \lambda_{t} = i_{t} + \mu^{2}(1+p_{t})^{2}i_{t} + \lambda_{t} \left[\alpha(1-2i_{t}) - (b+\delta) - \mu(1+p_{t})(1-2i_{t})\right] + \eta_{t} \left[\theta \alpha(1-2i_{t}) + \mu(1+p_{t})p_{t}\right], \\
-\dot{\eta}_{t} + \rho \eta_{t} = \mu^{2}i_{t}^{2}(1+p_{t}) + \omega p_{t} - \lambda_{t}\mu i_{t}(1-i_{t}) + \eta_{t} \left[-(\eta + b - d) + \mu i_{t}(1+2p_{t})\right], \\
\lambda_{T} = \frac{\phi}{T}i_{T}\left[1 + \mu^{2}(1+p_{T})^{2}\right], \\
\eta_{T} = \frac{\phi}{T}\left[\mu^{2}i_{T}^{2}(1+p_{T}) + \omega p_{T}\right], \\
i_{t=0} = i_{0}, \\
p_{t=0} = p_{0}.
\end{cases}$$
(13)

Solving explicitly the above system is not possible due to high degree of nonlinearity involved, however it is possible to solve it numerically to visualize the behavior of the optimal policy and dynamics and to explore how they depend on some key parameters. We test the reliability of our numerical algorithm by comparing the numerical solution with the explicit solution obtained in a specific case of our general model (i.e., the early epidemic stage setting), showing that the two solutions almost perfectly coincide (see Appendix A for further details). In our numerical analysis we calibrate the model's parameters according to the daily data based on the Italian COVID-19 experience during the first epidemic wave (February - July 2020). We consider a weekly planning horizon by setting T=7. The birth and the death rates are determined according to demographic research as follows: b = 0.007/365 and d = 0.011/365 (World Bank, 2021). The infectivity and the recovery rates are set from Italian epidemiological studies as $\alpha = 0.1328$ and $\delta = 0.0476$, respectively (La Torre et al., 2021b). Some works show that the probability of dying from COVID-19 increases by 15% by living in areas with one extra unit of particulate matter, from which we determine $\mu = 0.15$ (Wu et al., 2020). The time preference and the pollution decay rate are set according to traditional macroeconomic and environmental economics papers, that is $\rho = 0.04/365$ and $\eta = 0.01$ (Mullingan and Sala-i-Martin, 1993; Economides and Philippopoulos, 2008). Finally, we arbitrarily set the degree of sustainability concern, the relative weight of the environmental loss in the social cost function and the environmental inefficiency of individual response to the epidemic to show how different values of these parameters may affect our results. Specifically, as a benchmark we rely on the following parametrization: $\phi = 1$, $\omega = 0.6$ and $\theta = 0.07$. We also arbitrarily set the initial conditions for the pollution stock and the level of disease prevalence to show how the optimal policy changes with different initial health and environmental conditions. In our benchmark parametrization we set: $p_0 = 0.04$ and $i_0 = 0.2$. The next figures present the results of our numerical analysis.

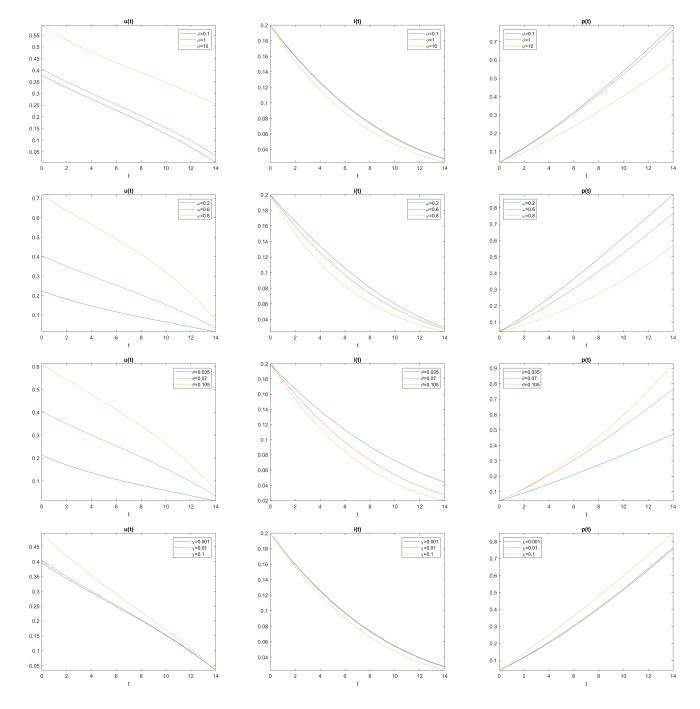


Figure 1: Evolution of social distancing (left), disease prevalence (center) and pollution (right) for different values of sustainability concerns (top), relative weights of environmental loss (mid), and environmental inefficiencies of individual response (bottom).

Figure 1 shows the dynamics of the social distancing intensity (left panels), of the share of infectives (central panels) and of the pollution stock (right panels), for different values of the degree of sustainability concern (top panels), the relative weight of the environmental loss in the social cost function (mid panels), and the environmental inefficiency of the individual response (bottom panels). In all scenarios the qualitative behavior of the variables is the same and in particular social distancing is initially high to then monotonically decrease over time and this generates a monotonic reduction in prevalence which however is not enough to reverse the pollution growth pattern, which instead monotonically increases over time. The effect of the different parameters are quite intuitive. A higher weight for long-run outcomes requires a stronger mitigation policy, which slows down disease incidence reducing both disease prevalence and pollution. Also a higher

relative importance for environmental outcomes with respect to social ones needs for a stronger policy intervention, which thus decreases prevalence and pollution. A higher dirtiness of individual response to the epidemic leads to higher pollution which thus demands for a stronger mitigation policy to limit the extra deaths due to pollution; by reducing disease prevalence a more stringent social distancing policy induces a lower incidence which tends to reduce pollution; however, this effect is not enough to compensate for its higher environmental inefficiency which instead tends to increase pollution; this latter effect dominates and thus pollution increases with the degree of dirtiness.

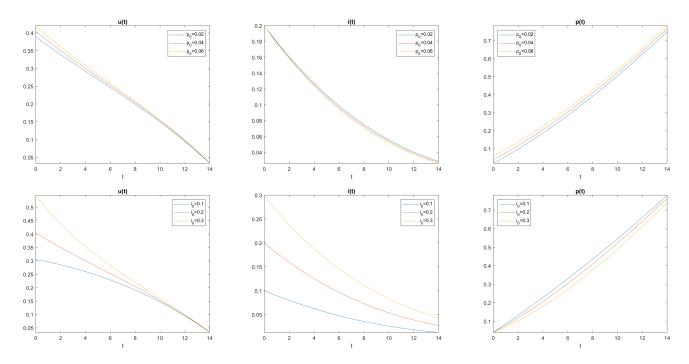


Figure 2: Evolution of social distancing (left), disease prevalence (center) and pollution (right) for different initial conditions for pollution (top), and disease prevalence (bottom).

Figure 2 shows the dynamic evolution of the variables for different initial conditions for the pollution stock (top panels) and the level of disease prevalence (bottom panels). An initially higher pollution stock requires stricter social distancing, which allows for a lower prevalence; despite the lower prevalence tends to reduce pollution, this effect is not enough to compensate for its larger initial value, thus the higher the initial pollution stock the higher the environmental degradation at any moment in time. The effects of the initial prevalence are more complicated. If prevalence is high (low) this requires stricter (softer) social distancing at the beginning of the epidemic management program which, because of its large (small) effects on incidence and thus prevalence and pollution, is then reduced by a large (small) amount towards the planning horizon; at any moment in time though the higher the initial prevalence the higher the share of infectives and pollution.

Consistent with previous works, these figures show that over a finite time horizon it is not possible to achieve disease eradication by employing social distancing measures, even if policy intervention allows for a monotonic reduction in disease prevalence (La Torre et al., 2021b). However, different from previous works which completely abstract from environmental considerations they also suggest that social distancing cannot be used to reduce the side effects generated by the epidemic on the environment. Indeed, social distancing alone is not enough to reverse the growth pattern of both disease prevalence and pollution. Despite social distancing reduces disease incidence and thus can be effectively used to improve both epidemiological and environmental outcomes, our results surprisingly suggest that it is not optimal to do so but rather it is convenient to rely on social distancing to contain the disease spread reducing its prevalence at the cost of tolerating a higher level of pollution. Therefore, in order to properly managing the pollution problem

another policy instrument (i.e., taxes to finance abatement) is needed. This suggests that the strong emphasis that has been placed on epidemic management during the ongoing COVID-19 pandemic, in which environmental issues have been to a large extent neglected from policy considerations, is likely to leave us with a high environmental bill which by deteriorating environmental and climatic conditions will require massive interventions in the near future in order to promote long-run sustainability.

4 The Model with Strategic Interactions

We now extend our baseline model to allow for strategic interactions between economies in order to understand how free-riding opportunities may affect the optimal social distancing policy. Several works have analyzed the role of strategic interactions in determining the relation between the spread of COVID-19 and macroeconomic outcomes (Cui et al., 2020; Bouveret and Mandel, 2021; La Torre et al., 2021a), but none has thus far considered the role played by environmental considerations. All these works discuss how the externality generated by disease dynamics affects the choice of single players while encompassing also environmental dynamics requires to account also for the presence of a pollution externality, thus different from extant literature in our setting both disease prevalence and pollution are transboundary and we wish to characterize how such transboundary features affect the single economy's policy intensity and the joint health-economy-environment outcome.

Specifically, we consider two neighbor economies (i.e., two regions) indexed by j = 1, 2 in the absence of interregional movement restrictions, and we focus on the non-cooperative equilibrium in which each region takes its own decision regarding social distancing. Therefore, each region decides independently its social distancing intensity $0 < u_{jt} < 1$. In the absence of restrictions on interregional movements, the disease can spread freely between regions which thus share the same level of disease prevalence, along with the same pollution stock. Individual region's social distancing choice partly contributes to reduce disease incidence, which thus ultimately depends on the average of the social distancing policy between the two regions, which drives also emissions determining thus the evolution of pollution. It follows that the disease and pollution dynamics, common to both regions, is given by the following equations:

$$\dot{i}_t = \alpha \left(1 - \frac{u_{1t} + u_{2t}}{2} \right) (1 - i_t) i_t - i_t [b + \delta + \mu (1 + p_t)(1 - i_t)], \tag{14}$$

$$\dot{p}_t = \theta \alpha \left(1 - \frac{u_{1t} + u_{2t}}{2} \right) (1 - i_t) i_t - [\eta + b - d - \mu (1 + p_t) i_t] p_t. \tag{15}$$

Therefore, the epidemic management problem in region j can be summarized as follows:

$$\min_{u_{jt}} \quad \mathcal{C} = \int_{0}^{T} \left\{ \frac{i_{t}^{2} [1 + u_{jt}^{2} (1 - i_{t})^{2} + \mu^{2} (1 + p_{t})^{2}]}{2} + \omega \frac{p_{t}^{2}}{2} \right\} e^{-\rho t} dt + \frac{\phi}{T} \left\{ \frac{i_{T}^{2} [1 + \mu^{2} (1 + p_{T})^{2}]}{2} + \omega \frac{p_{T}^{2}}{2} \right\} e^{-\rho T} \right. \\
s.t. \quad \dot{i}_{t} = \alpha \left(1 - \frac{u_{1t} + u_{2t}}{2} \right) (1 - i_{t}) i_{t} - i_{t} [b + \delta + \mu (1 + p_{t}) (1 - i_{t})], \qquad (16)$$

$$\dot{p}_{t} = \theta \alpha \left(1 - \frac{u_{1t} + u_{2t}}{2} \right) (1 - i_{t}) i_{t} - [\eta + b - d - \mu (1 + p_{t}) i_{t}] p_{t}.$$

Consistent with the recent COVID-19 experience in which policymakers have announced which level of social distancing would be implemented for a certain short period of time (i.e., usually one or two weeks), we assume that regions determine the policy intensity at the beginning of the planning horizon and commit to such a level for the entire duration of the epidemic management program. Therefore, we characterize the open-loop equilibrium outcome in which the individual region's optimal policy depends only upon time. As in the previous section, by defining the variable $\xi_{jt} = (1 - i_t)i_tu_{jt}$ the region j's optimization problem can

be recast as:

$$\min_{\xi_{jt}} \quad \mathcal{C} = \int_{0}^{T} \left\{ \frac{i_{t}^{2} + \xi_{jt}^{2} + \mu^{2} i_{t}^{2} (1 + p_{t})^{2} + \omega p_{t}^{2}}{2} \right\} e^{-\rho t} dt + \frac{\phi}{T} \left\{ \frac{i_{T}^{2} [1 + \mu^{2} (1 + p_{T})^{2}]}{2} + \omega \frac{p_{T}^{2}}{2} \right\} e^{-\rho T} \right.$$

$$s.t. \quad \dot{i}_{t} = \alpha \left((1 - i_{t}) i_{t} - \frac{\xi_{1t} + \xi_{2t}}{2} \right) - i_{t} [b + \delta + \mu (1 - i_{t}) (1 + p_{t})], \qquad (17)$$

$$\dot{p}_{t} = \theta \alpha \left((1 - i_{t}) i_{t} - \frac{\xi_{1t} + \xi_{2t}}{2} \right) - [\eta + b - d - \mu (1 + p_{t}) i_{t}] p_{t}.$$

The current Hamiltonian associated with region j's problem $\mathcal{H}_j(i_t, \xi_{1t}, \xi_{2t}, p_t, \lambda_{jt}, \eta_{jt})$, in which λ_{jt} and η_{jt} denote region j's costate variable associated with the share of infectives and the pollution stock respectively, follows:

$$\mathcal{H}_{j}(i_{t}, \xi_{1t}, \xi_{2t}, p_{t}, \lambda_{jt}, \eta_{jt}) = \frac{i_{t}^{2} + \xi_{jt}^{2} + \mu^{2} i_{t}^{2} (1 + p_{t})^{2} + \omega p_{t}^{2}}{2} + \lambda_{jt} \left(\alpha \left((1 - i_{t}) i_{t} - \frac{\xi_{1t} + \xi_{2t}}{2} \right) - i_{t} [b + \delta + \mu (1 - i_{t}) (1 + p_{t})] \right) + \eta_{jt} \left(\theta \alpha \left((1 - i_{t}) i_{t} - \frac{\xi_{1t} + \xi_{2t}}{2} \right) - [\eta + b - d - \mu (1 + p_{t}) i_{t}] p_{t} \right).$$

The optimality conditions for j = 1, 2 read as follows:

$$\begin{cases} \frac{\partial \mathcal{H}_{j}}{\partial \xi_{jt}} = \xi_{jt} - \frac{\alpha}{2}\lambda_{jt} - \frac{\alpha}{2}\theta\eta_{jt} = 0, \\ \dot{i}_{t} = \alpha\left((1-i_{t})i_{t} - \frac{\xi_{1t}+\xi_{2t}}{2}\right) - i_{t}[b+\delta + \mu(1-i_{t})(1+p_{t})], \\ \dot{p}_{t} = \theta\alpha\left((1-i_{t})i_{t} - \frac{\xi_{1t}+\xi_{2t}}{2}\right) - (\eta+b-d-\mu(1+p_{t})i_{t})p_{t}, \\ -\dot{\lambda}_{jt} + \rho\lambda_{jt} = i_{t} + (1+p_{t})^{2}\mu^{2}i_{t} + \lambda_{jt}((\alpha-(1+p_{t})\mu)(1-2i_{t}) - (b+\delta)) + \eta_{jt}\theta\alpha(1-2i_{t}) + \eta_{jt}\mu p_{t}(1+p_{t}), \\ -\dot{\eta}_{jt} + \rho\eta_{jt} = \mu^{2}i_{t}^{2}(1+p_{t}) + \omega p_{t} - \lambda_{jt}\mu i_{t}(1-i_{t}) - (\eta+b-d-\mu(1+2p_{t})i_{t})\eta_{jt}, \\ \lambda_{jT} = \frac{\phi}{T}i_{T}[1+\mu^{2}(1+p_{T})^{2}], \\ \eta_{jT} = \frac{\phi}{T}\left[\mu^{2}i_{T}^{2}(1+p_{T}) + \omega p_{T}\right], \\ i_{t=0} = i_{0}, \\ p_{t=0} = p_{0}. \end{cases}$$

Since the dynamic game is perfectly symmetric we characterize a symmetric open-loop Nash equilibrium in which $\xi_{jt} = \xi_t$, $\lambda_{jt} = \lambda_t$ and $\eta_{jt} = \eta_t$ for j = 1, 2. Taking into account that $\xi_{jt} = \frac{\alpha}{2}(\lambda_{jt} + \theta \eta_{jt})$, after some simple computations, it is possible to derive the following system of backward-forward differential equations which describes the symmetric open-loop Nash equilibrium of the dynamic game:

$$\begin{cases} \dot{i}_t = -\frac{\alpha^2}{2}\lambda_t - \frac{\alpha^2}{2}\theta\eta_t - i_t(b+\delta) + i_t(1-i_t)(\alpha - \mu(1+p_t)), \\ \dot{p}_t = \theta\alpha(1-i_t)i_t - \theta\frac{\alpha^2}{2}\left(\lambda_t + \theta\eta_t\right) - (\eta + b - d)p_t + \mu(1+p_t)i_tp_t, \\ -\dot{\lambda}_t + \rho\lambda_t = i_t + \mu^2(1+p_t)^2i_t + \lambda_t\left((\alpha - \mu(1+p_t))(1-2i_t) - (b+\delta)\right) + \eta_t(\theta\alpha(1-2i_t) + \mu(1+p_t)p_t), \\ -\dot{\eta}_t + \rho\eta_t = \mu^2i_t^2(1+p_t) + \omega p_t - \lambda_t\mu i_t(1-i_t) + \eta_t(-(\eta + b - d) + \mu(1+2p_t)i_t), \\ \lambda_T = \frac{\phi}{T}i_T[1+\mu^2(1+p_T)^2], \\ \eta_T = \frac{\phi}{T}\left[\mu^2i_T^2(1+p_T) + \omega p_T\right], \\ i_{t=0} = i_0, \\ p_{t=0} = p_0. \end{cases}$$

By relying on the same parameter values employed in the previous section we numerically solve the above nonlinear boundary value problem. Figure 3 compares the dynamic evolution of the main variables for our baseline model with no strategic interactions (solid curve) and for the extended model with strategic interactions (dashed curve) in the benchmark parameter configuration. Intuitively, because of free-riding effect social distancing is lower and thus both disease prevalence and pollution are higher in the game than in baseline framework. Apart from the quantitative effects induced by free-riding opportunities, in both setups the variables present the same qualitative behavior. Comparing the two models for different values of the degree of sustainability concern (ϕ) , the relative weight of the environmental loss in the social cost function (ω) , the environmental inefficiencies of individual response to the epidemic (θ) , as well as for different initial conditions for the pollution stock (p_0) and the level of disease prevalence (i_0) , leads to qualitatively the same conclusions as those illustrated in Figure 3.

In order to assess the inefficiency induced by free-riding, the following tables quantify the outcome differences (in terms of social distancing intensity, disease prevalence and pollution) between the game and the baseline frameworks at the end of the weekly planning horizon measured as a percentage with respect to the baseline model. Table 2 focuses on how the results change for different parameter values, while Table 3 on the effects of different initial conditions.

	$\phi = 0.1$	$\phi = 1$	$\phi = 10$	$\omega = 0.2$	$\omega = 0.6$	$\omega = 1.8$
Δu_T	-34.40	-43.07	-38.20	-44.07	-43.07	-37.83
Δi_T	4.58	5.55	11.62	2.38	5.55	10.04
Δp_T	9.56	11.05	22.99	4.69	11.05	23.08

Table 1: Inefficiency induced by free-riding for different values of ϕ and ω .

	$\theta = 0.035$	$\theta = 0.07$	$\theta = 0.105$	$\chi = 0.001$	$\chi = 0.01$	$\chi = 0.1$
Δu_T	-38.43	-43.07	-40.03	-43.07	-43.07	-42.90
Δi_T	4.26	5.55	1.30	5.55	5.55	5.38
Δp_T	3.94	11.05	19.35	10.91	11.05	12.41

Table 2: Inefficiency induced by free-riding for different values of θ and χ .

	$i_0 = 0.1$	$i_0 = 0.2$	$i_0 = 0.4$	$p_0 = 0.02$	$p_0 = 0.04$	$p_0 = 0.06$
Δu_T	-44.61	-43.07	-40.65	-42.98	-43.07	-43.15
Δi_T	5.63	5.55	5.43	5.44	5.55	5.65
Δp_T	10.11	11.05	11.81	10.92	11.05	11.18

Table 3: Inefficiency induced by free-riding for different initial conditions.

Overall, free-riding generates sizeable efficiency losses in term of the final prevalence and pollution levels, increasing them by about 15% and 5% respectively. The impact of most parameters on the size of inefficiency is quite limited, apart from the effects of the degree of sustainability concern which generates the largest effects on the estimates of the final prevalence level. These results suggest that allowing individual economies to independently determine the intensity of disease mitigation policies is not an effective approach to reduce final prevalence and pollution levels. This also confirms what stated in extant literature regarding the importance of promote coordination across different economies in order to reduce the losses induced by free-riding (Barrett, 2003; La Torre et al., 2022).

5 Conclusion

The ongoing COVID-19 pandemic has shown more clearly than ever that economy, environment and health are mutually related and that exogenous epidemic shocks may affect them all at once. Despite a growing body

of the literature analyzes the nature of the trade off between epidemiological and macroeconomic outcomes involved in disease containment policies, very little has been done to explore the role of environmental factors on optimal mitigation policies. However, this is particularly important since the regulations and recommendations implemented to contain the spread of COVID-19 have led individuals to modify their behavior generating an important increase in emissions and waste deteriorating environmental conditions, and augmented pollution substantially contributes to increase the probability of severe health consequences (including death) following an infection from COVID-19. We thus analyze the extent to which environmental considerations may affect the design of optimal disease containment policy, in the form of social distancing, which by reducing disease incidence allows to decrease emissions and prevalence eventually improving health and environmental outcomes. In particular, we develop a pollution-extended macroeconomic epidemiological model with bilateral health-environment feedback effects through emissions and mortality. By focusing on a calibration based on the Italian COVID-19 experience during the first epidemic wave, we characterize how the optimal social distancing policy depends on the main environmental factors, showing that social distancing alone is not enough to reverse the growth pattern of both disease prevalence and pollution. Indeed, the optimal policy allows for a reduction of disease prevalence only at a cost of a deterioration in environmental outcomes, suggesting that placing too much emphasis on epidemic management as done in the policy arena risks to leave us with a high environmental bill which will require massive efforts in the near future to improve environmental conditions in order to achieve long-run sustainability. We also extend our baseline model to account for the role of strategic interactions between two-neighbor economies in which both pollution and disease prevalence are transboundary. In this context we show that free-riding induces important efficiency losses, quantifiable in about 15% excess disease prevalence and 5% excess pollution at the end of the epidemic management program. This suggests that policy coordination is essential in order to effectively mitigate the consequences of infectious diseases.

To the best of our knowledge, ours is the first paper exploring how environmental factors may affect the intensity of disease containment policies, thus we have considered a simple and intuitive framework to make our arguments as clear as possible. However, this has precluded us from the possibility to consider some important aspects of the problem. Apart from its effects on mortality, by driving climate change pollution may also affect the likelihood of an epidemic outbreak which is likely to increase the relevance of environmental considerations in determining the optimal policy intensity (Brock and Xepapadeas, 2020). Moreover, a very short term effect of the mitigation policies against COVID-19 has consisted of a dramatic reduction in emissions due to their disruptive consequences on economic activities, which thus by decreasing mortality and the likelihood of a disease outbreak may act as a counteracting force in the determination of the optimal policy (Venter et al., 2020). It would thus be interesting to extend our analysis in order to enrich the nature of the feedback health-environment effects and analyze their implications on epidemiological, environmental and macroeconomic outcomes. This is left for future research.

A The Early Epidemic Stage

In this section we present a special case of our model which allows for a closed-form solution of the optimal policy and the optimal dynamics of the infectives share and pollution stock. Such explicit expressions allow us to test the reliability of the numerical approach that we have implemented in order to derive the optimal policy in our general setup. Specifically, we consider an early epidemic stage in which following the initial outbreak the disease spreads freely among the population and thus the number of infectives grow at a constant rate such that the evolution of the disease can be described through an exponential growth dynamics (Chowell et al., 2016; Ma, 2020). This setup can be formally derived via a linear approximation of the epidemic dynamics around the disease-free equilibrium (La Torre et al., 2021b), which thus requires to account also for the pollution dynamics.

A.1 The Baseline Model

In our baseline model without strategic interactions between neighbor economies, by approximating the epidemic and environmental dynamics in the neighborhood of the disease-free equilibrium, we get:

$$\dot{i}_t = \alpha (1 - u_t)(1 - i_t)i_t - i_t[b + \delta + \mu(1 + p_t)(1 - i_t)] = F(i_t, p_t),
\dot{p}_t = \theta \alpha (1 - u_t)(1 - i_t)i_t - [\eta + b - d - \mu(1 + p_t)i_t]p_t = G(i_t, p_t),$$
(18)

and, therefore

$$\frac{\partial F}{\partial i_t} = \alpha (1 - u_t)(1 - 2i_t) - (b + \delta) - \mu (1 + p_t)(1 - 2i_t),$$

$$\frac{\partial F}{\partial p_t} = -i_t \mu (1 - i_t),$$

$$\frac{\partial G}{\partial i_t} = \theta \alpha (1 - u_t)(1 - 2i_t) + \mu (1 + p_t)p_t,$$

$$\frac{\partial G}{\partial p_t} = -(\eta + b - d) + \mu i_t (1 + 2p_t),$$
(19)

and then the linearized system in the neighborhood of the disease-free equilibrium reads as:

$$\dot{i}_t = \frac{\partial F}{\partial i_t}(0,0)i_t + \frac{\partial F}{\partial p_t}(0,0)p_t = \alpha(1-u_t)i_t - i_t(b+\delta+\mu),
\dot{p}_t = \frac{\partial G}{\partial i_t}(0,0)i_t + \frac{\partial G}{\partial p_t}(0,0)p_t = \theta\alpha(1-u_t)i_t - (\eta+b-d)p_t.$$
(20)

Under the assumption that $s_t = (1 - i_t) \simeq 1$ and $1 + p_t \simeq 1$, the problem (10) boils down to the following:

$$\min_{u_t} \quad \mathcal{C} = \int_0^T \left\{ \frac{i_t^2 [1 + u_t^2 + \mu^2]}{2} + \omega \frac{p_t^2}{2} \right\} e^{-\rho t} dt + \frac{\phi}{2T} [i_T^2 (1 + \mu^2) + \omega p_T^2] e^{-\rho T},$$

$$s.t. \quad \dot{i}_t = \alpha (1 - u_t) i_t - i_t (b + \delta + \mu)$$

$$\dot{p}_t = \theta \alpha (1 - u_t) i_t - (\eta + b - d) p_t.$$
(21)

Similar to what we have done in our general framework, through the substitution $\xi_t = u_t i_t$ the model can be rewritten as:

$$\min_{\xi_t} \quad \mathcal{C} = \int_0^T \left\{ \frac{i_t^2 (1 + \mu^2) + \xi_t^2}{2} + \omega \frac{p_t^2}{2} \right\} e^{-\rho t} dt + \frac{\phi}{2T} [i_T^2 (1 + \mu^2) + \omega p_T^2] e^{-\rho T}
s.t. \quad \dot{i}_t = \alpha (i_t - \xi_t) - i_t (b + \delta + \mu),
\dot{p}_t = \theta \alpha (i_t - \xi_t) - (\eta + b - d) p_t.$$
(22)

Since the problem above is characterized by a linear-quadratic structure it is possible to solve in closed-form (La Torre et al., 2021b). The current Hamiltonian associated with the problem above reads as:

$$\mathcal{H} = \frac{(1+\mu^2)i_t^2 + \xi_t^2}{2} + \omega \frac{p_t^2}{2} + \lambda_t \left[\alpha i_t - \alpha \xi_t - i_t (b+\delta + \mu)\right] + \eta_t \left[\theta \alpha (i_t - \xi_t) - (\eta + b - d)p_t\right],\tag{23}$$

and the optimality conditions as:

$$\begin{cases}
0 = \xi_t - \alpha \lambda_t - \theta \alpha \eta_t, \\
\dot{i}_t = \alpha i_t - \alpha \xi_t - i_t (b + \delta + \mu), \\
\dot{p}_t = \theta \alpha (i_t - \xi_t) - (\eta + b - d) p_t, \\
-\dot{\lambda}_t + \rho \lambda_t = (1 + \mu^2) i_t + \lambda_t (\alpha - b - \delta - \mu) + \eta_t \theta \alpha, \\
-\dot{\eta}_t + \rho \eta_t = \omega p_t - (\eta + b - d) \eta_t, \\
\lambda_T = \frac{\phi}{T} i_T (1 + \mu^2), \\
\eta_T = \frac{\phi}{T} \omega p_T, \\
\dot{i}_{t=0} = i_0, \\
p_{t=0} = p_0.
\end{cases}$$
(24)

After some algebra it is possible to obtain the following system of backward-forward differential equations characterizing the optimal social distancing policy:

social distancing policy:
$$\begin{cases} \dot{i}_t = (\alpha - b - \delta - \mu)i_t - \alpha^2 \lambda_t - \alpha^2 \theta \eta_t, \\ \dot{p}_t = \theta \alpha i_t - (\eta + b - d)p_t - \alpha^2 \theta \lambda_t - \alpha^2 \theta^2 \eta_t, \\ \dot{\lambda}_t = -(1 + \mu^2)i_t - \lambda_t (\alpha - b - \delta - \mu - \rho) - \eta_t \theta \alpha, \\ \dot{\eta}_t = -\omega p_t + (\eta + b - d + \rho)\eta_t, \\ \lambda_T = \frac{\phi}{T}i_T(1 + \mu^2), \\ \eta_T = \frac{\phi}{T}\omega p_T, \\ i_{t=0} = i_0, \\ p_{t=0} = p_0. \end{cases}$$
 (25)

which characterizes the system of backward-forward differential equations determining the optimal social distancing policy. By defining the following vector Ξ and matrix Θ :

$$\Xi_{t} = \begin{bmatrix} i_{t} \\ p_{t} \\ \lambda_{t} \\ \eta_{t} \end{bmatrix}, \qquad \Theta = \begin{bmatrix} (\alpha - b - \delta - \mu) & 0 & -\alpha^{2} & -\alpha^{2}\theta \\ \theta \alpha & -(\eta + b - d) & -\theta \alpha^{2} & -\alpha^{2}\theta^{2} \\ -(1 + \mu^{2}) & 0 & -(\alpha - b - \delta - \mu - \rho) & -\theta \alpha \\ 0 & -\omega & 0 & \eta + b - d + \rho \end{bmatrix}$$
(26)

then the optimality conditions can be written in compact form as follows:

$$\dot{\Xi}_t = \Theta \Xi_t, \tag{27}$$

whose closed-form solution follows:

$$\Xi_t = Ce^{\Theta t}. (28)$$

In order to simplify this expression, let us define

$$\nu := \alpha - b - \delta - \mu,$$

and

$$\chi := \rho^2 + \left(-2\,\nu + 2\,(\eta + b - d) \right) \rho + 2\,(\eta + b - d)^2 + \left(2\,\mu + 2 \right) \alpha^2 + 2\,\omega\,\sigma^2 + 2\,\nu^2,$$

where

$$\sigma := \alpha \theta$$
,

and

$$\kappa := (1 + \mu)^2 \alpha^4 + \left(-2 \left(\nu + (\eta + b - d) \right) (1 + \mu) \rho + (2 \mu + 2) \nu^2 + (-2 \mu - 2) (\eta + b - d)^2 + 2 \sigma^2 (-1 + \mu) \omega \right) \alpha^2 + 8 \sigma^2 \omega \left(\nu - \rho/2 \right) \alpha + \left((-\nu - (\eta + b - d)) \rho - \omega \sigma^2 + \nu^2 - (\eta + b - d)^2 \right)^2.$$

Since the matrix Θ has four different eigenvalues, given by $\tau_1 := \frac{\rho}{2} + \frac{1}{2}\sqrt{\chi + 2\sqrt{\kappa}}$, $\tau_2 := \frac{\rho}{2} - \frac{1}{2}\sqrt{\chi + 2\sqrt{\kappa}}$, $\tau_3 := \frac{\rho}{2} + \frac{1}{2}\sqrt{\chi - 2\sqrt{\kappa}}$ and $\tau_4 := \frac{\rho}{2} - \frac{1}{2}\sqrt{\chi - 2\sqrt{\kappa}}$ respectively, the corresponding eigenvectors are v_1, v_2, v_3 and v_4 . The solution (28) can be then written as:

$$\Xi_t = Ce^{\Theta t} = C_1 v_1 e^{\tau_1 t} + C_2 v_2 e^{\tau_2 t} + C_3 v_3 e^{\tau_3 t} + C_4 v_4 e^{\tau_4 t}. \tag{29}$$

Despite the expression above is particularly cumbersome and thus does not allow to perform any sort of comparative statics, it is nevertheless useful to test the validity of our numerical algorithm. Figure 4 compares the evolution of the main variables as derived from the above closed-form expressions (top panels) and from our numerical approach (bottom panels), by relying on the parameter values we employed in our earlier benchmark parametrization, with the only exception that the final time horizon has been reduced (i.e., T=1) in order to better visualize the eventual differences. We can see that the two solutions display the same qualitative behavior, and also from a quantitative point of view their differences are very small. This suggests that even in the absence of analytical results and despite the high model nonlinearity, our numerical approach can be reliably used to determine and quantify the optimal intensity of the social distancing policy.

A.2 The Model with Strategic Interactions

In our extended model with strategic interactions between neighbor economies, by approximating the epidemic and environmental dynamics, given by (14) and (15), in the neighborhood of the disease-free equilibrium, and under the assumption that $s_t = (1 - i_t) \simeq 1$, $1 + p_t \simeq 1$, we get the following problem:

$$\min_{u_{jt}} \quad \mathcal{C} = \int_{0}^{T} \left\{ \frac{i_{t}^{2} [1 + u_{jt}^{2} + \mu^{2}]}{2} + \omega \frac{p_{t}^{2}}{2} \right\} e^{-\rho t} dt + \frac{\phi}{2T} [i_{T}^{2} (1 + \mu^{2}) + \omega p_{T}^{2}] e^{-\rho T}
s.t. \quad \dot{i}_{t} = \alpha \left(1 - \frac{u_{1t} + u_{2t}}{2} \right) i_{t} - i_{t} [b + \delta + \mu],$$

$$\dot{p}_{t} = \theta \alpha \left(1 - \frac{u_{1t} + u_{2t}}{2} \right) i_{t} - (\eta + b - d) p_{t}.$$
(30)

By defining the variable $\xi_{jt} = u_{jt}i_t$, the model above can be rewritten as:

$$\min_{\xi_{jt}} \quad \mathcal{C} = \int_{0}^{T} \left\{ \frac{(1+\mu^{2})i_{t}^{2} + \xi_{jt}^{2}}{2} + \omega \frac{p_{t}^{2}}{2} \right\} e^{-\rho t} dt + \frac{\phi}{2T} [i_{T}^{2} (1+\mu^{2}) + \omega p_{T}^{2}] e^{-\rho T}
s.t. \quad \dot{i}_{t} = \alpha \left(i_{t} - \frac{\xi_{1t} + \xi_{2t}}{2} \right) - i_{t} (b + \delta + \mu),$$

$$\dot{p}_{t} = \theta \alpha \left(i_{t} - \frac{\xi_{1t} + \xi_{2t}}{2} \right) - (\eta + b - d) p_{t}.$$
(31)

We now look for an open-loop Nash equilibrium of the dynamic game. The current Hamiltonian associated with region j's problem reads as:

$$\mathcal{H}_{j}(i_{t}, \xi_{1t}, \xi_{2t}, p_{t}, \lambda_{jt}, \eta_{jt}) = \frac{(1 + \mu^{2})i_{t}^{2} + \xi_{jt}^{2}}{2} + \omega \frac{p_{t}^{2}}{2} + \lambda_{jt} \left(\alpha \left(i_{t} - \frac{\xi_{1t} + \xi_{2t}}{2}\right) - i_{t}(b + \delta + \mu)\right) + \eta_{jt} \left(\theta \alpha \left(i_{t} - \frac{\xi_{1t} + \xi_{2t}}{2}\right) - (\eta + b - d)p_{t}\right).$$

The optimality conditions for j = 1, 2 follow:

$$\begin{cases} \frac{\partial \mathcal{H}_{j}}{\partial \xi_{jt}} = \xi_{jt} - \frac{\alpha}{2} \lambda_{jt} - \frac{\alpha}{2} \theta \eta_{jt} = 0, \\ \dot{i}_{t} = \alpha \left(i_{t} - \frac{\xi_{1t} + \xi_{2t}}{2} \right) - i_{t} (b + \delta + \mu), \\ \dot{p}_{t} = \theta \alpha \left(i_{t} - \frac{\xi_{1t} + \xi_{2t}}{2} \right) - (\eta + b - d) p_{t}, \\ -\dot{\lambda}_{jt} + \rho \lambda_{jt} = (1 + \mu^{2}) i_{t} + \lambda_{jt} (\alpha - b - \delta - \mu) + \eta_{jt} \theta \alpha, \\ -\dot{\eta}_{jt} + \rho \eta_{jt} = \omega p_{t} - (\eta + b - d) \eta_{jt}, \\ \lambda_{jT} = \frac{\phi}{T} (1 + \mu^{2}) i_{T}, \\ \eta_{jT} = \frac{\phi}{T} \omega p_{T}, \\ i_{t=0} = i_{0}, \\ p_{t=0} = p_{0}. \end{cases}$$

From the first equation it follows that $\xi_{jt} = \frac{\alpha}{2}(\lambda_{jt} + \theta \eta_{jt})$, which substituted in the second and third equations leads us to the following two-boundary problems, a system of six linear first-order differential equations, that must be satisfied by any open-loop Nash equilibrium of the dynamic game:

$$\begin{cases} \dot{i}_t = (\alpha - b - \delta - \mu)i_t - \frac{\alpha^2}{4} \left(\lambda_{1t} + \lambda_{2t} + \theta(\eta_{1t} + \eta_{2t})\right), \\ \dot{p}_t = \alpha\theta \left(i_t - \alpha \frac{\lambda_{1t} + \lambda_{2t}}{4} - \alpha\theta \frac{\eta_{1t} + \eta_{2t}}{4}\right) - (\eta + b - d)p_t, \\ \dot{\lambda}_{jt} = -(1 + \mu^2)i_t - \lambda_{jt}(\alpha - b - \delta - \mu - \rho) - \eta_{jt}\theta\alpha, \quad j = 1, 2, \\ \dot{\eta}_{jt} = -\omega p_t + (\eta + b - d + \rho)\eta_{jt}, \quad j = 1, 2, \\ \lambda_{jT} = \frac{\phi}{T}(1 + \mu^2)i_T, \quad j = 1, 2, \\ \eta_{jT} = \frac{\phi}{T}\omega p_T, \quad j = 1, 2, \\ i_{t=0} = i_0, \\ p_{t=0} = p_0. \end{cases}$$

Because the dynamic game is completely symmetric, we focus on a symmetric open-loop Nash equilibrium, where $\xi_{1t} = \xi_{2t} = \xi_t$, $\lambda_{1t} = \lambda_{2t} = \lambda_t$, $\eta_{1t} = \eta_{2t} = \eta_t$. Any symmetric open-loop Nash equilibrium of the dynamic game satisfies:

$$\begin{cases} \dot{i}_t = (\alpha - b - \delta - \mu)i_t - \frac{\alpha^2}{2}\lambda_t - \frac{\alpha^2}{2}\theta\eta_t, \\ \dot{p}_t = \theta\alpha i_t - (\eta + b - d)p_t - \frac{\alpha^2}{2}\theta\lambda_t - \frac{\alpha^2}{2}\theta^2\eta_t, \\ \dot{\lambda}_t = -(1 + \mu^2)i_t - \lambda_t(\alpha - b - \delta - \mu - \rho) - \eta_t\theta\alpha, \\ \dot{\eta}_t = -\omega p_t + (\eta + b - d + \rho)\eta_t, \\ \lambda_T = \frac{\phi}{T}(1 + \mu^2)i_T, \\ \eta_T = \frac{\phi}{T}\omega p_T, \\ [[]i_{t=0} = i_0, \\ p_{t=0} = p_0. \end{cases}$$

In matrix form the above system reads as:

$$\dot{\Xi}_t = \Theta \Xi_t, \tag{32}$$

where:

$$\Xi_{t} = \begin{bmatrix} i_{t} \\ p_{t} \\ \lambda_{t} \\ \eta_{t} \end{bmatrix}, \qquad \Theta = \begin{bmatrix} \alpha - b - \delta - \mu & 0 & -\frac{\alpha^{2}}{2} & -\frac{\alpha^{2}}{2}\theta \\ \alpha\theta & -(\eta + b - d) & -\frac{\alpha^{2}}{2}\theta & -\frac{\alpha^{2}}{2}\theta^{2} \\ -(1 + \mu^{2}) & 0 & -(\alpha - b - \delta - \mu - \rho) & -\theta\alpha \\ 0 & -\omega & 0 & \eta + b - d + \rho \end{bmatrix}.$$
(33)

As in the previous section, by defining $\nu = \alpha - b - \delta - \mu$ and $\sigma = \alpha \theta$, the closed-form solution of system (32) is given by:

$$\Xi_t = Ce^{\Theta t} = C_1 v_1 e^{\tau_1 t} + C_2 v_2 e^{\tau_2 t} + C_3 v_3 e^{\tau_3 t} + C_4 v_4 e^{\tau_4 t}, \tag{34}$$

with v_i , i = 1, ..., 4 the eigenvectors associated to the eigenvalues τ_i , i = 1, ..., 4 of matrix Θ and C_i , i = 1, ..., 4 constants to be determined from the boundary conditions. The eigenvalues of Θ are given by:

$$\tau_1 = \frac{\rho}{2} + \frac{1}{2}\sqrt{\chi + 2\sqrt{\kappa}}, \quad \tau_2 = \frac{\rho}{2} - \frac{1}{2}\sqrt{\chi + 2\sqrt{\kappa}}, \quad \tau_3 = \frac{\rho}{2} + \frac{1}{2}\sqrt{\chi - 2\sqrt{\kappa}}, \quad \tau_4 = \frac{\rho}{2} - \frac{1}{2}\sqrt{\chi - 2\sqrt{\kappa}},$$

where:

$$\chi := \left(\frac{\alpha}{2} - (\eta + b - d) + \nu - \rho\right)^2 + (\eta + b - d + \nu + \frac{\alpha}{2})^2 + \alpha^2(1 + \mu^2) + 4\sigma^2\omega,$$

and:

$$\kappa := \left(\frac{\alpha^2}{4}(3+2\mu^2) + \frac{\alpha}{2}(2\nu - \rho) - (\eta + b - d + \nu)(\eta + b - d - \nu + \rho) - 2\sigma^2\omega\right)^2 + 8\alpha(\frac{\alpha}{2}(1+\mu^2) + 2\nu - \rho)\sigma^2\omega.$$

As for our baseline model, the above analytical solution allows us to test the reliability of our numerical analysis. Figure 5 compares the evolution of the main variables as derived from the above closed-form expressions (top panels) and from our numerical approach (bottom panels), by relying on the parameter values earlier employed. As for the baseline model, we can observe that the two solutions display the same qualitative behavior, and also from a quantitative point of view their differences are very small, suggesting that our numerical approach works quite well in determining and quantifying the optimal intensity of the social distancing policy.

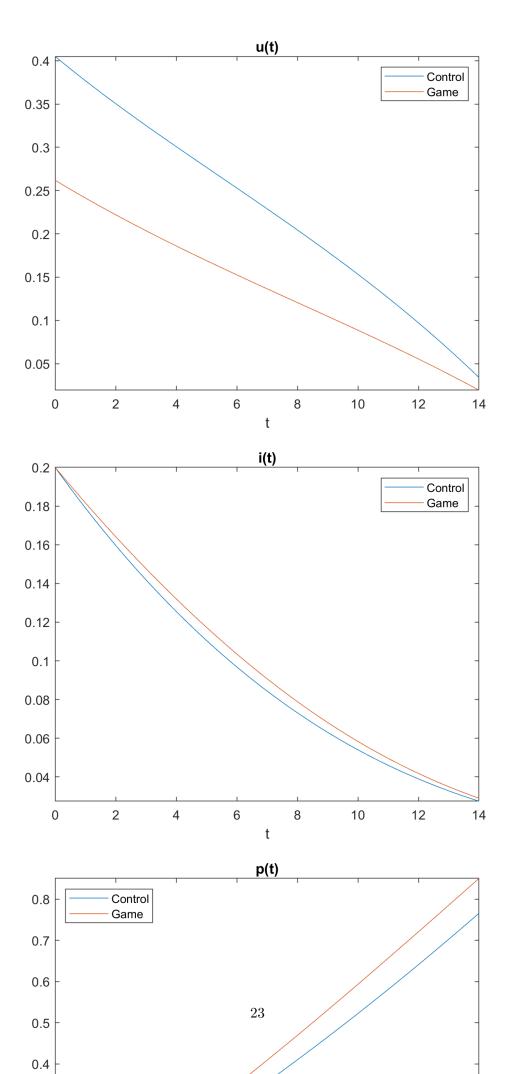
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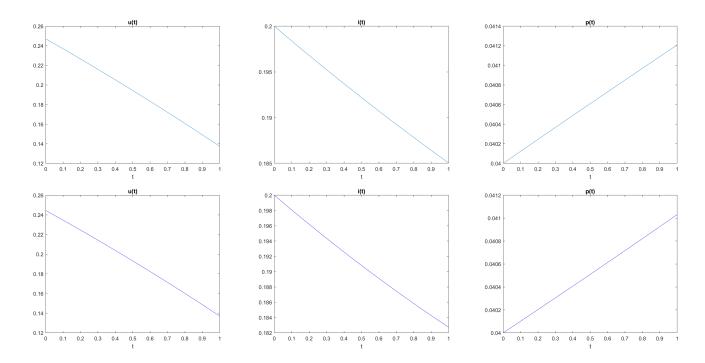


Figure 4: Comparison of the analytical (top) and numerical (bottom) solutions in an early stage epidemic context in the baseline framework.

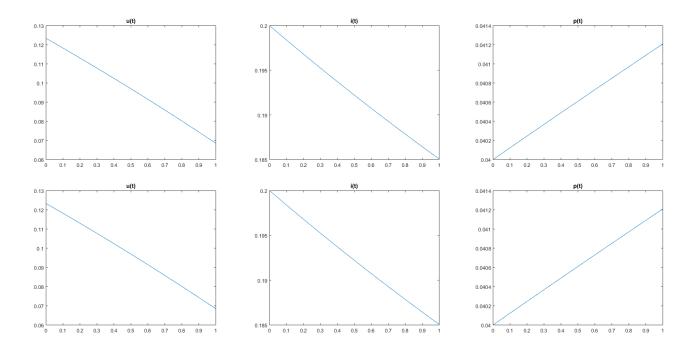


Figure 5: Comparison of the analytical (top) and numerical (bottom) solutions in an early stage epidemic context in the strategic interactions framework.